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Aerobic group training improves exercise capacity and heart rate variability in elderly patients with a recent coronary event

A randomized controlled study

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Aims Reduced heart rate variability is associated with an unfavourable prognosis in patients with ischaemic heart disease. Whether physical training can modify this risk factor is not definitely proven. Our hypothesis was that training might increase both physical capacity and heart rate variability in elderly patients recovering from an acute coronary event, i.e. acute myocardial infarction (n=38) or an episode of unstable angina (n=27).

Methods and Results 24 h ambulatory ECG recordings were obtained from 65 patients randomized to either a 3 months supervised outpatient group training programme 50 min three times a week (n=29) or to a control group (n=36). The two groups were well balanced as regards demographic data and pharmacological treatment at the time of randomization. Body mass index and pharmacological therapy remained unchanged during the study. Heart rate variability was analysed in the time and frequency domains. At the 3 month follow-up, exercise tolerance had increased from 103 to 120 W in the training group ($P<0{\cdot}001$), and from 102 to 106 W in the control group (ns). The time–domain heart rate variability measures SDNN (standard deviation of all filtered RR intervals over the analysed time period) and SDANN (standard deviation of the means of all filtered RR intervals for all 5 min epochs of the analysed time period) increased significantly during the daytime in the training group ($P<0{\cdot}01$ and $P<0{\cdot}05$, respectively), but not in the control group. A significant improvement in night-time heart rate variability was observed among controls. There was a statistically significant correlation ($P<0{\cdot}05$) between changes in 24 h overall power (frequency domain measure) and changes in maximal exercise capacity in the training group.

Conclusion A regular aerobic group training programme after an acute coronary event can significantly improve exercise capacity and modify heart rate variability in a prognostically favourable direction in elderly low-to-intermediate risk patients, recovering from an acute coronary event.

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Key Words: Heart rate variability, elderly, ischaemic heart disease, training, physical therapy, cardiac rehabilitation, randomized controlled study.

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Introduction

Heart rate variability, which is assumed to reflect autonomic nervous system modulation of cardiac activity[1] decreases with age[2] and in disease states, accompanied by autonomic neuropathy such as diabetes mellitus[3]. Reduced heart rate variability is also a risk factor associated with impaired prognosis in post myocardial infarction patients and in patients with congestive heart failure[4–8]. Treatment with drugs known to affect the prognosis of these disorders positively, such as angiotensin converting enzyme inhibitors, and negatively, such as class Ic anti-arrhythmic drugs, is associated with an increase and a decrease in heart rate variability, respectively[9–12]. There is also a relationship between degrees of physical activity and fitness and degrees of heart rate variability and risk for cardiovascular disease, respectively[13].

In a non-randomized study on relatively young (40 to 66 years old) post myocardial infarction patients,

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Table 1 Characteristics of the patients in the intervention group (Group I) and the control group (Group C) and in the subgroups participating in the heart rate variability part (bold letters) at baseline. Variables are presented as mean (age) and number (n) of patients

	All patients (n=101)	Group I (n=50)	Group C (n=51)	**Group I (n=29)**	**Group C (n=36)**
Age (years; mean (SD))	71 (4)	71 (4)	71 (5)	**71 (4)**	**72 (5)**
Range	65–84	65–84	65–83	**65–84**	**65–83**
Sex (M/F)	81/20	41/9	40/11	**22/7**	**28/8**
Diabetes mellitus	16	10	6	**6**	**5**
Hyperlipidaemia	17	9	8	**5**	**5**
Hypertension	32	18	14	**10**	**9**
Previous AMI	29	18	11	**11**	**8**
Previous angina pectoris	41	20	21	**11**	**14**
Previous PTCA	12	7	5	**2**	**4**
Previous CABG	18	9	9	**5**	**6**
Congestive heart failure	7	2	5	**2**	**1**

AMI=acute myocardial infarction; PTCA=percutaneous transluminal coronary angioplasty; CABG=coronary artery bypass graft surgery.

exercise training alone (n=19) or in combination with beta-blocker therapy (n=20) modified heart rate variability in a direction associated with better prognosis[14]. In contrast, no significant effect was found after a 6 week moderately intense supervised hospital-based training programme in 49 patients of similar age with an acute myocardial infarction[15]. In patients with ischaemic heart disease there is, thus, still a question of whether physical training can modify heart rate variability. This issue was addressed in the present study, which included a larger cohort of elderly patients randomized after an acute coronary event either to a 3 month supervised hospital-based aerobic group-training programme or to a control group. Based on the established positive relationship between physical fitness and heart rate variability, we assumed that an increase in exercise capacity, if large enough, would be accompanied by an increase in heart rate variability. Furthermore, if we could observe a correlation at the level of the individual between improvement in exercise capacity and increase in heart rate variability, that would strengthen the case for training induced heart rate variability improvement, provided confounding factors were not affected.

Material and Methods

Study patients

This study was part of a larger randomized study on the overall effects of physical training in a cohort of 109 consecutive elderly patients aged ≥65 years (mean 71; SD 4) admitted to the Coronary Care Unit at Karolinska Hospital, Stockholm, because of an acute coronary event between October 1994 and June 1997[16]. An acute coronary event was defined as either an acute myocardial infarction according to conventional criteria (n=64) or an episode of unstable angina pectoris (n=45) with anginal chest pain and dynamic electrocardiographic (ECG) changes at rest (transient/manifest T-wave inversion and/or ST depression >1 mm in more than two adjacent leads), but without any release of cardiac enzymes. To be eligible for the study the patients had to be able to perform a pre-discharge exercise test at a workload ≥70 W in males and ≥50 W in females; for patients with unstable angina pectoris, an ST60 depression of >1 mm in at least two adjacent leads had to be documented during the exercise test.

Prior to discharge, all patients received verbal and written information about the importance of regular physical activity after the acute cardiac event. They were recommended to take a daily walk at a comfortable speed, and to increase the time, length and speed of the walk gradually.

The patients were stratified according to diagnosis (acute myocardial infarction or unstable angina pectoris) to be randomized into an intervention group (Group I; n=56) or a control group (Group C; n=53) after discharge and after a baseline exercise test conducted within 6 weeks of the acute event. This protocol was chosen so that allocation to a group would not influence performance at the baseline investigation. The median time between initial hospitalization and the time of randomization was 18 days. Eight patients were withdrawn during the study because of coronary artery bypass graft surgery (CABG) (n=4), lack of time (n=2), they had moved from the area (n=1), or for an orthopaedic reason (n=1). In all, 101 patients completed the study, 50 in Group I and 51 in Group C. Patient characteristics are presented in Table 1. Out of these 101 patients, satisfactory ambulatory ECG recordings, according to our quality requirements (see below), were obtained from 65 patients, 29 Group I and 36 Group C. Twenty-seven of the 65 patients were included after an episode of unstable angina pectoris (13 with a previous

Table 2 Reasons for exclusion from heart rate variability analysis. Variables are presented as number of patients

	Group I	Group C
Quality requirements not fulfilled	11	11*
Technically deficient recording	3	2
Atrial fibrillation	2	1
Technical hitch	2	1
Pacemaker dependent	2	0
A-V block I–II	1	0
Total number (%)	21 (42%)	15 (29%)

*2 patients with suspected sinus node dysfunction.

Table 3 Pharmacological treatment of the patients in the intervention (Group I; n=29) and the control group (Group C; n=36) at baseline and after 3 months of follow-up. Variables are presented as number of patients

Type of drug	Baseline		Follow-up	
	Group I	Group C	Group I	Group C
Beta-blocker	25	32	27	30
Digitalis	0	1	0	0
Long-acting nitrate	20	19	16	19
Diuretic	7	7	8	7
ACE-inhibitor	5	5	5	5
Calcium antagonist	8	6	7	7
Aspirin	27	34	28	34
Lipid-lowering drug	6	5	5	7
Sedatives	2	3	1	4

acute myocardial infarction) and 38 after an acute myocardial infarction. Reasons for exclusion are presented in Table 2.

There was no significant difference in body mass index ($kg \cdot m^{-2}$) between groups at randomization (Group I 26·5; Group C 25·2) and no changes at follow-up (Group I 26·3; Group C 25·2).

The pharmacological treatment did not differ significantly between the two groups at baseline and there were no major changes during the study period (Table 3).

All patients gave their informed consent to participate, and the study was approved by the local ethics committee.

Training programme

The training programme consisted of 50 min outpatient group training three times a week over 3 months, described in detail elsewhere[16]. In brief, it consisted of interval training with three 4-min peaks at >85% of the individual maximal heart rate observed at the baseline exercise test[17]. The training sessions were supervized by a physiotherapist specialized in cardiac rehabilitation.

Exercise testing

Maximal exercise capacity was assessed on two occasions, before randomization and 3 months thereafter. The tests were symptom limited and performed on an electrically braked bicycle ergometer (Siemens Elema, Ergomed 840, Sweden) starting at 30 W with workload stepwise increased by 10 $W \cdot min^{-1}$[18]. Using a computerized electrocardiograph (Siemens Megacart, Sweden) a 12-lead ECG was continuously monitored during the test. Systolic blood pressure was recorded every minute, as were subjective symptoms. Perceived exertion was rated according to a 6–20 graded scale, Borg's Ratings of Perceived Exertion (RPE) scale[19], while chest pain, shortness of breath and leg fatigue were assessed using a 0–10 graded scale, Borg's Category Ratio scale, (CR 10 scale)[20]. The test was terminated due to fatigue (defined as an RPE score of 15–17/20), severe angina (grade ≥ 5 of 10), severe symptomatic arrhythmia, a fall in blood pressure >10 mmHg at two consecutive workloads or an ST60 depression >2 mm. All exercise tests were supervised by the same medical technologist, who had no knowledge about group allocation.

Long-term ECG recording

Twenty-four hour ambulatory ECG monitoring was performed on a Spacelab two-channel tape recorder type 90205 or 90208 (SpaceLabs, Inc. Redmond, Washington, U.S.A.) (lead positions were approximately V_3 and V_5) just prior to randomization and after the baseline exercise test, and 3 months thereafter. The patients were encouraged to follow their ordinary daily living habits during the recording period.

Analysis of heart rate variability

Heart rate variability was analysed in the time and frequency domains by means of a commercially available system (Oxford Instruments Medical Systems Division, software version 7.5, United Kingdom). The recordings were first edited by an experienced medical technologist, kept unaware of clinical data, and normal and non-normal beats were identified. The following quality criteria were applied: for each 5-min epoch of the recording, $\geq 96\%$ normal beats were required for inclusion of that epoch. For each of the three time periods analysed, 24 h, daytime (0900h–2100h), and night (midnight to 0600h)[21], respectively, at least 75% of the 5-min epochs had to fulfil these quality requirements for inclusion in the statistical analysis.

The RR intervals which included a non-normal beat were deleted before the time–domain analysis, which included the following measures[1]: SDNN defined as the standard deviation of all filtered RR intervals over the analysed time period; SDANN defined as the standard deviation of the means of all filtered RR intervals for all

5-min epochs of the analysed time period; rMSSD defined as the square root of the mean of the sum of squares of differences between adjacent filtered RR intervals of the analysed time period.

For the frequency–domain analysis the rejected intervals were replaced by linear interpolation. The FFT (fast Fourier transform) algorithm of this software was then applied to the raw data tachogram without preceding detrending or resampling. The spectral frequency ranges were the conventional: ultra-low frequency 0·0000–0·0033, very low frequency 0·0033–0·0400, low frequency 0·0400–0·1500, and high frequency 0·1500–0·4000 Hz, respectively[1].

Running 10 recordings through the entire editing and subsequent heart rate variability analysis procedure twice at about the 6 month interval, has tested the reproducibility of this procedure. The coefficients of variation calculated from the intra-individual standard deviations were less than 5% for the time- and frequency-domain measures used in this study.

Statistical methods

Data are presented as mean, SD (standard deviation), and range, or as median and range depending on the type of data. The distribution of all heart rate variability measures was analysed for skewness with the Shapiro–Wilk W test and, if positively skewed, data were ln-transformed before statistical analysis. Analysis was performed using a two-way ANOVA (analysis of variance) with repeated measures on one factor. When a significant difference was found further statistical analysis was performed using two-sided Student's paired and unpaired t-test. Correlation analysis was performed using Spearman's rank correlation coefficient. Statistically significant differences were assumed when $P<0·05$.

Results

Exercise capacity

Exercise capacity did not differ between the two groups at baseline. There was no significant difference in resting heart rate before or maximal heart rate after the baseline exercise test. The average compliance to the training programme (actually performed training sessions divided by possible sessions) was 88% (range 64–100%). There was no complication of any kind during these sessions.

Exercise capacity increased on average by 17 W (14%, from 103 to 120 W; $P<0·001$) in Group I, and 4 W (2·9%; from 102 to 106 W; ns) in Group C, (Table 4). The changes in exercise capacity between the two groups were significantly different ($P<0·01$). Resting heart rate before the follow-up exercise test decreased significantly in comparison with the control group ($P<0·05$), while the maximal heart rates at the end of this test did not change from baseline.

Heart rate and heart rate variability

Of the patients included in the analysis of 24 h heart rate variability 27 were in Group I and 33 in Group C. At least 24 and 32, respectively were included for day-time analysis, at least 27 and 35 for night-time analysis, and at least 23 and 31 for the ratio between day and night, respectively.

Baseline heart rate — 24 h as well as day and night heart rate — was slightly, but not significantly lower in Group I, with a 24 h mean heart rate of 63 beats . min^{-1} (SD 6), than in Group C (mean 66, SD 7 beats . min^{-1}). There were no significant changes in heart rate, or in the relationship between the heart rate during the day and night time periods, respectively, over the study period (Table 4).

The time–domain analysis, which included all patients, showed a consistent pattern of increased 24 h and day-time heart rate variability in Group I, while the results for Group C varied. In the former group there was a statistically significant increase in day-time SDNN ($P<0·01$) and SDANN ($P<0·05$). The night-time heart rate variability was slightly higher at baseline in Group I than in Group C, but did not change during the training period. In contrast, night-time heart rate variability increased in Group C, and was statistically significant for rMSSD ($P<0·001$), (Table 4).

The frequency–domain analysis pattern was similar to the time–domain analysis, and showed a — non-significant — increase in overall power in Group I, consistent with the increase in SDNN. A consistent pattern in the frequency domain analysis was a statistically significant difference in the directional changes of the day-to-night ratio in the two groups. While this ratio increased in Group I, it decreased in Group C. The night-period power increased in both groups, while the day-period power increased only in the training group. Except for the ultra-low frequency band, these ratios were <1, i.e. there was more power in the shorter night than in the day period. The relative contribution of the different frequency bands to the overall power was similar in the two groups and remained stable over time. At baseline, the respective percentage powers were: ultra-low frequency 14% in Group I and 13% in Group C, very low frequency 61% and 62%, low frequency 16% and 17%, high frequency 9% and 8%.

In order to analyse the changes in exercise capacity and the changes in heart rate variability on an individual level (overall power and SDNN), the relationships had to be studied for the 'spontaneous' and 'training induced' changes in the entire population, and those in the training group alone (Fig. 1(a) and (b)). There was a statistically significant ($P<0·05$), but biologically weak correlation between changes in 24 h ln overall power and maximal exercise capacity in the training group.

Table 4 Exercise capacity (W), heart rate and heart rate variability at baseline and after three months of follow-up in trained (Group I) and control (Group C) subjects. Values are presented as mean and standard deviation (SD). Heart rate variability values are presented as ln-transformed data in ms in the time domain and in ms^2 in the frequency domain analogues. For number of patients see text

	Baseline		Follow-up	
	Group I	Group C	Group I	Group C
Watts	103 (19)	102 (28)	120 (22)	106 (36)
Heart rate; 24 h	63 (6)	66 (7)	62 (8)	67 (8)
Heart rate; daytime	68 (8)	69 (8)	67 (9)	71 (9)
Heart rate; night-time	57 (6)	61 (7)	56 (7)	60 (6)
Heart rate; d/n	1·20 (0·1)	1·14 (0·1)	1·20 (0·1)	1·19 (0·1)
SDNN; 24 h	4·85 (0·2)	4·76 (0·3)	4·93 (0·3)	4·84 (0·3)
SDNN; daytime	4·63 (0·2)	4·65 (0·3)	4·73 (0·2)	4·63 (0·3)
SDNN; night-time	4·45 (0·2)	4·41 (0·3)	4·45 (0·3)	4·49 (0·3)
SDANN; 24 h	4·72 (0·3)	4·63 (0·2)	4·78 (0·3)	4·70 (0·3)
SDANN; daytime	4·47 (0·2)	4·49 (0·3)	4·58 (0·2)	4·48 (0·3)
SDANN; night-time	4·11 (0·3)	4·08 (0·4)	4·09 (0·3)	4·13 (0·3)
rMSSD; 24 h	3·36 (0·4)	3·33 (0·4)	3·41 (0·4)	3·38 (0·4)
rMSSD; daytime	3·21 (0·4)	3·28 (0·4)	3·33 (0·5)	3·24 (0·3)
rMSSD; night-time	3·47 (0·4)	3·30 (0·4)	3·45 (0·3)	3·47 (0·5)
Overall power; 24 h	7·94 (0·5)	7·93 (0·5)	8·00 (0·4)	8·0 (0·5)
Overall power; daytime	7·83 (0·4)	7·87 (0·5)	7·97 (0·4)	7·97 (0·5)
Overall power; night-time	7·95 (0·6)	7·88 (0·6)	8·03 (0·6)	8·13 (0·6)
Overall power; d/n	0·97 (0·0)	0·99 (0·1)	0·99 (0·1)	0·96 (0·1)
ULF; 24 h	5·92 (0·5)	5·87 (0·5)	6·02 (0·5)	5·87 (0·5)
ULF; daytime	5·93 (0·5)	6·0 (0·6)	6·11 (0·5)	5·99 (0·6)
ULF; night-time	5·56 (0·6)	5·31 (0·7)	5·6 (0·7)	5·52 (0·8)
ULF; d/n	1·06 (0·1)	1·12 (0·1)	1·10 (0·1)	1·08 (0·1)
VLF; 24 h	7·44 (0·4)	7·45 (0·5)	7·53 (0·4)	7·52 (0·5)
VLF; daytime	7·35 (0·4)	7·40 (0·5)	7·50 (0·4)	7·41 (0·6)
VLF; night-time	7·51 (0·6)	7·45 (0·6)	7·59 (0·6)	7·62 (0·6)
VLF; d/n	0·98 (0·1)	0·99 (0·1)	0·99 (0·1)	0·97 (0·1)
LF; 24 h	6·04 (0·6)	6·10 (0·6)	6·08 (0·5)	6·26 (0·7)
LF; daytime	5·75 (0·6)	5·92 (0·6)	5·90 (0·6)	5·87 (0·6)
LF; night-time	6·07 (0·7)	6·16 (0·7)	6·14 (0·8)	6·47 (0·9)
LF; d/n	0·93 (0·1)	0·96 (0·1)	0·95 (0·1)	0·91 (0·1)
HF; 24 h	5·31 (0·8)	5·23 (0·7)	5·34 (0·7)	5·31 (0·8)
HF; daytime	4·96 (0·9)	4·96 (0·8)	5·14 (0·9)	4·84 (0·6)
HF; night-time	5·34 (0·7)	5·18 (0·8)	5·40 (0·7)	5·44 (1·0)
HF; d/n	0·90 (0·1)	0·96 (0·2)	0·93 (0·1)	0·90 (0·1)
LF/HF; 24 h	1·15 (0·1)	1·17 (0·1)	1·15 (0·1)	1·19 (0·1)
LF/HF; daytime	1·18 (0·1)	1·21 (0·1)	1·17 (0·2)	1·22 (0·1)
LF/HF; night-time	1·15 (0·1)	1·20 (0·1)	1·44 (0·1)	1·21 (0·2)

SDNN=standard deviation of all filtered RR intervals over the analysed time period; SDANN=standard deviation of the means of all filtered RR intervals for all 5-min epochs of the analysed time period; rMSSD=the square root of the mean of the sum of squares of differences between adjacent filtered RR intervals; ULF=ultra-low frequency; VLF=very low frequency; LF=low frequency; HF=high frequency.

There was, however, no correlation between changes in exercise capacity and changes in day- or night heart rate variability measures in the two groups.

Discussion

In this study on elderly patients, regular physical training after an acute coronary event increased the maximal exercise capacity and heart rate variability in the training group. The increase in heart rate variability in this group was mainly a reflection of increased day-time heart rate variability, while there was no change in mean heart rate. Since the night-time heart rate variability only increased minimally, the result was a — seemingly paradoxical — decrease in the day-to-night ratio, which has previously been associated with disease rather than health in subjects approximately 20 years younger[21]. Importantly, our observations are consistent with those from a study comparing elderly athletes (n=11, 73 (3) years) with age- and sex-matched healthy but more sedentary controls (n=12, 75 (3) years)[22]. In this study,

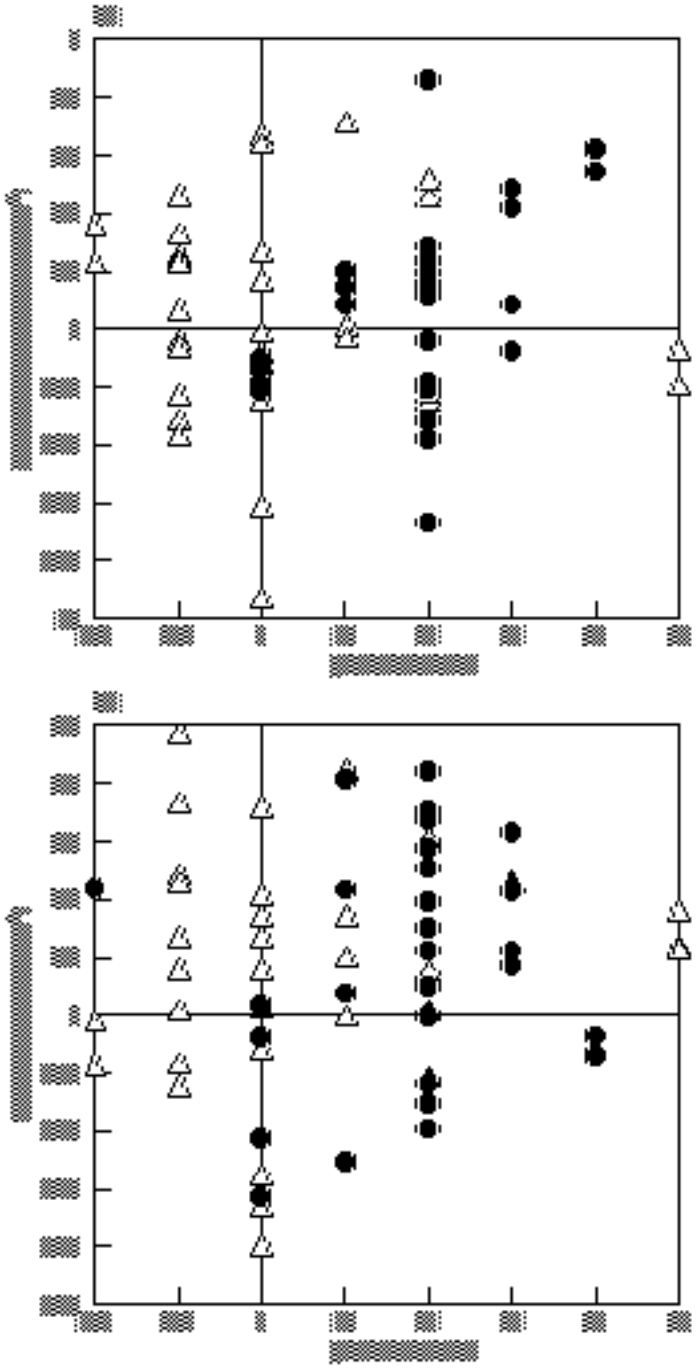

Figure 1 **(a) Correlation between changes in exercise capacity and changes in heart rate variability (overall power 24 h) from baseline to follow-up in Group I (n=27) and Group C (n=33). r_s were 0·38 ($P<0·05$) and −0·01 (ns), respectively. (b) Correlation between changes in exercise capacity and changes in heart rate variability (SDNN 24 h) from baseline to follow-up in Group I (n=29) and Group C (n=36). r_s were 0·16 and 0·20, respectively (both ns). ●=intervention; △=control.**

there was little difference in nightly overall power, but on average there was approximately 80% more daytime overall power among the athletes. Thus, it seems that physical training, at least at the intensity in the cited and the present studies, increases the autonomic modulation responsiveness of heart rate during daily activities. The 'paradox' seems thus resolved, and our coronary patients responded in a healthy fashion.

Methodological aspects

In the classical paper by Bigger and co-workers[6] on post-myocardial infarction patients (<70 years old), tape or cassette recordings were included if at least 50% of the recording time was analysable and ≥50% was sinus rhythm; 152 of 867 patients [18%; (95% CI: 15–21%)] were excluded because of inadequate, technically deficient or missing recordings. We applied harder criteria, and had to exclude 36 of our patients [36%; (95% CI: 27-45%)], fortunately leaving a representative sample of patients with analysable recordings (Tables 1–3). The reason for using such strict criteria was to optimize the possibility of detecting a change in heart rate variability associated with the intervention. For the same reason we chose a significance level of $P<0{\cdot}05$, without correction for multiple analyses. The guidelines issued jointly by the NASPE and ESC Task Force recommend that 18 analysable hours out of 24 should be required, adding that the entire night recording should be adequate; our criteria come close to this recommendation. These guidelines also mention the possible impact of non-normal beats, but provides no recommendation as to the highest proportion acceptable. Huikuri and co-workers[21] required >90% normal beats, while we were even more strict when requiring ≥96% normal beats. Recommendations on this issue would be welcome. We chose the same day- and night-time periods used by Huikuri *et al.*[21], whose study was also based in Scandinavia.

Physiological correlates to heart rate variability measures — lack of knowledge

The results are presented and analysed in ln-transformed values, due to the skewed distribution, but we have not calculated so-called normalized units, and only provided in tabular form the ratio between the powers of the low frequency and high frequency bands. This is a reflection of the presently limited understanding of the physiological correlates to different measures of heart rate variability, as discussed in recent publications[1,23–25], which explains our hesitation to attempt interpretation of our observations in terms of interaction between the sympathetic and parasympathetic limbs of the autonomic nervous system.

Heart rate variability — as a phenomenon — presumably reflects cardiac autonomic modulation, provided the sino-atrial function is unaffected by any disease process, a generally assumed but usually not tested prerequisite[26]. We excluded from the heart rate variability analysis two patients with suspected sinus node dysfunction based upon the presence of exaggerated variations of their sinus node-related RR intervals. The understanding of the physiological — not to mention the pathophysiological — correlates to different heart rate variability measures is incomplete and controversial[23,25]. From this perspective, the heart rate variability response to systematic physical training, successful as judged from improvement in physical fitness, becomes particularly interesting in this group of elderly patients with clinically significant coronary artery disease. Increasing age and coronary artery disease are both associated with decreased heart rate variability[2,27].

The degree of heart rate variability is, in different populations, correlated with the level of physical fitness. One previous non-randomized study of post myocardial infarction patients showed an increase in heart rate variability during rehabilitation[28], while in another randomized study such an effect was not revealed[15]. Since there is a spontaneous increase in heart rate variability in the first months after an acute coronary event[29,30], randomization is required. It is, however, difficult to prove that physical training per se improved heart rate variability. Indirect evidence to support this assumption is that training significantly increased exercise capacity and decreased the standardized assessment of heart rate at rest before the exercise test in the training group, while these variables remained unchanged in the control group. Importantly, there were no significant changes in body mass index or pharmacological therapy that might have influenced heart rate variability. Along the same lines, it is crucial to analyse any correlation on an individual level, between changes in physical capacity and heart rate variability measures — occurring with or without association with systematic aerobic training. We did observe such a relationship. With regard to the extreme complexity of the influence on heart rate variability, it is not surprising that this relationship was weak, and not detectable in most heart rate variability measures. Taken together, the above evidence suggests that physical training was the cause for the increase in heart rate variability. Whether this improvement in heart rate variability translates into a more favourable prognosis was not addressed in the present study. According to a review on this subject, several thousands of patients would be required to prove a 20% reduction in cardiovascular-related mortality by a rehabilitation programme which included physical training[31].

Study group

Although there are problems with comparisons of frequency–domain measures between different studies, time–domain measures can be compared more reliably. The baseline average SDNN in our patients was of the

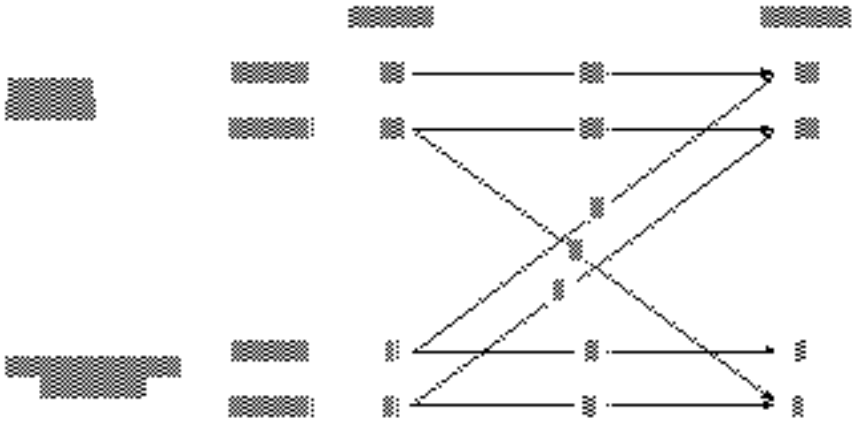

Figure 2 **Risk-group classification in heart rate variability at baseline and follow-up in Group I and Group C, and changes over time. SDNN strata according to Kleiger[4] were used. No patient was in the high risk stratum, SDNN <50 ms, while two patients, one from each group, had a SDNN <70 ms, which was the cut-off in the ATRAMI (Autonomic Tone and Reflexes After Myocardial Infarction) study[40].**

same magnitude as in the study by Huikuri *et al.*[32], and when applying the same risk stratification as Kleiger *et al.*[4] similar results were found. During the study period, 96% of the low-risk patients remained in the same risk group, while 50% of the intermediate-risk patients improved in this aspect, Fig. 2.

Physical training in elderly coronary patients

In the early years of exercise rehabilitation of patients with coronary heart disease, an age exceeding 65 years was mostly, and on arbitrary grounds, considered to be an exclusion criterion[33]. Although older coronary patients may improve exercise capacity to an extent similar to that of younger patients[34–36], they are less likely to be referred to cardiac rehabilitation programmes[37,38]. Low physical fitness is an important risk factor in both men and women, and higher levels of physical fitness appear to delay all-cause mortality primarily due to lowered rates of cardiovascular disease[13]. A change in physical fitness also changes the mortality risk in men, as reported by Blair *et al.* in 1995, who found that men who maintained adequate physical fitness were less likely to die from all causes and from cardiovascular disease than unfit men[39].

Based on the recently published ATRAMI (Autonomic Tone and Reflexes After Myocardial Infarction) study, showing independent and additive predictive values of heart rate variability and vagal reflexes, assessed as baroreceptor sensitivity on prognosis post acute myocardial infarction[40], physical training has been recommended in such patients[8]. This study proves that physical training can be performed without complications in an elderly population after an acute coronary event and with a successful modification of exercise capacity and heart rate variability as a result. However, whether this is due to improved vagal sensitivity, to a modified sympathetic response, to an increased overall responsiveness to autonomic modulation, or to other mechanisms, remains to be established.

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