# Coupling between variations in strength and baroreflex latency of sympathetic discharges in human muscle nerves

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1. Pulse-synchronous multiunit muscle nerve sympathetic activity was recorded simultaneously from two nerves together with ECG in eleven healthy subjects; seven recordings were made from the two peroneal nerves during prolonged expiratory apnoeas and four from a radial and a peroneal nerve during lower body negative pressure of 10–40 mmHg. The neural records were displayed in mean voltage neurograms (time constant 0·1 s) and for each mean voltage burst the following measures were taken and related to each other: amplitude, duration, rise time, decay time and baroreflex latency (from the appropriate R-wave of the ECG to the peak of the burst).

2. Average baroreflex latencies were 1·3 s in the peroneal nerves and 0·9 s in the radial nerve. There were significant positive correlations between both the amplitudes and the baroreflex latencies of corresponding bursts in peroneal–peroneal recordings and in radial–peroneal recordings.

3. In all nerves baroreflex latency shortened significantly when burst amplitude increased. The correlation between burst amplitude and baroreflex latency was weaker in the radial than in the peroneal nerve. The average variation of baroreflex latency in peroneal–peroneal recordings was $0{\cdot}20 \pm 0{\cdot}02$ s in both legs, and in radial–peroneal recordings the variation was $0{\cdot}09 \pm 0{\cdot}01$ s in the radial nerve and $0{\cdot}12 \pm 0{\cdot}02$ s in the peroneal nerve.

4. When peroneal burst amplitudes increased, burst duration increased. This was due to increases of both the rise time and the decay time of the burst, the latter being the greater.

5. We suggest that the variation of baroreflex latency is due to changes of supraspinal synaptic delays or pathways and/or to recruitment of faster conducting sympathetic neurones when bursts become stronger.

In microneurographic recordings, sympathetic activity in nerve fascicles innervating human muscle (MSA) consists of intermittent bursts of vasoconstrictor impulses which are time-locked to the cardiac rhythm and occur predominantly during transient reductions in blood pressure (Delius, Hagbarth, Hongell & Wallin, 1972). The cardiac rhythmicity is due to modulation of the activity by the arterial baroreflex, i.e. pauses between successive bursts correspond to systolic blood pressure peaks inhibiting the sympathetic nerve traffic. In agreement with this there is a relatively reproducible reflex latency from the start of the systolic pressure wave (or the R-wave in the ECG) to the peak of corresponding burst in the mean voltage neurogram (the peak being taken as the start of inhibition) (Delius *et al.* 1972; Fagius & Wallin, 1980). In a given subject at rest the mean baroreflex latency of MSA is reproducible, but Fagius, Sundlöf & Wallin (1987) found that changes may occur with certain manoeuvres or experimental interventions. Both prolongations of and reductions in latency were seen, the latter being more common than the former. The most marked change was a reduction of mean latency by 120 ms (a change of approximately 8–10 %) during the Valsalva manoeuvre. No satisfactory explanation for this latency variability was advanced.

In the study of Fagius *et al.* (1987) the main emphasis was on mean changes of latency under different conditions. During the Valsalva manoeuvre, however, clear changes of reflex latency were also observed between individual bursts of MSA and often there was a progressive decrease in latency during the first part of the manoeuvre as the strength of activity increased. This observation prompted the present study, in which we have examined whether there is a systematic relationship between the strength of individual bursts of MSA and their baroreflex latency. At rest and during some manoeuvres there is normally a marked similarity between the strengths of the corresponding,

simultaneously recorded bursts of MSA directed to different extremities (Sundlöf & Wallin, 1977; Wallin, Victor & Mark, 1989; Wallin, Burke & Gandevia, 1992). If variations in baroreflex latency are coupled to variations in burst strength they would be expected to occur in parallel in different nerves. For this reason we have analysed records from two recent studies (Rea & Wallin, 1989; Wallin *et al.* 1992), which involved simultaneous recordings of MSA in two nerves in different extremities.

## METHODS

With the approval of the Ethics Committees of the University of New South Wales, Australia and the University of Göteborg, Sweden and after informed consent of each subject, experiments were made on eleven healthy volunteers (eight males and three females) aged 22–42 years (mean 29 years). They were not taking any medication.

### Nerve recordings

Tungsten microelectrodes, lacquer insulated except for a few micrometres at the tip, were used to record MSA. Recordings were made with seven subjects simultaneously from both peroneal nerves at the fibular head and with four subjects simultaneously from the left peroneal nerve at the fibular head and the right radial nerve at the spiral groove in the upper arm. The reference electrodes had large uninsulated tips and were inserted a few centimetres away from the recording electrodes. After amplification (× 50000) the signal was filtered (bandpass 500–2000 Hz) and passed through a resistance–capacitance circuit (time constant 0·1 Hz) to obtain a mean voltage display of the neurogram which was stored together with the ECG signals on analog tape. The filtered nerve signals were also monitored throughout the experiment on a loudspeaker and a storage oscilloscope.

Muscle nerve fascicles were localized by weak electrical shocks through the recording electrode and then small electrode adjustments were made until a site was found in which MSA could be recorded. Criteria for an acceptable recording site and evidence that the recorded activity was of sympathetic origin have been published previously (Sundlöf & Wallin, 1977).

The ECG was recorded via surface electrodes on the chest.

### Experimental procedure

Subjects were supine. In the recordings from the two peroneal nerves the primary aim of the experiments was to study effects of isometric muscle contraction on MSA (Wallin *et al.* 1992). Records obtained during two successive voluntary expiratory apnoeas and a short intervening rest period were analysed for reflex latencies. In the recordings from the peroneal and radial nerves the primary aim was to study effects of lower body negative pressure (LBNP) on MSA (Rea & Wallin, 1989). Records obtained during negative pressures of 10–40 mmHg were analysed for reflex latencies.

### Analysis

For analysis the mean voltage neurograms and the ECG were replayed from the tape onto a chart using an ink-jet recorder (Mingograf EEG Junior or Mingograf 1000, Siemens-Elema, Solna, Sweden) with a paper speed of 30 mm $s^{-1}$. The method for analysis of reflex latency has been described in detail previously (Fagius & Wallin, 1980; Fagius *et al.* 1987). In short, the interval from the R-wave of the ECG to the peak of the corresponding burst of MSA in the mean voltage neurogram was measured manually from the chart. When measured in this way the latency will include both haemodynamic events and neural conduction times, and in the peroneal nerve at the fibular head the overall latency will be in the range of 1·2–1·5 s depending on body height (Fagius & Wallin, 1980). Similar measurements have not been reported from the radial nerve, but in the median nerve at the elbow latencies are approximately 0·9–1·1 s (Fagius & Wallin, 1980). In each subject latencies, burst amplitudes and burst durations were determined for forty-two to fifty corresponding bursts in each nerve using a digitizing board connected to a personal computer, which was used for calculations and statistical analysis. Bursts occurring in only one nerve were excluded. To determine if there were significant changes in burst morphology the shape of the burst in the mean voltage neurogram was assumed to be a triangle. The amplitude of each burst was then plotted against both the rise time and the decay time of the burst and the degree of correlation calculated.

### Statistics

Values are given as means ± S.E.M. Linear regression analysis was used to quantify the relationship between variables. Probability values $< 0·05$ were considered significant.

## RESULTS

### Reflex latencies in the peroneal nerves

In a given subject the mean baroreflex latency in a peroneal nerve was 1·15–1·46 s (Table 1). Mean values (± S.E.M.) for the whole material obtained from simultaneous recordings in both peroneal nerves were 1·33 ± 0·03 s for the left leg and 1·31 ± 0·04 s for the right leg, values consistent with previous studies (Delius *et al.* 1972; Fagius & Wallin, 1980). There were parallel changes in latency of corresponding bursts in the two nerves with a significant linear relationship (Fig. 1*B*) and correlation coefficients of 0·72 ± 0·06 (Table 1). Similarly, there was a significant linear relationship between the amplitudes of corresponding bursts in the two nerves (Fig. 1*C*), with correlation coefficients of 0·86 ± 0·04.

When the oscilloscope sweep was triggered by the R-wave of the ECG during a period of increasing sympathetic activity (such as during apnoea), it was obvious that baroreflex latency shortened as burst amplitude increased (Fig. 2). As shown in Fig. 2 the decrease in latency affected the start of the burst to a much greater extent than the end of the burst. As a result, when superimposed as in the figure, the falling phases tended to superimpose while the rising phases were displaced to the left, dependent on burst size. Plots of burst amplitude *versus* baroreflex latency (Fig. 1*A*) showed highly significant linear relationships in the two nerves for all subjects, with correlation coefficients of −0·71 ± 0·05 for the left leg and −0·72 ± 0·04 for the right leg (Table 1). If the regression line for the relationship between burst amplitude and burst latency was used to calculate the mean latency difference between the smallest and largest bursts (the mean latency variation in Table 1) the result was 0·20 ± 0·02 s for each leg.

**Table 1. Reflex latencies, burst amplitudes and their correlation in the two peroneal nerves**

| | Mean reflex latency (s) | | Correlation coefficient: latency | Mean latency variation (s) | | Correlation coefficient | | |
|---|---|---|---|---|---|---|---|---|
| | | | | | | Amplitude | Amplitude–latency | |
| Subject | Left | Right | Left/right | Left | Right | Left/right | Left | Right |
| M.Cl. | 1·31 | 1·30 | 0·89* | 0·25 | 0·24 | 0·96* | 0·85* | 0·88* |
| G.Y. | 1·35 | 1·33 | 0·86* | 0·23 | 0·25 | 0·84* | 0·70* | 0·75* |
| S.G. | 1·28 | 1·28 | 0·71* | 0·16 | 0·15 | 0·93* | 0·65* | 0·67* |
| K.B. | 1·20 | 1·15 | 0·71* | 0·21 | 0·25 | 0·88* | 0·73* | 0·76* |
| M.B. | 1·36 | 1·31 | 0·47* | 0·10 | 0·14 | 0·70* | 0·47* | 0·61* |
| G.M. | 1·44 | 1·46 | 0·57* | 0·19 | 0·16 | 0·77* | 0·75* | 0·60* |
| M.Ce. | 1·37 | 1·36 | 0·82* | 0·26 | 0·21 | 0·96* | 0·85* | 0·78* |
| Mean | 1·33 | 1·31 | 0·72 | 0·20 | 0·20 | 0·86 | 0·71 | 0·72 |
| S.E.M. | 0·03 | 0·04 | 0·06 | 0·02 | 0·02 | 0·04 | 0·05 | 0·04 |

*$P < 0·001$.

## Burst morphology in the peroneal nerves

As shown in Fig. 2 most bursts had an approximately triangular shape with a distinct peak in the mean voltage record at all burst amplitudes. Occasionally, however, the maximum was a short plateau (cf. Fig. 4). The mean burst duration was $0·62 \pm 0·01$ s in both legs (Table 2). In all nerves except one there was a significant linear increase in burst duration with increasing burst amplitude and mean correlation coefficients were $0·55 \pm 0·07$ for the left leg and $0·61 \pm 0·07$ for the right leg. (Fig. 1*D*, Table 2). When the

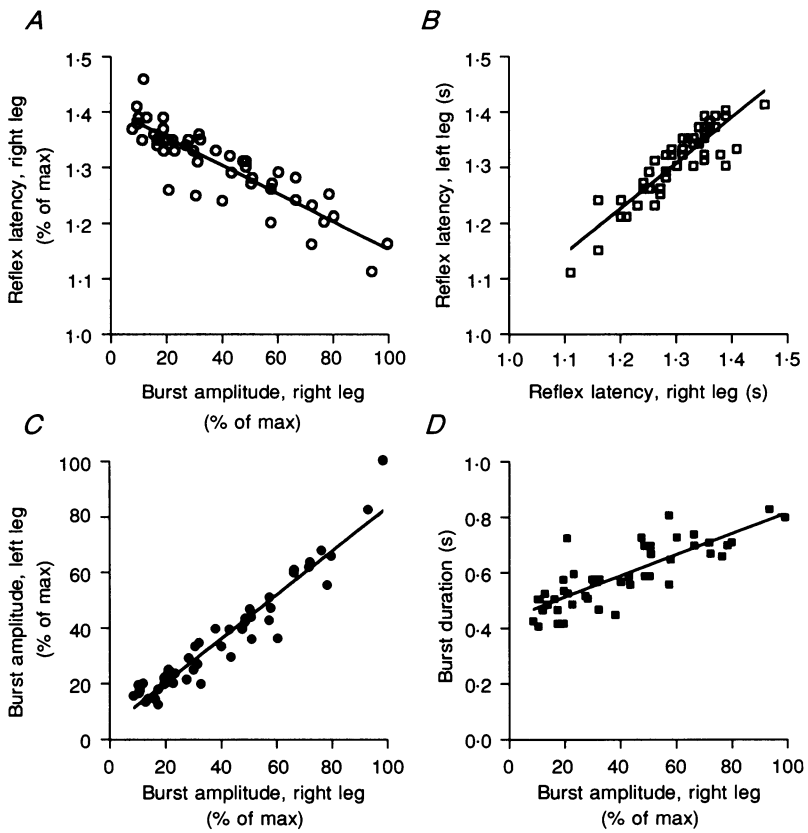


**Figure 1.**
Relationships between mean voltage amplitudes of sympathetic bursts recorded from the right peroneal nerve and corresponding baroreflex latencies (*A*) or burst durations (*D*). *B*, relationship between baroreflex latencies for corresponding bursts recorded simultaneously in the two peroneal nerves. *C*, corresponding relationship for burst amplitudes. Data from subject M.Cl.

**Table 2. Burst durations and their correlation to burst amplitudes in the two peroneal nerves**

| | Burst duration (s) | | | | | | Correlation coefficient: amplitude–duration | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Total | | Rising phase | | Falling phase | | Total | | Rising phase | | Falling phase | |
| Subject | Left | Right | Left | Right | Left | Right | Left | Right | Left | Right | Left | Right |
| M.Cl. | 0·58 | 0·61 | 0·22 | 0·24 | 0·35 | 0·37 | 0·71* | 0·82* | 0·49* | 0·46* | 0·73* | 0·83* |
| G.Y. | 0·63 | 0·61 | 0·26 | 0·26 | 0·37 | 0·35 | 0·68* | 0·63* | 0·33‡ | 0·42† | 0·69* | 0·61* |
| S.G. | 0·61 | 0·61 | 0·24 | 0·25 | 0·36 | 0·35 | 0·55* | 0·72* | 0·17§ | 0·46† | 0·64* | 0·70* |
| K.B. | 0·60 | 0·62 | 0·26 | 0·27 | 0·34 | 0·35 | 0·74* | 0·79* | 0·29§ | 0·49* | 0·77* | 0·80* |
| M.B. | 0·59 | 0·61 | 0·25 | 0·26 | 0·33 | 0·35 | 0·53* | 0·41† | 0·38† | 0·30‡ | 0·48* | 0·32‡ |
| G.M. | 0·58 | 0·58 | 0·24 | 0·26 | 0·35 | 0·32 | 0·25§ | 0·60* | 0·02§ | 0·44† | 0·40† | 0·45† |
| M.Ce. | 0·69 | 0·69 | 0·28 | 0·31 | 0·41 | 0·38 | 0·36† | 0·29‡ | 0·07§ | 0·13§ | 0·44† | 0·51* |
| Mean | 0·62 | 0·62 | 0·25 | 0·26 | 0·36 | 0·35 | 0·55 | 0·61 | 0·25 | 0·39 | 0·59 | 0·60 |
| S.E.M. | 0·01 | 0·01 | 0·01 | 0·01 | 0·01 | 0·01 | 0·07 | 0·07 | 0·06 | 0·05 | 0·06 | 0·07 |

*$P < 0·001$; †$P < 0·01$; ‡$P < 0·05$; § n.s.

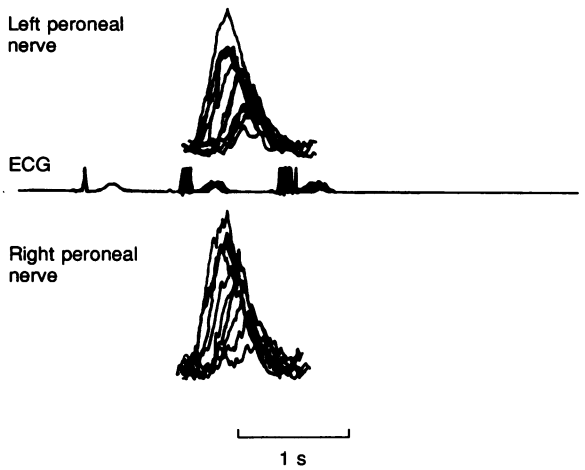


**Figure 2.**
Superimposed mean voltage records of ten sympathetic bursts recorded simultaneously from the two peroneal nerves during an expiratory apnoea in subject M.Ce. Oscilloscope sweep triggered on the R-wave of the ECG.

regression line for the relationship between burst amplitude and burst duration was used for the calculation, the average difference in duration between the biggest and the smallest burst was $0·26 \pm 0·04$ s in the left leg and $0·32 \pm 0·04$ s in the right leg.

In all subjects and all nerves the rising phase of the burst had a shorter duration than the falling phase (Table 2), a finding which in part may have been due to the time constant of the 'integrator' (0·1 s). Mean values for all nerves were $0·26 \pm 0·01$ for the rising and $0·36 \pm 0·01$ for the falling phase ($P < 0·001$). There was a significant linear relationship between burst amplitude and the duration of the falling phase of the burst in all nerves ($r = 0·60 \pm 0·04$, $n = 14$; Fig. 3). The corresponding relationship for the rising phase of the burst was significant in nine of fourteen nerves ($r = 0·32 \pm 0·04$). The slope of the regression line for the falling phase was steeper than that for the rising phase in all nerves ($P < 0·001$, $n = 14$).

In one female subject (K.B.) the last burst occurring during prolonged expiratory apnoeas had clearly aberrant properties. This is illustrated in Fig. 4, which shows the last parts of two expiratory apnoeas. In each apnoea all bursts

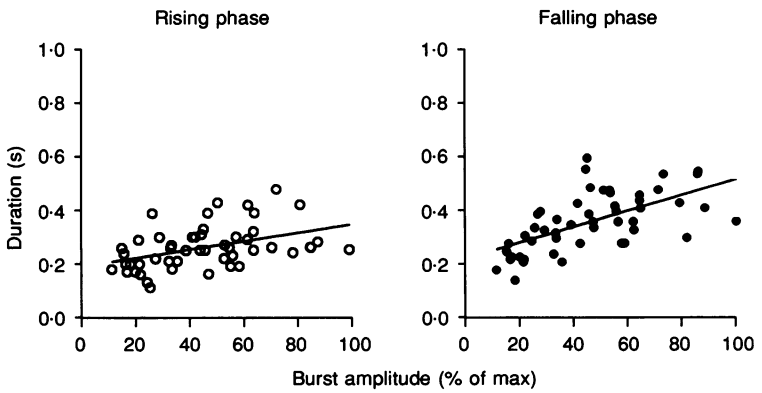


**Figure 3.**
Relationship between sympathetic burst amplitude and the duration of the rising (left) and the falling (right) phase of the burst. Data from right peroneal nerve in subject G.Y.

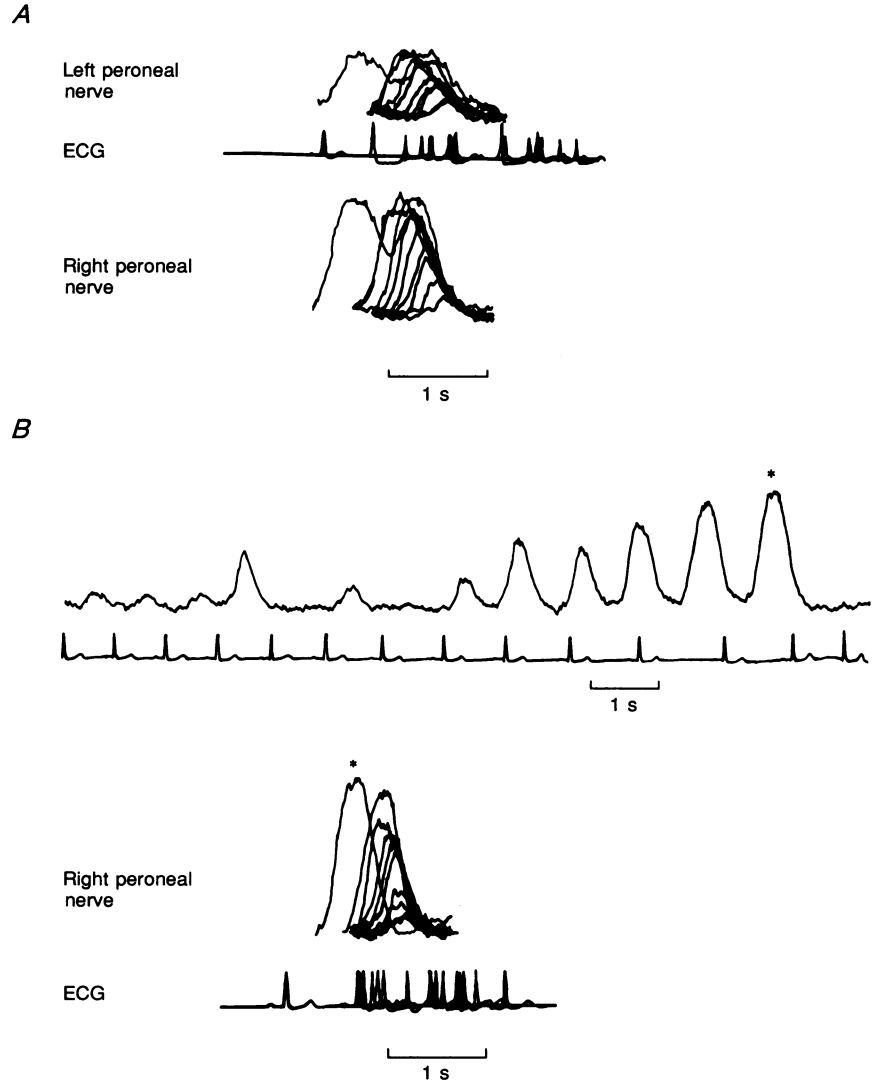


**Figure 4. Aberrant properties of sympathetic bursts derived during prolonged expiratory apnoeas in subject K.B.**
*A*, superimposed sympathetic bursts recorded simultaneously from the two peroneal nerves. Oscilloscope sweep triggered on the R-wave of the ECG. Note double-peaked burst with short latency of first component. *B*, mean voltage neurogram and ECG shown as original tracings (upper) and after superimposition obtained by triggering on the R-wave of the ECG (lower). Note short latency of last high-amplitude burst (indicated by asterisk).

except the last display the usual systematic relationship between burst amplitude and burst latency. In Fig. 4*B* the last burst appears to be uncoupled from the ECG and in Fig. 4*A* the burst morphology differed and there was a double-peaked burst, the first peak of which had an unusually short latency. The second peak, on the other hand, occurred at an appropriate latency compared with other bursts of similar amplitude.

**Table 3. Comparison of reflex latencies and burst durations in the radial and peroneal nerves**

| | Mean reflex latency (s) | | Correlation coefficient: latency | Max latency variation (s) | | Mean latency variation (s) | | Burst duration (s) | | Correlation coefficient | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | | | | Ampl.–ampl. | Ampl.–latency | |
| Subject | Rad | Per | Rad/Per | Rad | Per | Rad | Per | Rad | Per | Rad/Per | Rad | Per |
| K.A. | 0·87 | 1·24 | 0·50* | 0·26 | 0·26 | 0·11 | 0·15 | 0·52 | 0·54 | 0·77* | 0·58* | 0·72* |
| J.W. | 1·00 | 1·28 | 0·31‡ | 0·25 | 0·36 | 0·09 | 0·12 | 0·52 | 0·52 | 0·57* | 0·42† | 0·53* |
| L.R. | 1·01 | 1·41 | 0·39† | 0·37 | 0·24 | 0·07 | 0·07 | 0·58 | 0·56 | 0·69* | 0·29‡ | 0·48* |
| E.R. | 0·89 | 1·29 | 0·45† | 0·20 | 0·26 | 0·08 | 0·14 | 0·55 | 0·53 | 0·70* | 0·53* | 0·67* |
| Mean | 0·94 | 1·31 | 0·41 | 0·27 | 0·28 | 0·09 | 0·12 | 0·54 | 0·54 | 0·68 | 0·46 | 0·60 |
| S.E.M. | 0·04 | 0·04 | 0·04 | 0·04 | 0·03 | 0·01 | 0·02 | 0·01 | 0·01 | 0·03 | 0·06 | 0·06 |

Rad, radial nerve; per, peroneal nerve; ampl., amplitude; *$P < 0·001$; †$P < 0·01$; ‡$P < 0·05$.

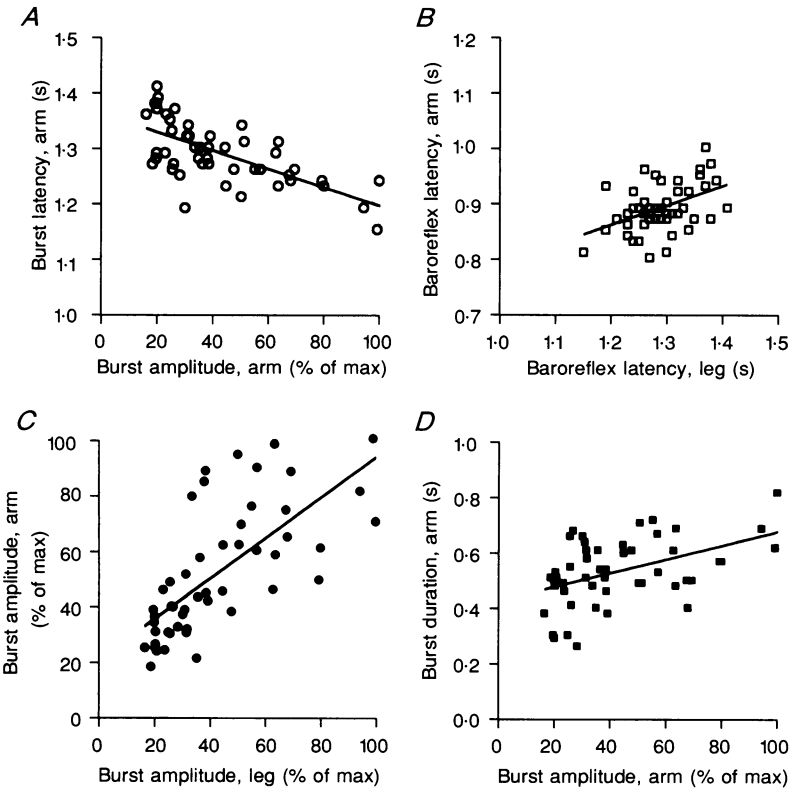


**Figure 5.**
Relationships between mean voltage amplitudes of sympathetic bursts recorded from the right radial nerve and corresponding baroreflex latencies (*A*) or burst durations (*D*). *B*, relationship between baroreflex latencies for corresponding bursts recorded simultaneously in the right radial and left peroneal nerves. *C*, corresponding relationship for burst amplitudes. Data from subject E.R.

### Comparison of reflex latencies in the radial and peroneal nerves

In simultaneous recordings from the radial and peroneal nerves ($n = 4$, all measurements summarized in Table 3) reflex latency was $0{\cdot}94 \pm 0{\cdot}04$ s in the radial nerve and $1{\cdot}31 \pm 0{\cdot}04$ s in the peroneal nerve. There was a significant correlation between latencies for corresponding bursts in the two nerves (Fig. 5*B*) in all subjects ($r = 0{\cdot}41 \pm 0{\cdot}04$, range 0·31–0·50). However, this correlation was weaker than that obtained in the simultaneous recordings from two peroneal nerves ($r = 0{\cdot}72 \pm 0{\cdot}06$, range 0·47–0·89). Also, the degree of correlation between burst amplitudes in the radial and peroneal nerves ($0{\cdot}68 \pm 0{\cdot}03$, Fig. 5*C*) was weaker than that between burst amplitudes in the two peroneal nerves ($0{\cdot}86 \pm 0{\cdot}04$).

Plots of burst amplitude *versus* reflex latency in the radial nerve (Fig. 5*A*) showed significant negative correlations in all subjects, but the strength of the correlation was always higher in the peroneal nerve than in the radial nerve (Table 3). The mean latency difference between the smallest and biggest bursts (calculation based on the regression line for the relationship between amplitude and latency) was smaller in the radial than in the peroneal nerve in three subjects and equal in both nerves in the fourth subject. Mean values were $0{\cdot}09 \pm 0{\cdot}01$ s in the radial and $0{\cdot}12 \pm 0{\cdot}02$ s in the peroneal nerve.

## DISCUSSION

The principal finding in the present study is the negative correlation between the mean voltage amplitude of a sympathetic burst and its baroreflex latency. In addition, there was a positive correlation between burst amplitude and burst duration. The findings imply that the stronger a sympathetic discharge the shorter its latency and the longer its duration.

The mean latency difference between the smallest and biggest bursts in the peroneal nerves were 0·12 and 0·20 s during LBNP and apnoea, respectively. Whether the difference between these figures is due to a greater variability of burst amplitudes during apnoea than during LBNP or reflects a difference in the underlying mechanisms between the manoeuvres is unclear. As measured in the present study, baroreflex latency can be divided into several components: (a) the time for the cardiovascular events from the R-wave of the ECG until the pulse wave reaches the baroreceptors; (b) conduction time in afferent baroreceptor fibres; (c) the time for central processing; (d) descending spinal conduction time; and (e) peripheral efferent conduction time (pre- and postganglionic). The variability of reflex latency may be due to variations in several of these components.

The time for cardiovascular and afferent events is less than 0·1–0·15 s (Fagius & Wallin, 1980), its variability is

probably small and it is unlikely to be an important factor in the variability of reflex latency. Variation in the time for central baroreflex processing is a more likely possibility. Only two to three brainstem synapses are thought to be involved (Ross, Ruggiero & Reis, 1985) but the central delay has nevertheless been estimated using different methods to be 0·25–0·30 s in human subjects (Fagius & Wallin, 1980; Borst & Karemaker, 1983). To our knowledge intra-individual variations of the central sympathetic delay of the baroreflex arc have not been published. However, Coote & MacLeod (1974) have reported a variation of approximately 60–70 ms for the central delay of a cardiac sympathetic reflex evoked by intercostal nerve stimulation. For the vagal baroreflex limb fairly large variations of the central delay have also been reported in cats and rats (McAllen & Spyer, 1978; McCloskey & Potter, 1981). Our results could be explained by such variations if, for example, a decrease in the arterial baroreceptor input (resulting in a strong sympathetic discharge) reduced synaptic delays or opened alternative central pathways. On the other hand, the finding that the mean latency difference between the smallest and the largest burst was longer in the peroneal than in the radial nerve in the same manoeuvres (0·12 *vs.* 0·09 s) strongly suggests that supraspinal mechanisms cannot account for the whole latency variability.

Another explanation for the variations in reflex latency would be recruitment of neurones with faster conduction velocity (spinal or peripheral) as the intensity of the sympathetic burst became greater. This would be consistent with recruitment according to the 'size principle', well established for skeleto-motor neurones (Hennemann & Mendell, 1981; Burke, 1981). The size principle implies that neurones with a low threshold of activation have smaller diameters and lower conduction velocities than neurones with high thresholds of activation. If valid also for MSA, one would expect small bursts (which occur following small reductions in blood pressure, i.e. have a low threshold) to be dominated by impulses in neurones with a low conduction velocity. In high amplitude discharges, on the other hand, both fast and slow conducting impulses are present which should lead to a longer total duration of large than of small bursts. The finding of a significant positive correlation between burst amplitude and burst duration is consistent with this alternative. Only fairly small differences in peripheral conduction velocity between 'fast' and 'slow' neurones would be needed to account for a (peroneal) latency difference of 0·12–0·20 s. The mean conduction velocity for MSA in the peroneal nerve is 1·1 m $s^{-1}$ (Fagius & Wallin, 1980) and if there were an increase of the maximal conduction velocity from 1·0 to 1·2 m $s^{-1}$ between the smallest and the largest bursts, conduction time would decrease by 0·13 s over a peroneal conduction distance of 0·8 m. In the spinal cord much larger differences in conduction velocity would be needed to explain the results. The mean conduction velocity for MSA in the spinal cord has been estimated to be 2·9 m $s^{-1}$ (Wallin & Rea, 1988). To account for a latency difference of 0·15 s over a spinal conduction distance of 0·5 m a velocity increase from 2·5 to 10 m $s^{-1}$ would be needed, a possibility which seems less likely.

When recording in the peroneal nerve at the fibular head (i.e. after a total spinal and peripheral conduction distance of more than 1 m) one would expect impulses with the highest conduction velocities to occur in the first part and those with the lowest velocities in the last part of the bursts. Recently, the probability for multiple firing in the same fibre was found to increase with increasing strength of the burst (V. G. Macefield, B. G. Wallin & Å. B. Vallbo, unpublished observation). Multiple firing should probably cause greater dispersion in time of low than of high conduction velocity impulses. Our findings that the falling phase of the bursts had longer mean duration and increased more in duration with increasing burst amplitudes than the rising phase agree with these considerations.

The moment-to-moment variations in the strength of MSA discharges can be expected to reflect the balance between the central sympathetic drive and several excitatory and inhibitory reflex mechanisms, such as arterial and low pressure baroreflexes, stretch reflexes from the lung and/or the chest wall and chemoreflexes. The present findings suggest that in addition to the strength of a sympathetic discharge, the baroreflex latency may provide useful information about the balance between excitation and inhibition. Occasionally, the latency of a discharge may even be a better indicator than the strength. This is illustrated by the burst with aberrant properties (which were more pronounced for latency than for amplitude) occurring at the end of several prolonged apnoeas in one subject (Fig. 4). In this case chemoreceptor activation, together with the absence of inhibition from lung receptors, may have led to an increase of the excitatory drive which was sufficiently potent to overcome arterial baroreflex inhibition, the net effect being a transient loss of cardiac rhythmicity. The occurrence of bursts of MSA related to K complexes during stage 2 sleep may also be caused by an increased (in this case central) excitatory drive overriding arterial baroreflex inhibition (Hornyak, Cejnar, Elam, Matousek & Wallin, 1991; Okada, Iwase, Mano, Sugiyama & Watanabe, 1991). In other situations in which the cardiac rhythmicity of MSA has been lost (Fagius & Wallin, 1983; Fagius, Wallin, Sundlöf, Nerhed & Englesson, 1985; Stjernberg, Blumberg & Wallin, 1986; Sellgren, Pontén & Wallin, 1990, 1992), the effect was due mainly to a weakened inhibitory baroreflex influence.

MSA records obtained simultaneously from different extremity nerves have been reported previously to be remarkably similar at rest (Sundlöf & Wallin, 1977; Wallin *et al.* 1989), during LBNP (Rea & Wallin, 1989) and voluntary apnoea (Wallin *et al.* 1992). The present data show, however, that the similarity was less in simultaneous arm–leg recordings than in leg–leg recordings. For correlations involving radial burst latencies part of the difference may be technical (due to lesser resolution when measuring the shorter radial reflex latencies). This is unlikely to be the whole explanation, and since it does not apply to amplitude measurements, an underlying physiological mechanism must be inferred. That conclusion agrees with previous data showing differences between peroneal

and radial burst amplitude spectra at rest (Wallin *et al.* 1989). Whether the differences are due to supraspinal, spinal or ganglionic mechanisms is unclear.

Changes of MSA in a given electrode site are often quantified as the product of the number of bursts per minute and the mean voltage amplitude of the bursts (= 'total MSA'). Our finding that the burst duration increases significantly when burst amplitude increases implies that this method of quantitation underestimates changes of 'total MSA'. The product of the number of bursts and the mean area of the bursts in the integrated neurogram probably represents a more appropriate method of quantitation.

In conclusion, it is likely that a number of factors contribute to the inverse covariation of burst amplitude and baroreflex latency. It would be possible to explain the entire latency variability on the recruitment of faster conducting peripheral sympathetic efferents as bursts become stronger, but supraspinal mechanisms and perhaps even changes in the spinal transmission may contribute.

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