ELSEVIER

Biological Psychology 49 (1998) 123–135

BIOLOGICAL PSYCHOLOGY

Cardiorespiratory effects of breathing and relaxation instruction in myocardial infarction patients

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Abstract

The effect of individual instruction in relaxation and breathing, additional to an exercise training program, was investigated in 76 post-myocardial infarction patients after rehabilitation and at 3 months follow-up. Respiration rate (RR), heart rate (HR) and respiratory sinus arrhythmia (RSA) were the outcome variables used to compare experimental (exercise plus relaxation) and control (exercise without relaxation) groups. HR and RR decreased slightly during 20-min sessions of supine measurement. This response did not vary between sessions (pre-rehabilitation, post-rehabilitation and after 3-month follow-up). RSA tended to decrease during the sessions. The within-session reduction in RSA became more apparent in the control group after treatment and less so in the experimental group. RR decreased in the experimental group after rehabilitation, but not in the control group. HR decreased for all patients, but the decrease was larger in the experimental group. This effect was associated with the lower RR. RSA did not change in the control group but increased in the experimental group, during both normal and deep breathing. This effect was also associated with a slower RR and became marginally significant when RR was statistically controlled for. We conclude that the relaxation intervention induced a slower breathing pattern which was associated with beneficial effects on resting HR and RSA. Further study is warranted to clarify the degree to which reduced respiration rate is an indicator of lower sympathetic arousal or merely a concomitant of the learned breathing technique. © 1998 Elsevier Science B.V. All rights reserved.

Keywords: Relaxation; Breathing therapy; Physical exercise; Myocardial infarction; Respiration; Respiratory sinus arrhythmia

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PII S0301-0511(98)00031-3

1. Introduction

Autonomic imbalance in cardiac regulation has received increasing attention as a possible mechanism for cardiac complaints and as a prognostic factor in heart disease (Niemelä et al., 1990; Coumel et al., 1991; Cripps et al., 1991; Lombardi et al., 1992; Buchanan et al., 1993; Detollenaere et al., 1993). Conversely, improvement of autonomic imbalance may influence prognosis of and recovery from acute heart disease and may partly explain the benefit of physical exercise, psychosocial stress management and relaxation training (Frasure-Smith and Prince, 1989; Buchanan, 1992; Kiilavuori et al., 1995). Previous reports have indicated that breathing and relaxation instruction added to a program of exercise rehabilitation improved psychological and physical outcome of rehabilitation after myocardial infarction (MI) and reduced the occurrence of cardiac events over a 2-year follow-up period (van Dixhoorn et al., 1987). The mediating physiological processes that account for such effects need further investigation. In this report the effects of breath relaxation on respiration, resting heart rate and respiratory sinus arrhythmia (RSA) are examined in post-MI patients, after rehabilitation and at 3 months follow-up.

2. Methods

2.1. Patients

The study population consisted of 156 myocardial infarction patients, referred for rehabilitation soon after hospital discharge. At intake they were asked to participate in the study and upon consent they were randomly assigned to either the usual exercise rehabilitation program only (control group, CG), or to exercise plus additional sessions for relaxation therapy (experimental group, EG). For this report complete physiological data of 76 patients were available. They were 74 men and two women, 39 in the EG and 37 in the CG.

2.2. Interventions

The exercise training consisted of 5 weeks of daily bicycle-ergometer interval training for 30 min. After each session there was a possibility to change, sit together and talk. Training was done in groups of four supervised by two physical therapists. Each patient exercised up to 70% of the heart rate reserve, established by submaximal exercise testing.

Relaxation training was provided once a week in six individual 1-h sessions by five specially trained persons, three psychologists, one physician and one physiotherapist. Electromyographic (EMG) feedback of M. frontalis was used to clarify the concept of muscle relaxation, during exercises which entailed alternate active muscular contraction and passive release of tension. EMG feedback was also employed to monitor excessive motor responses during breathing exercises (van Dixhoorn and Duivenvoorden, 1989).

The basic procedure for breathing instruction involved alternating periods of passive attention to spontaneous breathing and active regulation of respiration. Respiratory movement was first brought to the attention of the patient in a passive way. With one hand on the abdomen the patient was asked to follow the breathing pattern through the body, without any active interference. Then respiration was modified voluntarily, as described below. Thereafter, the patient once again breathed normally and was asked to monitor respiratory movement passively and to perceive differences between active influence and passive attention to breathing. Several such alternations of passive and active attention took place during the session. Active modification of breathing included: (1) making the passage of air audible for five to six respiratory cycles by breathing through slightly pursed lips; and (2) employing indirect movements of the body that facilitated respiration, e.g. by flexing the feet with exhalation. The latter manipulation was either performed by the patient alone or with the aid of the therapist who used it as a manual technique. It was emphasized that the patient should *not* try to breathe slowly and abdominally all the time. Patients were asked to practice relaxation daily and to use breath monitoring and regulation as often as they wished.

2.3. Measurements

Measurements were made during three sessions, before rehabilitation, after exercise rehabilitation and once again after 3 months follow-up. Experimental subjects received relaxation training during the rehabilitation phase whereas control patients had no such treatment during this period.

Recordings of physiological data for each session took place during a test introduced to the patient as a physiological measurement of the resting condition (additional to an exercise test). The research assistant did not refer to the relaxation therapy and made it a point to avoid any special attention to breathing. When the patient appeared to practice breath control, he was told to not pay attention to his breathing and just breathe normally. Respiratory movements were recorded by stretch-sensitive bands strapped around the chest and abdomen, and a finger plethysmograph used to measure heart rate which was converted by a cardiotachometer into beat-to-beat heart rate. Both signals were recorded on a polygraph (ZAK). The paper tracings were kept for analysis. Respiration rate was counted in cycles per min (cpm) and heart rate as the average beat-to-beat rate per min (bpm). Respiration sinus arrhythmia was quantified according to the peak-to-trough procedure (Grossman and Kollai, 1993), converting heart rate to heart period in ms. Inspiratory minimum heart period was subtracted from expiratory maximal heart period and this difference was averaged across respiratory cycles during one min.

Once the measurement equipment had been attached in the standing position, the patient was then asked to lie down in the supine position. After 3 min, a mouthpiece and noseclip were attached for assessment of end-tidal CO_2 (via capnography). The patient was first asked to breathe normally, then slowly and deeply for five consecutive respiratory cycles. The patient subsequently breathed

into a spirometer for 1 min, and again repeated capnographic measurement with slow deep breathing. In the next phase, the subject was asked to lie quietly still for some time. This rest phase took 6 min. Then capnographic measurement was repeated, i.e. first normal breathing and then slow and deep respiration. The total duration of a session was about 20 min. For this report, data were used from the following four 1-min measurement periods: (1) the second min after lying down; (2) slow, deep breathing during capnography at the beginning; and at (3) end of the test; and (4) the last minute of the 6-min supine rest period. The square root of RSA were used for analysis which served to normalize distribution of RSA values.

2.4. Data analysis

Each of the three variables was analysed with a repeated-measure Manova with one grouping level (CG vs. EG) and three repeated measures: 'time', to compare changes before and after rehabilitation, and at follow-up, 'rest', to compare the beginning and the end of the test, and 'breath', to compare normal and deep breathing during capnography. Roy-Bargman stepdown F-tests were used to test differences in linear and quadratic trends over time. The cardiovascular data were also analysed with respiration rate as a covariate, in order to control for changes in respiratory pattern.

3. Results

3.1. Clinical data

This report is based on 39 patients out of the original experimental group (EG) of 76 (51%) and 37 out of the control group (CG) of 80 (46%). Complete physiological data were unavailable in 80 patients, largely because the test was not carried out in the initial period of the study, but also either because some measurements did not yield adequate recordings, or patients did not return for the follow-up measurement. All patients had survived a recent myocardial infarction, 24 (32%) had a large infarction (EG 26%, CG 38%), 26 (34%) had an anterior infarction (EG 38%, CG 30%) and 12 patients (16%) had mild heart failure in hospital (EG 10%, CG 22%). There was no difference in these baseline clinical variables between treatment groups, nor between the study group and the original population. At discharge from hospital, 24 patients were using β-blocking medication (EG 28%, CG 35%), eight patients heart glucosides (EG 13%, CG 8%) and 21 patients diuretics (EG 28%, CG 27%). At follow-up, β-blocking medication had increased a little (EG 33%, CG 41%). There were no significant differences in medications between experimental and control groups or between the study group and the original population. Thus the use of β-blockers or other drugs did not show a different pattern between the groups and could not account for any cardiovascular differences found.

The rehabilitation program started in the 1st month after hospital discharge and ended in the 3rd month. Follow-up measurements were done 3 months after that, which was on an average of 6–7 months after hospital discharge. Exercise tolerance improved among 57% of the patients (EG 61%, CG 51%), according to a previously described criterion derived from exercise testing (van Dixhoorn et al., 1989); among 26% there was no change, and 17% (EG 13%, CG 22%) worsened in exercise tolerance after rehabilitation or stopped training for medical reasons. The original study population contained more patients with a training failure (25%, EG 20%, CG 33%) and less patients with a successful outcome (52%, EG 55%, CG 46%), χ^2 for trend, 5.1, $df = 1, p < 0.05$. Thus, there were fewer patients who worsened or stopped training in the study group. However, outcome did not differ between the treatment groups: both in the study group and the original population, patients in the experimental group tended to have more often a successful and less often a negative outcome of training.

The outcome of the relaxation intervention was rated by the therapists at the last session. A total 16 patients (41%) appeared to be able to apply the instructions well, 17 patients (44%) were only partially successful, whereas six patients (15%) could not master the techniques at all. These percentages were very similar to those of the original population (43, 43 and 14%, respectively). Most patients reported to practice regularly: 79% practised daily, 10% a few times a week and 11% rarely practised or never at all. The same percentages were found in the original population, 77, 12.5 and 10.5%, respectively.

Thus, the study population consisted of fewer patients with an unfavourable outcome of exercise rehabilitation, but did not differ in base-line characteristics, nor in success of the relaxation intervention from the original population. No significant differences were found between the two treatment groups that could account for possible differences in the physiological outcome.

3.2. Respiration

After treatment respiration rate showed a reduction of about 2 cpm in the experimental group, whereas it did not change in the control group (Table 1). The pattern of change appeared in a linear trend and differed statistically significantly between the groups, Roy-Bargman Stepdown $F(1,74) = 11.7$, $p < 0.001$. The effect of 'rest' was significant, $F(1,74) = 43.6$, $p < 0.001$. There was a small but consistent decrease in respiration rate, of about 1 cpm, during the test. This respiratory response to rest occurred in both treatment groups. It did not change after treatment, time × rest: $F(1,74) = 0.14$, $p = 0.89$, nor did the intervention modify it, time × rest × treatment: $F(1,74) = 0.37$, $p = 0.69$.

Respiration rate fell to about 7 cpm when the patients were instructed to breathe slowly and deeply during capnography, breath: $F(1,74) = 3.10$, $p < 0.001$. The effect was present in both groups. It did not change after treatment, time × breath: $F(1,74) = 0.36$, $p = 0.55$. The contrast was smaller at the end of the test in both groups, because breathing had already become slower, rest × breath: $F(1,74) = 63.0$, $p < 0.001$. Also, the contrast between normal and deep breathing decreased in the

experimental group after the intervention, because their normal breathing pattern had become slower, time × breath × treatment: $F(1,74) = 11.7$, $p < 0.001$.

The results are summarized in Table 4. The intervention induced a slower respiratory pattern in the experimental group, which remained present after the intervention had ended, and did not interfere with the responsiveness of breathing to the situation of relatively reduced energy requirement in the supine position (rest).

3.3. Resting heart rate

At the start of rehabilitation average heart rate was under 70 bpm, which is relatively low. After rehabilitation it decreased in both groups in a linear trend, Roy-Bargman Stepdown $F(1,74) = 10.7$, $p = 0.002$, and in a curvilinear trend, Roy-Bargman Stepdown $F(1,73) = 5.4$, $p < 0.05$. Inspection of the data (Table 2) showed that in the control group heart rate decreased by about 3 bpm post treatment, whereas the difference with baseline resulted in less than 2 bpm at follow-up. This is a curvilinear trend. By contrast, heart rate reduction in the experimental group continued post treatment and was 5.5 bpm at follow-up. The difference between the groups was statistically significant, time × treatment: Roy-Bargman Stepdown $F(1,74) = 4.6$, $p < 0.05$. The difference lost statistical significance when respiration rate was included in the analysis as a covariate, $F(1,72) = 2.5$, $p = 0.12$.

There was a significant effect of 'rest' on heart rate, independent of treatment group, $F(1,74) = 16.2$, $p < 0.001$. Heart rate slowed down in the course of the test. This response did not change after rehabilitation, rest × time: $F(1,74) = 1.5$, $p = 0.22$, nor was it influenced by the relaxation intervention, rest × time × treatment: $F(1,74) = 1.27$, $p = 0.26$. The effect of 'breath' was also significant, $F(1,74) = 7.2$, $p < 0.01$, and independent of the treatment group. Heart rate decreased about 1 bpm with deep breathing. The effect did not change after rehabilitation, breath ×

Table 1
Respiration rates for patients with and without relaxation instruction

		Begin		Capno		Rest		Capno	
	n	X	SD	X	SD	X	SD	X	SD
Pre-rehabilitation									
With relaxation	39	14.5	3.7	7.1	1.2	13.1	3.7	7.3	1.4
Without relaxation	37	14.8	4.7	7.3	1.3	13.0	4.4	7.2	0.9
After rehabilitation									
With relaxation	39	12.8	4.4	6.8	1.2	11.6	4.3	6.9	0.8
Without relaxation	37	15.1	3.5	7.3	1.0	13.4	3.4	7.0	0.8
Follow-up									
With relaxation	39	12.4	4.8	6.8	1.1	11.2	4.0	6.7	1.0
Without relaxation	37	15.5	4.0	7.0	0.9	13.9	3.1	7.0	0.7

Table 2
Resting heart rate-for patients with and without relaxation instruction

		Begin		Capno		Rest		Capno	
	n	X	SD	X	SD	X	SD	X	SD
Pre-rehabilitation									
With relaxation	39	69.1	14.1	68.1	14.2	67.9	13.2	66.9	12.4
Without relaxation	37	67.3	12.7	66.7	12.8	66.1	12.0	65.5	12.0
After rehabilitation									
With relaxation	39	64.8	13.0	64.6	12.7	63.8	12.0	63.6	11.7
Without relaxation	37	64.3	13.0	64.3	12.4	63.2	11.3	62.2	11.6
Follow-up									
With relaxation	39	63.3	10.5	61.9	10.1	62.6	11.4	62.2	10.4
Without relaxation	37	65.9	12.2	65.0	12.4	64.9	11.5	63.5	11.1

time: $F(1,74) = 0.54$, $p = 0.46$, and it was not influenced by the intervention, breath × time × treatment: $F(1,74) = 0.33$, $p = 0.57$. Thus, despite relatively low initial values, resting heart rate was lower after rehabilitation and at follow-up particularly in the experimental group (Table 4). This effect of the intervention was associated with the reduced respiration rate. There was a consistent response of heart rate to the period of lying supine, as well as to deep breathing.

3.4. Respiratory sinus arrhythmia

Average RSA did not change in the control group after rehabilitation, but increased in the experimental group, both during normal and deep breathing (Table 3). The pattern of change showed a linear trend and was statistically significant between the groups, time × treatment: Roy-Bargman Stepdown $F(1,74) = 7.7$, $p < 0.01$. The difference in RSA was largest at follow-up, with values of about 25% higher in the experimental group. When respiration rate was employed as a covariate to the analysis, the difference between groups lost statistical significance, $F(1,72) = 2.28$, $p = 0.14$.

Despite the fact that respiration and heart rate slowed down during rest, RSA did not increase but tended to become smaller in both groups, $F(1,74) = 3.39$, $p = 0.07$. This response did not change after treatment, time × rest: $F(1,74) = 1.84$, $p = 0.18$, but tended to become more apparent in the control group and disappeared in the experimental group. Thus, the groups differed in this respect, time × rest × treatment: $F(1,74) = 3.97$, $p = 0.05$. When respiration rate was controlled statistically, the change in RSA response to rest between the groups remained marginally significant, $F(1,72) = 3.39$, $p < 0.07$.

There was a strong effect of 'breath' on RSA, as was expected. Voluntary deep and slow breathing at 7 cpm increased RSA greatly, to about 30–50% of spontaneous breathing values, $F(1,74) = 205.0$, $p < 0.001$. The effect did not change after rehabilitation, time × breath: $F(1,74) = 0.87$, $p = 0.35$ and was independent of rest,

rest × breath: $F(1,74) = 0.89$, $p = 0.35$. Despite the higher RSA values during spontaneous breathing after the intervention, this response to deep breathing was unaffected in the experimental group, time × breath × treatment: $F(1,74) = 1.93$, $p = 0.17$. When the effect of relaxation was analysed separately for the controlled deep breathing measurement, a significant difference appeared: time × treatment: Roy-Bargman Stepdown $F(1,74) = 4.81$, $p = 0.03$. This effect remained significant when the respiration rate was controlled for statistically, Roy-Bargman Stepdown $F(1,72) = 3.95$, $p = 0.05$.

RSA, therefore, was not influenced by exercise training but the relaxation intervention did increase RSA values (Table 4). This effect was associated with the slower respiratory pattern in the experimental group. The RSA response to rest was small, but changed in opposite directions for the two groups: decreasing in the control group and increasing in the experimental group. The response of RSA to deep breathing was also increased by the intervention.

4. Discussion

4.1. Limitations of the study

The results of this study suggest the existence of a long-term effect of breathing and relaxation instruction on cardiorespiratory parameters in MI patients. When six sessions of individual instruction in breath relaxation were added to an exercise rehabilitation program, patients showed, at 3 months follow-up, lower resting heart rates, slower breathing and greater respiratory sinus arrhythmia.

These changes may play a role as an intermediate process to account for the clinical benefit of stress management and relaxation instruction. A shift towards improved autonomic cardiac balance is mentioned in the literature as a mechanism for many types of intervention (Niemelä et al., 1990; Coumel et al., 1991; Lombardi

Table 3
Respiratory Sinus Arrhythmia, square root of IBI-for patients with and without relaxation instruction

		Begin		Capno		Rest		Capno	
	n	X	SD	X	SD	X	SD	X	SD
Pre rehabilitation									
With relaxation	39	6.6	2.4	9.8	3.4	6.4	2.3	9.4	3.4
Without relaxation	37	6.4	2.6	9.0	3.0	6.6	2.7	8.9	3.0
After rehabilitation									
With relaxation	39	7.7	2.8	10.3	3.2	7.3	2.4	10.4	3.0
Without relaxation	37	6.4	1.S	8.8	2.7	6.4	1.9	8.4	2.4
Follow-up									
With relaxation	39	8.0	3.0	10.9	3.6	8.2	3.1	10.6	3.5
Without relaxation	37	6.6	2.2	8.9	2.6	6.0	1.7	8.4	2.1

Table 4
Summary statistics of Manovas for three dependent variables

Factor	Respiration rate		Heart rate		RSA	
	F	*p*	*F*	*p*	*F*	*p*
Time	4.4	0.038	10.7	0.002	3.74	0.057
Time × treatment	11.7	0.001	4.6	0.05	7.7	0.01
Respiration as covariate			2.5	0.12	2.28	0.14
Rest	43.6	0.001	16.2	0.001	3.39	0.07
Tlme × rest	0.14	ns	1.5	ns	1.84	0.18
Time × rest × treatment	0.37	ns	1.27	ns	3.97	0.050
Respiration as covariate					3.39	0.069

Timc: effect of repeated measurement after rehabilitation and at 3 months follow-up. Rest: effect of supine position for about 20 min. Treatment: effect of relaxation intervention.

et al., 1992; Detollenaere et al., 1993). However, determination of underlying processes accounting for these findings is not obvious, and firm conclusions are not allowed. The main limitation of the study is that data were taken from laboratory measurements, and covered a limited timespan of four 1-min periods in each recording session. As such, our findings provide an insufficient basis for statements regarding permanent changes in a physiological state. A replication study will therefore be necessary preferably with on-line recording of heart and respiration rate during an extended period of time (Stein et al., 1994). Nevertheless, the physiological measurements showed several consistent and statistically significant patterns. Moreover, this is to our knowledge the only study on long term physiological cardiorespiratory effects of breathing and relaxation instruction in cardiac patients.

4.2. General response patterns

The 20-min period in the supine position during testing led to small but consistent reductions in heart rate and respiration rate. This pattern remained present after rehabilitation. The test appeared to be a period of physical rest, that simulated relaxation. The 'relaxation response' involves a reduction of metabolic energy requirement and results in a shift in autonomic balance towards reduced sympathetic and increased parasympathetic activity (Benson et al., 1974; Hoffman et al., 1982). Although the changes were small, the reduction in heart rate and respiration rate corresponded to the relaxation response. The increase in RSA reported by Sakakibara et al. during physical and mental rest, however, was not consistently found (Sakakibara et al., 1994). RSA values tended to decrease rather than increase during rest, in particular after treatment in the control group. The intervention group responded more favourably to physical rest in this respect (i.e. showing a modest increase at follow-up), also when respiratory changes were

controlled for statistically. This may have been due to decreases in tidal volume, which became more apparent in the control group after rehabilitation, as was shown in a previously reported analysis (van Dixhoorn and Duivenvoorden, 1989). Another possibility is that control of mental activity during physical rest, which is necessary to obtain the full relaxation response (Benson et al., 1974) occurred more often in the experimental group and less often in the control group.

The response to voluntary slow and deep breathing at 7 cpm led to a large increase in RSA and a small decrease in heart rate. The respiration induced changes in RSA represent a normal, short-term phasic pattern of parasympathetic influence upon the heart, which is not necessarily associated with changes in cardiac vagal tone (Grossman and Kollai, 1993). They illustrate the need to control for respiration in order to interpret RSA as an indicator of cardiac vagal tone. The magnitude of RSA at fixed levels of rate and depth of respiration does seem to be a reliable index of autonomic and cardiac vagal tone (Grossman and Kollai, 1993). For instance, it has been used clinically in the evaluation of diabetic autonomic neuropathy. In this study respiration was experimentally controlled during deep breathing. Patients in the intervention group had still larger RSA, which seems to indicate an effect on parasympathetic tone. However, it may also have been due to the fact that they were better able to follow instruction in slow deep breathing.

Another noteworthy response pattern was a reduction in resting heart rate after rehabilitation, despite already relatively low values. This may be interpreted as a physical training effect, or 'training bradycardia', which is also assumed to be due to a shift in autonomic cardiac balance (Maciel et al., 1985; de Geus et al., 1990; Kiilavuori et al., 1995). Interestingly, exercise without relaxation did not result in lower respiration rate or higher RSA. The absence of a training effect on RSA confirms the finding of de Geus et al.

4.3. Specific response patterns

The association between the respiratory and cardiovascular effects of relaxation is of special interest to this study of breath relaxation, because the intervention included a form of breath control. Several alternative interpretations need to be considered: (1) the intervention did not induce a change in habitual respiratory pattern, but taught the patients to modify their breathing at will, which they did during the recording session; thus cardiovascular changes were momentary responses to breath control. (2) The intervention elicited a general relaxation response or familiarized the patient with the situation of quiet repose, which led to a situation-specific lower arousal during testing. (3) The intervention successfully changed habitual breathing pattern, possibly reducing unnecessary effort; this differential or active physical relaxation led possibly to general relaxation. (4) The regular elicitation of the relaxation response, together with less effortful habitual breathing tended to generalize and to integrate within the individual psychophysiologic system, resulting in a shift in cardiac autonomic balance. For explanations (1) and (3) above, the training would result in a form of active relaxation, largely a voluntary technique (trick) in (1), more of an integration into the habitual breath-

ing in (3). In (2), relaxation may be active or passive, but it mainly served to help the patient tolerate, or even enjoy, physical and mental rest. Alternative (4) represents the most successful possible outcome, which is not necessarily feasible for all patients.

Each of these four possibilities may have actually occurred in some patients. It is important to realize that patients may respond differently to the same instruction. It is sensible therefore in clinical practice to monitor the response of the patients in order to interpret what actually happens and to tailor the instruction to the response of the individual patient. Instruction in that way becomes 'patient-driven' and makes the most of it for each individual.

Possibility (1) would apply to patients who exhibited a constant, very slow respiration pattern during the test. Voluntary application of slow, diaphragmatic breathing usually results in large tidal volumes and respiration rates around 6 cpm (Fried, 1987; Tibbets and Peper, 1993), particularly when patients are asked to show their skill. In this study the researcher tried to discourage this because we were interested in changes in spontaneous breathing. The reductions found were indeed quite modest, around 2–3 cpm on average. Moreover, the respiratory response to physical rest remained intact. Nevertheless, some patients may have practised breath control during the test. Taking as a criterion a respiration rate below 8 cpm at the beginning of the test, this occurred before rehabilitation in one patient of the control group, after treatment in two patients in the experimental group, and at follow-up in six patients, five of whom in the experimental group. Consequently breath control may have been practised during the test by several experimental group patients.

Alternative (2) was one of the purposes of the intervention, i.e. to familiarize patients with a situation of physical rest and to enhance the conditions for a relaxation response. One of the ideas that prompted the study was that cardiac patients are prone to overactivity and averse to passivity (Type A) and that relaxation would be beneficial but difficult to achieve. According to their self-reports, 31 patients from the intervention group (79%) practised relaxation daily and another four (10%) a few times per week. At follow-up 68% of subjects receiving relaxation training reported a pleasant feeling at the end of the test, in contrast to 35% in the control subjects. Thus, a substantial number of relaxation-trained patients had learned to enjoy rest. However, as stated above, it is not certain to what degree or for how many patients the physiological effects of rest also are maintained in daily life.

Possibility (3) would apply to patients who found the procedure for breath regulation beneficial without practising passive relaxation. Some patients reported using the breathing procedure mainly for active tension control, in particular to cope with angina pectoris and dyspnea during activity. The procedure contained an explicit passive phase, a period where the patient was asked to stop regulating breathing and to notice how the body was breathing by itself. The alternation of active and passive phases was included to promote general relaxation and because continued active breath control can easily lead to dysregulation (Schwartz, 1981), as for instance hyperventilation. The ability to stop deep breathing is just as important. Reduction of respiratory effort is seen in more smooth transitions between

inspiration and exhalation, a more even distribution of volume changes over the whole of the trunk and soundless passage of air. A fine-grain analysis on some of the data of the experimental group showed that their reduced respiration rate was mainly due to the lengthening of exhalation pause. This applied both to the effect of rest and to the effect of the intervention. Longer pauses after exhalation are a sign of more relaxed breathing (Umezawa, 1992) and this seems to have happened in many patients. Alternative (4) was the major purpose of the intervention. Both active breath regulation and passive relaxation were to become tools for the patient in dealing with tension, and their effect were hoped to generalize and integrate within the fabric of real world living. The very association of the effect of the intervention on heart rate, RSA and respiratory rate suggests the possibility that some degree of integration occurred. The increased effect of the intervention at follow-up also supports this idea, because the process of physiological integration needs time. As far as such generalization may have occurred, it probably took place in a limited number of patients. After six sessions of instruction only 41% of the patients were well able to apply the techniques. These patients had a lower respiration rate and higher RSA at pre-treatment than patients who did not master the techniques. Within this subgroup of successful subjects, no extra reduction in respiration rate was found, although they tended to manifest greater increases in RSA and larger heart rate reductions during rest, independent of respiration rate.

In conclusion, beneficial changes in cardiovascular variables have been found as a result of breath relaxation, but the data are insufficient to conclude that a shift in autonomic balance has occurred. It is not clear to what degree the reduced respiration rate is an indicator of lowered sympathetic arousal or an effect of habituated breathing technique. Further study is necessary to clarify these issues and to establish the possibility and desirability of modifying respiration as a tool in stress management. So far to the best of our knowledge, no such study on long-term respiratory changes has been initiated.

Acknowledgements

The help of the following persons is gratefully acknowledged: G. van Poppelen for the data acquisition, Tj. Versteeg for data coding, P. Grossman for advice on RSA analysis, C. Wientjes and H.J. Duivenvoorden for methodological advice. This study has been made possible by financial support of the National Heart Foundation.

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