The Benefit of Graded Physical Exercise in Chronic Heart Failure*

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A new program of rehabilitation is less demanding on cardiac output than standard programs. Twenty-five patients with chronic heart failure (ejection fraction [EF]: 0.26 ± 0.10) were randomized into 2 groups: a control group with 13 patients and a rehabilitation group of 12 patients. In the control group, 2 did not complete the study (cancer, cardiac transplantation). For the 11 others, the different parameters studied were comparable at day 0 with group R and did not significantly change over 3 months outside of a spontaneous improvement in endurance performance by 22%. In the rehabilitation group (40 sessions over 90 days; specialized equipment) there were no incidents. Tolerance was excellent (heart rate during sessions <115 bpm) and all functional parameters improved. Training did not modify the isotopic ejection fraction. The quality of life score increased respectively by 52% ($p<0.0001$ in comparison with the control group) and by 63% ($p<0.0001$); 80% of the patients requested that training be prolonged. The functional improvement obtained by purely peripheral effect had no adverse effect on the heart.

The balance of circulatory gas exchange, as described by Wasserman et al,[1] is upset in patients with chronic heart failure. Therefore, there is a disparity between cardiac output and the needs of the peripheral vessels in oxygen. The cardiogenic component of this imbalance is extremely difficult to treat. The limited number of active therapeutic treatments is witness to this. On the other hand, the peripheral component is much easier to approach. A number of cardiotonic treatments have, with use, proved to be very powerful vasodilators.

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Cardiac rehabilitation is one of the active therapeutic possibilities here, as most of the authors emphasize.[2-5] In addition, the action it has on the weak muscle is complementary to the drug therapy used. However, this retraining poses a risk problem for those patients whose hemodynamic profile is often very much impaired. It is for this reason that we have developed a "gentle" retraining method based on muscle building in graded stages. The hypothesis was that the demand put on the cardiovascular system by the training of one muscle group at a time would be such that even patients with chronic heart failure would be able to fill it.

Method

A trial was decided comprising 25 patients with chronic heart failure, divided at random into 2 groups: 13 in the control group and 12 in the group of patients to undergo rehabilitation treatment. The paraclinical explorations were carried out in simple blind fashion by independent observers. Drug treatment was continued throughout the trial and modified as necessary. This was a pragmatic trial.[6] The criteria for inclusion were as follows: patients with heart failure at stage 2 or stage 3 of the New York Heart Association classification with an isotopic ejection fraction at rest of less than 40%, and whose condition had been stable for more than 3 months while receiving the same drug therapy. Only patients with dilated or ischemic cardiomyopathies not limited by angina were chosen. Patients with associated respiratory failure or a history of sustained ventricular arrhythmia were excluded from the trial.

Before randomization, a detailed explanation of the program was given to each patient who was asked if he agreed to take part and they then drew lots.

The rehabilitation program (Fig 1) consisted of 40 sessions of an hour and a half each over a period of 90 days. The sessions were carried out on specially designed equipment (KOCH bench,ref. 320—Genin Médical). They consisted of the building up of a small number of muscle groups at a time and simultaneously so as to avoid

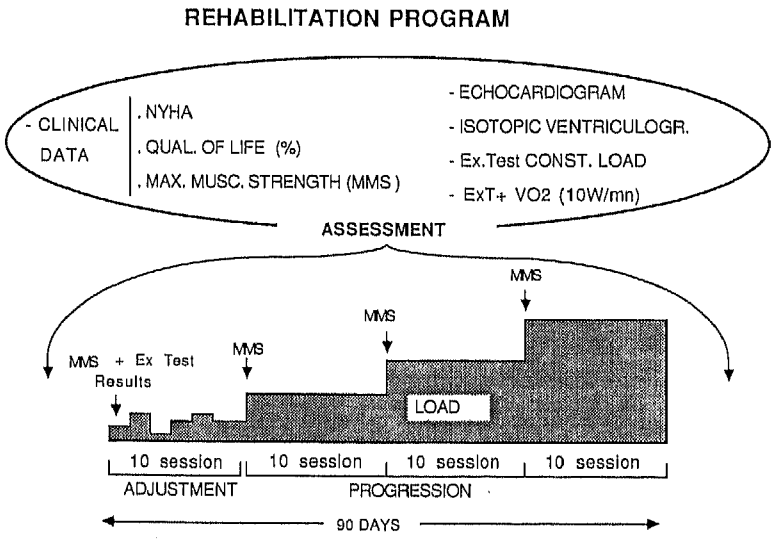


FIGURE 1. Rehabilitation program.

Table 1—*Comparability of Samples**

Variables	Rehab Group (n = 12)	Control Group (n = 13)	Significance
1			
Age, yr	56 ± 11	54 ± 10	NS
Sex, No. of men	8/12	11/13	$p<0.01$
Weight, kg	72 ± 16	68 ± 14	NS
NYHA, 3/2	5/7	5/8	NS
Yale scale	6.5 ± 2	7 ± 2.5	NS
EDD, mm	65 ± 12	69 ± 8	NS
% of shortening	21 ± 6	20 ± 6	NS
LA diameter, mm	43 ± 8	43 ± 8	NS
2			
Vo_2, ml/kg/min	18.3 ± 5	19.5 ± 8	NS
Duration, increasing load, min	8 ± 3	10 ± 5	NS
HR at rest, bpm	90 ± 16	80 ± 7	NS
SBP at rest, mm Hg	125 ± 21	118 ± 18	NS
Duration, constant load, min	10 ± 13	13 ± 15	NS
Ejection fraction at rest, %	26.5 ± 10	26 ± 12	NS
Ejection fraction during exercise, %	28 ± 11	27 ± 12	NS
P/V†	121 ± 16	120 ± 27	NS

*The two groups are comparable except for sex (more women in the rehabiliation group). Two patients were excluded from the control group (1 for neoplasia and 1 for cardiac transplantation).
†P/V = SBP/ESV = contractility index.

too much pressure on the heart. The load of each patient was chosen according to the results of the exercise test and the measurement for maximum muscular strength which was measured by a dynamometer for each muscle group on position on the training bench.

Using stress performance, the patient was classified at a given level and the total exercise load for that particular patient was set according to that level. Measuring maximum muscular strength enabled this load to be adapted to a particular muscle group. The sessions started on this basis but the physiotherapist had a period of adjustment of 10 sessions in which the load could be adjusted (Fig 1). The program was then divided into 3 groups of 10 sessions separated by a period in which muscular strength was measured, enabling the successive loads to be adapted according to the patient's progress. The number of series of movements remained constant throughout the program.

Assessment, which was carried out a few days before day 0 and on day 90, consisted of an echocardiogram, an exercise test with increasing load (ergometer, stages: 10 W/1 min, symptom-limited, under treatment) and an exercise test at constant load (as in the protocol of Sullivan et al[3]; load at 2 stages under maximal stage of test with increasing load, an isotopic ventriculogram, and a compilation of the quality of life score determined as a percentage on a visual scale (percentage of overall improvement as estimated by the patient).

Results

Two patients had to be excluded from the control group: 1 for the onset of pulmonary neoplasia and the other had to have a cardiac transplantation. The comparability of the concomitant treatment between the 2 sample groups was correct, whether for clinical and echocardiographic or for ergometric variables (Table 1). Concomitant treatment was on the whole more important in the control group, and these treatments were not modified for either group during the trial (Table 2).

The left ventricular function at rest was not significantly modified by rehabilitation (Fig 2), whether for the end diastolic diameter of the left ventricle, the percentage of shortening of the left ventricle, or the ejection fraction at rest.

During the exercise test there were no significant bradycardiac effects, either at rest (Table 3) or during exercise (heart rate for the same effort in the group undergoing rehabilitation was as follows: day 0 = 136 ± 25; day 90 = 132 ± 24; for the control group it was, respectively: 137 ± 24; 138 ± 16).

The systolic blood pressure at rest was not modified by the program either. Performance during the exercise test with increasing load was improved by 34% in the group undergoing rehabilitation while it did not improve in the control group (Fig 3).

Total muscular strength went from 77 ± 20 kg to 112 ± 24 kg (Fig 4). The improvement in the quality of life was estimated by the patients to be 63%, while the spontaneous variation in the control group was only 4% (Fig 4).

It must be emphasized that throughout the program, and for the patients undergoing rehabilitation, there were no side effects: no cardiac arrhythmia and the heart rate remained under 115 beats per minute during exercise, with

Table 2—*Associated Treatment**

	Rehab Patients, %		Control Patients, %	
	Day 0	Day 90	Day 0	Day 90
Diuretics	50	50	83	83
CEI	90	90	100	100
Digitalis and associated drugs	50	50	50	50
Vasodilators	33	33	45	45
PDI	8	8	18	18
Antiarrythmic drugs and/or β-blockers	33	33	45	45

*CEI = conversion enzyme inhibitors; PDI = phosphodiesterase inhibitors.

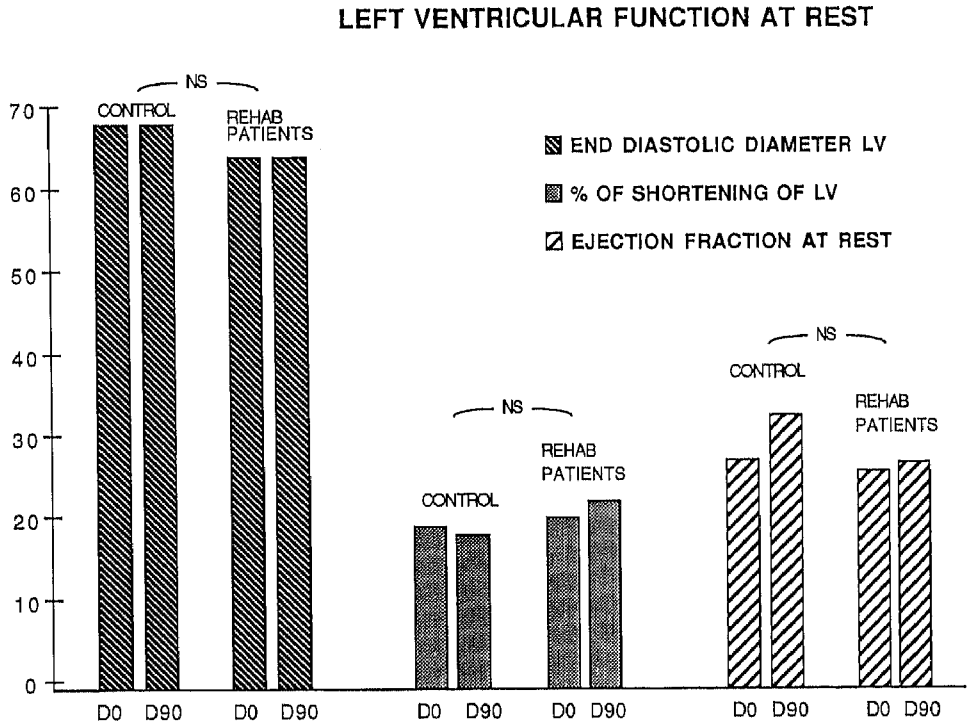


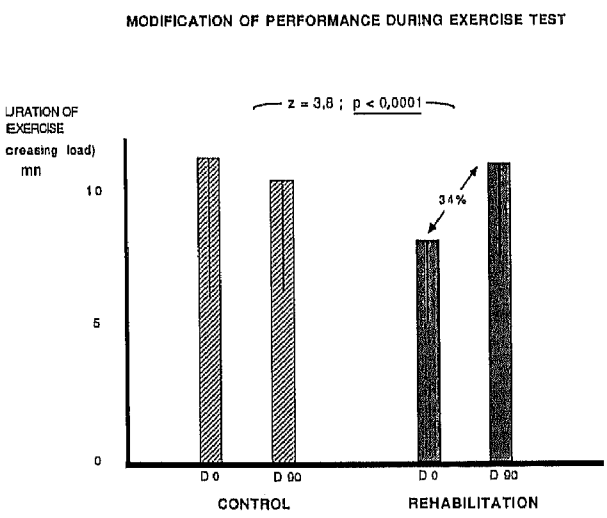
FIGURE 2. The parameters of left ventricular contractility are not modified by physical training.

e exception of 1 patient who showed signs of permanent ricular fibrillation.

DISCUSSION

The imbalance that exists between the heart and the rrounding vessels is sustained by a vicious circle syndrome ig 5) at the center of which lies a state of physical unfitness. vo essential causes for this can be found: (1) muscular tigue, which seems to be initially due to a decrease in cal output[7]; and (2) dyspnea, which has multiple causes, nong which are lactic acidosis and hyperpressure in the ngs.

This physical deconditioning is itself responsible for real ructural muscular modifications that have been described in depth by Mancini et al.[8] These modifications include morphology as much as cellular histochemistry and are responsible for a considerable decrease in muscular performance. They are also the cause of an aggravation of the initial imbalance from excessive loss of peripheral energy. Training enables this vicious circle to be broken by giving the muscle back its ability to perform.



IGURE 3. Modification of performance during exercise test.

Graded rehabilitation does not cause any bradycardiac effects at rest or during effort since it is done in stages and is not a question of stamina.

The improvement in performance (34%) is one of the best to be found in the literature.[2-5,9] It is probably due to the effect of training on peripheral vessels[8,10] since the function of the left ventricle is not improved. Training had no adverse effect on the left ventricular systolic function at rest.

Classic rehabilitation programs do not seem to have caused any accidents in the trials already published.[2-5] However, these series concern only very small numbers and, given the overall character of the exercises and the very poor cardiac condition of the patients, we have been reluctant to set up such programs.

Graded training in stages means less pressure on the heart. In addition, the patient's training is harmonious and enables considerable improvement in muscular strength at all levels. This enables patients to gain in autonomy and in quality of life. The proof is that 80% of the patients asked for training to be continued.

CONCLUSION

Physical training for patients with chronic heart failure means an increase in muscular strength and better adaptation to effort through a purely peripheral action. The risks appear to be very slight if the exercises are adapted to patients who have been carefully selected and who are

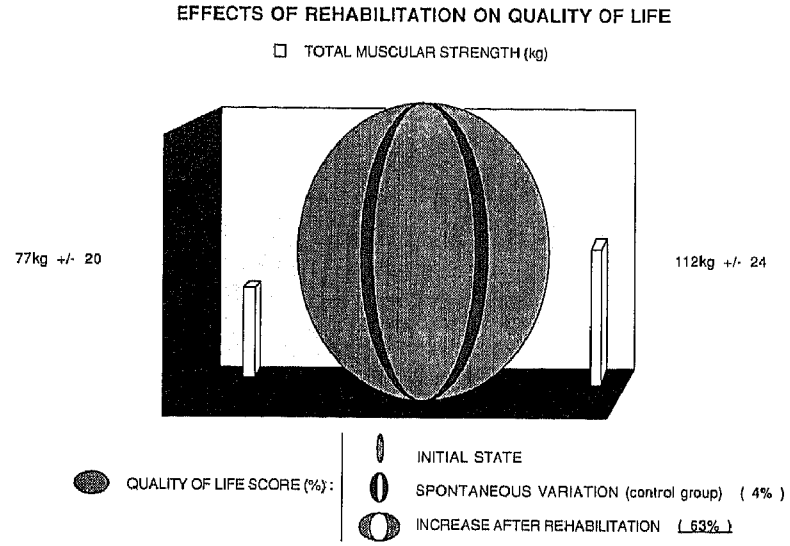


FIGURE 4. Effects of rehabilitation on quality of life.

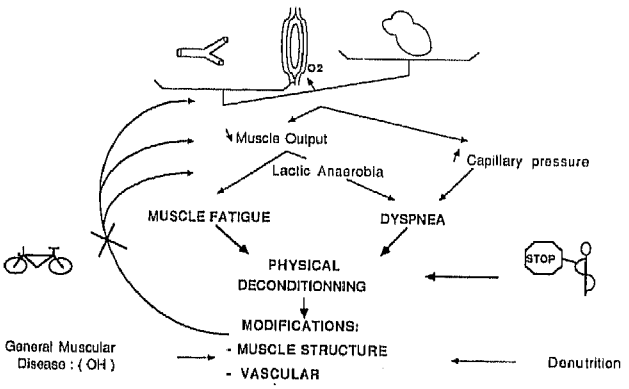


FIGURE 5. Physiopathologic approach to the functional handicap of the patient with chronic heart disease. It is a vicious circle at the center of which one finds physical deconditioning that has 3 causes: muscle fatigue, dyspnea (due to the inadaptation of the heart to peripheral needs), and sedentarity (often due to advice given by physicians). This deconditioning causes modifications to the muscle structure that helps in the general deterioration of muscle performance and leads to an increase in the imbalance: heart muscle/muscle requirement for oxygen.

carefully monitored. The results of the functional improvement of patients with chronic heart failure greatly outweigh those of any drug therapy.

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Table 3—*Mean Values of Ergometric Variables**

	Exercise Test Increasing Load					
	Duration, min	Heart Rate at Rest, bpm	Max Heart Rate at Same Performance, bpm†	Systolic BP at Rest, mm Hg	Max SBP at Same Performance mm Hg‡	Exercise Test Constant Load Duration, min
Rehab patients						
Day 0	8.2±3	89±16	135.7±24.7	125±21	151.7±40	9.8±13
Day 90	11±3.4	91±21	132±24.2	130±12	154.6±29	19.2±14
Control subjects						
Day 0	11.3±4.8	77±6	137.5±23.6	121±18	162.7±28.7	15.3±1.6
Day 90	10.4±4.1	85±9	137.9±15.7	125±16	162.7±31.5	18.7±17

*Physical training does not significantly modify heart rate and systolic blood pressure during effort. However performance is significantly improved whether under increasing load or endurance (constant load).
†Maximum heart rate for the same maximum performance (the lower of the two exercise tests).
‡Maximum systolic blood pressure for the same maximum performance (the lower of the two exercise tests).

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