Left Ventricular Function During Transluminal Angioplasty: A Haemodynamic and Angiographic Study

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ABSTRACT. The response of left ventricular function, was studied in a series of patients undergoing percutaneous transluminal coronary angioplasty (PTCA). From 4 to 6 balloon inflations procedures per patient were performed with an average duration per occlusion of 51 ± 12 sec (mean ±SD), total occlusion time 252 ± 140 sec. Analysis of left ventricular (LV) haemodynamics showed that the relaxation parameters peak negative rate of change in pressure and the early time constant of relaxation responded earliest to acute coronary occlusion while other parameters such as peak pressure, LV end-diastolic pressure, and peak positive rate of change of pressure responded more gradually and suggested a progressive depression in myocardial mechanics during the entire procedure. LV angiogram available in 14 patients indicate an early onset of asynchronous relaxation concurrent with the early response in peak -dP/dt and the time constant of early relaxation. All haemodynamic parameters fully recovered within minutes after the end of PTCA.

The results of this study indicate no permanent dysfunction to global or regional myocardial mechanics, after PTCA with 4 to 6 coronary occlusions each lasting 40 to 60 seconds.

INTRODUCTION

Until recently the measurement in man of left ventricular geometry and haemodynamics early after an abrupt occlusion of a major coronary artery has not been feasible. Percutaneous transluminal coronary angioplasty (PTCA) however, now provides an unique opportunity to study the time course of these variables during the transient interruption of coronary flow in the balloon occlusion sequence in patients with single vessel disease and without angiographycally demonstrable collateral circulation (1, 2). We report here the dynamic changes in left ventricular haemodynamics and the concurrent left ventricular geometry changes assessed by angiography in 14 patients during PTCA. This study was undertaken in order to investigate the sequence of events during transient ischaemia induced by transluminal angioplasty and to determine whether the effects of ischaemia after repeated occlusions were reversible or not.

STUDY POPULATION AND PROTOCOL

Fourteen patients were selected from 356 consecutive attempted angioplasty procedures. These patients met the criteria of an isolated obstructive
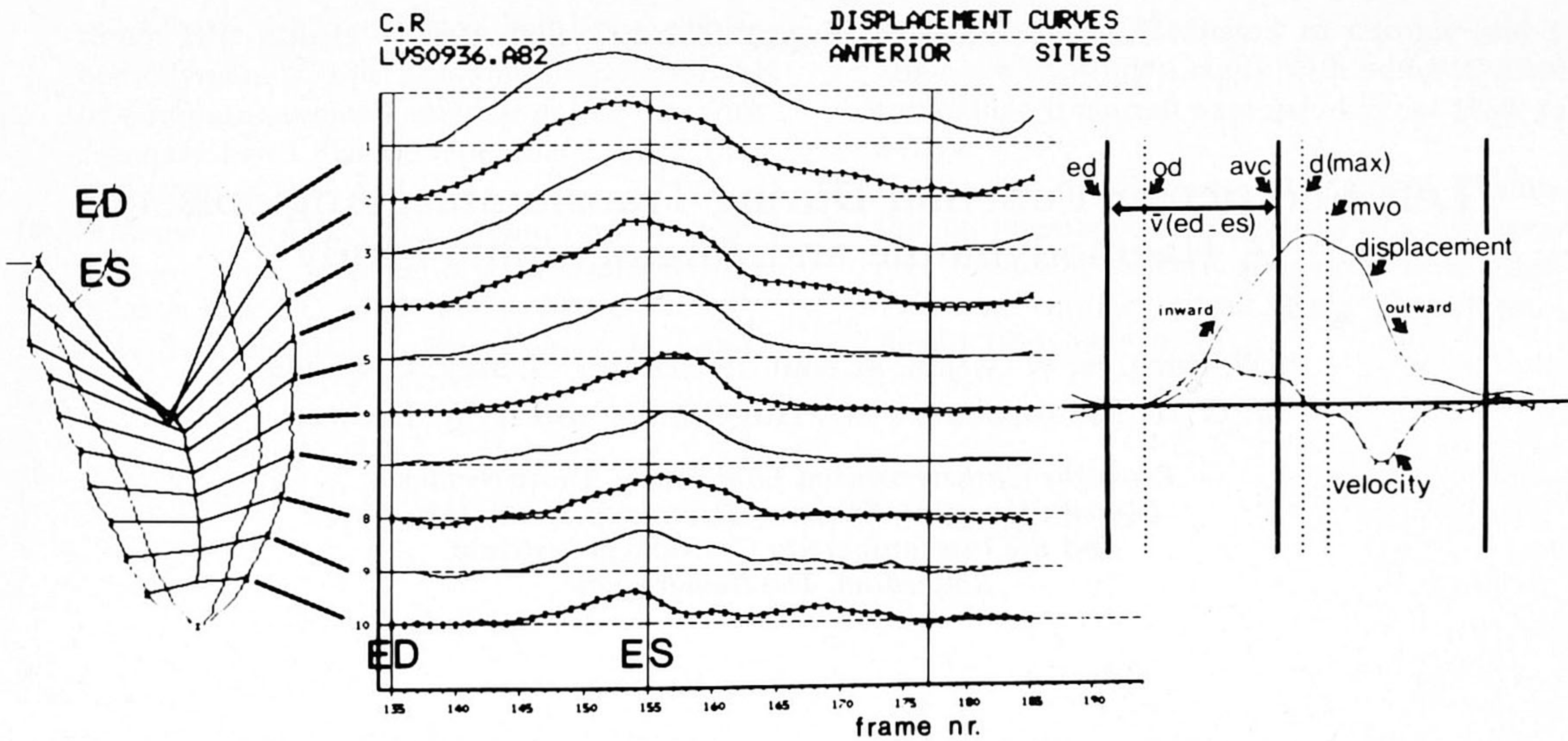
Fig. 1. End-diastolic and end-systolic left ventricular contours, as detected by the automated analysis system. On these silhouettes a system of coordinates is superimposed, along which segmental left ventricular wall displacement is detected. Left ventricular wall velocity – first derivative of wall displacement – is derived from these data. Abbreviations: ed: end-diastole; es: end-systole; od: onset of displacement; v (ed-es): mean ejection phase wall velocity; avc: aortic valve closure; d (max): maximal inward wall displacement; mvo: mitral valve opening.

lesion of one coronary vessel (left anterior descending artery in ten patients, right coronary in four, left circumflex in one) having a normal resting left ventricular function and wall motion. Four patients had mild essential hypertension and elevated left ventricular filling pressures (EDP ≥ 25 mmHg). During the PTCA procedure the number of transluminal occlusions performed per patient was 4.9 ± 2.2 (mean ± SD).

The average duration of each occlusion was 51 ± 12 sec and the total occlusion time during the whole procedure was 252 ± 140 sec (mean ± SD). With a tipmanometer on a 8F pigtail catheter, pressures were recorded and derived variables were calculated off-line by a computer system (3, 4). Three to four ventriculograms (30 degrees RAO at 50 frames/sec) were obtained by injection of 0.75 ml/kg of a non ionic contrast medium (metrizamide, Amipaque[R]). The haemodynamic and angiographic investigations were performed before the PTCA procedure was begun, after 20 sec of occlusion during the second dilatation, after 50 sec of occlusion during the fourth dilatation and again 5 minutes after completion of the PTCA procedure. These sequential LV angiograms were made only after the values for left ventricular end-diastolic pressure and the various isovolumic parameters had returned to those recorded before the initial angiogram. In all cases the interval between two angiograms was at least 10 minutes. Care was taken to maintain the patient's position unchanged
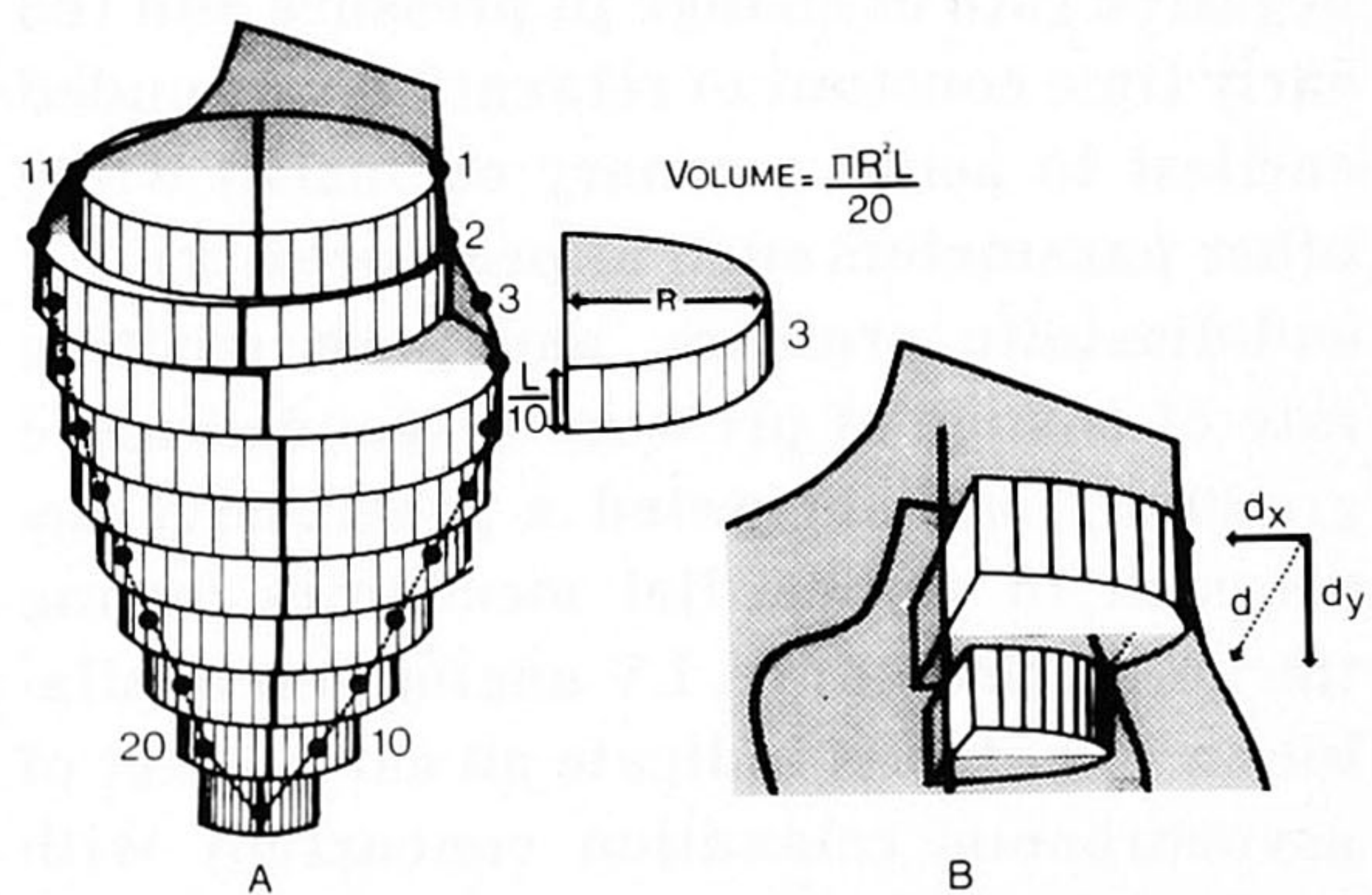

Fig. 2. Method for computing regional contibution to ejection fraction (CREF): volume of each segment (slice volume) is computed according to the formula shown in the figure. The systolic volume change is derived from the regional displacement and is mainly a consequence of the decrease of radius (R) of a half slice, which is expressed by the x-component (dx) of the displacement vector (d). L: left ventricular long axis length extending from base to apex.

Table I. *Haemodynamic variables before PTCA, at 20 and 50 sec after occlusion, and after the PTCA procedure.*

	before PTCA		during PTCA		after PTCA	
			20 sec occlusion	50 sec occlusion		
	Total group (n = 14)	Subgroup (n = 9)	Total group (n = 14)	Subgroup (n = 9)	Total group (n = 14)	Subgroup (n = 9)
HR (bpm)	62 ± 16	59 ± 18	61 ± 13	62 ± 14	64 ± 11	63 ± 11
EDV (ml/m²)	81 ± 15	79 ± 14	81 ± 15	81 ± 16	77 ± 11	78 ± 11
ESV (ml/m²)	31 ± 9	29 ± 7	37 ± 9**	41 ± 9**	27 ± 7*	26 ± 15
SV (ml/m²)	50 ± 11	49 ± 11	44 ± 12*	39 ± 14*	50 ± 9	52 ± 10
EF (%)	61 ± 8	62 ± 6	54 ± 8**	48 ± 12**	64 ± 7	66 ± 6
peak LVP (mmHg)	154 ± 30	151 ± 35	142 ± 29	145 ± 37	147 ± 21	148 ± 25
peak +dP/dt (mmHg·s⁻¹)	1403 ± 304	1356 ± 257	1312 ± 320	1278 ± 317	1412 ± 333	1442 ± 384
Vmax (s⁻¹)	39 ± 9	40 ± 8	39 ± 9	34 ± 10*	42 ± 11	43 ± 12
ESP (mmHg)	95 ± 18	92 ± 22	90 ± 19	98 ± 24	90 ± 14	91 ± 15
peak −dP/dt (mmHg⁻¹)	1727 ± 322	1614 ± 267	1268 ± 355**	1404 ± 370*	1664 ± 243	1665 ± 296
T₁ (msec)	55 ± 8	55 ± 6	79 ± 17**	68 ± 16**	54 ± 7	56 ± 7
T₂ (msec)	44 ± 7	43 ± 7	51 ± 8*	59 ± 8**	45 ± 9	45 ± 8
Pmin (mmHg)	10 ± 5	8 ± 3	11 ± 4	16 ± 6**	8 ± 4	8 ± 5
EDP (mmHg)	22 ± 8	18 ± 6	22 ± 7	29 ± 5**	20 ± 6	21 ± 5
K ln P/V (ml⁻¹)	.0244 ± .099	.0239 ± .008	.0314 ± .016	.0431 ± .018*	.0339 ± .013	.0349 ± .016

Abbreviations: PTCA = Percutaneous transluminal coronary angioplasty, HR = heart rate, bpm = beats per minute, EDV = end distolic volume indexed, ESV = end systolic volume indexed, SV = stroke volume indexed, EF = ejection fraction, LVP = left ventricular pressure, dP/dt = rate of change of pressure, Vmax = maximal velocity of the contractile element (dP/dt/P linearly extrapolated to P = 0), ESP = end systolic pressure, T = time constant of relaxation, Pmin = left ventricular minimal diastolic pressure, EDP = left ventricular end diastolic pressure, K = slope of diastolic pressure-volume relation. *p < 0.05, **p < 0.005 as compared to values before PTCA. Student t-test for paired observations.

in relation to the X-ray equipment during the consecutive angiograms. Diaphragm movement was reduced to a minimum by shallow inspiration taking care to prevent the Valsalva maneuvre.

METHODS

A. Analysis of pressure-derived indices during systole and diastole

Left ventricular pressure was measured with a Millar micromanometer cateter and digitized at 250 samples/sec. Combined analog and digital filtering resulted in an effective time constant of less than 10 msec. This employed an updated version of the beat-teat program described previously (3, 4).

Peak LV pressure, LV end-diastolic pressure, peak negative dP/dt, peak positive dP/dt and the relation between dP/dt/pressure and pressure linearly extrapolated to pressure = O (V_{max}) were computed on line after a data acquisition of 20 seconds.

Determination of relaxation parameters

A new technique has been implemented for the off-line beat-to-beat calculation of the relaxation parameters (5, 6, 7), using a semilogarithmic model:

$P(t) = P_0 e^{-t/T}$ The P_0 and T parameters are estimated from a linear least squares fit of $LnP = -t/T + LnP_0$, starting from the time of peak-dP/dt (7).

a) fit of first 40 msec (n 8), T_1, bi-exponential (7).
b) fit after 40 msec (n 3), T_2, bi-exponential (7).
c) fit of all points (n 8), T, mono-exponential.

B. Analysis of global and regional left ventricular function, during systole and diastole

A complete cardiac cycle was analyzed frame by frame from all cineangiograms. The ventricular contour was detected automatically (8). For each analyzed cineframe left ventricular volume was computed according to Simpson's rule. After the end-diastolic and end-systolic frames were determined, stroke volume, global ejection fraction and total cardiac index were computed. End-diastolic (ED) pressure was defined at that point on the pressure trace at which the derivative of the pressure first exceeds 200 mmHg/sec (3) and in all cases coincided with the maximal measured LV volume.

End-systole (ES) was defined, with reference to the pressure tracing, at the occurrence of the dicrotic notch of the central aortic pressure. To analyze the regional left ventricular function, the computer generated a system of coordinates along which the left ventricular displacement is determined frame by frame in 20 segments (Fig. 1). The definition of the 20 segmental coordinates was derived from the mean trajectories of endocardial sites in 23 normal individuals (9) and generalized as a mathematical expression amenable to automatic data processing (10, 11).

Segmental wall velocity was computed as the first derivative of the instantaneous displacement function. Mean ejection phase wall velocity (V) for each segment was calculated from end-diastole to end-systole (Ved-es), (Fig. 1). Segmental volume was computed from the local radius (R) and the height of each segment (1/10 of left ventricular long axis length L) according the formula: 1/20 $\pi R^2 L$. When normalized for end-diastolic volume, the systolic segmental volume change can be considered as a parameter of regional pump function (Fig. 2).

During systole this parameter expresses quantitatively the contribution of a particular segment to global ejection fraction, termed regional contribution to global ejection fraction or CREF (10). The sum of the values for all 20 segments equals the global ejection fraction. Diastolic function was analyzed in terms of volume stiffness. Pressure-volume relations were determined from the lowest diastolic pressure to the beginning of the »a» wave. The natural logarithm of pressure was used

in a linear regression analysis of pressure and volume from which a slope K was derived. Changes in K were taken as changes in volume stiffness (12).

RESULTS

A. Global left ventricular function during systole and diastole

The left ventricular pressures and volumes measured before, during, and after angioplasty are shown in Table I.

There was no important change in heart rate during the PTCA procedure. The pattern of change in peak LVP, LVEDP, peak +dP/ dt, and V_{max} however, suggest a progressive
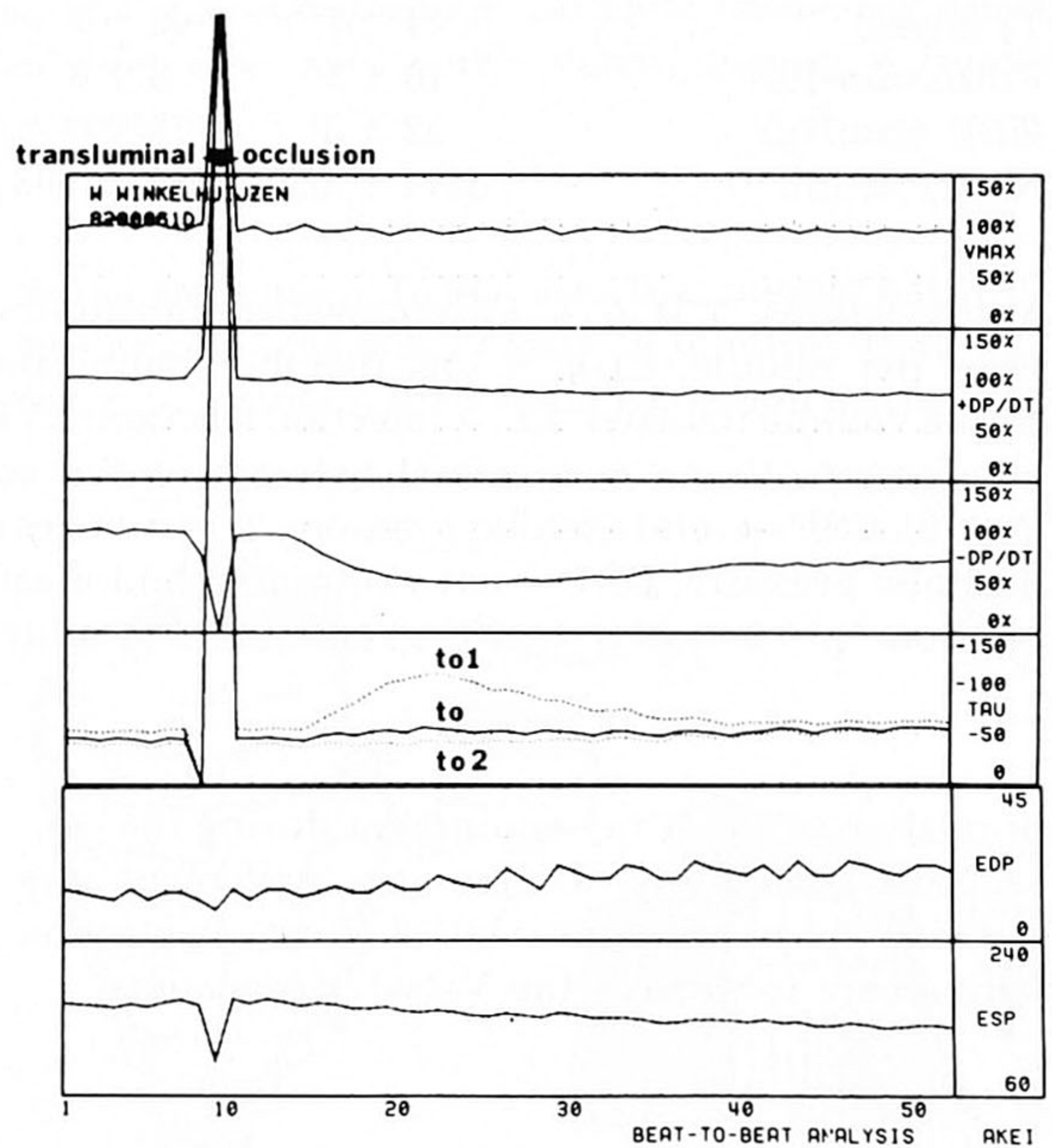

Fig. 3. Haemodynamic measurements in a patient during percutaneous transluminal coronary angioplasty (PTCA). From top to bottom, maximal velocity of the contractile elements (Vmax), peak + and - dP/dt expressed as a percentage of control values, the time constants of relaxation To_1 dashed line, To solid line, To_2 dotted line (scale 50 msec), end-diastolic pressure (EDP, scale 15 mmHg), peak systolic pressure (ESP, scale 60 mmHg, with 60 mmHg offset). The break in the data at beat 10 corresponds in inflation of the PTCA balloon.
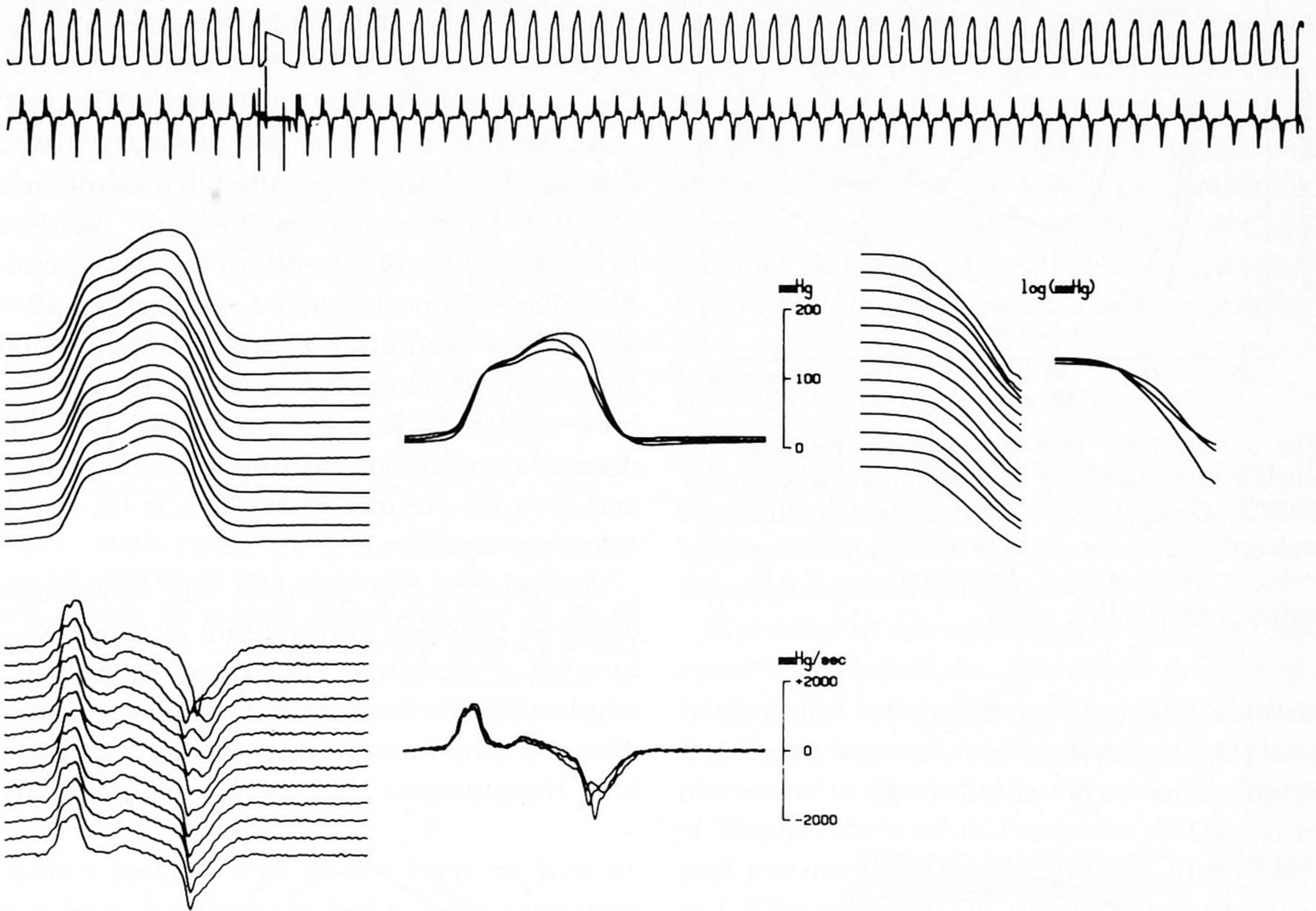
Fig. 4. Effects of coronary artery occlusion on left ventricular pressure (mmHg) and + and - dP/dt (mmHg/sec). The break in the recording at beat 15 corresponds the inflation of the balloon. On the left hand side, the left ventricular pressure and + and -dP/dt of individual beats are displayed (15, 18, 21, and so forth) while the natural logarithm of the pressure is shown on the right hand side. Notice decrease in -dP/dt associated with an irregularity in the upstroke of the negative dP/dt curve. After 30 sec (beat 42) peak -dP/dt starts to return toward a more normal shape of the signal.

depression in myocardial mechanics without any indication of an early peak (Fig. 3).

In contrast, within four of five beats after occlusion, a deformation appeared in the ascending limb of the negative dP/dt curve (Fig. 4) and in the next ten seconds this deformation in the negative dP/dt curve gradually increased so that the irregularity in the negative dP/dt curve reached the same height as peak −dP/dt which had progressively decreased to its nadir. In the next 20−50 sec, peak −dP/dt began to return towards control levels with a resolution of the irregularity in the ascending limb of −dP/dt. At 50 sec, peak −dP/dt

recovered to 77 % of the preocclusion value and the deformity was no longer present.

This deformation of the negative dP/dt signal at the early phase of the occlusion, means that the time course of left ventricular pressure decay deviates substantially from the mono-exponential model usually proposed, and it means also that asynchronous contraction or relaxation may be involved at the very beginning of the transluminal occlusion. Therefore bi-exponential fitting of the pressure curve was computed during the isovolumic relaxation, primarily on the basis that the pressure curve when plotted on semilog-
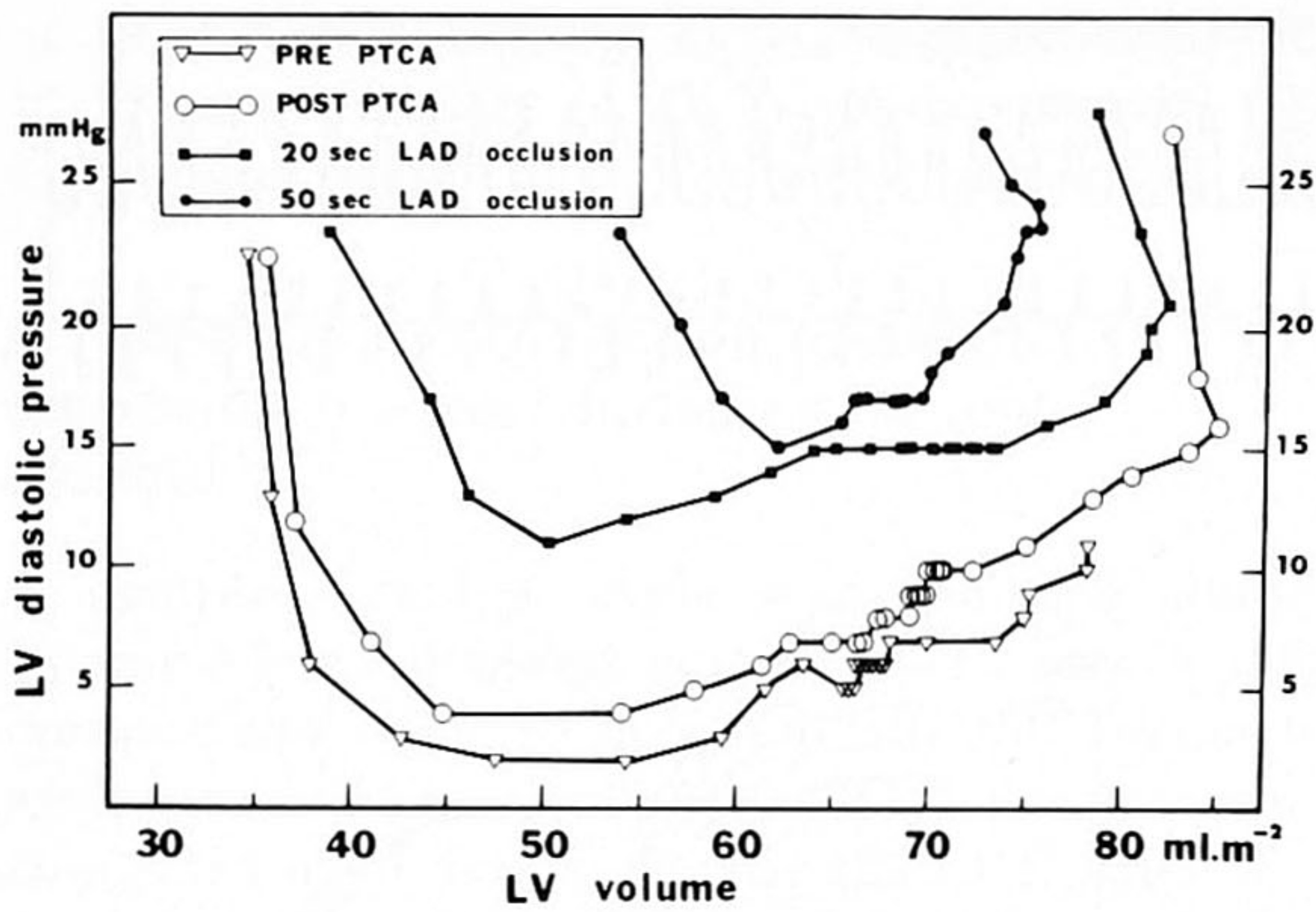
Fig. 5. Diastolic pressure volume relationships during percutaneous transluminal angioplasty (PTCA). During occlusion, there is a gradual shift upward and to the right of the diastolic pressure volume relationship. Abbreviations: LAD: left anterior descending artery.

arithmic paper was noted to follow two straight lines rather than the one predicted by the mono-exponential mode.

The second half of Table I summarizes the results of the relaxation parameters.

The behaviour of the two time constants (T_1, T_2) during PTCA is illustrated in Figure 3.

An occlusion of a major coronary artery during 20 sec already resulted in a significant $(p < 0.005)$ increase in end-systolic volume (from 31 ± 9 to 38 ± 9 ml/m²) while the end-diastolic volume remained unchanged after 20 sec and even after 50 sec of transluminal occlusion. At 50 sec the ejection fraction decreased from 62 % to 48 % $(p < 0.005)$ and this decrease was essentially due to an increase in end-systolic volume from 29 ± 7 to 41 ± 9 ml/m² $(p < 0.005)$.

The relationship between left ventricular diastolic pressure and volume during transluminal occlusion is illustrated by one example (Fig. 5). It is evident that the entire diastolic pressure-volume relationship during transluminal occlusion is gradually
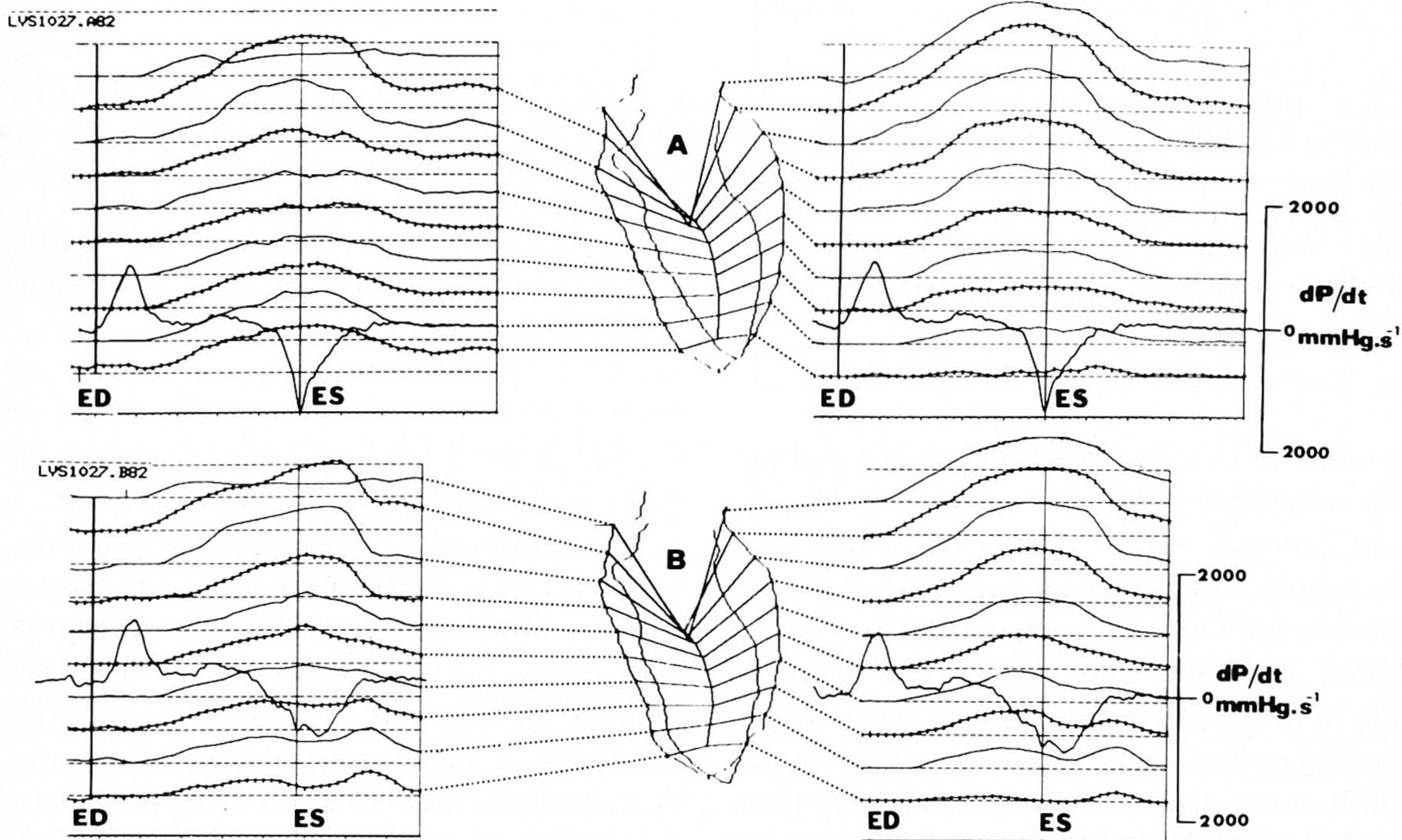

Fig. 6. Left ventricular wall displacement studied in twenty separate segments, ten in the anterior (right) and ten in the inferoposterior wall (left). A typical example of the relation between segmental wall displacement and dP/dt curve is observed before PTCA (A) and after 20 sec (B) of left anterior descending artery occlusion: after 20 sec of occlusion, the notch in the dP/dt curve corresponds to a second wave of inward wall displacement in the antero- and inferoapical segments.
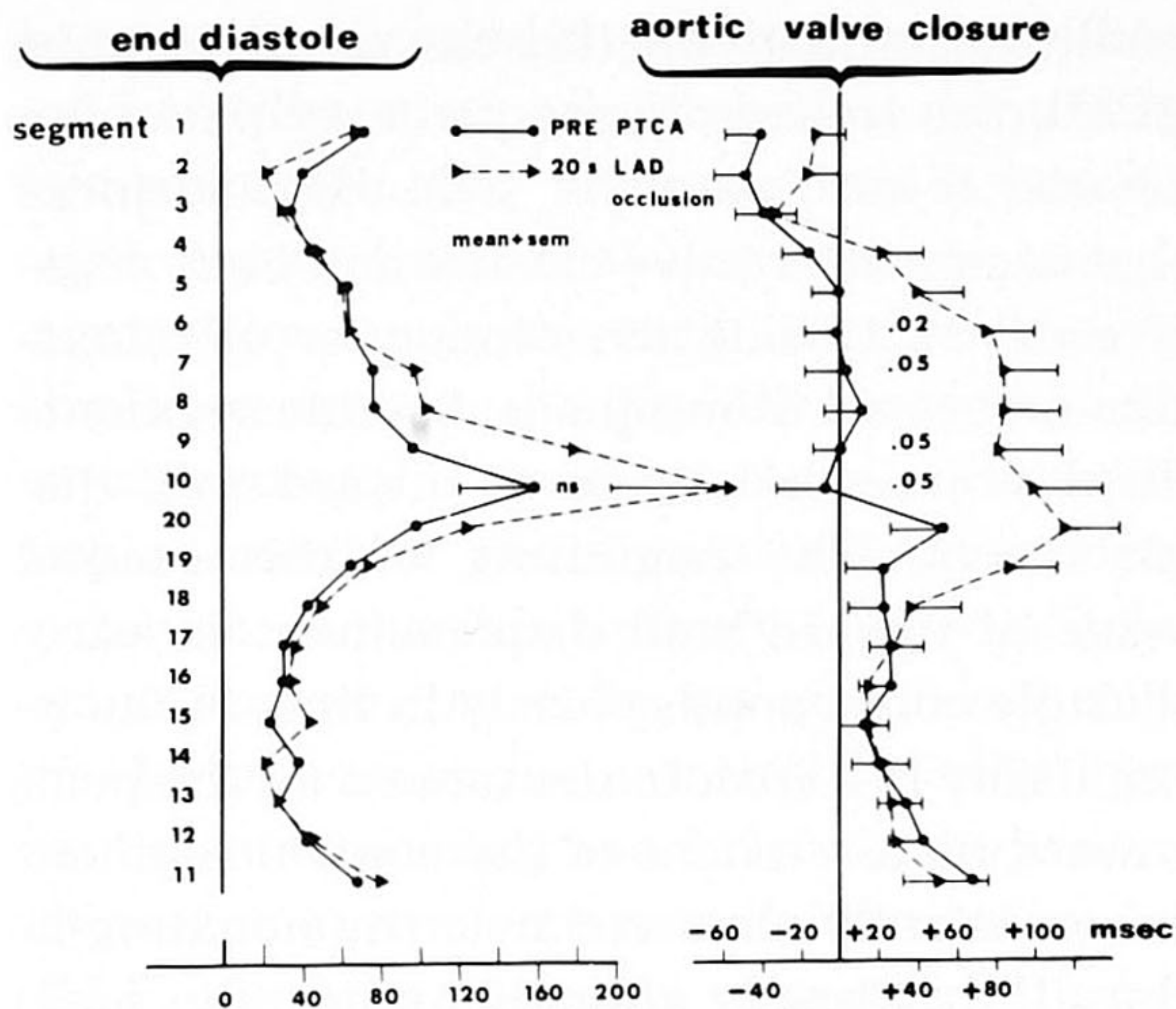
Fig. 7. Delay (msec) in onset of displacement for the 20 individual wall segments with respect to end-diastole (time zero) before and after 20 sec of LAD occlusion. Time relationship between aortic valve closure (time zero) and the occurrence of maximal wall displacement before and after 20 sec of LAD occlusion.

the K constant considered as an index of volume stiffness was significantly increased after 50 sec of transluminal occlusion (Table I). Nevertheless, the haemodynamic and cineangiographic investigations performed after completion of the PTCA procedure demonstrated the perfect reversibility of these changes in volume as well as the normalization of the different pressure derived indices.

B. Regional left ventricular function

The profound effect of a 20 sec occlusion of the left anterior descending artery (LAD) on left ventricular wall motion and its time sequence is shown in Figure 6.

The delay in onset of displacement with respect to end-diastole as well as the timing relationship between the aortic valve closure and the occurrence of the maximal wall displacement is illustrated in Figure 7. The onset of displacement of the anterior and inferior wall was not significantly affected after 20 sec of LAD occlusion. On the contrary, the moment of maximal wall displacement for the anterior wall shifted from end-systole to early

shifted upward and to the right so that at any given volume, the diastolic pressures were higher. This effect was consistently observed after 50 sec of occlusion. Furthermore,
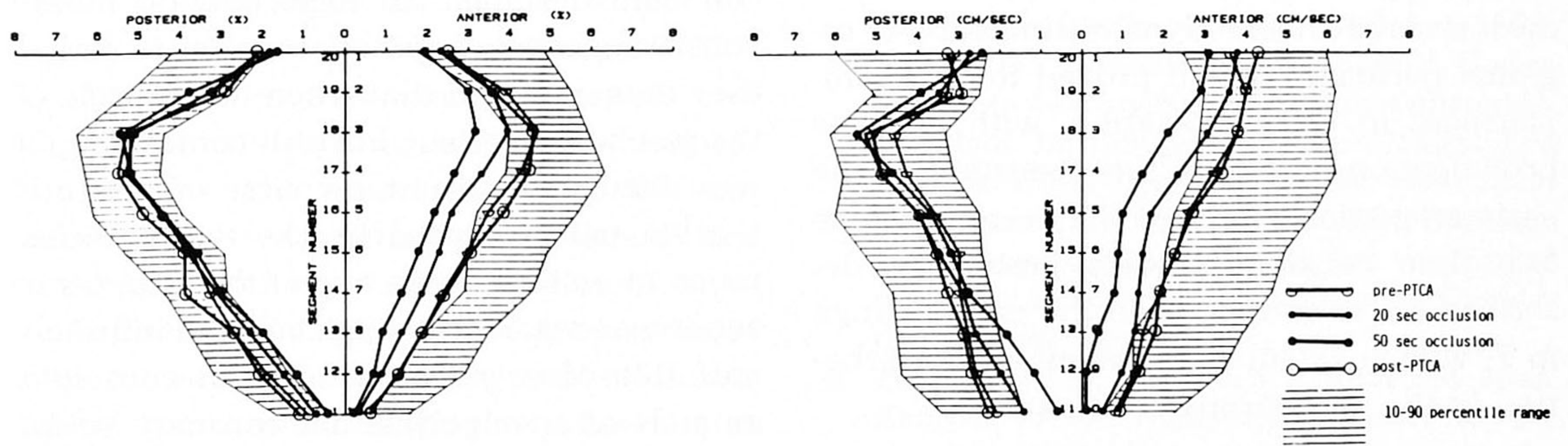

Fig. 8. A) Display of the computed regional contribution to global ejection fraction (CREF) values after a 20 or 50 sec occlusion of the left anterior descending artery. On the X-axis the CREF values of the anterior and inferoposterior wall areas are displayed (%), while on the Y-axis the segment numbers of the anterior wall (1–10) and of the inferoposterior wall (11–20) are depicted. The shaded zones represent the 10th–90th percentile area of CREF values in normal individuals.
B) Mean ejection phase velocity: pre PTCA versus 20 sec and 50 sec occlusion of the left anterior descending artery. On the X-axis the velocity values of the anterior and inferoposterior wall areas are displayed (cm/sec) while on the Y-axis the segment numbers of the anterior wall (1–10) and of the inferoposterior wall (11–20) are depicted. The shaded zones represent the 10th–90th percentile area in normal individuals.

disatole. The anterolateral segment (no 6 and 7) and the apical segment (no 9 and 10) of the anterior wall, as well as the apical segment (no 20 and 19) of the inferior wall appeared to be most affected.

The measurement of mean ejection phase velocity, after 20 and 50 sec of LAD occlusion, showed a decrease which was again more pronounced in the anterior wall segments (Figure 8). The regional wall motion and wall velocity (Figure 8A and B) show a similar response to LAD occlusion. These data clearly demonstrate a progressive myocardial depression affecting specifically the anterolateral and apical segments.

It must be emphasized that all these ischaemic changes were transient and perfectly reversible as demonstrated by the regional analysis of the last cineangiogram performed after completion of the whole procedure.

DISCUSSION

Global and regional left ventricular performance

The earliest (1 to 15 sec after occlusion) and most sensitive haemodynamic indicator of regional perfusion deficit proved to be an impairment in early relaxation, with extreme prolongation of T_1, the time constant of the early relaxation phase. If the premise of the two time constant models previously described (7), is correct, then the early change in T_1 with constant T_2 represent an exacerbation in the asynchrony of relaxation.

This is illustrated by the change in negative dP/dt and wall displacement induced by a 20 sec coronary occlusion (Fig. 6b). Within four or five beats after occlusion, a distinct deformation appears in the ascending limb of the negative dP/dt curve and in the next ten seconds this deformation reaches the same height as peak −dP/dt which in the meantime has progressively decreased to its

nadir. Accompanying this change in negative dP/dt, the ischaemic segments exhibit a biphasic inward-outward wall displacement that occurs after valve closure and peak negative dP/dt. During the remainder of relaxation and rapid filling the ischaemic segments display a second wave of inward wall displacement. The beginning of this second wave of inward wall displacement in early diastole corresponds closely in time to the irregularity in dP/dt. In the same way, the peak inward displacement of the control segment is consistently observed near the notching in the dP/dt. Shortly after this point, the pressure ceases to have a relaxation time constant T_1 and abruptly switches to T_2. On the other hand, after 50 sec of occlusion the majority of the ischaemic segments were akinetic exhibiting an increased regional stiffness, whereas T_1, the time constant of the early relaxation phase tended to return toward less abnormal values. At 50 sec, the deformity in −dP/dt was no longer present.

The connection between transient asynergy, myocardial ischaemia and alteration in the time course of relaxation was ponted out as early as 1969 by Tyberg et al. (13) who designed an experimental model consisting of two papillary muscles in series; they demonstrated that when one muscle of the pair was hypoxic, but still contracting, it was disturbing the time course of the total tension fall generated by the two muscles, much more than when one of the muscles in series was not contracting at all and infinitely stiff (13). More recent studies in conscious animals after experimental coronary occlusion have indicated that ventricular dyssynchrony due to late systolic contraction and relaxation in different regions can produce marked effects on the linearity and maximal rate of pressure fall in the left ventricle (14, 15, 16).

The present study suggests that a similar phenomenon may occur in the intact human heart during acute ischaemia. At 20 sec, the

late systolic outward displacement of the ischaemic segment is probably passive and due to a simultaneously increased and active inward displacement of the nonischaemic segments. Conversely the early diastolic inward displacement of the ischaemic segments must correspond to an accelerated outward displacement of the normal segment. Ultimately the ischaemic zone after 20 sec of ischaemia appears to act as an additional elastic element, in series with the actively contracting and relaxing non-ischaemic segment. This mechanism is consistent with the model of LV pressure relaxation recently proposed by our group (7) which assumes that the observed time constant T_1 results from the combined action of that fraction of the myocardium in the process of relaxing and the remainder yet to initiate relaxation.

CLINICAL IMPLICATIONS

Experimental data on atherosclerotic vessel segments have shown that volume reduction of atherosclerotic tissue is related to the duration of pressure application. These findings have led many clinicians to use longer inflation durations (30–60 sec) during PTCA (17, 18). On the other hand, Braunwald and Kloner (19) have recently addressed the question whether the myocardium can become chronically, even permanently »stunned» as a consequence of repetitive episodes of myocardial ischaemia. Although most episodes of transient ischaemia occurring in our patients during transluminal angioplasty are not as severe as those of the animal studies (14, 15, 20), the total duration of occlusive episodes during PTCA has increased considerably since our initial experience: the median is now four minutes and a few cases exceed ten minutes in our laboratory (2). This total occlusion time of four minutes might be excessive since it has been demonstrated in conscious dogs that the return of myocardial

function is delayed after periods of coronary occlusion as brief as 100 seconds. Here, the reactive hyperaemia which occurs normally during reperfusion is prevented by a residual subtotal occlusion (21) a situation which does not apply after successful PTCA. In this respect, the results of the present study seem to be reassuring since there is no evidence of global or regional myocardial dysfunction, even after 4 to 6 coronary occlusions, each of them lasting for 40 to 60 seconds.

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