THE SIGNIFICANCE OF ABNORMAL POLYGRAPHIC FINDINGS IN ISCHEMIC HEART DISEASE AND PRIMARY MYOCARDIAL DISEASE. CORRELATION OF LEFT VENTRICULAR SYSTOLIC TIME INTERVALS WITH CORONARY AND LEFT VENTRICULAR ANGIOGRAPHIC FINDINGS.

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Abstract

The results of coronary angiography (CAG) and left ventriculography (LVG) were correlated with measurements of left ventricular systolic time intervals (STI) in ischemic heart disease (IHD) and primary myocardial disease (PMD) in 35 subjects.

All 7 patients with IHD and abnormal wall motion, and all 7 patients with PMD showed significantly (p < 0.01) low values of ET/PEP and significantly (p < 0.01) high values of \triangle PEP. These polygraphic parameters varied widely in IHD patients with normal wall motion, with angina pectoris, or with myocardial infarction, and did not correlate with the number of vessels involved. Q-II and ET were poorly correlated with the severity or type of myocardial disease.

The correlation of ejection fraction with ET/PEP and with PEP was good: correlation coefficients were r=+0.66 and r=-0.70, respectively.

From these results it is concluded that 1) if a patient has an ET/PEP of more than 2.45 or a \triangle PEP of less than +13 msec, IHD with abnormal wall motion or PMD is most unlikely, and that 2) abnormal values of ET/PEP and \triangle PEP are related to the abnormally low ejection fractions which are found in IHD with abnormal wall motion or in PMD.

INTRODUCTION

Left ventricular systolic time intervals (STI) have been generally useful in the evaluation of cardiac function but they vary widely in patients with ischemic heart disease (IHD). In the present study we

tried to define the significance of abnormal STI's in patients with IHD and primary myocardial disease (PMD) by correlating STI values with the results of coronary angiography (CAG) and left ventriculography (LVG).

SUBJECTS AND METHOD

LVG and CAG were carried out in 22 cases of IHD, in 7 cases of PMD and in 6 control cases. All MI patients were studied more than two months after the onset of their acute episodes. The PMD cases included 2 cases of the congestive type and 4 cases of the hypertrophic type; cases of the obstructive type were excluded. As a special control group, 6 subjects with the mid-systolic click syndrome were selected who showed no abnormality on CAG or LVG. None of the patients had arrhythmias or conduction disturbances at the time of study. The sex and age distribution of all cases were as shown in Table 1.

TABLE 1.

The sex and age distribution of the subject studied

Classification	No. of Cases	Sex	Age	
A) IHD	22	M=21	34-59	
1) 0-VD	5			
1-V D	9			
2-V D	4			
3-V D	4			
2) M I	11			
ΑP	11			
3) $A(+)$	7			
A(-)	15			
B) PMD	7	M=6	26-49	
C) Control	6	M=1	22-56	
Total	(35)	(M = 28)	(22-56)	

IHD=ischemic heart disease, MI=myocardial infarction, AP=angina pectoris, VD=vessel disease, A(+)=with abnormal wall motion, A(-)=without abnormal wall motion, PMD=primary myocardial disease, M=male

The IHD cases were subdivided into those with 0-, 1-, 2- and 3-vessel disease (VD). Significant lesions were defined as those with 75% stenosis or more of the vascular lumen. On the basis of clinical data, the patients were also divided into those with myocardial infarction (MI) and those with angina pectoris (AP). In addition, the IHD group was divided into two groups according to the presence [A(+)] or the absence [A(-)] of abnormal wall motion (AWM) by angiography, using the criteria for dyssynergy established by Herman and Gorlin¹⁾.

Each polygraphic tracing was recorded immediately before the left heart catheterization. Measurements of STI's (Q-II, ET and PEP) were made as shown in Fig. 1. The difference between the measured value and the normal value (msec) was expressed as ΔQ -II, ΔET and ΔPEP^2 , and the ET/PEP ratio was calculated. All STI parameters were correlated with the site, number and degree of involved vessels on CAG as well as with the presence or absence of AWM on LVG. These data were compared with ejection fractions (EF) estimated from LVG by the area-length method as described by Chapman³⁾.

All data were subjected to chi-square analysis.

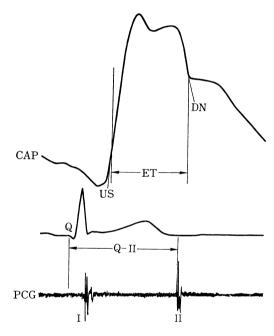


Fig. 1. The measurement of left ventricular systolic time intervals, from simultaneous tracings of carotid artery pulse (above), electrocardiogram (middle) and phonocardiogram (bottom). ET=ejection time, PEP=preejection period (QII-ET), I=first heart sound, II=second heart sound, US=upstroke, DN=dicrotic notch.

RESULTS

1) ET/PEP (Fig. 2).

Normal values (mean \pm 1SD) in our laboratory are 2.95 \pm 0.5. There was no significant difference between the MI and AP groups, but ET/

PEP values were below 2.45 (our lower limit of normal) in 9 (82%) of 11 cases of the MI group and in 8 (73%) of 11 cases of the AP group. All 7 cases of the [A(+)] group showed a significantly low value whereas 8 (53%) of 15 cases in the [A(-)] group had a low value. The difference between these groups was significant (p < 0.01). All 7 cases with PMD revealed a significantly low value (p < 0.01). In the click-murmur syndrome all values but one were within normal limits. In the primary myocardial disease group, none showed a normal value of ET/PEP.

	ET/ŖEP							
	1.0	1.5	2.0	2.5	3.0	3.5		
M I			8 00 000)O O	0			
A P			80 6	3 0	8			
A (+)			9.8 0 0	•				
A (-)			000	80∞0	&			
P M D		8	× % ××	××				
Control				ΔΔΔ	Δ	Å		

Fig. 2. ET/PEP values in patients with ischemic heart disease and primary myocardial disease. MI=myocardial infarction, AP=angina pectoris, A(+)=with abnormal wall motion of the left ventricle, A(-)=without abnormal wall motion of the left ventricle, PMD=primary myocardial disease. Controls=patients with click-murmur syndrome. Normal values (mean ±1SD) of ET/PEP in our laboratory are indicated by thick bar.

- 2) ΔPEP (Fig. 3). Normal values are 0 ± 13 msec (mean ±1 SD). ΔPEP exceeded +13 msec in 73% of the MI group, 55% of the AP group, 100% of the PMD group (p < 0.01) and in 17% of the click-syndrome group. None of the primary myocardial disease group had a normal ΔPEP .
 - 3) ΔET . Normal values are 0 ± 12 msec. (mean ±1 SD). ΔET was less than -12 msec in 55% of the MI group, 27% of the

AP group, 57% of the [A(+)] group, 33% of the [A(-)] group, 86% of the PMD group and in 33% of the click group. Only one of 22 cases with IHD has a Δ ET of more than +12 msec.

ΔΡΕΡ								
	-10	0	10	20	30	40	50	60 (msec)
M I		@	00 0	80	© (9090		
A P		00	0 @	000) C	0 0		
A (+)				•	• •	8 00		
A (-)		000	000	800) (0		
P M D				** *	×	⊗ ×		8
Control	ΔΔ	ΔΔ ,	۵	Δ				

Fig. 3. \(\Delta\)PEP values in patients with ischemic heart disease and with primary myocardial disease. Abbreviations as in Fig. 2.

4) ΔQ -II. Normal values are 0 ± 14 msec (mean ±1 SD).

 ΔQ -II was greater than +14 msec in 45% of the MI group, 64% of the AP group, 71% of the [A(+)] group, 47% of the [A(-)] group, 43% of the PMD group and in 17% of the click group. ΔQ -II was less than -14 msec were in 75% of the PMD group but in only one (5%) of the IHD group.

5) Relation of the number of involved coronary vessels to ET/PEP (Fig. 4).

Figure 4 shows the relation of the number of involved coronary vessels to ET/PEP in the IHD group. For reference, the PMD and click groups are also shown. ET/PEP was less than 2.45 in 4 of 5 cases with 0-VD, in 5 of 9 cases with 1-VD, in all 4 cases with 2-VD and in 3 of 4 cases with 3-VD.

6) Relation of vessel pathology to $\triangle PEP$ (Fig. 5).

 $\triangle PEP$ was more than +13 msec in all 5 patients with 0-VD, in 3 of 9 with 1-VD, in all 4 with 2-VD and in 3 of 4 with 3-VD.

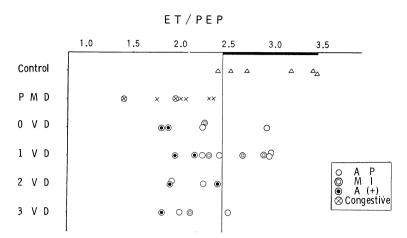


Fig. 4. Relation of the number of involved coronary vessels to ET/PEP. VD=vessel disease, congestive=congestive type of PMD. Other abbreviations, see Fig. 2. Other sub-groupings of patients indicated by symbols as defined in the enclosed square.

△PEP								
	-10	0	10	20	30	40	50	60 (msec)
Control	ΔΔ	ΔΔ Δ	`	Δ				
P M D				<i>x</i> × ×	×	⊗ >	<	8
0 V D				@O ()	••		
1 V D		000	o 0	0	•	•		
2 V D				∞		• 0		
3 V D			0	0		○ •		
	I							

Fig. 5. Relation of the number of involved coronary vessels to ΔPEP . Abbreviations see Fig. 2 and 4.

7) Relation of vessel pathology to ΔET .

 Δ ET was less than -12 msec in 3 out of 4 cases with 0-VD, in one of 9 with 1-VD, in one of with 2-VD and in 3 of 4 with 3-VD.

8) Relation of vessel pathology to 4Q-II.

4Q-II was less than +14 msec in 2 out of 4 cases with 0-VD, in 4 of 9 with 1-VD, in 3 out of 4 with 2-VD and in 1 of 4 with 3-VD.

9) Correlation between ejection fraction and STI.

Although left heart catheterization and non-invasive measurements were not carried out together, the correlation of EF with ET/PEP and with Δ PEP in the IHD and PMD cases was good. Correlation coefficient: r=+0.66 (p < 0.01) with ET/PEP and r=-0.70 (p < 0.01) with Δ PEP.

DISCUSSION

In the present study, abnormal values of ET/PEP and PEP were found in a high percentage of patients with IHD. The incidence of abnormal values was not clearly affected by the number of involved coronary arterial branches, and was not significantly different in the MI vs AP groups. However, in the group with abnormal wall motion abnormalities ET/PFP and ΔPEP were consistently abnormal (p < 0.01).

Stack et al.4 have reported abnormality of PEP/ET in less than 30% of their cases with 1-VD, but in over 80% of the cases with 2-Meng et al.5 reported similar results. Sarma et al.6 also found that patients with 3-VD showed a significant difference in △PEP from patients with 0-VD. The present study revealed that these results are probably more related to the presence of abnormal wall motion than to the number of vessels involved. In our group with AWM all had a low ET/PEP and a prolonged \(\Delta \text{PEP}, \) and the relationship between abnormal STI values and AWM has been previously documented⁷⁻¹⁰. This relationship is mediated by the low ejection fractions observed in the patients with AWM, and the high correlation which exists between STI's and the ejection fraction 11). Since patients with multivessel disease do not necessarily have AWM, and since patients with old MI do not necessarily show more marked wall motion abnormality than patients with AP, as attested by our study, STI values may commonly be normal in the presence of old MI or 3-VD.

Our PMD cases showed as high an incidence of ET/PEP and \triangle PEP abnormality (p < 0.01) as did the group with AWM in IHD. Due to the small numbers of cases, differences between the congestive and hyper-

trophic types of PMD could not be evaluated. In the present study, the STI parameter that distinguished between PMD and IHD was $\Delta Q-II$, which tended to be high in IHD with AWM, but low in the PMD group. This is an interesting difference which requires further investigation.

In the present study, as well as in those already reported by others⁴⁻¹⁰⁾, the ratio ET/PEP (or its reciprocal) and PEP were the indices most sensitive and reliable, among all STI's, for excluding AWM. From our results it is concluded that if an individual has an ET/PEP of more than 2.45 or a PEP of less than +13 msec IHD, AWM and PMD can be ruled out.

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