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ORIGINAL ARTICLE

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# Changes in R–R variability before and after endurance training measured by power spectral analysis and by the effect of isometric muscle contraction

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**Abstract** The long-term conditioning effects of physical training on cardiorespiratory interaction in 11 young healthy males were studied. Significant increases in maximum oxygen uptake ($\dot{V}O_{2max}$) ($P < 0.05$) and decreases in heart rate ($P < 0.05$) were achieved in all subjects following a 6-week training programme consisting of cycling for 25 min each day at a work level that increased heart rate to 85% of maximum. Heart rate variability, measured as the differences between the maximum and minimum R–R interval in a respiratory cycle, increased in nine of the subjects and decreased in two. The respiratory-cycle-related high-frequency peak in the power spectral plot of R–R variability also showed significant increases in the same nine subjects and decreases in two. The latter result was similar after normalisation of the data for changes in heart rate by calculating the common coefficient of variance ($CCV = \frac{HF}{R\text{–}R} \times 100$), where HF is the high-frequency component of the power spectral plots, using a further measure of vagal tone it was shown that, for all subjects, the R–R interval change in response to isometric contractions of the arm flexors in one respiratory cycle were significantly greater after training. These data suggest that cardiac vagal tone is increased by aerobic training for all subjects and that this is accompanied by a respiratory sinus arrhythmia (RSA) in most, but may be associated with a decrease in RSA in subjects with a very low ($< 50$ beats·min$^{-1}$) heart rate.

**Key words** Respiratory sinus arrhythmia · Cardiac vagal tone · Aerobic training

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## Introduction

Human subjects at rest show a degree of sinus arrhythmia in which heart rate increases during inspiration and decreases during expiration, as illustrated in Fig. 1A. This variation in beat-to-beat interval (R–R variability) during each respiratory cycle is dependent on cardiac parasympathetic activity since it is absent after peripheral cholinergic blockade with atropine (Eckberg 1983). Therefore R–R variability could be considered as an index of cardiac vagal tone. The R–R variability is important in healthy young subjects, since it acts as a compensatory mechanism to reduce the variability in blood pressure caused by the thoracic-pressure-induced alteration in cardiac output, as shown by Toska and Eriksen (1993).

Endurance training is accompanied by a significant drop in resting heart rate (Lewis et al. 1980; Katona et al. 1982; Maciel et al. 1985; Barney et al. 1988; Reiling and Seals 1988; De Meersman 1992) and therefore an increase in R–R variability might be expected. However, there are conflicting reports in the recent literature. One report shows that training increases heart rate variability, suggesting a change in the cardiac vagal neurone activity (De Meersman 1992). In contrast others could not demonstrate a difference in the R–R variability between a group of endurance athletes and non-athletes, therefore concluding that respiratory sinus arrhythmia (RSA) is not increased by training (Lewis et al, 1980; Katona et al. 1982; Maciel et al. 1985; Reiling and Seals 1988).

In view of these reports, the present longitudinal study was undertaken in an attempt to clarify the effect

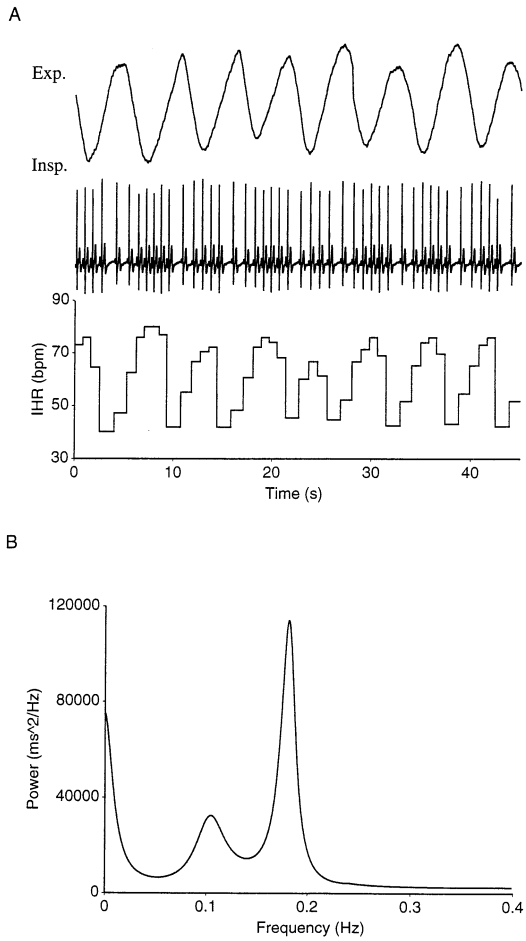


**Fig. 1A, B** Respiratory-related heart rate variability in one resting young male subject with depth and rate of breathing voluntarily controlled within prescribed limits. **A** *Top trace* shows respiratory movements. (*Exp.* Expiration, *Insp.* inspiration) *Second trace* is the ECG recorded via chest leads. *Bottom trace* is instantaneous heart rate (*IHR*) in beats per minute (*bpm*) on the *vertical axis*, plotted against time. **B** Power spectral plot of R–R interval variability for the data illustrated in **A**

of endurance training on parasympathetic activity to the heart. Two methods were used to evaluate RSA or cardiac parasympathetic activity. One was the difference between the maximum and minimum R–R interval in one respiratory cycle (VHP), suggested by Katona and Jih (1975) and used by many contributors (Lewis et al. 1980; Maciel et al. 1985; Reiling and Seals 1988). The other was power spectral analysis (PSA), illustrated in Fig. 1B, which gives a more sensitive estimation of the variation in R–R interval (Karemaker 1993) and which has not been used so far to evaluate the effects of endurance training on heart rate variability. As a further measure of vagal tone, we also used a method we have recently developed which examines the early changes in the R–R interval occurring in one respiratory cycle elicited by a brief isometric voluntary muscle contraction (Al-Ani et al. 1995). These changes are vagally mediated since they are abolished by atropine (Freyschuss 1970; Maciel et al. 1987).

## Methods

### Subjects

Eleven healthy young members of our Institute, one female and ten males, of ages 20 (1) years [(mean (SE))] participated in this study. None of them had any cardiac or respiratory-related illnesses, as assessed by medical history and an electrocardiogram (ECG) at rest. They all had full knowledge of the experimental protocol and gave informed consent to participate in the study, which had been approved by the local ethics committee.

### Training programme

The 11 subjects were subjected to a 6-week endurance training programme. Under supervision they were instructed to cycle daily for 25 min continuously at a work level that increased heart rate to 85% of the maximum heart rate recorded during a test of maximal oxygen uptake ($\dot{V}O_{2max}$). Their ECG or pulse was continuously monitored (Favor heart rate monitor, Polar) to provide a visual display of heart rate. As fitness improved, work load was increased so as to maintain the heart rate at 85% of the maximum throughout each exercise period.

### Determination of $\dot{V}O_{2max}$

$\dot{V}O_{2max}$ was determined using an incremental work test to exhaustion on a bicycle ergometer (Siemens Elema 380B, auto-tracking model). $\dot{V}O_2$ was measured by passing the expired air through a gas analyser (Cardiokinetics-Oxycon) which provided a computer printout of the oxygen uptake. ECG was measured using electrodes placed on the chest, and from this heart rate was derived and printed out (Cardiorater-monitor). Two such tests on different days were performed by each subject prior to their entering the training programme. Two similar tests were repeated to measure $\dot{V}O_{2max}$ at the end of the 6-week training period.

### Determination of RSA

The subjects were rested in a semi-supine position for at least 30 min before any recording occurred, and they were instructed not to eat or consume any caffeine for at least 2 h prior to the experiment. The subjects were well familiarised with the technique and the environment before the experiment took place.

Both ECG and respiratory movement were recorded via chest leads, amplified and digitised via an A/D converter at 125 sample/s, then stored by computer (Apple Mac llci). The subject was provided with a visual display of these two signals on a VDU.

The magnitude of RSA in beat-by-beat variation in R–R interval is closely dependent on the respiratory frequency and tidal volume (Hirsch and Bishop 1981; Eckberg 1983), therefore each of these variables was closely controlled. We were aware that chest impedance does not provide an absolute measure of tidal volume, but we avoided spirometry during the tests because we were concerned that tidal volume and respiratory frequency were kept close to the natural resting level throughout each test. Furthermore the ability to detect changes was most important and the method of chest impedance provides a high level of sensitivity to change. The subjects were instructed to breathe as naturally as possible and the frequency

and depth measured first using spirometry for absolute values and kept the same throughout the two tests before, and in the two tests after, the training period. Since ventilatory and cardiovascular variables are known to exhibit diurnal variation, the experiments were performed at the same time of day for each individual.

After the resting period, when the subjects were comfortable with their respiratory frequency and tidal volume, a recording period of 10 min commenced, during which time the subjects were left undisturbed.

Instantaneous heart rate and R–R intervals were analysed by computer (Apple Mac llci). Care was taken, when detecting the R–R intervals, to exclude other peaks. Heart rate variability (HRV) was measured from these data by determining the difference between the maximum and minimum R–R intervals in a respiratory cycle (Katona and Jih 1975).

Power spectral analysis of R–R variability was performed off-line. The spectral components were computed from tachograms of 512 R–R intervals using an autoregressive algorithm (Burg 1975). The model order was chosen by the method known as AKAIKE information criterion. Two peaks were identified, a high-frequency (HF) component centred at the respiratory frequency around 0.25 Hz that reflects cardiac vagal activity, and a low frequency (LF) component at 0.1 Hz reflecting both sympathetic and vagal activity to the heart (Aguirre et al. 1990). Care was taken to exclude all recordings in which ectopic beats or movement artefacts occurred. Stationarity of the time series of R–R intervals was tested by calculation of the mean variance of the first and last 256 beats of each recording period, made in order to verify a difference of $< 10\%$ in the values of each time series. Quantification of the power of each underlying frequency was performed by sorting the total variability signal into individual components using the method suggested by Zetterberg (1969). Because total power varies greatly between individual subjects, the power of each component was compared in terms of both absolute power (area under the component curve), as well as the power in normalised units (NU). The power in NU was calculated by dividing the absolute power of a given component by the total variance. As an additional control we have also normalised for the different heart rates before and after training. For this purpose we have expressed RSA as the coefficient of variance of the R–R interval at a constant respiratory frequency using the HF component of the power spectral plot. Thus for each individual we have calculated the common coefficient of variance, CCV as $\frac{\text{HF}}{\text{R–R}} \times 100$ and averaged the values for all subjects.

### Determination of the change in R–R interval induced by isometric muscle contraction

Isometric contraction of the flexors of the upper arm was performed, with the arm fixed, at the elbow and wrist, by a frame attached to a couch on which the subject reclined in a semi-supine position. Isometric force was measured by a strain gauge attached to a wrist clamp. The tension signal was amplified digitised at 125 samples/s and displayed on the VDU. Each subject was first asked to perform a maximum isometric contraction of the upper arm flexors (maximum voluntary contraction, MVC) and from this a 60% proportion of MVC was determined and indicated on the VDU. This could be achieved without causing a Valsalva manoeuvre, as indicated by the chest impedance measurement on the VDU and checked in a separate series of tests using a respiratory flowmeter (Butler and Woakes 1987) connected to a mouthpiece. During the test procedure the subject was instructed to perform a contraction at a sound initiated by a signal from the computer at the beginning of an expiratory phase (phase 2, Fig. 4A) or at the beginning of an inspiratory phase (phase 6 Fig. 4B) and the contraction was maintained for up to five respiratory cycles. Only the changes in the first respiratory cycle in which the contraction was elicited were considered for the present study.

### Experimental protocol

The subjects attended on two separate occasions and were instructed not to eat or drink for at least 2 h before the experiment. Before the start of each of the tests a 30-min rest period was allowed to enable ventilation and heart rate to stabilise. All experiments were carried out in a quiet room with temperature maintained at 21–22°C. The subject practiced breathing at a frequency synchronised to a metronome, the frequency of which was adjusted to suit the subject's natural resting mean breathing frequency which was then kept constant throughout the experiments. This usually lay between 0.2 Hz and 0.25 Hz. The subjects also had a visual display of the respiratory movements and they were asked to maintain tidal volume within a narrow range ( $\pm$ 5%) of their mean resting values indicated by traces on the VDU (set for each subject). Power spectral analysis of this set of data was performed.

For the examination of the effects of muscle contraction on R–R intervals, methods similar to those described previously were used (Al-Ani et al. 1995). On each visit baseline data were collected over 60 respiratory cycles (control line Fig. 4). A muscle contraction test was then repeated five times with a recovery period lasting 2 min between each test. For the purpose of analysis the respiratory cycle was divided into 10 phases (0 to 9) of equal duration from the beginning of expiration to the end of inspiration. The duration of consecutive R–R intervals occurring in any test respiratory cycle was measured by the computer and that value consigned to the respiratory phase in which the R–R interval finished. This procedure gave at least three values in any one of the phases. These were measured and the standard error of the mean (SEM) calculated. The graphs presented in Fig. 4 show the pooled data obtained from 11 subjects, so for each point $n = 11$. Measurements were repeated during two visits before, and two visits after, training. Values were not significantly different for the two visits either before or after training.

### Statistical analysis

After establishing that the data were normally distributed, a direct comparison of paired data was made using analysis of variance. The data are presented as means (SEM). Significance was established at $P$ values $< 0.05$.

## Results

The pre- and post-training results for both $\dot{V}O_{2max}$ and heart rate are listed in Table 1A. $\dot{V}O_{2max}$, expressed as $l \cdot min^{-1}$ or $ml \cdot kg^{-1} \cdot min^{-1}$, increased significantly by 13% and 15% respectively as a consequence of the training ($P < 0.05$). Resting heart rate also showed a significant drop of 12 beats $\cdot min^{-1}$ with training ($P < 0.05$).

HRV for nine of the subjects increased significantly from 110 (45) ms to 180 (61) ms ($P < 0.05$) with training. A typical example of data from one of the subjects is shown in Fig. 2. This subject had a mean heart rate before training of 62 beats $\cdot min^{-1}$ and a mean HRV of 169 ms, whereas after training mean heart rate was 50 beats $\cdot min^{-1}$ and mean HRV was 247 ms. Power spectral analysis further confirmed this training effect for the nine subjects. The power spectral plots of data from one of these subjects is shown in Fig. 2B, D, which illustrates the increase in the power spectral density curve in the region around the breathing frequency

**Table 1 A** Training data values of $\dot{V}O_{2max}$ ($l \cdot min^{-1}$ and $ml \cdot kg^{-1} \cdot min^{-1}$), resting heart rate ($f_c$) and maximum heart rate $f_{c\,max}$ for 11 subjects before (*Pre*) and after (*Post*) training. Values presented are mean (SEM). $P < 0.002$, comparing post to pre values for 11 subjects. **B** Power spectral data values for the high frequency (*HF*) and low frequency (*LF*) components in the power spectral plots of R–R variability (*i*) for the nine subjects and (*ii*) for the two subjects. Values presented here are absolute power (i.e. area under the curve) as mean (SEM). $P < 0.05$ for both HF and LF comparing post to pre values for the nine subjects

| Parameter | Period | |
|---|---|---|
| | Pre | Post |
| **A** Training data | | |
| $\dot{V}O_{2max}$ ($l \cdot min^{-1}$) | 3.23 (0.2) | 3.66 (0.3)* |
| $\dot{V}O_{2max}$ ($ml \cdot kg^{-1} \cdot min^{-1}$) | 45 (2) | 51 (2)* |
| $f_c$ ($beat \cdot min^{-1}$) | 69 (3) | 57 (2)* |
| $f_{c\,max}$ ($beat \cdot min^{-1}$) exercise | 196 (3) | 194 (2) (*$P < 0.002$) ($n = 11$) |
| **B** Power spectral data | | |
| (i) 9 subjects | | |
| HF ($ms^2$) | 2051 (541) | 5131 (905)* |
| LF ($ms^2$) | 912 (147) | 2646 (621)* (*$P < 0.05$) ($n = 9$) |
| (ii) 2 subjects | | |
| | Pre | Post |
| HF ($ms^2$) | 8281 (706) | 2602 (813) |
| LF ($ms^2$) | 1766 (544) | 1339 (689) |

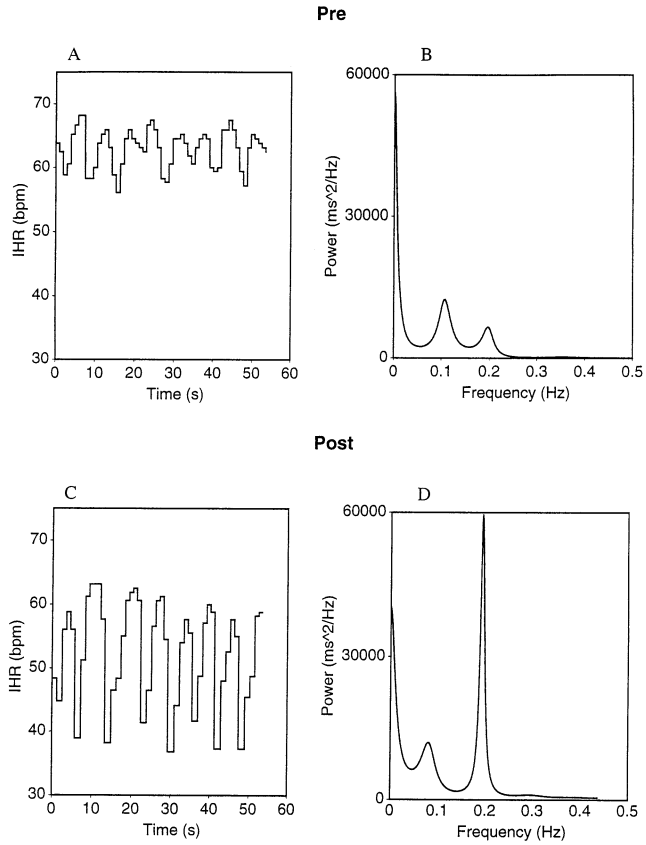


**Fig. 2A–D** Instantaneous heart rate (*IHR*) in beats per min (*bpm*) and power spectral plot of R–R interval for one resting subject with controlled voluntary breathing before (*Pre*, **A, B**) and after (*Post*, **C, D**) a period of endurance training. This subject was typical of nine of the subjects

(0.2 Hz), suggesting that cardiac vagal tone has increased as a consequence of training.

The group mean data for these nine subjects are listed in Table 1B(i) and show that both LF and HF components significantly increased, by some twofold.

In the remaining two subjects the opposite effect in the HRV and power spectral curves was observed, since, surprisingly, HRV and the LF and HF components in the power spectral plots were reduced with training (Fig. 3). In these latter subjects, following training the resting heart rate was remarkably low at a mean of 43 $beats \cdot min^{-1}$ for one and 45 $beats \cdot min^{-1}$ for the other, reduced from 68 $beats \cdot min^{-1}$ and 55 $beats \cdot min^{-1}$ respectively. The mean power spectral data for these two subjects are shown in Table 1B(ii).

Since spectral powers of R–R variability at respiratory frequencies may be heart rate dependent we have normalised for mean R–R interval and heart rate in a further analysis of the power spectral data. For this purpose we expressed RSA as the common coefficient of variance (CCV) of the R–R interval for the HF component in each subject. The CCV was calculated from $\frac{\text{HF}}{\text{R–R}} \times 100$. The mean CCV for the same nine subjects that showed an increase in HF was also significantly increased from 4.8 (0.5)% before training to 6.8 (0.7)% after training ($P < 0.05$). In the two subjects with a low heart rate, the CCV significantly decreased from 9.3 (0.6)% before training to 4.0 (0.8)% after training.

## The effect of isometric muscle contraction before and after training

In all 11 subjects brief voluntary isometric contraction of the arm flexors (60% MVC) performed on command during phase 2 (expiration) or phase 6 (inspiration) caused a significant ($P < 0.01$) reduction of the R–R interval in all the subsequent phases of the first respiratory cycle (Fig. 4). Muscle contraction in the expiratory phase decreased the R–R interval by a maximum of 115 (22) ms and in the inspiratory phase by 98 (18) ms in the pre-training tests. Following training, similar contractions elicited more marked decreases in the R–R interval [160 ms in expiration, 145 (24) ms in inspiration, Fig 4C, D], which were significantly

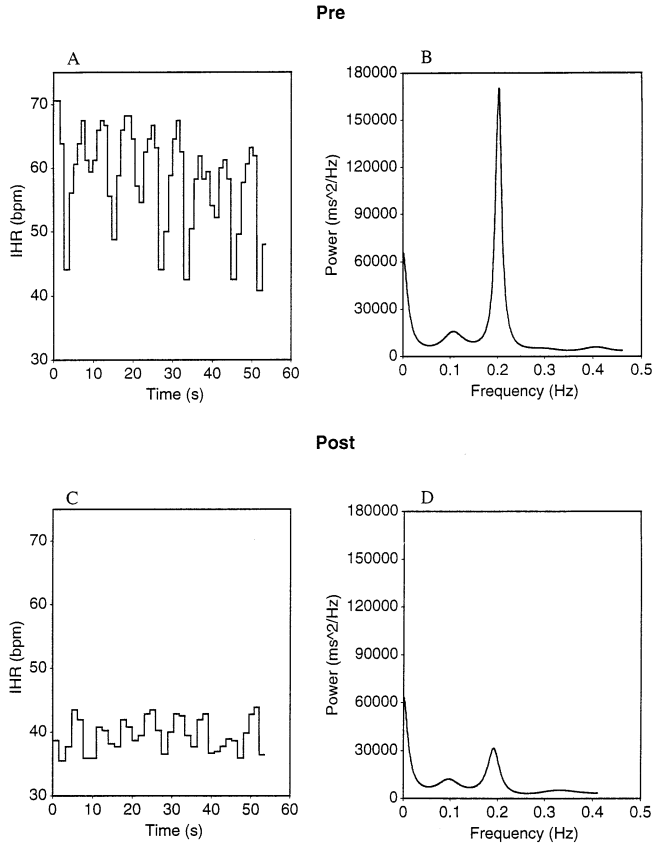


**Fig. 3A–D** Instantaneous heart rate (*IHR*) in beats per min (*bpm*) and power spectral plot of R–R interval for one resting subject with controlled voluntary breathing before (*Pre*, **A, B**) and after (*Post*, **C, D**) a period of endurance training. This subject was one of two subjects showing a low resting heart rate and a reduced sinus arrhythmia

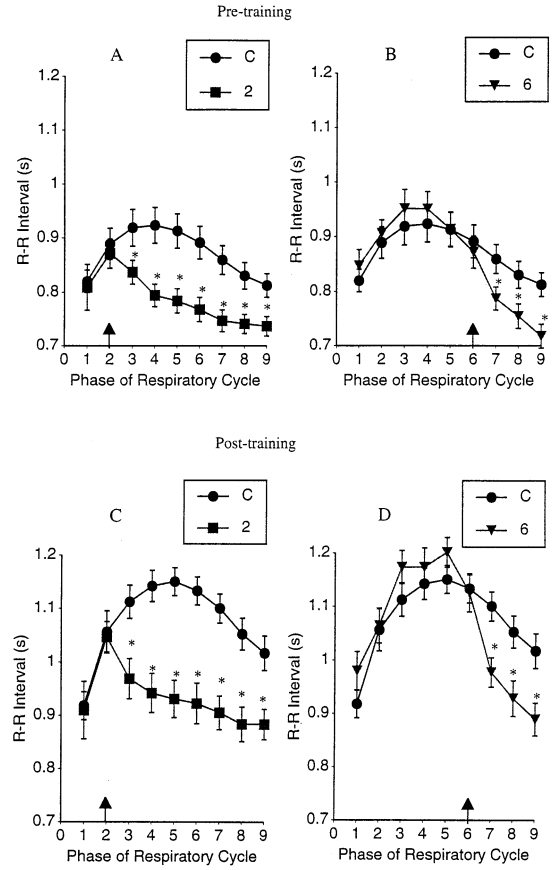


**Fig. 4** Graphs showing the effect of a 60% maximum voluntary contraction (MVC) on R–R intervals throughout one respiratory cycle, pre-training (**A, B**) and post-training (**C, D**). Each contraction was repeated 5 times during expiration and 5 times during inspiration for each subject and the mean data for 11 subjects are plotted in each graph. (*C* Control plot of R–R intervals throughout 60 respiratory cycles without muscle contraction. **A** R–R intervals before (phases 0–2) and following (phases 3–9) muscle contraction initiated in expiration at *arrow*. **B** R–R intervals before (phases 0–6) and following (phases 7–9) muscle contraction initiated in inspiration at *arrow*. R–R intervals for control and before and following 60% MVC (*arrows*) post-training are shown in **C** for contractions initiated in expiration and **D** for contractions initiated in inspiration. $P < 0.05$ compared to control value *C* in same phase

different in magnitude from pre-training values ($P < 0.05$). Despite this, the absolute value of heart rate achieved during such muscle contractions post-training was 73 (4) beats $\cdot$ min$^{-1}$, which was lower than that of 88 (2) beats $\cdot$ min$^{-1}$ initiated before training. This was because the resting R–R intervals from which the change occurred (measured in the period immediately before contraction) were so much longer after training [mean 1052 (36) ms] than the values before training [mean 833 (36) ms].

## Discussion

The increase in $\dot{V}O_{2max}$ and drop in resting heart rate as a consequence of aerobic training are well agreed upon (see review Clausen 1977; Scheuer and Tipton 1977). The issue addressed by the study was whether the fall in heart rate is associated with an increase in RSA. At present there is not a consensus in favour of training-induced RSA. A reason may be that studies have been structured differently. One approach has been the so-called Transverse Study, whereby groups of trained and untrained subjects have been compared (Lewis et al. 1980; Maciel et al. 1985; Barney et al. 1988; Reiling and Seals 1988). Although these studies are important in so far as they provide data on populations, they also hide the fact that RSA varies quite markedly from one subject to another even amongst "fit" subjects. Furthermore, unless factors like posture, environmental temperature, time of day, respiratory frequency and tidal volume are controlled, comparing RSA between individuals may be misleading.

To overcome these possible drawbacks we have used a longitudinal design for our study in which the changes in the same individual are compared under strictly controlled conditions. The results show that young subjects of either sex, starting from a "fairly" unfit baseline ($\dot{V}O_{2max} = 3.23\,l \cdot min^{-1}$), decreased their resting heart rate significantly after a period of intense training and in the majority there was also a significant increase in RSA, which supports the conclusions of Barney et al. (1988) and De Meersman (1992). However, they also show that training may reduce RSA if cardiac vagal tone is increased above a critical level. Thus, two trained subjects with an unremarkable $\dot{V}O_{2max}$ (of 4.6 and $3.3\,l \cdot min^{-1}$) but, most importantly, with a very low resting heart rate had a reduced RSA. In these two subjects it was almost as though cardiac vagal excitability was so high that respiratory influences were less able to decrease RSA. We did not measure arterial blood pressure although there is evidence that endurance training can reduce blood pressure. Therefore we cannot rule out a smaller fluctuation in baroreceptor feedback in phase with respiration occurring in these individuals although we might expect this to be offset by an increase in baroreflex sensitivity in highly fit subjects, as reported by some authors (Fiocchi et al. 1985; Barney et al. 1988; Somers et al. 1991). Nonetheless the marked reduction in RSA in two individuals points to one reason why studies of a population of athletes are unlikely to provide a clear answer as to whether RSA is increased by training.

In recent years power spectral analysis of heart rate or R–R variability has been used to indicate the degree of cardiac vagal tone. We observed marked changes in the power spectra which were still highly significant after normalisation for the change in resting R–R intervals by calculating the common coefficient of variance. The data conform the conclusion drawn from measurements of RSA. First, the power spectral density curves for the nine subjects with a high RSA showed an increase in the HF component. This peak reflects the variation induced by respiration (peripherally or centrally) in cardiac vagal activity since it is blocked by atropine (Aguirre et al. 1990). Second, the same two subjects with a reduced RSA following training also showed a reduced HF peak in the power spectral density plots, although their resting heart rates were lower than those of the other nine subjects. Despite this paradoxical evidence from RSA measurements and power spectral analysis, we feel it is justified to conclude that the lowering of resting heart rate in all subjects is mainly due to an increase in cardiac vagal tone. We favour this because of the finding of increased changes in R–R interval induced immediately following the start of a brief isometric muscle contraction. These are purely vagally mediated, occurring too rapidly to be sympathetic and they are abolished by atropine (Freyschuss 1970; Weiling et al. 1985; Maciel et al. 1987). This increased effect was not only present in the same nine subjects showing increased RSA, but also strongly apparent in the two subjects with very low resting heart rate, but with decreased RSA post-training. In addition it seems unlikely that a decrease in sympathetic activity played any significant part since, for all 11 subjects, the LF peak of the power spectra, which reflects sympathetic and parasympathetic activity, was either unchanged or increased following training. Therefore the best explanation of the data arising from these several tests is that the lowering of resting heart rate following training reflects an increase in cardiac vagal tone, as concluded by numerous studies (Raab et al. 1960; Frick et al. 1967; Ekblom et al. 1973; Clausen, 1977; Scheuer and Tipton 1977).

The results, demonstrating that the initial changes in the R–R interval in response to muscle contraction are greater than pre-training changes, are of further interest. This effect is not an action of training on the arm muscles because these were not involved in the training protocol and the tension developed by the arm muscle contractions were similar before and after training. Furthermore even taking into account the relatively poor resolution of the measurements which were $\pm 8$ ms (since the R–R intervals were sampled at 125 Hz) the effect of training on the initial changes in R–R interval is still highly significant compared to pre-training values. We interpret the effect as due to an inhibition of cardiac vagal neurones either by muscle afferents (McMahon and McWilliam 1992) or by central command (Secher 1985; Decety et al. 1993; Gandevia et al. 1993). The results show that this inhibition has greater efficacy following endurance training when the vagal tone is higher. Furthermore the values for changes during expiration when vagal tone is greater are higher than those during inspiration. Thus an inhibitory input from muscle afferents is seeming to have a greater effect when the excitability of cardiac vagal neurones is high.

The present study also measured the maximum heart rate that could be achieved when performing intense exercise both before and after the training period. Numerous earlier studies indicate that the maximum heart rate is not altered by training (see Astrand and Rodahl 1986), but there are some studies that report a decrease (Lewis et al. 1980; Maciel et al. 1985). We found no change in the maximum heart rate [196 (3) beats $\cdot$ $min^{-1}$ pre-training, 194 (2) beats $\cdot$ $min^{-1}$ post-training] despite a significant increase in $\dot{V}O_{2max}$, which was similar to, or more than, that reported by Lewis et al. (1980) and Maciel et al. (1985). Bearing this is mind, it is interesting that although the changes in R–R intervals induced by muscle contraction were greater after training, the heart rate achieved was still less than that evoked before training. This means that following training all the subjects performed the same level of muscle contraction at a lower heart rate. Since the maximum heart rate was the same they therefore had

a greater range over which they could increase their heart rate, thus confirming for isometric contraction what has long been known for dynamic exercise (Astrand and Rodahl 1986).

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