Radionuclide Ventriculography and Central Aorta Pressure Change in Noninvasive Assessment of Myocardial Performance

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Systolic pressure-volume diagrams were obtained noninvasively by measuring the systolic central aortic pressure with a new device and by combining the pressure measurements, thus obtained, with absolute volume measurements obtained by radionuclide ventriculography during ejection. By dividing the peak power by the time elapsed from the beginning of ejection to the peak power point, the ejection rate of change of power (ERCP) was calculated. The ability of this index to assess left ventricular function at rest and exercise was evaluated in ten healthy subjects. ERCP proved to be more sensitive than global left ventricular ejection fraction increasing fivefold from rest to exercise compared with only 20% increase in global ejection fraction. ERCP increased dramatically postexercise from 3411 ± 2173 to 18 162 ± 14 633 gm/sec^2, median 12 750, 95% confidence interval 9700–29 600, in healthy, while in patients it increased twofold from 2637 ± 824 to 5062 ± 1897 gm/sec^2, median 4070, 95% confidence interval 2800–7030, $p < 0.001$. ERCP had an excellent discriminative power in differentiating healthy subjects from patients, having 100% sensitivity, 90% specificity, 95% accuracy, 95% positive predictive value, and 90% negative predictive value. Thus, this noninvasive index seems to have a more comprehensive ability to evaluate changes in left ventricular function and shows a promising potential for clinical applications.

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Major efforts were made in the last decades to develop a reliable index to evaluate left ventricular performance. These efforts included the development of invasive indices obtained during cardiac catheterization such as maximal left ventricular DP/DT, as well as noninvasive indices such as left ventricular ejection fraction at rest and during exercise. In recent studies (*1–3*) pressure volume diagrams were generated for diagnostic purposes for the evaluation of left ventricular performance using radionuclide ventriculography for volume measurements and left ventricular intracavitary pressure recordings. In spite of their invasive nature, these studies demonstrate the clinical importance of pressure-volume loops and provide a new insight into the left ventricular function. Stein and Sabbah (*4,5*) in a series of studies used left ventricular systolic power and the ejection rate of change of power (ERCP) to evaluate left ventricular performance. They demonstrated the superiority of their index in animal studies and in catheterization studies in patients. The invasive nature of this method prevented it from becoming an everyday clinically accepted diagnostic tool.

In the present study an attempt was made to generate noninvasively the power indices described by Stein and Sabbah and to assess their ability to evaluate left ventricular function in healthy subjects under varying conditions. The noninvasive measurements were made possible by a new instrument allowing noninvasive measurement of the central aortic pressure (*6*). This method was found to yield a good correlation with the ascending aortic pressure wave as measured by a Millar tipped manometer in patients during heart catheterization. By combining this method with radionuclide measurement of left ventricular absolute volume, pressure-volume curves were generated and the left ventricular systolic work and power were calculated. The mean ejection

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rate of change of power (ERCP) in early systole was calculated from the power-time curve. We attempted to determine whether this index would be more sensitive than other ejection indices (ejection fraction, mean power, and peak power) in assessing left ventricular function at rest and after exercise.

METHODS

Patient Population

The study was performed in nine male patients 3 to 6 mo after an acute myocardial infarction (MI), mean age 56 ± 5 yr (patients) and ten healthy subjects (male, mean age 56 ± 12 yr). The patients were included if they had a documented MI by elevation of total CK above 120 IU/l (>90 IU normal value), and CK-MB fraction above 4%, and had a normal or near normal ejection fraction (>44%). There were six patients with an inferior MI and three patients with a non-Q wave MI, mean total CPK being 655 ± 160 IU with 6 ± 2% CK-MB. Global left ventricular ejection fraction (LVEF) measured by radionuclide ventriculography was 57.11 ± 9.3%, ranging between 44% to 73%. No regional wall abnormalities were detected in any of the patients.

The normal group included ten subjects with no symptoms of angina pectoris. Mean global LVEF in the healthy subjects was 61.1 ± 7%, ranging from 51% to 75%, p = N.S., when compared to the study group. All subjects underwent supine exercise radionuclide ventriculography which was stopped after 3 min at 100 W. The increase in heart rate and blood pressure are summarized in Table 1. None of the subjects experienced chest pain, arrhythmias, or ST-T wave changes. Three patients complained of fatigue and shortness of breath. Measurements were taken before (at rest) and immediately after exercise. Absolute LV volumes were measured by the count rate methods as described (*3*). At the same time, noninvasive measurements of central aortic pressure were made by the device developed by us (*6*) which is described briefly in the following paragraphs. Pressure-volume-time systolic curves were generated and the initial systolic work and power were calculated for the first half of the stroke volume. The rate of change of power was calculated by dividing the peak power by the time elapsed from the beginning of ejection to the peak power point. The changes, from rest to exercise, in mean power, peak power, and the rate of change of power were compared with the change in global LVEF.

The Device

The components and the theoretic principle of the device and its elements were reported in a previous work (*6*) and are briefly summarized here. The device is composed of four elements:

(a) a standard sphygmomanometric cuff with an internal transducer measuring the intracuff pressure. The cuff is provided with an automatic deflecting device controlled by a microprocessor allowing gradual and constant deflation;

(b) an additional narrow sphygmomanometer cuff connected to a high sensitivity pressure transducer placed 1–3 cm below the occlusive cuff;

(c) a standard ECG monitoring system; and

(d) all three elements are connected through an analog to digital converter to a central processing unit (CPU) consisting of an INTEL 8088 micro-processor. The output is displayed on a monitor screen (Fig. 1).

The method is based on the creation of a standing fluid column from the aorta to the occluded brachial artery during the entire process of pressure measurements. By applying an occlusive pressure on the brachial artery during systole using an inflatable cuff, a temporary standing fluid column is created in which the rising intraaortic pressure is transmitted to the periphery with minimal distortion. The time intervals needed for the aortic pressure wave to overcome a given occlusive brachial pressure applied by the inflatable cuff on the arm are equal to the time intervals needed to reach the same pressure in the central aorta plus the propagation time to the brachial point, which is constant in the same patient throughout the measurements. Time intervals are measured from the onset of depolarization (QRS complex serving as a reference system) to the detection of the pressure wave by an external transducer at the brachial artery level. Application of multiple, successive, occlusive pressures on the brachial artery decreasing sequentially from peak systolic to diastolic pressures, and plotting their values against the above described time intervals results in the reconstruction of the central aortic pressure curve.

The validity of the noninvasive method was documented by two different approaches (*6*).

1. The pressure values measured by the device were superimposed on the simultaneously measured central intraaortic pressure waves in patients undergoing cardiac catheterization

TABLE 1
Blood Pressure and Heart Rate at Rest and Postexercise in Normals and in Patients*

	Rest			Postexercise		
	HR beats/sec	BP mm/Hg	Double product	HB beats/sec	BP mm/Hg	Double product
Normals n = 10	70 ± 10	102 ± 17	7140	121 ± 5	124 ± 16	15 004
Patients n = 9	70 ± 8	103 ± 9	7210	114 ± 8	128 ± 19	14 592
P value	NS	NS	NS	NS	NS	NS

* Note: There was no significant difference in the postexercise response of heart rate, blood pressure and double product between the healthy and the patients.

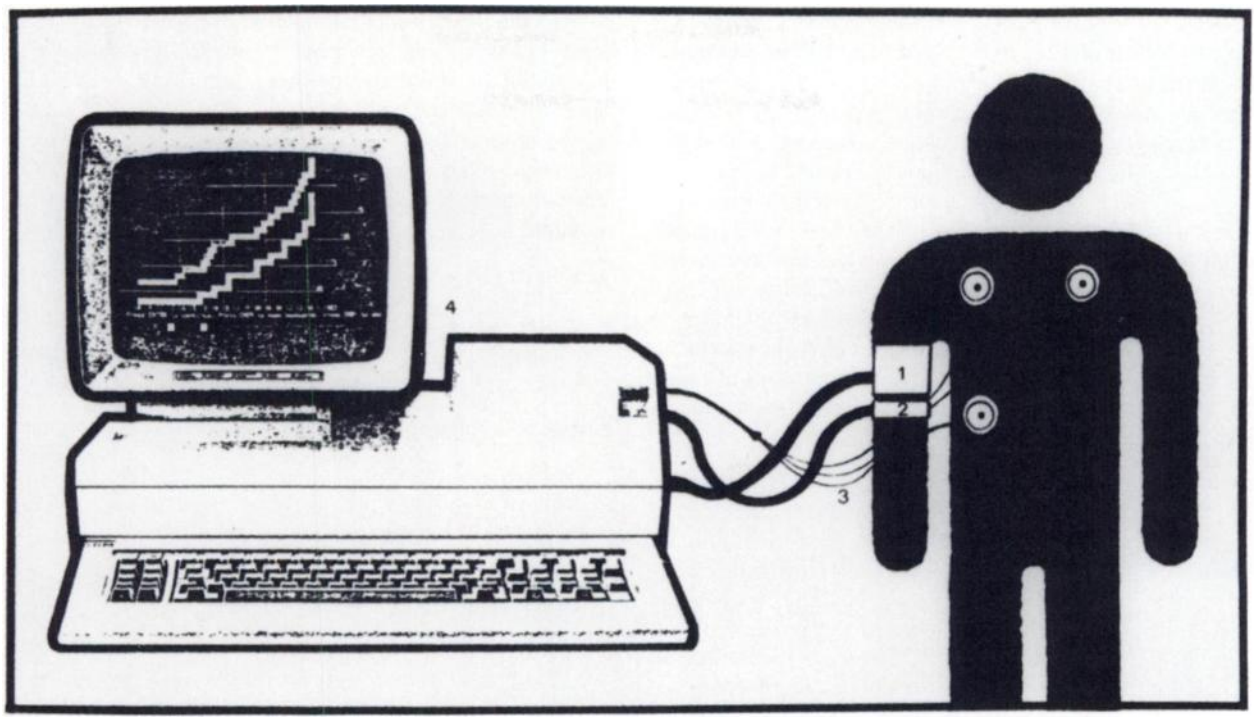

FIGURE 1
Schematic representation of the noninvasive pressure device. A. Sphygmomanometer cuff with internal transducer measuring the intracuff pressure. 2. Narrow cuff connected to a high sensitivity pressure transducer. ECG and computer are also schematically represented.

using Millar micromanometers. The typical invasive pressure time curve was generated from ~40 cycles, and the mean s.d. curves were calculated. All values measured by the device fell within 1 s.d. from the central intraaortic recordings in 14 out of the 15 patients studied.

2. Using linear regression analysis, an excellent correlation of r = 0.97 was found in each patient (in a total group of 15 patients) between the invasive and noninvasive digitized pressure values, obtained in the same patient for the same time intervals.

Generation of Systolic Pressure Volume Curves and Power Calculation

Absolute ventricular volumes were determined by gated radionuclide ventriculography according to the count rate method (*3*). Using red blood cells labeled in vivo with 20 mCi of technetium-99m (^{99m}Tc), a standard field-of-view gamma camera was interfaced to a dedicated minicomputer with a low-energy, medium resolution, parallel hole collimator. Data were collected in 45° left anterior oblique (LAO) position with a 15° caudal angulation. The cardiac cycle was divided into 20 frames. A total of 5 million counts were collected. Time-activity curves were generated for the left ventricle. After the aquisition of left ventricular volume points a smoothing algorithm was applied using a fast Fourier regression analysis with 16 harmonics. The ejection flow was calculated as the first derivative of the above described Fourier fit. After the simultaneous acquisition of pressure and volume points they were aligned in such a way that for every pressure point a corresponding volume point was found from the Fourier fit. All pressure points were moved leftward on the horizontal axis to correct for the time lag between the pressure and volume curves. The beginning of the initial pressure rise was aligned with the beginning of the decrease in left ventricular volume (negative DV/DT) when the initial end diastolic point signifies the beginning of ejection. In order to eliminate errors resulting from changes in heart rate during acquisition of data the study was aborted and the patients were excluded when changes of 5 bpm or more were found. The pressure curves recorded by the device and the time-activity curves obtained by the gamma camera were plotted against each other and systolic pressure-volume diagrams were generated. As the peak power occurs early in ejection (*4*), and as we were interested in deriving indices on the initial phase of ejection, only the first half of the ejection phase (in terms of stroke volume) was generated, namely, the portion of the diagram during the first half of the ventricular emptying. Left ventricular work at half stroke volume, mean power, peak power, and ERCP were derived according to the following formulas:

$$W = 0.0136 \int_{V=V_{ed}}^{V=V_{ed}-1/2SV} p \cdot dv \qquad (1)$$

$$P = \frac{W}{T} \qquad (2)$$

$$ERCP = \frac{PP}{TI}, \qquad (3)$$

where W = systolic work, V_{ed} = the end diastolic volume in milliliters, SV = stroke volume in milliliters, p = instantaneous pressure in milliliters of mercury and 0.0136 is constant to express the work in gram*meter, P = the mean power in

g*m/sec, PP = peak power, T = time in milliseconds, T1 = time to peak power in seconds, and ERCP = ejection rate of change of power in g*m/sec^2.

A new index for the assessment of rate of change of power was developed. In order to calculate this index, peak power was divided by the time to peak power, namely we calculated the slope of the line connecting the power at the beginning of ejection to the peak power (Fig. 2). The reason for the generation of this index is that it reflects an average estimate of rate of change of power, unaffected by the variability associated with measurements of the instantaneous power values. A possible noisiness in the instantaneous power measurements may result from the MUGA technique of measuring relatively few volume values. This index is independent of instantaneous pressure and flow variability.

Statistical Analysis

The ability of the subjects to perform and to increase the ejection power is dependent on their physical condition, especially in the healthy subjects (sedentary and physically trained subjects participated in the study). The population thus displayed a skewed distribution, the trained subjects outstanding in their performance. Therefore the method of calculating confidence intervals for a population median was applied rather than a parametric approach with t-tests and standard deviation. After calculating a 95% confidence interval the nonparametric rank test, Mann-Whitney, was applied. This approach is recommended in medical studies dealing with small and nonhomogeneous populations (*7,8*). For the purpose of presentation and comparison with ejection fraction mean and standard deviation were calculated, although these parameters were not used in the statistical analysis, as the population is not normally distributed.

Receiver operator characteristic (ROC) analysis stands for receiver operator characteristic curves and it is derived from a graph in which the sensitivity is plotted versus 1-specificity

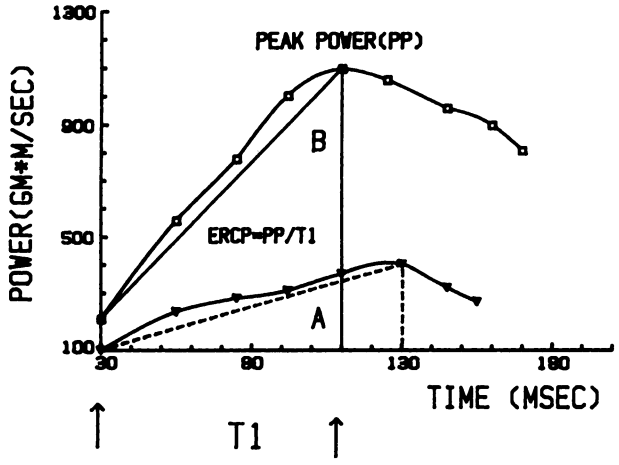

FIGURE 2
Schematic representation of calculation of ERCP from the power time curve at rest and after exercise in a healthy subject. A: Power-time curve at rest. B: Power-time curve postexercise. ERCP as shown in the figure is calculated by dividing the peak power by the time needed to reach peak power in early systole, and represents the slope of the line connecting the power at the beginning of the ejection and peak power.

of a specific test using multiple thresholds to distinguish between normal and abnormal groups. ROC analysis was done to test the sensitivity, specificity, accuracy and predictive values of each of the methods studied. The threshold with the highest accuracy was selected to represent the best possible threshold delineating normal from pathological response. The following indices were derived according to the following equation:

$$\text{sensitivity (\%)} = \text{true positive}/(\text{true positive} + \text{false negative}) \times 100$$

$$\text{specificity (\%)} = \text{true negative}/(\text{true negative} + \text{false positive}) \times 100$$

$$\text{predictive accuracy of a positive test (\%)} = \text{true positive}/(\text{true positive} + \text{false positive}) \times 100$$

$$\text{predictive accuracy of a negative test (\%)} = \text{true negative}/(\text{true negative} + \text{false negative}) \times 100.$$

RESULTS

All subjects performed supine exercise stress tests starting with 25 W with an increment of 25 W every 3 min and reaching 100 W in all subjects, global ejection fraction increasing in the healthy subjects from 61.1 ± 7% to 66 ± 7%, p = N.S., and in the patients from 57.1 ± 9% to 58 ± 6%, p = N.S. By using 95% confidence interval and nonparametric rank test, resting ejection fraction in healthy subjects had a median value of 63% with a confidence interval of 52–65%. It increased after exercise to a median value of 67%, 95% confidence interval of 59–71%, $p < 0.03$. In patients the median was 59% at rest (95% confidence interval 47–68%), while after exercise the median was 60% (95% confidence interval 48–70%), p = N.S. When the two groups were compared there was an overlapping in the confidence intervals, rank test showing non significant difference between the groups. The indices of myocardial performance in the control group (healthy subjects) and in the patient group are detailed in Tables 2, 3. Mean power increased in the healthy from 297 ± 169 g*m/sec to 695 ± 279 g*m/sec, while in the patients from 245 ± 67 to 441 ± 147 g*m/sec. In the patients the peak power increased only slightly from 346 ± 78 to 561 ± 129 g*m/sec. The ejection rate of change of power (ERCP) increased in the healthy fivefold from 3411 ± 2173 to 18 162 ± 14 633 g*m/sec^2 (Table 2), median 12750 (95% confidence interval 9700–29 600) while in the patients it increased twofold from 2637 ± 824 to 5062 ± 1897 g*m/sec^2 (Table 3), median 4070 (95% confidence interval 2800–7030). As shown, the two confidence intervals are completely different, $p < 0.001$. To further demonstrate the discriminative power of this parameter the percent change from rest to effort was calculated: ERCP increased in the healthy group by 376% (95% confidence interval 304–625%) while in the patients it increased only by 84% (95% confidence

TABLE 2
Myocardial Performance Indices at Rest and after a 100W Exercise in Healthy Subjects

	A. Rest				
Subject no.	Work [gm]	LVEF [%]	Power [g*m/sec]	Peak power [g*m/sec]	ERCP [g*m/sec^2]
1	36	51	324	533	4517
2	65	60	462	604	4027
3	32	53	203	354	2360
4	95	75	733	1110	9250
5	34	64	293	431	3748
6	31	52	207	333	2220
7	27	64	236	330	2750
8	26	65	182	271	1936
9	20	65	145	200	1429
10	29	62	193	300	1875
AVG	39	61	298	447	3411
STD	22	7	169	249	2173
			B. Postexercise		
Patient no.					
1	47	54	1010	1089	14918
2	88	62	1208	2072	29601
3	23	59	556	604	10067
4	107	71	1071	1402	58417
5	60	67	670	1318	15506
6	46	64	534	759	9731
7	55	67	597	1128	14100
8	45	70	540	674	11424
9	36	70	400	619	10317
10	45	77	372	604	7550
AVG	55	66	696	1027	18163
STD	24	6	278	453	14634

interval 67–217%). The individual changes in ejection fraction and in the ejection rate of change of power in both groups are summarized in Tables 2, 3. Note that the change in ejection fraction was relatively small in both groups while the change in ERCP was markedly higher in the normal group compared to the patient group. ROC analysis of the studied indices showed that LVEF at rest had a low sensitivity and a low negative predictive value (Table 4, Fig. 3). Mean power, peak power, and ERCP also had low discriminative power at rest, but ERCP showed an excellent discriminative power at exercise with a 100% sensitivity; 90% specificity; 90% positive predictive power and 100% negative predictive power (Table 5, Fig. 4). This contrasted with the ejection fraction which showed 67% sensitivity, 70% specificity with positive and negative predictive powers of 62% and 63%, respectively (Figs. 3, 4). As illustrated in Figure 4, ERCP after exercise separates accurately the healthy from the patients, while ejection fraction at rest and after exercise and ERCP at rest were unsuccessful in differentiating between the two groups. Even when the change in ejection fraction was considered (Fig. 5), its specificity reached only 60% and its positive predictive value only 40%. In contrast, when the change in ERCP was used, a 100% sensitivity and 90% specificity was found, its positive predictive value being 88% and the negative predictive value being 90% (Fig. 5).

DISCUSSION

The function of the left ventricle as a pump is best assessed during ejection, when simultaneous changes in pressure and volume with respect to time take place. Most of the methods devised to measure left ventricular function are based on changes of one parameter, either volume (circumferential fiber shortening, ejection fraction) or pressure (dp/dt) as a function of time. These methods give an incomplete information, each of them assessing only one aspect of the left ventricular performance, ignoring the simultaneous changes in the other parameter. An index taking into account end-systolic pressure-volume relationship has been used more recently as a relatively load independent and sensitive measure of ventricular contractile state (*9*). However, this index, measured invasively, was shown to be relatively insensitive to changes in the inotropic state (*10*). The only systolic index based on all three parameters (pressure, volume, and time) is the left ventricular power. In physical terms the power is the most impor-

TABLE 3
Myocardial Performance Indices at Rest and After a 100W Exercise in Patients

Subject no.	Work [gm]	LVEF [%]	Power [g*m/sec]	Peak power [g*m/sec]	ERCP [g*m/sec²]
			A. Rest		
1	32	49	266	429	3900
2	28	52	178	303	2020
3	32	47	262	388	3180
4	30	44	276	360	3273
5	34	73	306	361	2888
6	36	68	225	360	2118
7	16	59	91	150	882
8	44	63	315	420	2819
9	35	59	292	346	2662
AVG	32	57	246	346	2638
STD	7	9	68	78	825
			B. Postexercise		
Patient no.					
1	36	50	400	598	7035
2	36	60	318	542	3613
3	42	48	410	626	6260
4	53	42	408	363	2792
5	46	73	575	638	5317
6	35	70	295	514	4283
7	30	63	252	420	2800
8	72	57	720	514	4673
9	54	65	597	835	8789
AVG	45	59	442	516	5063
STD	12	10	148	129	1898

tant parameter which describes the function of a pump. The left ventricular power is an expression of the rate at which the left ventricle does work, and it was used before to characterize the performance of the left ventricle in dogs (*11*) and in man (*12–15*). It was measured invasively and calculated as the product of the aortic pressure and flow (*13–15*). Another invasive method that was used to calculate left ventricular power was the product of aortic pressure and the rate of change of left ventricular volume during ejection (*16*).

The reason ejection power did not become popular as an index of left ventricular contractility is its invasive nature and therefore the inability to use it in a clinical setting for the measurement of myocardial reserve or ischemia during exercise tests. In the present study we present a noninvasive method of measuring ventricular power as the product of aortic pressure and the rate of change of left ventricular volume during ejection. We recently described a method to measure the ascending limb of the aortic pressure noninvasively (*6*). The pressure wave of the ascending aorta during early ejection phase represents the pressure wave of the left ventricle in the absence of aortic stenosis. We utilized this method together with the absolute ventricular volume curve obtained by radioventriculogram to obtain ventricular instantaneous power during ejection. The left ventricular power increases rapidly during early ejection, reaches a peak volume and then declines, therefore

TABLE 4
ROC Analysis at Rest in the 19 Subjects

	LVEF	Mean power	Peak power	ERCP
Sensitivity	33	89*	100*	100*
Specificity	100	30*	30*	30*
Accuracy	68	58	63	63
Threshold	50	31	459	3957
Positive predictive value	100	35*	63*	63*
Negative predictive value	63	65	100*	100*

* Statistically significant ($p < 0.05$).

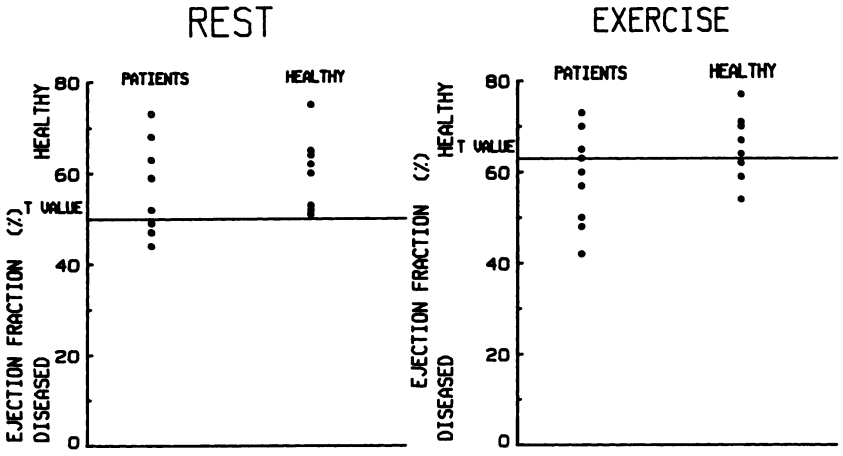

FIGURE 3
Discriminative power of ejection fraction at rest and exercise.

we considered the early ejection period only (till the volume reached half the end diastolic volume, Fig. 2). In order to assess myocardial reserve we measured the left ventricular power at rest and postexercise.

Ejection Rate of Change of Power

We calculated the mean rate of change of power during early ejection by dividing the peak power to the time from beginning of ejection to peak power. Mathematically, the rate of change of power is the first derivative of power and the second derivative of work with respect to time. Physiologically, the rate of change of power during ejection means the acceleration of work generated by the left ventricle during ejection. A similar index had already been measured invasively by Stein and Sabbah (*4,5*). It was shown to be sensitive to drug-induced changes of the inotropic state in dogs, while affected little by alterations in preload or afterload, in contrast to other ejection indices (ejection fraction, circumferential fiber shortening, ventricular work and power, etc) which are markedly influenced by loading conditions (*4*). The ejection rate of change of power was also shown to separate patients with abnormal and normal ventricular performance (categorized on the basis of the ejection fraction, mean velocity of circumferential fiber shortening and left ventricular end diastolic volume index) with no overlap of values between categories of patients ($p < 0.001$) (*5*).

The noninvasive nature of our method enables us to use this index in ambulatory patients and to measure changes in ERCP at rest and exercise, in order to assess myocardial performance and myocardial reserve.

The index developed in the present study (the slope of the line connecting the power at the beginning of ejection to peak power, Fig. 2) constitutes an average estimate of the rate of change of power during early ejection. This index is similar to the index measured by Stein and Sabbah (*4,5*)—the peak rate of change of power—both assessing the rate of change of power. However, our index represents an average estimate of the rate of change of power and not a single value of rate of change of power as Stein and Sabbah's index.

Another difference between our method and the index measured by Stein and Sabbah is the noninvasive and indirect nature of our method. The noninvasive approach has inherent difficulties and errors in measurements of instantaneous volume and pressure. The pressure measurements were validated invasively (*6*). The radionuclide technique was chosen for volume measurements because it is free of geometric assumptions and was extensively validated in the past (*17–20*).

Patient Selection and Exercise Testing

The aim of this study was to assess the usefulness of power and ERCP indices in the evaluation of left ventricular performance. Patients after MI with preserved

TABLE 5
ROC Analysis Postexercise in the Studied Population

	LVEF (%)	Mean power (%)	Peak power (%)	ERCP (%)
Sensitivity	67	67	56	100*
Specificity	70	80	100*	90*
Accuracy	68	74	79	95
Positive predictive value	62	75	100	90*
Negative predictive value	63	62	71	100
Threshold value	63	489 g*m/sec	564 g*m/sec	8970 g*m/sec^2

* Statistically significant ($p < 0.05$).

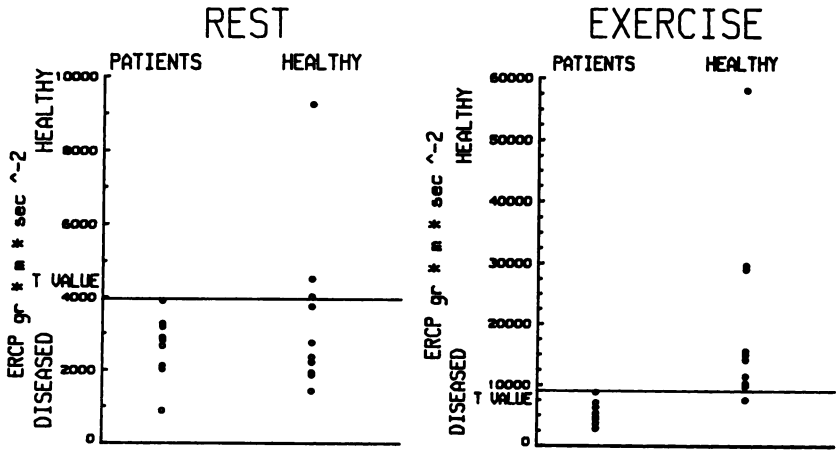

FIGURE 4
Discriminative power of ERCP at rest and exercise.

or slightly reduced left ventricular function (as judged by the ejection fraction at rest) were chosen. We deliberately chose these patients, who in spite of being after documented myocardial injury were diagnosed as healthy by LVEF at rest and at exercise, failing to differentiate between them and the healthy subjects (Fig. 3, Tables 2A, 3A, and 4). LVEF increased from 61 ± 7% to 66 ± 6.2% in the healthy subjects (Table 2) and from 57 ± 9% to 58 ± 9.8% in patients, p = N.S. (Table 3). Ejection fraction did not differentiate between healthy subjects and patients as shown by the rank test and the 95% confidence intervals, which were almost identical in the two groups at rest and after exercise. In this respect, this group of patients with minimal myocardial damage served as the reference system when comparing left ventricular ejection fraction performance to ERCP performance in discriminating the healthy from the diseased. These patients had myocardial damage (documented enzymatic infarction). Peak power, mean power, and ERCP did not separate the two groups at rest. We concluded that the myocardial damage was too small to affect ejection indices at basal conditions.

ERCP at the postexercise measurement was 18 162 ± 14 633 g*m/sec^2 in the healthy group and 5062 ± 1897 g*m/sec^2 in the patient group (Tables 2B and 3B), and it differentiated the two groups with 100% sensitivity, 90% specificity, 90% accuracy and had a very small overlap of values (Tables 5, Fig. 4). Using 95% confidence interval a clear differentiation between the healthy subjects and the patients was obtained, ERCP increasing in the healthy by 376%, 95% confidence interval 304–625% while in the patients it increased only by 84% (95% confidence interval 67–217%).

The difference in ERCP from rest to exercise also separated the two groups with high sensitivity and specificity (100% and 90%, respectively) (Fig. 5). It increased fivefold in the healthy group while less than twofold in the patients.

We conclude that ERCP is a useful index of myocardial performance. It can be measured noninvasively. Its physiologic meaning is the acceleration of energy expended upon the production of useful work by the ventricle during ejection. It was shown to be sensitive to change in contractile state, while relatively independent of loading conditions (*5*). It increases markedly during exercise, and has a high sensitivity and specificity for detecting myocardial reserve. In order to assess its clinical importance as an indicator of left ventricular performance or as a detector of myocardial ischemia

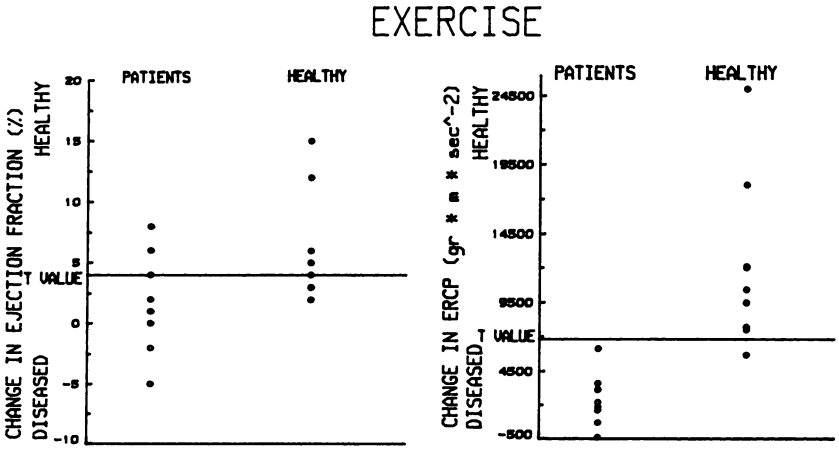

FIGURE 5
Discriminative power of the change in ejection fraction and in ERCP after exercise.

further studies in different groups of patients with ischemic heart disease have to be performed.

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