EUROPEAN JOURNAL OF CARDIOLOGY, 1976, 4/SUPPLEMENT, 131–141. EXCERPTA MEDICA

Postextrasystolic potentiation in man

Tjeerd van der Werf, Rube van Poelgeest, Han H. Herbschleb and Frits L. Meijler

University Hospital, Utrecht, The Netherlands

VAN DER WERF, Tj., VAN POELGEEST, R., HERBSCHLEB, H.H. and MEIJLER, F.L. (1976): Postextrasystolic potentiation in man. *Europ. J. Cardiol., 4/Suppl.,* 131–141.

Postextrasystolic potentiation (PESP) is a pre- and afterload-independent property of the myocardium. Therefore it has been used to study left ventricular (LV) contractile state in patients with coronary heart disease and normals. Using a computer-controlled cardiac stimulator, regular rhythms with just above sinus frequency were applied to the right atrium and interrupted after every 20th beat by one shorter interval after varying delays. The next stimulus interval was corrected in such a way that the resulting R–R interval was the same as the basic R–R interval. Control of the postextrasystolic R–R interval is a prerequisite for meaningful and quantitative evaluation of PESP.

Of 166 basic and postextrasystolic beats in 12 patients LV pressures (P), obtained with a catheter tipmanometer, were analyzed. There was no significant difference in enddiastolic P between pre- and postextrasystolic beats.

Peak dP/dt, time to peak dP/dt, (dP/dt)/P at a left ventricular P of 50 mm Hg and V_{max} were used as indices of left ventricular contractile state. It was found that on increasing prematurity of the extrasystolic beat, there was a progressive and reproducible increase of these indices (selfevidently a decrease in the case of the time to reach peak dP/dt). Using V_{max} as an index of contractile state, no significant PESP could be demonstrated in the two patients with very enlarged left ventricular volumes and depressed ejection fractions.

PESP, elicited in this (only allowable) way, is an integral, quantitative and reproducible method for LV contractility estimation in man.

postextrasystolic potentiation; left ventricular contractility; coronary heart disease

Introduction

After a brief and spasmodic clinical life in the middle of the sixties [1,2] postextrasystolic potentiation had almost faded away from the cardiological clinical scene. These last two years, however, the study of postextrasystolic potentiation has enjoyed a revival [3–6], obviously as a result of the fact that a quantitative description of left ventricular contractile behavior is badly needed, especially in relation to surgical interventions in patients with coronary heart disease. Postextrasystolic potentiation, as a part of the so-called interval–strength relationship [7], affects almost exclusively the contractile state of the myocardium [8]. For that reason we have developed a technique enabling us to use postextrasystolic potentiation to quantify the contractile reserve of the human left ventricular myocardium.

Methods

Patients

After giving their verbal consent 12 patients with complaints of chest discomfort were studied using right and left heart catherization, including biplane left ventricular cinecardiograpy, selective coronary arteriography and right atrial artificial electrical stimulation. No systematic selection procedure was applied; the patients were chosen depending on a number of circumstances. Each patient served as his

oped. For that reason a computer-programmed current source stimulator with highly set safeguards has been constructed [9]. One of the advantages of this stimulator is that the heart can be stimulated continuously while premature beats are initiated. For instance in the present study after each twentieth regular beat a premature beat is evoked. A diagrammatical representation of the cycle length patterns as used in this study is given in Figure 1.

The postextrasystolic stimulation interval was chosen in such a way, that the increase of the extrasystolic P–R duration was corrected, which resulted in a postextrasystolic R–R interval, being identical (accuracy of approximately 1%) with the basal R–R interval. The desired correction of the postextrasystolic interval depends of course on the prematurity of the extrasystolic stimulus.
For instance:

A 20	B	A 0
last regular interval	Extrasystolic interval	Post extrasystolic interval
600 (R–R 600)	525 (R–R 535)	610 (R–R 600)
600 (R–R 600)	450 (R–R 465)	620 (R–R 605)
600 (R–R 600)	375 (R–R 400)	630 (R–R 600)

The accuracy of the interval duration is 1 msec. Stimulus duration and stimulus strength are also selected via the computer program [9]. As a rule the stimulation characteristics were: duration 1 msec; strength 4 mAmp. The basal stimulation rate was at least 10 beats $\cdot$ min^{-1} above the autochtonous sinus frequency of the patient under study.

Pressure measurements and derivations

Left ventricular pressures were measured by means of Millar Instuments 7F tipmanometer catheters with reference to zero at mid chest level. The output of the micromanometer was amplified by a Hewlett Packard (7868A) direct inkrecording system using high paperspeed (200 mm $\cdot$ sec^{-1}) and also stored on an Ampex FR 1300 magnetic tape recorder at a tapespeed of $3\frac{3}{4}$ inch $\cdot$ sec^{-1}. The high fidelity left ventricular pressure tracings were calibrated by matching the signal with a predetermined electronic calibration constant. The first derivative of the left ventricular pressure was obtained with an analog differentiator and recorded on an additional channel of the Hewlett Packard recorder. Calibration was obtained with a saw-teeth generator. The pressure tracings were analyzed by hand, taking measurement samples at 5-msec intervals during the isovolumetric contraction phase. R–R intervals were also measured by hand. Later on it was possible to do the measurements with the aid of a PDP 15-computer with sample intervals of 2.5 msec. Figure 2 shows an example of a recording.

The following indices were derived or calculated from the measurements mentioned above:

A. $P_{lv\ ed}$ = left ventricular enddiastolic pressure in mm Hg defined as the pressure at the moment of the beginning of the steep post-a-wave rise of the dP/dt tracing. The average of all beats of the same category of pre- and postextrasystolic beats has been taken.

B. $P_{lv\ max}$ = peak of left ventricular pressure in mm Hg. The average of all beats of the same category has been used.

C. $P_{ao\ ed}$ = enddiastolic aortic pressure in mm Hg. The average of all beats of the same category was taken into account. This determination has only be performed in the patients No. 8 to 12 where we were able to use a double-tipmanometer catheter.

D. dP/dt_{max} = peak value of the rate of rise of left ventricular pressure in mm Hg $\cdot$ sec^{-1}. The average of all beats of the same category has been used.

E. $t_o - t_{dP/dt_{max}}$ = time to peak dP/dt in msec [10]. The average of all beats of the same category has been used.

F. (dP/dt)/P at P = 50 mm Hg in sec^{-1}. Of each pre- and postextrasystolic beat a curve relating (dP/dt)/P to P was constructed using the isovolumic measurements. The curves obtained in this way were averaged per category enabling us to read the value of (dP/dt)/P at P = 50 mm Hg.

G. V_{max} in sec^{-1} was obtained by rectilinear extrapolation to P = 0 (zero) mm Hg of the averaged curves as outlined in F. The latter was calculated with the aid of a digital computer.

Results

The results are summarized in Table II. Every number is the average of the measurements of 2–4

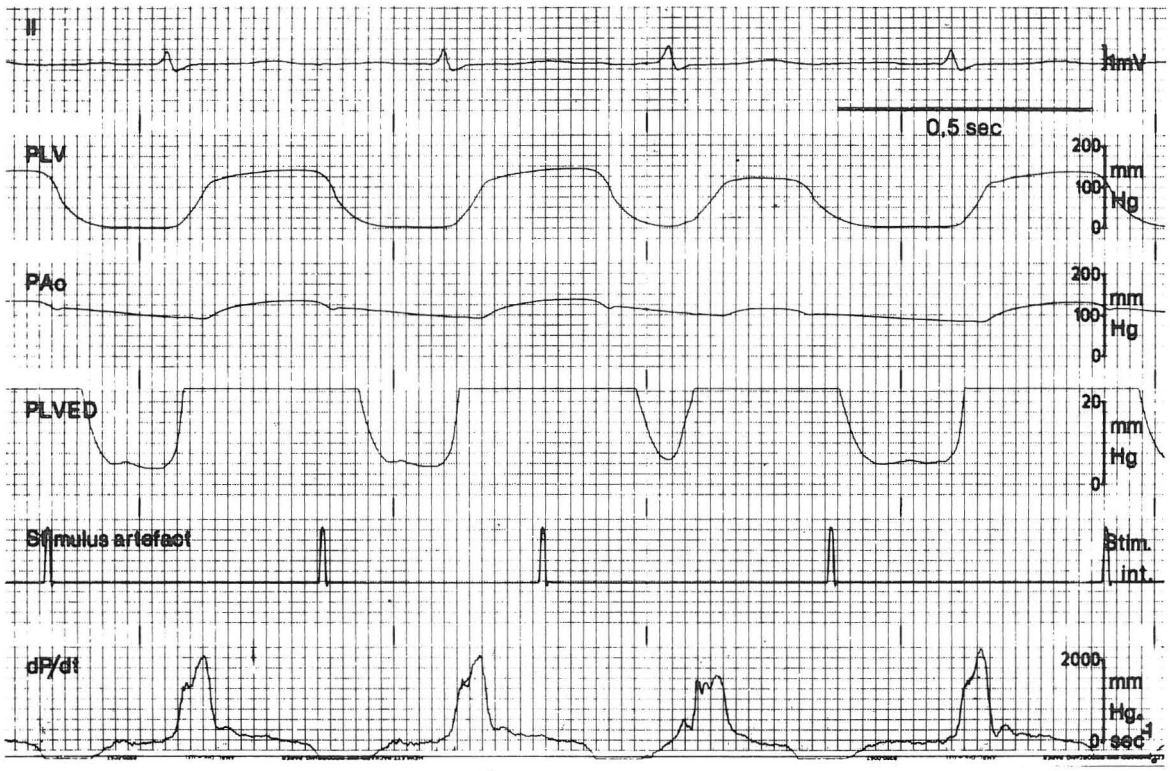

Fig. 2. An example of a multichannel recording during postextrasystolic potentiation.

The paperspeed used was 200 mm · sec^{-1}. From above to below the following signals have been recorded: the electrocardiogram lead II (II), the left ventricular pressure (PLV), the pressure in the ascending aorta (PAo), a blow up of left ventricular pressure, to facilitate the reading of enddiastolic pressure (PLVED), the stimulus artefact and the first derivative of left ventricular pressure (dP/dt), recorded in such a way that only the positive part is shown.

beats with an identical extrasystolic interval. In the description of the results the values are considered for the total population and – if possible – for three subgroups, namely (1) the patients with normal coronary arteries (Nos. 1, 2, 4, 9, 11 and 12), called 'normals'; (2) the patients with coronary heart disease without dilated ventricles and without depressed ejection fractions (patients 3, 5, 8 and 10), called CHD I patients, and (3) the patients with coronary heart disease complicated by enlarged left ventricles and depressed ejection fractions (patients 6 and 7), called CHD II patients.

The variations in extrasystolic interval with respect to the basic interval ranged from 41 to 260 msec (= 7 to 38% of the basic interval). There were no significant differences between these values in the three subgroups.

The left ventricular enddiastolic pressure ($P_{lv\ ed}$) varied from 5 to 21 mm Hg. The difference in $P_{lv\ ed}$ between pre- and postextrasystolic beats ranged from −1.9 to +2.8 mm Hg with a mean of +0.3 mm Hg (post > pre) and a standard error of ±0.22 mm Hg. This difference is not significant, nor is there any significant difference between the subgroups.

The peak left ventricular pressure ($P_{lv\ max}$) ranged from 95 to 149 mm Hg. The difference in $P_{lv\ max}$ between pre- and postextrasystolic beats varied from −11 to +9 mm Hg with a mean of −2 mm Hg (post < pre) and a standard error of ±0.8 mm Hg. This difference is also not significant nor does there exist any significant difference between the subgroups.

Enddiastolic aortic pressure ($P_{ao\ ed}$) was measured in 5 patients. The values ranged from 62 to 105 mm Hg. The difference in $P_{ao\ ed}$ between pre- and post-

extrasystolic beats varied from −1 to −10 mm Hg with a mean of −5 mm Hg (post < pre) and a standard error of ±1.1 mm Hg; $P < 0.1$. Significant differences between patients, belonging to the subgroups 'normal' and 'CHD I' were not present.

The maximal value of the first derivative of left ventricular pressure (dP/dt_{max}) ranged from 889 to 2250 mm Hg · sec^{-1}. The differences in dP/dt_{max} between pre- and postextrasystolic beats varied from −4 to +354 mm Hg · sec^{-1}, with a mean of +170 mm Hg · sec^{-1} (post > pre) and a standard error of ±18 mm Hg · sec^{-1}; $P < 0.001$. From Figure 3 it can be concluded that the shorter the extrasystolic interval, the more the rise in dP/dt_{max}. Linear regression lines were computed with the least squares method of the total population and of the three subgroups. In Table III the regression coefficients with their standard errors and the correlation coefficients of these four regression lines are stated. In Figure 3 the regression lines for the three subgroups are drawn. All regression lines differ significantly from zero. There are no significant differences between the subgroups.

The time to reach the peak value of the first derivative of left ventricular pressure ($t_0 - t_{dP/dt_{max}}$) ranged from 104 till 59 msec. The differences in $t_0 - t_{dP/dt_{max}}$ between pre- and postextrasystolic

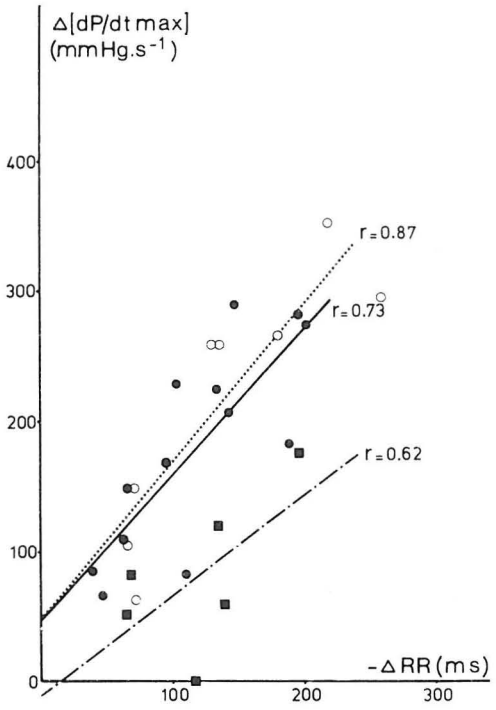

Fig. 3. Postextrasystolic changes in the peak value of the first derivative of left ventricular pressure.

Horizontal axis: differences between the basic R–R interval and the extrasystolic R–R interval (−ΔRR in msec).

Vertical axis: change in the peak value of the first derivative of left ventricular pressure in the postextrasystolic beats with respect to the preextrasystolic beats ($\Delta[dP/dt_{max}]$ in mm Hg · sec^{-1}).

Closed circles represent the group normals, open circles the group CHD I patients, and squares the group CHD II patients. The continuous line is the regression line of the normals, the dotted line the regression line of the CHD I patients, and the broken line the regression line of the CHD II patients.

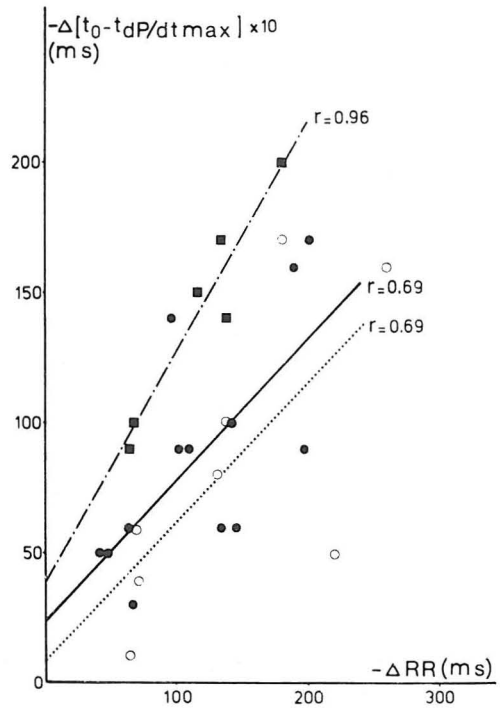

Fig. 4. Postextrasystolic changes in the time to reach dP/dt_{max}.

Horizontal axis: difference between the basic R–R interval and the extrasystolic R–R interval (−ΔRR in msec).

Vertical axis: change in the time to reach dP/dt_{max} from the enddiastolic moment in the postextrasystolic beats ($-\Delta[t_0 - t_{dP/dt_{max}}]$ in msec).

The meaning of the symbols and lines used, is the same as in Fig. 3.

TABLE II Results of measurements during postextrasystolic potentiation

Nr.	prepreceding R–R interval (msec)	−Δ id. (msec)	$P_{lv\ ed}$ (mm Hg)	Δ id. (mm Hg)	$P_{lv\ max}$ (mm Hg)	Δ id. (mm Hg)	$P_{ao\ ed}$ (mm Hg)	Δ id. (mm Hg)
1. pre	670		6.8		107			
post	560	110	7.3	+0.5	108	+ 1		
post	480	190	8.3	+1.5	106	− 1		
2. pre	602		10.7		137			
post	460	142	11.7	+1.0	130	− 7		
post	401	201	10.8	+0.1	129	− 8		
3. pre	676		9.0		147			
post	416	260	9.2	+0.2	138	− 9		
4. pre	599		12.3		123			
post	535	64	12.3	0.0	126	+ 3		
post	465	134	11.7	−0.6	122	− 1		
post	402	197	14.0	+1.7	119	− 4		
5. pre	704		12.8		125			
post	633	71	13.0	+0.2	129	+ 4		
post	485	219	12.7	−0.1	123	− 2		
6. pre	602		12.4		106			
post	538	64	12.0	−0.4	105	− 1		
post	485	117	10.5	−1.9	95	−11		
post	463	139	13.0	+0.6	108	+ 2		
7. pre	601		18.0		111			
post	533	68	20.3	+2.3	120	+ 9		
post	467	134	19.0	+1.0	109	− 2		
post	405	196	20.8	+2.8	106	− 5		
8. pre	601		5.9		114		79	
post	535	66	5.8	−0.1	113	− 1	77	− 2
post	470	131	7.0	+1.1	114	0	74	− 5
post	419	181	6.5	+0.6	104	−10	69	−10
9. pre	549		6.8		117		72	
post	508	41	6.8	0.0	116	− 1	70	− 2
post	447	102	6.7	−0.1	120	+ 3	70	− 2
post	403	146	6.8	0.0	112	− 5	62	−10
10. pre	601		12.8		121		81	
post	532	69	11.8	−1.0	121	0	79	− 2
post	463	138	13.3	+0.5	120	− 1	76	− 5
11. pre	601		11.3		149		105	
post	534	67	10.0	−1.3	147	− 2	100	− 5
12. pre	541		4.8		143		92	
post	494	47	5.5	+0.7	143	0	91	− 1
post	444	97	5.0	+0.2	133	−10	82	−10

$P_{lv\ ed}$ = enddiastolic pressure as defined in text; $P_{lv\ max}$ = peak left ventricular pressure; $P_{ao\ ed}$ = enddiastolic aortic pressure; dP/dt_{max} = peak value of the first derivative of left ventricular pressure; $t_0 - t_{dP/dt_{max}}$ = time to reach dP/dt_{max} from the end-

dP/dt_{max} (mm Hg · sec^{-1})	Δ id. (mm Hg · sec^{-1})	$t_0 - t_{dP/dt_{max}}$ (msec)	−Δ id. (msec)	$\frac{dP/dt}{P}$ at P = 50 mm Hg (sec^{-1})	Δ id. (sec^{-1})	V_{max} (sec^{-1})	Δ id. (sec^{-1})
1300		90		22.5		42.8	
1383	83	81	9	24.7	2.2	48.5	5.7
1483	183	74	16	27.8	4.8	52.7	9.9
1550		83		24.3		72.6	
1758	208	73	10	28.0	3.7	75.5	2.9
1825	275	66	17	29.0	4.7	84.0	11.4
1620		83		23.8		68.4	
1915	295	67	16	32.6	8.8	78.2	9.8
1583		74		28.1		50.7	
1692	109	68	6	30.7	2.6	52.0	1.3
1808	225	68	6	32.7	4.6	55.2	4.5
1867	284	65	9	33.7	5.6	54.5	3.8
1763		75		31.3		49.8	
1825	62	71	4	31.7	0.4	54.0	4.2
2117	354	70	5	36.0	4.7	56.6	6.8
889		104		15.9		32.0	
940	51	95	9	16.5	0.6	32.8	0.8
885	− 4	89	15	17.0	1.1	32.6	0.6
948	59	90	14	17.5	1.6	32.0	0.0
1028		80		18.4		27.3	
1165	83	70	10	20.0	1.6	27.1	−0.2
1203	121	63	17	21.0	2.6	27.4	0.1
1260	178	60	20	21.0	2.6	26.9	−0.4
1590		82		28.6		52.7	
1695	105	81	1	31.2	2.6	56.1	3.4
1850	260	74	8	33.2	4.6	61.4	8.7
1855	265	65	17	32.7	4.1	59.3	6.6
1765		68		32.3		69.7	
1850	85	63	5	34.3	2.0	73.8	4.1
1995	230	59	9	36.7	4.4	76.0	6.3
2055	290	62	6	37.5	5.2	76.5	6.8
1570		75		24.6		35.8	
1720	150	69	6	27.3	2.7	40.2	4.4
1830	260	65	10	29.5	4.9	39.8	4.0
1625		89		24.5		34.6	
1775	150	86	3	27.8	3.3	40.6	6.0
2080		82		32.3		65.4	
2145	65	77	5	33.5	1.2	64.7	−0.7
2250	170	68	14	34.5	2.2	68.6	3.2

diastolic moment (t_0); Δ id. = a change between pre- and postextrasystolic beat in the index concerned; pre = the last normal beat = control beat; post = the postextrasystolic beat.

TABLE III Regression analysis of the results of the measurements during postextrasystolic potentiation (the regression equation is $Y = a_0 + a_1X$; in all cases $X = -\Delta RR$ in msec)

Y	Group	n	a_0	a_1	r	P
$\Delta[dP/dt_{max}]$ (in mm Hg · sec^{-1})	total	27	24 ± 31	1.17 ± 0.22	0.73	<0.001
	normals	13	48 ± 34	1.12 ± 0.26	0.73	<0.01
	CHD I	8	48 ± 43	1.21 ± 0.28	0.87	<0.01
	CHD II	6	−10 ± 63	0.77 ± 0.49	0.62	<0.1
$-\Delta[t_0 - t_{dP/dt_{max}}] \times 10$ (in msec)	total	27	33 ± 20	0.52 ± 0.14	0.59	<0.001
	normals	13	24 ± 23	0.54 ± 0.17	0.69	<0.01
	CHD I	8	8 ± 36	0.54 ± 0.23	0.69	<0.1
	CHD II	6	38 ± 17	0.89 ± 0.13	0.96	<0.01
$\Delta\left[\frac{dP/dt}{P}\right] \times 100$ at P = 50 mm Hg (in sec^{-1})	total	27	32 ± 58	2.38 ± 0.42	0.75	<0.001
	normals	13	106 ± 55	2.12 ± 0.43	0.83	<0.001
	CHD I	8	6 ± 108	2.84 ± 0.69	0.86	<0.01
	CHD II	6	23 ± 69	1.21 ± 0.54	0.75	<0.1
$\Delta V_{max} \times 100$ (in sec^{-1})	total	27	59 ± 138	2.89 ± 1.00	0.50	<0.01
	normals	13	51 ± 174	3.81 ± 1.34	0.65	<0.05
	CHD I	8	239 ± 129	2.53 ± 0.82	0.79	<0.05
	CHD II	6	79 ± 49	−0.53 ± 0.39	0.57	N.S.

Symbols in column Y are declared in text; for the definition of groups see text; n = number of observations; a_0 and a_1 are the regression coefficients ± standard error, r = correlation coefficient.

beats varied from 1 to 20 msec (post < pre) with a mean of 10 msec and a standard error of ±1.0 msec; $P < 0.001$. In the same way as for $\Delta(dP/dt_{max})$, in Table III the regression coefficients and the correlation coefficients of the regression lines of the total population and the three subgroups are outlined. In Figure 4 the regression lines of the three subgroups are drawn. All regression lines differ significantly from zero. Between the subgroups are no significant differences.

The values of (dP/dt)/P at P = 50 mm Hg ranged from 15.9 till 37.5 sec^{-1}. The differences between pre- and postextrasystolic beats varied from 0.4 to 8.8 sec^{-1} with a mean of 3.3 sec^{-1} (post > pre) and a standard error of ±0.36 sec^{-1}; $P < 0.001$. In Table III the regression coefficients and the correlation coefficients of the regression lines of the total population and the three subgroups are outlined. In Figure 5 the regression lines of the three subgroups are drawn. All regression lines differ significantly from zero. There are no significant differences between the subgroups.

The values of V_{max} ranged from 27.1 to 76.5 sec^{-1}. The differences in V_{max} between pre- and postextrasystolic beats varied from −0.7 to +11.4 sec^{-1} with a mean of +4.2 sec^{-1} (post > pre) and a standard error of ±0.65 sec^{-1}; $P < 0.001$. In Table III the regression coefficients and the correlation coefficients of the regression lines of the total population and the three subgroups are outlined. In Figure 6 the regression lines of the three subgroups are drawn. All regression lines, except the one for the CHD II patients, differ significantly from zero. There is a significant difference between the regression line of CHD II on one side and the regression lines of the normals and CHD I on the other side ($P < 0.02$); between the two last-mentioned groups the difference was not significant. Using V_{max} as a parameter of the contractile state of the left ventricular myocardium, patients with dilated ventricles and depressed ejection fractions seem to have lost their ability to show the phenomenon of PESP. In this respect they clearly differ from normals and from patients with coronary heart disease without dilated ventricles and depressed ejection fractions.

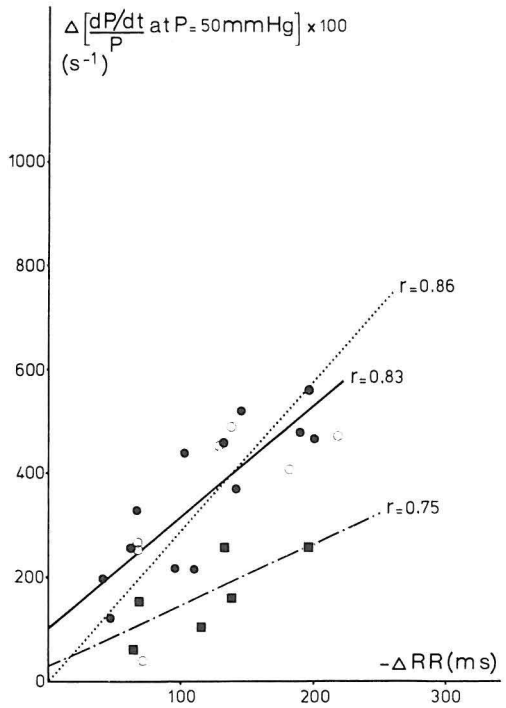

Fig. 5. Postextrasystolic changes in the value of (dP/dt)/P at a pressure of 50 mm Hg.

Horizontal axis: difference between the basic R–R interval and the extrasystolic R–R interval (–ΔR–R in msec).

Vertical axis: change in the value of (dP/dt)/P at a left ventricular pressure of 50 mm Hg in the postextrasystolic beats with respect to the preextrasystolic beats (Δ[(dP/dt)/P at P = 50 mm Hg] in sec^{-1}).

The meaning of the symbols and lines used, is the same as in Fig. 3.

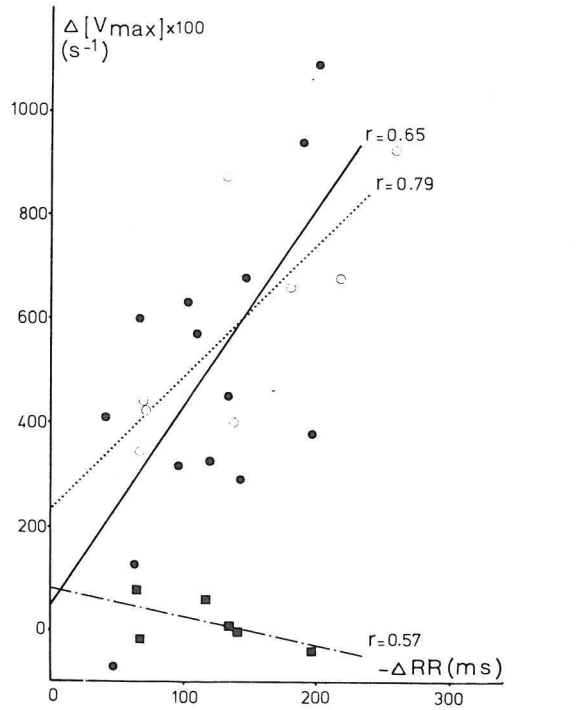

Fig. 6. Postextrasystolic changes in V_{max}.

Horizontal axis: difference between the basic R–R interval and the extrasystolic R–R interval (–ΔR–R in msec).

Vertical axis: change in V_{max} in sec^{-1}.

The meaning of the symbols and lines used, is the same as in Fig. 3.

Discussion

In this paper a stimulation procedure to evoke postextrasystolic potentiation has been presented which differs substantially from the procedure used by others [3–6]. Dyke et al. [3] made use of one ventricular premature beat induced by an R wave coupled pacemaker, connected to a stimulation catheter lying with its tip in the right ventricle. The extrasystolic interval was as on average 400 msec; the premature beat was followed by a compensatory pause, at least by an R–R interval, longer than the basic interval. Cohn et al. [4] made use of a ventricular premature beat provoked by the injection of X-ray contrast material into the left ventricle, the measurements being performed during left ventriculography. The short extrasystolic interval was followed by a compensatory pause. Hamby et al. [5] used one to three premature beats arising during injection of contrast material into left ventricle. They stress in their publication the importance of a compensatory pause which should be at least one and a half time the basic interval. Schwarz et al. [6] made use of two stimulation catheters, one lying with its tip in the right atrium to give a basic rhythm of 125 beats per minute and a second stimulation catheter lying with its tip in the apex of the right ventricle to give one premature beat after a controllable interval, again followed by a compensatory pause.

These four methods have in common that at least two changes in the rhythm are introduced, namely the shorter interval preceding the premature beat and the longer interval following the premature beat. The

so-called postextrasystolic beat is thus a beat potentiated by the prepreceding shorter interval and, at the same time, an augmented rest contraction [7] due to the directly preceding longer interval. In our stimulation procedure, as outlined above, this summation of two effects on the postextrasystolic beat is avoided. We were able to keep the postextrasystolic R–R interval within 1% identical to the basic interval. It should be mentioned that a small variation of the P–R duration is introduced and hence of the hemodynamic coupling between atrium and ventricle. Due to its prematurity the P–R interval of the premature beat is longer than the P–R interval of the basic beat. This effect is also present in the next beat, the postextrasystolic beat, be it less than in the premature beat. Another difference between the cited methods and our procedure is that we avoid the use of ventricular premature beats.

We are not able to give exact values for preload and afterload changes during the procedure. However, no significant difference between left ventricular enddiastolic pressure in the control beats and the postextrasystolic beats could be demonstrated, nor was there any significant difference in peak left ventricular pressure between the control beats and the postextrasystolic beats. In the last five patients we could demonstrate a small but significant ($P < 0.1$) lowering of enddiastolic aortic pressure in the postextrasystolic beats with respect to the control beats. Since we only use isometric contraction indices this may not effect our results.

Postextrasystolic potentiation should be preferred over steady-state atrial pacing tests [11,12] because during the atrial pacing tests the total circulation may adapt itself to the altered conditions. Recorded changes in left ventricular performance are then a mixture of altered left ventricular contractility and the adaption of the peripheral circulation.

The intervention to be chosen for the study of left ventricular contractility may be more important than the kind of measurement to be performed. Nevertheless it seems worthwhile to consider the choice of indices derived from left ventricular isovolumic pressure. Peterson et al. [13] have shown that parameters, derived from the isovolumic part of the left ventricular pressure curve are less sensitive in dividing patients with normal hearts from patients with severely damaged myocardium than parameters derived from left ventriculography during the ejection phase. The study of left ventricular contractile mechanisms under controlled conditions on several beats with different degrees of shortening of the extrasystolic interval is virtually impossible with the aid of left ventriculography. Moreover, we wanted to avoid the depressing effects of repeated injections of X-ray contrast material [14].

As shown in the presentation of our results V_{max} seems to be the most sensitive parameter. The use of V_{max} as a parameter of contractility has several disadvantages: (a) the theoretical and physiological background of this parameter is still under discussion [15]; (b) the influence of enddiastolic pressure on the extrapolation to zero; (c) the influence of the zero level of the pressure measuring device on V_{max} as clearly demonstrated by Peterson et al. [14]; (d) the existance of nonuniform activation of left ventricular myocardium; and (e) the possible presence of mechanical asynergy. In our setup using changes in V_{max} the arguments b–e are not applicable. As mentioned before, there was no significant difference between left ventricular enddiastolic pressures in pre- and postextrasystolic beats. Using supraventricular stimuli the activation of the ventricle was identical in pre- and postextrasystolic beats.

All four parameters chosen demonstrate significant postextrasystolic potentiation, considering, the total group. With the aid of V_{max} postextrasystolic potention could be demonstrated in patients with normal coronary arteries and in patients with coronary heart disease with normal left ventricular volumes and normal ejection fractions. Between these two groups – in this small series – no differences could be demonstrated. The patients with enlarged ventricles and depressed ejection fractions did not, however, show postextrasystolic potentiation. It is tempting to conclude that these patients did not have any inotropic reserve left. Further investigations have to confirm these assumptions. Furthermore we intend to compare the postextrasystolic potentiation in patients before and after coronary surgery. Using a computer-controlled stimulator, it is possible to apply postoperatively exactly (within 1 msec) the same sequence of stimuli to the right atrium as preoperatively.

In summary we can state that our method, in

which only one shorter interval is introduced and in which the postextrasystolic beat is preceded by an R–R interval, identical to the basic interval with the additional advantage of keeping left ventricular end-diastolic pressure constant, is the only allowable way to study postextrasystolic potentiation.

Acknowledgements

The authors wish to express their gratitude to Mrs. P. van der Weegen-Wassenaar and Mrs. M. van Hessen-van der Vegt for typing the manuscript and preparing the figures.

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