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Diastolic aortic pressure rise during percutaneous transluminal coronary angioplasty: an index of left ventricular systolic dysfunction

Ioannis A Paraskevaidis, Zenon S Kyriakides, Antonis K Kassimatis, Thomas P Apostolou, George K Kalopisis, Dimitrios Th Kremastinos

Abstract

***Objectives*—To investigate the relation between diastolic aortic pressure response and left ventricular systolic dysfunction during percutaneous transluminal coronary angioplasty.**

***Background*—The abnormal diastolic blood pressure rise during exercise in patients with coronary artery disease probably reflects left ventricular systolic dysfunction rather than the number of stenosed coronary arteries.**

***Methods*—Aortic blood pressures and left ventricular systolic function indices were estimated in 26 patients with single proximal stenosis of the left anterior descending coronary artery both before and during angioplasty.**

***Results*—During coronary angioplasty all patients presented an increase in diastolic aortic pressure ($P \ll 0{\cdot}001$), 8–12 s before intracoronary electrocardiographic changes. During acute ischaemia there was a decrease in left ventricular ejection fraction ($P \ll 0{\cdot}001$) and stroke volume ($P \ll 0{\cdot}001$) and an increase in end systolic volume ($P \ll 0{\cdot}001$) and left ventricular end diastolic pressure ($P \ll 0{\cdot}001$). No statistically significant changes were observed in systolic blood pressure or heart rate. The aortic diastolic pressure increase was correlated with the decrease in ejection fraction ($r = -0{\cdot}95$, $P \ll 0{\cdot}001$) and with the increases in end systolic volume ($r = 0{\cdot}86$, $P \ll 0{\cdot}001$) and left ventricular end diastolic pressure ($r = 0{\cdot}85$, $P \ll 0{\cdot}001$).**

***Conclusions*—The rise in diastolic aortic pressure during percutaneous transluminal coronary angioplasty occurs earlier than intracoronary electrocardiographic changes and is related to ischaemic left ventricular systolic dysfunction.**

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Keywords: diastolic aortic pressure; left ventricular systolic dysfunction; coronary angioplasty

Cardiac Department, Onassis Cardiac Surgery Centre, Athens, Greece
I A Paraskevaidis
Z S Kyriakides
G K Kalopisis
D Th Kremastinos

Athens General Hospital, Athens, Greece
A K Kassimatis
T P Apostolou

Correspondence to:
Dr I A Paraskevaidis, Onassis Cardiac Surgery Centre, Department of Cardiology, 356 Sygrou Ave, 176 74 Athens, Greece.

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Although systolic blood pressure behaviour has been extensively studied in patients with coronary artery disease[1 2] and well correlated with myocardial ischaemia[3 4] there are few data on diastolic blood pressure response during ischaemia. We and others have recently shown that the rise of diastolic blood pressure during stress testing in patients with coronary artery disease is an index of latent left ventricular systolic dysfunction[5 6] and is not correlated with the number of stenotic arteries, as others have previously suggested.[7 8] In all studies diastolic blood pressure was measured during stress induced ischaemia, while the indices of left ventricular systolic function were measured at rest. Percutaneous transluminal coronary angioplasty offers a unique opportunity to study the sequential changes in left ventricular function during transient occlusion of the vessel and thus during acute myocardial ischaemia. It has been well documented that during coronary angioplasty there is an impairment of the haemodynamic indices of left ventricular systolic function.[9–12] Although an increase in diastolic blood pressure has been observed during coronary angioplasty[13] no relation between diastolic blood pressure and left ventricular haemodynamic variables was found.

The purpose of this study was to investigate the relation between diastolic aortic blood pressure response and left ventricular systolic dysfunction during percutaneous transluminal coronary angioplasty.

Methods

STUDY PATIENTS

Twenty six patients (22 male and four female), aged 56 (SD 10) years with left anterior descending coronary artery disease were included in this study. All patients had a history of stable angina (Canadian Heart Association, classes III-IV) and were in New York Heart Association functional class I. All patients were receiving oral antianginal treatment (stopped 24 hours before the procedure), sublingual glyceryl trinitrate (when necessary), and aspirin, while 23 of them were also taking β blocker medication which was stopped three days before the procedure. None of the patients had had angioplasty of a totally occluded artery or had a history of myocardial infarction or hypertension. Coronary angiography, performed two to five weeks before angioplasty, showed significant proximal left anterior descending artery stenosis (defined as > 85% of lumen diameter).

CARDIAC CATHETERISATION AND ANGIOPLASTY PROTOCOL

All patients underwent left heart catheterisation by the Seldinger technique. A pigtail

catheter was introduced into the left ventricle via the left femoral artery. Evaluations immediately before angioplasty included initial haemodynamic recordings (with the use of fluid filled catheters) consisting of left ventricular and aortic pressures (ascending aorta) followed by contrast left ventricular angiography (30° right anterior oblique). Special care was taken to ensure that the fluid in the cannula, tubing, and transducer dome was free of air bubbles. Without premedication, percutaneous transluminal coronary angioplasty (90 s; 8 atm) was performed after left ventricular and aortic pressures had returned to preventriculography values (11–15 min to be certain). The guiding catheter for the coronary angioplasty procedure was introduced through the right femoral artery. Patients were monitored during the procedure with a three-lead electrocardiogram (ECG leads D_1, D_2, and intracoronary). Continuous ECG, left ventricular pressure, and aortic pressure recordings were made simultaneously during angioplasty. Balloon dilatation catheters and guide wires of variable sizes were used as clinically indicated. During angioplasty, and more specifically 40–50 s after balloon inflation, haemodynamic variables and the second left ventriculography[11 14 15] from the same view were obtained using the same procedure. In patients who developed angina (n = 4) earlier than 50 s during angioplasty, haemodynamic variables and left ventriculography were obtained immediately. All patients gave their written informed consent to the procedure. The study was approved by the hospital's ethics committee.

DERIVED CALCULATION

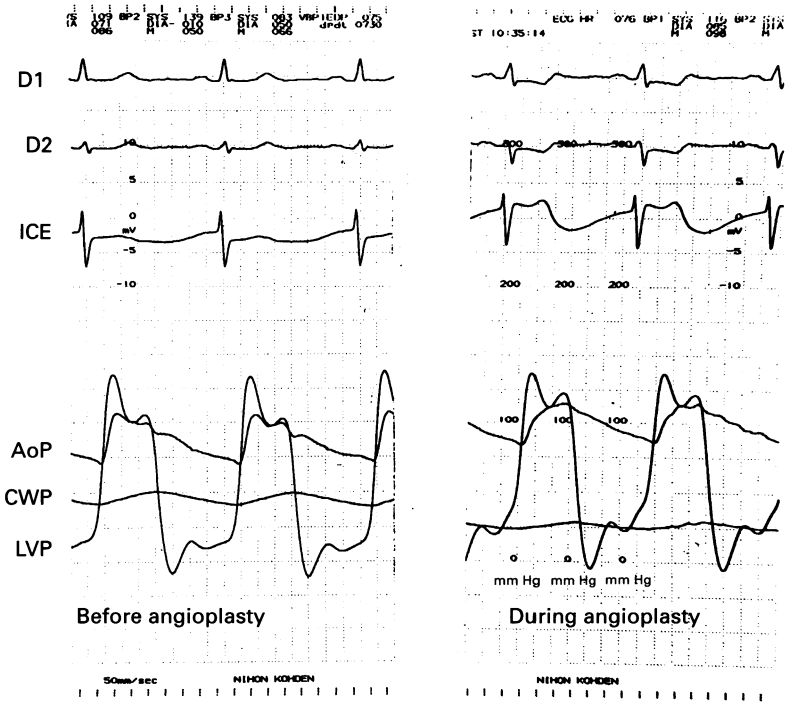
Left ventriculography was performed for the determination of end systolic and end diastolic volume and ejection fraction both before and during angioplasty. Stroke volume was measured by subtracting end diastolic from end systolic volume. Left ventricular end diastolic and systolic pressures, and aortic systolic and diastolic pressures were also estimated both before and during angioplasty. All haemodynamic variables were determined before left ventriculography was performed.

STATISTICS

All values are expressed as mean (SD). The *t* test for paired data was used for values before and during angioplasty. Linear regression analysis was used to correlate the diastolic blood pressure difference (before-during acute ischaemia) with the haemodynamic variables. A P value <0·05 was considered statistically significant.

Results

The main haemodynamic findings before and after coronary angioplasty are shown in table 1. All patients had a statistically significant increase (13·9 (5·9) mm Hg) in diastolic aortic pressure (figure). Fifteen out of 26 patients developed angina during coronary angioplasty and an ST segment deviation (seven with elevation; eight with depression) 8–12 s after the increase in diastolic aortic pressure. During angioplasty there were decreases in ejection fraction (14·2 (6·3)%) and in stroke volume (13 (6·5) ml/m²) whereas there were increases in left ventricular end diastolic pressure (13·1 (7·0) mm Hg), end



Increase in aortic diastolic pressure during percutaneous transluminal coronary angioplasty recorded simultaneously with an increase in left ventricular end diastolic pressure. AoP, aortic pressure; CWP, coronary wedge pressure; D1, D2, ICE, electrocardiographic leads D1, D2 and intracoronary; LVP, left ventricular pressure

Table 1 Haemodynamic findings before and during coronary angioplasty (n = 26). Values are means (SD)

	Before	*During*	*P value*
SAP (mm Hg)	134 (14)	134·8 (12)	NS
DAP (mm Hg)	73·4 (10·6)	89·6 (16·1)	<0·00001
EF (%)	59·6 (11·3)	45·4 (13·5)	<0·00001
LVEDP (mm Hg)	10·3 (1·7)	23·4 (7·9)	<0·00001
ESV (ml/m2)	28·9 (2·3)	54·6 (8·4)	<0·00001
EDV (ml/m2)	85·6 (4·7)	95·5 (5·7)	<0·00001
SV (ml/m2)	55·9 (5·1)	42·8 (8·0)	<0·00001
mBP (mm Hg)	95 (12·5)	104·6 (12·9)	<0·00001
HR (beats/min)	77·7 (4·2)	78·1 (3)	NS

SAP, systolic aortic blood pressure; DAP, diastolic aortic blood pressure; EF, ejection fraction; LVEDP, left ventricular end diastolic pressure; ESV, end systolic volume; EDV, end diastolic volume; SV, stroke volume; mBP, mean blood pressure; HR, heart rate.

Table 2 Correlation coefficients of difference in aortic diastolic pressure before and during coronary angioplasty compared with differences in the haemodynamic variety (n = 26)

		dEF	*dESV*	*dLVEDP*
dDAP	*r*:	−0·95	0·86	0·85
	P:	<0·0001	<0·0001	<0·0001

d, difference between values before and during angioplasty; EF, ejection fraction; ESV, end systolic volume; LVEDP, left ventricular end diastolic volume.

Table 3 Haemodynamic findings from the individual patients

Patient	SAP_1 (mm Hg)	DAP_1 (mm Hg)	SAP_2 (mm Hg)	DAP_2 (mm Hg)	dSAP (mm Hg)	dDAP (mm Hg)	mBP_1 (mm Hg)	mBP_2 (mm Hg)	EF_1 (%)	EF_2 (%)	dEF (%)	dmBP (mm Hg)
1	137	72	145	84	8	12	93·6	104·3	65	55	10	10·7
2	140	75	140	76	0	1	96·6	97·3	80	75	5	1·3
3	120	72	125	85	5	13	88·0	98·3	57	43	14	10·3
4	150	90	153	132	3	12	130·0	139·0	45	35	10	9·0
5	130	55	138	59	8	4	80·0	85·3	52	48	4	5·3
6	113	63	119	75	6	12	79·6	89·6	47	37	10	10·0
7	118	75	120	90	2	15	89·3	100·0	65	48	17	11·7
8	135	75	137	85	2	10	95·0	102·3	70	62	8	7·3
9	118	75	123	93	5	18	89·3	103·0	50	30	20	3·7
10	135	67	131	75	−4	8	89·3	93·6	50	45	5	4·0
11	150	83	150	100	0	17	105·3	116·6	50	31	19	11·3
12	125	68	126	88	1	20	87·0	100·6	50	28	22	12·4
13	135	73	138	108	3	25	100·3	118·0	55	31	24	17·3
14	153	70	155	97	2	27	97·6	116·3	52	27	25	18·7
15	95	55	98	73	3	18	68·3	81·3	60	40	20	13·0
16	134	93	132	100	−2	7	106·6	110·6	60	52	8	4·0
17	153	75	148	98	−5	18	104·3	114·6	75	55	20	10·3
18	135	61	131	75	−4	14	85·6	93·6	80	65	15	8·0
19	140	86	135	97	−5	11	104·0	109·6	58	48	10	5·6
20	145	88	142	106	−3	18	107·0	118·0	54	34	20	11·0
21	155	85	140	112	−15	20	113·0	121·3	83	63	20	7·7
22	125	73	132	90	7	17	90·3	104·0	60	43	17	13·7
23	127	85	130	103	3	13	102·3	112·0	40	25	15	9·7
24	142	75	142	90	0	15	97·3	107·3	60	43	17	10·0
25	135	55	136	65	1	10	81·6	88·6	62	55	7	7·0
26	137	65	139	73	2	8	89·0	95·0	70	63	7	6·0
Mean (SD)	134·0 (14)	73·4 (10·6)	134·8 (12)	89·6 (16·1)	0·9 (4·9)	13·9 (5·9)	95 (12·5)	104·6 (12·9)	59·6 (11·3)	45·4 (13·5)	14·2 (6·4)	9·2 (4·1)

Subscript 1, before angioplasty; subscript 2, during angioplasty; SAP, systolic arterial pressure; DAP, diastolic arterial pressure; d, differences between values before and during angioplasty; mBP, mean blood pressure; EF, ejection fraction; LVEDP, left ventricular end diastolic pressure; ESV, end systolic volume; EDV, end diastolic volume; SV, stroke volume, HR, heart rate.

diastolic volume (9·8 (3·2) ml/m²) and end systolic volume (25·6 (6·4) ml/m²). Heart rate and systolic blood pressure did not change during angioplasty, whereas mean blood pressure increased (9·2 (4·1) mm Hg).

The correlation coefficients of the difference in aortic diastolic pressure before and during angioplasty compared with differences in the haemodynamic variables are shown in table 2. The difference in aortic diastolic pressure before and during angioplasty was not correlated significantly with baseline left ventricular function. The individual results for the whole study group are given in table 3.

Discussion

This study shows that during percutaneous transluminal coronary angioplasty there is an increase in mean aortic pressure because of an increase in diastolic aortic pressure. This increase in diastolic blood pressure precedes the intracoronary electrocardiographic changes and is well correlated with the impairment of left ventricular systolic function when myocardial ischaemia occurs.

DIASTOLIC AORTIC PRESSURE AND LEFT VENTRICULAR HAEMODYNAMIC CHANGES

Since the systolic aortic pressure before and during coronary angioplasty was similar in all patients, the increase in mean aortic pressure was related to the increase in diastolic aortic pressure. This increase was well correlated with the left systolic ventricular function, expressed by a decrease in ejection fraction and stroke volume, and an increase in left ventricular end systolic volume and end diastolic pressure. The impairment of left ventricular systolic function during coronary angioplasty has been well established in previous studies where left ventricular systolic dysfunction during angioplasty was reported in experimental animals[16 17] and humans.[8 11] More specifically, it has been suggested that during angioplasty there is a reduction of left ventricular systolic function which is characterised by a rightward displacement of the end systolic pressure-volume relation.[8]

MECHANISM OF BLOOD PRESSURE CHANGES

Although diastolic blood pressure is the primary determinant of arterial baroreflex neuroeffector responses in humans,[18] the exact mechanism by which diastolic blood pressure is raised is not totally clear. It has been suggested that there is a muscle afferent feedback mechanism, whereby signals are sent from receptors in contracting muscle to the brain, stimulating sympathetic centres,[19] or most likely excited through a metabolic mechanism.[20] Moreover, it has been shown that there is a greater increase in mean arterial pressure when the muscle is rendered ischaemic than when ischaemia is absent.[21] Inadequate cardiac output relative to local tissue demands, generating ischaemia, would therefore affect blood pressure responses.

When the heart is coupled to the circulation the resulting pressure and flow are determined by the mutual mechanical interaction of the ventricle and the circulation, and not by either one or other acting independently.[22] In this respect, the physiological terms "cardiac output" and "arterial pressure", which refer to the flow and pressure determined by the joint interaction of the heart and circulation, could be misleading. In the diseased ventricle the mutual interaction between the ventricular pump and the arterial load becomes more pronounced.[23] Maintenance of a normal or nearly normal level of arterial pressure is

$LVEDP_1$ (mm Hg)	$LVEDP_2$ (mm Hg)	DLVEDP (mm Hg)	ESV_1 (mm Hg)	ESV_2 (mm Hg)	dESV (mm Hg)	EDV_1 (mm Hg)	EDV_2 (mm Hg)	dEDV (mm Hg)	SV_1 (ml/m^2)	SV_2 (ml/m^2)	dSV (ml/m^2)	HR_1 (beats/min)	HR_2 (beats/min)
9	16	7	27	47	20	80	89	9	53	42	9	78	79
11	16	5	26	42	16	81	91	10	55	49	6	81	83
9	18	9	28	50	22	90	102	12	62	52	10	79	81
10	19	9	27	52	25	75	88	13	48	36	12	76	75
9	14	5	28	46	18	80	85	5	52	39	13	85	81
10	19	9	28	56	28	80	95	15	52	49	3	82	79
11	22	11	30	62	32	90	100	10	60	38	22	83	80
8	14	6	27	50	23	90	98	8	63	48	15	80	80
9	23	14	30	62	32	88	95	7	58	33	25	78	81
10	14	4	27	47	20	90	97	7	53	50	3	69	71
9	22	13	29	58	29	85	92	7	46	34	12	74	76
10	35	25	31	62	31	90	102	12	59	40	19	76	77
8	32	24	34	69	35	85	100	15	51	31	20	81	79
10	34	24	33	67	34	88	105	17	55	38	17	77	75
13	28	15	29	58	29	88	95	7	59	37	22	73	75
9	16	7	27	45	18	92	101	9	65	56	9	79	81
12	38	26	32	65	33	92	101	9	60	56	4	72	75
10	21	11	29	51	22	88	95	7	59	44	15	81	79
11	19	8	26	43	17	83	90	7	57	47	10	85	81
13	35	22	31	66	35	90	103	13	59	37	22	82	82
12	32	20	32	61	29	80	90	10	48	39	9	75	76
13	32	19	30	62	32	78	85	7	48	33	15	74	78
11	21	10	26	47	21	85	100	15	59	53	6	76	79
14	34	20	30	60	30	83	91	8	53	31	22	74	74
9	16	7	27	44	17	86	92	6	59	48	11	72	74
8	19	11	28	47	19	88	100	12	60	53	7	79	79
10·3 (1·7)	23·4 (7·9)	13·1 (7·0)	28·9 (2·3)	54·6 (8·4)	25·6 (6·4)	85·6 (4·7)	95·5 (5·7)	9·9 (3·3)	55·9 (5·1)	42·8 (8·0)	13 (6·5)	77·7 (4·2)	78·1 (3·0)

important, not only for pressure control itself, but also because it allows adequate blood flow through the tissues.[24] Thus, during percutaneous transluminal coronary angioplasty, where the ventricular pump function is reduced, the circulatory system has to respond accordingly. In this respect, in our study the attempt to maintain the mean blood pressure near normal might be due to systemic resistance increases, since systolic pressure was not significantly changed, stroke volume was decreased, and there was no change in heart rate. Recent studies support this view by showing that during coronary angioplasty, when a major coronary artery is totally blocked, there is a rise in peripheral resistance.[25] This is due to the flattening of the arterial pressure curve caused by the decreased pulse pressure, equivalent to the reduction of the arterial peak dP/dt.[25 26] Thus it could be assumed that during percutaneous transluminal coronary angioplasty the decrease in cardiac output led to an increase in peripheral resistance and to a rise in diastolic blood pressure.

RELATION TO INTRACORONARY ECG CHANGES

Finally, this study shows that during acute ischaemia the diastolic aortic blood pressure increased before intracoronary ECG changes. The earlier appearance of the rise in diastolic pressure is in accordance with previous studies[5 6] and is not paradoxical, since haemodynamic changes are known to precede the electrocardiographic findings.

LIMITATIONS OF THE STUDY

Firstly, systemic resistance was not measured since the changes in oxygen consumption and thus the measurement of cardiac output during a short period of time such as during coronary angioplasty are inaccurate. Secondly, stroke volume, velocity of ejection, cardiac output, and left ventricular performance might influence distensibility of the aorta[27 28] and subsequently the response of aortic blood pressure. In this respect, a multivariable analysis of the above variables in a large study population might clarify our findings further.

Conclusions

In this study it has been shown that during acute ischaemia there is an impairment of left ventricular systolic function, probably leading to an increase in systemic arterial resistance and thus to an increase in the diastolic aortic blood pressure. The increase in diastolic aortic pressure occurs before intracoronary ECG changes and is well correlated with ischaemic left ventricular dysfunction. These findings support the view that the increase in diastolic blood pressure is an index of left ventricular systolic dysfunction when myocardial ischaemia occurs.

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