# II. Clinical problems

## EARLY ATRIAL PACING TEST FOR MYOCARDIAL CONTRACTILITY EVALUATION IN PATIENTS WITH UNSTABLE ANGINA PECTORIS

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Echocardiography is an easily accessible method for evaluation of left ventricular function and has an essential importance in determination of prognosis and behaviour of ischemic heart disease (IHD) patients. It is known that some IHD patients have normal left ventricular function at rest (3). On the other hand, echocardiographic examination of patients during physical loading is practically very difficult (3, 7, 9, 10). That is why it is appropriate to perform echocardiographic determination of the parameters of left ventricular function during an atrial pacing test.

The purpose of this study is to ascertain how left ventricular contractility changes during atrial pacing in patients with unstable angina pectoris (UAP).

# Material and methods

6 patients (4 males and 2 females) with UAP (crescent angina on the background of an old stable one) aged between 48 and 66 years (mean age of 58.3 years) were studied. UAP patients were diagnosed after WHO modified criteria (4). Treatment of patients was carried out by modern medicines resulting in overcoming of endangering symptoms in an Intensive Coronary Unit and then—in complete stabilization of the state in a general cardiological division (5).

An atrial pacing test and a simultaneous recording of M-mode echocardiography, apexcardiography (ACG), phonocardiography (PCG) and ECG was performed 3 till 7 days after complete clinical stabilization (on the background of treatment administered on the corresponding day after examination). Atrial pacing test was performed with a bipolar electrode introduced in the right atrium through the right subclavian or femoral vein (6). Pacing was realized by means of portable pacemaker of the «Medtronic» 5375 model. We started with 10 beats per minute above the patients' heart rate and then increased to 20 beats of each step (1, 2, 6, 7). Every step was of 2 min duration. Electrocardiogram was recorded on 6-channel ECG NEK-4 prior to test at 12 leads and during the pacing and the restitution period at the end of every minute at 6 precordial leads (V<sub>1-6</sub>) with velocity of moving band of 50 mm/sec. Simultaneous echoapex-phonocardiographic recording was carried out at left half-side patient's position from a standard parasternal position prior to and after every step of

pacing. Arterial pressure was measured after Korotkov's method prior to, during pacing at the end of every step as well as during the restitution period at the end of every minute. Atrial pacing test was considered positive when ischemic ECG-changes appeared (horizontal ST-segment depression > 0.1 mV with time duration > 0.08 sec in one or more leads) and/or in case of angina pectoris attack (6). When ECG-changes were absent pacing was continued till the appearance of disorder of atrioventricular conduction and/or till the appearance of heart rate of 160-170 per minute.

Mean velocity of circumferential fiber shortening  $(V_{6f})$  and the ratio between systolic arterial pressure and telesystolic left ventricular volume index (sAP — TSLVVI) prior to stimulation, with prethreshold (one step of pacing prior to the appearance of is hemic ECG-changes) and threshold (at the step of pacing when ischemic ECG-changes appeared) heart rate were estimated to

evaluate the dynamics of myocardial contractility.

#### Results and discussion

Atrial pacing test is positive in all the patients — 5 patients react with ST-depression only and one patient with ST-depression combined with angina

pectoris attack.

Parameters characterizing myocardial contractility demonstrate a biph asic dynamics. With increasing heart rate (HR) from 65.8  $\pm$  12.8 up to 83.3  $\pm$  8.2,  $V_{cf}$  increases by 13.5 per cent (p > 0.10). With further HR increase  $V_{cf}$  begins to decrease exceeding its initial one only by 5.4 per cent at prethreshold HR but being below it by 4.5 per cent at threshold HR (table 1). After initial increase in all the patients V begins to decrease in 3 patients 2 steps prior to, in 2 ones — one step prior to, and in one patient at threshold HR.  $V_{cf}$  ex

Table 1

Parameters characterizing left ventricular function

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a tradition of the one of the one of the original o	TSLVD  x ± s  \[ \Delta \psi \text{ towards} \]  initial rates	$\frac{TDLVD}{\overline{x}} = s$	$x \pm s$ $\Delta$ % towards initial rates	sAP-TSLVVI ratio $x \pm s$ $\Delta\%$ towards initial rates
Initial rates	3.33**0.67	5.01±0.56	1.11±0.17	3.53±1.72 <sub>7 → 6</sub>
Maximal rates	e de la <del>la</del> com-	lige o <del>ya</del> on in La sentiri	$1.26 \pm 0.10 \ (+13.5\%)$	4.88±2.54 (+38.2%)
Heart rate	March of the		83,3 + 8.2	90.2 = 20.7
Minimal rates	2.94+0.56 (+11.7%)	7' 1'-0 =0 31, 1-0 =0		b, me <u> </u>
Heart rate	93.3 ± 16.3	rock <u>d</u> ryd	5 . 6 . 70	de sig <u>u</u> ne did
At prethreshold heart rate	$3.14 \pm 0.53 \\ (+5.7\%)$	4.37 = 0.60	$1.17 \pm 0.12$ $(+5.4\%)$	$3.94 \pm 1.60 \ (+11.6\%)$
At treshe heart rat	3.27±0.51 (+1.8%)	4.22±0.54	1.06±0.12 (-4.5%)	3.59±1.34 (₩1.7%)

Although of different quantitaive characteristique, the dynamics of sAP—TSLVVI ratio is analogous. With HR increase from 65.8 ± 12.8 till 90.2 ± 20.7 sAP—TSLVVI ratio increase es by 38.2 per cent (p > 0.10). With further HR increase sAP—TSLVVI ratio decreases thus exceeding the initial level by 11.6 per cent at pre threshold HR but by 1.7 per cent at threshold one (table 1). With initial HR increase sAP—TSLVVI ratio incerases in 5 patients but remains unchanged in one patient. This ratio decreases in one patient 2 steps prior to, in 3 patients one step prior to and in two patients at threshold HR when HR continues to increase. sAP—TSLVVI ratio is over the initial level in 2 ratio and the law it in the rest 2 area at threshold HR

in 3 patients and below it in the rest 3 ones at threshold HR.

This dynamics of contractility parameters is realized on the background of the following changes of left ventricular size. Telediastolic left ventricular dimension (TDLVD) progressively decreases with increasing HR as its change between pretoreshold and threshold HR is considerably less than that between the other neighbouring steps (table 1). Telesystolic left ventricular dimension (TSLVD) decreases by 11.7 per cent (p > 0.10) when HR increases from  $65.8 \pm 12.8$  till  $93.3 \pm 16.3$ . With further HR increase TSLVD increases and at pretreshold HR it is by only 5.7 per cent over the initial values while at threshold HR it by 1.5 per cent below them (table 1). TSLVD begins to increase 2 steps prior to threshold HR in 3 patients, one step prior to threshold HR in 2 ones, and at threshold HR in one patient. It exceeds initial values at treshold HR in 5 patients and lies under them in one patient only.

Atrial pacing test performed synchronously with an echocardiographic examination enables: first, to follow-up the changes of left ventricular function after loading, and second, to establish the degree of time correlation between

functional disturbances and ischemic ECG changes.

In concordance with other authors' data (7) we find out two phases in the dynamics of TSLVD and  $V_{ct}$  as well. Initial TSLVD reduction and  $V_{ct}$  increase, respectively, can be explained by an imporoved myocardial contractility. This is confirmed by the initial increase of sAP/TSLVVI ratio as a contractility index established in our study. This index depends to a smaller extent on the changes of opposite and preliminary load of the left ventricle as compared with

V<sub>cf</sub> and TSLVD both.

Supervening TSLVD increase and V<sub>cf</sub> and sAP-TSLVVI ratio decrease, respectively, reflect myocardial contractility diminution as related to beginning ischemia. Similarly to other authors (1, 2, 8, 10, 11), we also establish that in most patients contractility changes precede ischemic ECG ones. It stresses that sAP-TSLVVI ratio and V<sub>cf</sub> both remain over initial values till threshold HR inclusive, although progressively diminishing, in some of the patients while they decrease down to values below the initial ones in other patients. With the latter, it probably refers to certain manifestation of an existing latent contractility deficiency not detectable during patient's examination at rest. The determination of the significance of these two types of dynamics of contractility parameters for the further state of left ventricular function and for patients' prognosis requires additional prospective observations.

An other fact of interest is that one that «breaking» occurs earlier with TSLVD and  $V_{cf}$  than with sAP-TSLVVI ratio. It is probably due to discrete changes of the arterial pressure (systolic pressure increase by 3 per cent). The small number of patients under investigation, however, does not enable us

to draw an explicit conclusion about the additional loading which has induced ischemia having altered contractility and passed into ischemia provoking is-

chemic ECG changes.

We conclude that echocardiographic study of UAP patients during atrial pacing test demonstrates a biphasic dynamics of contractility indices. Contractility suppression precedes the appearance of ischemic ECG changes. In order to speifying the place of the method when determining left ventricular function and prognosis as well as when following-up of the effect of drug or surgical treatment of UAP one requires the investigation of a larger contingent of patients.

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### РАННИЙ ТЕСТ С ПРЕДСЕРДНОЙ СТИМУЛЯЦИЕЙ 'ДЛЯ ОЦЕНКИ' СОКРАТИМОСТИ МИОКАРДА У БОЛЬНЫХ С НЕСТАБИЛЬНОЙ СТЕНОКАРДИЕЙ

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#### РЕЗЮМЕ

Проведена оценка изменений сократимости мнокарда у шести больных с нестабильной стенокардией после их клинического стабилизирования. Посредством эхокардиографии М-типа был установлен телесистолический размер левого желудочка сердца, средняя скорост сокращения циркумферентных волокон (V°) и отношение между систолическим артериальным давлением и телесистолическим объемом левого желудочка. Параметры сократимости показывают двуфазную динамику. После первоначального повышения скорости сокращения циркумферентных волокон и отношения между систолическим артериальным давлением и телесистолическим объемом левого желудочка, респективно понижения телесистолического объема, при дальнейшем увеличении частоты стимуляции Vc и отношения систолическое артериальное давление — телесистолический объем левого желудочка начинают уменьшаться, а телесистолический объем левого желудочка начинают уменьшаться, а телесистолический объем левого желудочка начинают уменьшаться, а телесистолический объем левого желудочка начинает увеличнваться. Нарушения сократимости предшествуют ищемические ЭКГ-изменения (т. н. лороговая частота).