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Physical performance in the early phase after acute myocardial infarction evaluated by invasive and non-invasive stress testing*

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Symptom-limited maximal stress testing reveals any abnormal response to physical stress, thus identifying limiting factors of physical performance. In the early phase after myocardial infarction only low-level stress tests seem to be justified, with work rates approximating the degree of physical performance during everyday life. Following the introduction of computer techniques for the on-line analysis of ergospirometric and hemodynamic parameters [1] we launched a study of the feasibility and risk of stress testing in which invasive and noninvasive techniques were compared. For clinical purposes test procedures should be evaluated with regard to: identifying those at high coronary risk (early coronary angiography, by-pass surgery), improving individual myocardial performance by medical treatment (arrhythmias, influence on determinants of myocardial oxygen demand), and reaching a decision as to when to start physical training [2-4].

Patients and methods

After a period of increasing mobilization 36 men (mean age 55 years, range 40-67) underwent graded exercise testing [5] (nonsteady-state exercise, rectangular-triangular bicycle ergometry; 2-minute increment test; 20, 30, 50 and 75 watts). In 18 cases ergospirometric data during nonsteady-state exercise (Ergopneumotest, Fa. Jäger, Würzburg) and hemodynamic data during steady-state exercise were also obtained (20 and 30 watts; microheart catheterization, catheters being placed in the pulmonary and radial arteries; Fick's principle was used to calculate derived parameters on-line).

The first test was performed on the 29th day (mean; range 15-37) after admission to the hospital. The diagnosis of myocardial infarction was established by standard criteria. Due to the increase in pulmonary artery end-diastolic pressure (PAEDP) 2 subgroups were formed, group A (N = 7) and group B (N = 11) (see Table I). Data obtained from noninvasive stress-testing were analyzed to determine differences between these subgroups. Ergometric, ergospirometric and hemodynamic data were also compared with reference values [5].

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Table I Noninvasive stress-testing (nonsteady-state exercise)

Ergometry (2-min. increment test, bicycle ergometry; N = 36)			
Delta HR/work load relative (= W/kg b.w.): + 7 beats (myocardial infarction/normal person $p \leq 0.05$)			
Delta systolic blood pressure/work load relative: + 8 mm Hg ($p \leq 0.05$)			
ECG ST-depression (range 0.1-0.4 mV)	9/36	3/7	1/11
ST-elevation (range 0.1-0.8 mV)	11/36	3/7	1/11
PVC (> 1)	7/36	1/7	1/11
Angina pectoris (rate score III)	5/36	3/7	0/11
alarming symptoms (breaking off)	9/36	4/7	1/11
A/B χ^2 $\alpha \leq 0.05$	subgroup	A	B
Ergospirometry (N = 18; subgroups A and B)			
Oxygen uptake: + 15.6% (50 watts 2nd min.; $2p \leq 0.001$)			
Tidal volume NS			
Minute ventilation: + 16.9% (30 W), + 40.6% (50 W; $2p \leq 0.001$)			
Maximal work load (watts (χ))		57.1	63.6
Work output (watt·min)		232	293
Base excess (mmol/l)		-3	-4

HR = heart rate.

Results

During noninvasive and invasive stress testing (Table II) in the early phase after myocardial infarction (4th week) no serious complications occurred (72 tests). In 9 cases we had to interrupt the exercise test (ergometry) due to alarming symptoms, mainly the combination of abnormal responses to physical stress, such as arrhythmias, marked ST changes and angina pectoris. Patients with abnormal reactions to low-level exercise belonged to subgroup A, which was characterized by a pronounced increase in left ventricular filling pressure (index PAEDP) at work (20 watts). Even volume loading — lifting the legs to the pedals of the ergometer while in the recumbent position — led to the identification of the subgroup with low myocardial performance (PAEDP at rest: A: 13.4 mm Hg; B: 8.2 mm Hg; volume loading: A: 18.3 mm Hg, B: 12.7 mm Hg; control: mean 10.9 mm Hg, $2p \leq 0.01$). Analysis of ergospirometric parameters (18 patients) revealed signs of decondition (increase in oxygen uptake related to work output) and distinct hyperventilation, which probably reflected respiratory compensation of metabolic acidosis due to a diminution of endurance performance capacity. But ergospirometry did not help to

Table II Invasive stress testing (steady-state exercise)

Central hemodynamics (N = 18; subgroups A and B due to PAEDP)

	20 watts steady-state				
	MI	A	B	NP	2P MI/NP
N	18	7	11	14	
HR (beats/min) (χ)	98	n.s.		92	n.s.
BPpm (mm Hg)	118	130^{+}	110	106	+
PApm (mm Hg)	34	43^{+++}	29	22,3	+ + +
PAEDP (mm Hg)	22	$\underline{28}^{+++}$	$\underline{18}$	$\underline{12,3}$	+ + +
CI (l/min/m²)	$\underline{4,6}$	n.s.		$\underline{5,45}$	+ + +
SV (ml)	$\overline{89}$	n.s.		$\overline{116}$	+ + +
$avDO_2$ (ml/l)	76	n.s.		67	+ +
SWI (g·m/m²)	77	n.s.		88	+
PVR (dyn·sec·cm^{-5})	1116	1263^{+}	1023	833	+ + +
PulmVR (dyn·sec·cm^{-5})	331	418^{+++}	275	177	+ + +
$\dot{V}O_2$ (l/min)	0,66	n.s.		0,70	n.s.
$\dot{V}E$ (l/min)	17,0	n.s.		15,9	n.s.

Comparison within subgroups and with reference values. MI = myocardial infarction; NP = normal person; HR = heart rate; BPpm = mean arterial pressure; PApm = mean pulmonary artery pressure; PAEDP = pulmonary artery end-diastolic pressure; CI = cardiac index; SV = stroke volume; $avDO_2$ = arteriovenous oxygen difference; SWI = stroke work index; PVR = peripheral vascular resistance; PulmVR = pulmonary vascular resistance; $\dot{V}O_2$ = oxygen uptake; $\dot{V}E$ = minute ventilation volume; + = $2p \leqslant 0.10$; + + $\leqslant 0.05$; + + + $\leqslant$ 0.01.

discriminate between groups A and B. Determination of cardiac output at rest and during steady-state exercise (18 patients) revealed a low pump performance, and failed to discriminate between the 2 subgroups.

Early noninvasive stress testing (bicycle ergometry) can be recommended as a standard procedure after acute myocardial infarction to determine individual treatment and select handicapped patients for careful follow-up.

References

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