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Analysis of Ventricular Activation in Patients with Chronic Non-Q Wave Myocardial Infarction: Comparison with Left Ventricular Asynergy and Myocardial Perfusion Defects

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Summary

In this report, we dealt with ventricular activation abnormalities in 30 patients with previous non-Q myocardial infarction (MI) by means of the CARDIAG 128.1 device, which enables analysis of ECGs, VCGs and body surface potential maps. The diagnosis was verified by left ventriculography, echocardiography and perfusion scintigraphy. Twenty-nine healthy subjects served as the control group. Morphological findings confirmed the presence of a significant subgroup with serious left ventricular asynergy. Seven electrocardiological variables, which significantly differed from control values, disclosed that non-Q MI is responsible for localized activation time prolongation, and that inferoposterior scars tend to delay the entire activation of ventricles, and to cause disturbances of the terminal depolarization phase together with a decrease in voltage production during QRS. Lesions of the anterior wall and the apicomesial part of the inferoposterior wall affect the distribution of the Q wave more often than the posterior basal ones. The probability of such abnormalities increases with the degree of asynergy. Some VCG criteria increase the sensitivity of electrocardiological analysis. These parameters will be used for evaluating the diagnostic value of electrocardiological analysis in the chronic non-Q MI. Non-Q myocardial infarctions represent a heterogeneous group of infarctions from both electrophysiological and morphological aspects.

Key words

Non-Q myocardial infarction – Ventricular activation – Body surface potential mapping – Vectorcardiography

Introduction

Many publications have already dealt with the problems of non-Q myocardial infarction (MI). Nowadays, there is enough available evidence that there is no direct relation between the presence of diagnostic Q wave and transmurality of the lesion (Durrer *et al.* 1964, Savage *et al.* 1977, Phibbs 1984, Matsushita *et al.* 1990, Montague *et al.* 1986, Cagáň *et al.* 1987). It is obvious from the morphological point of view that the non-Q MIs are a heterogeneous group which includes cases with a normal function of the left ventricle (LV), cases of wall motion abnormalities in a single segment and also cases of multiple segment wall motion abnormalities with a greatly diminished global ejection fractions. In some reports each of these subgroups represented approximately one third of the cases (Montague *et al.* 1986).

From the electrocardiological point of view, the non-Q MIs can be divided into a subgroup with "missed" Q where the pathologic Q wave is outside the range of the standard lead system, or only the Q wave distribution is impaired, which is not discernible by the 12-lead ECG, and into a subgroup of "true" non-Q MIs, where these abnormalities do not exist. This knowledge, however, could be attained by introducing mapping techniques (body surface potential maps, BSPM). Hitherto, papers have described these data in the acute MI (Montague *et al.* 1986), or in old MIs, but only in certain localizations using a certain parameter (Bertoni *et al.* 1985, Hirai *et al.* 1984, Wen *et al.* 1990, Yabe *et al.* 1988). Nor have we any data about localized activation delay known in Q-MI cases (Ikeda *et al.* 1985).

New non-invasive diagnostic approaches in the recognition of non-Q MIs would also be desirable for clinical practice. One of the reasons is that many patients with atypical chest pain have a history of hospitalization for non-Q MI, without information about the kinetics of cardiospecific enzymes in the acute phase, and their diagnosis was based upon findings of transient T wave changes or changes not specified in time. In this situation, it would be desirable to recognize possible chronic changes of ventricular activation as a consequence of non-Q IM, especially in cases with no left ventricle wall motion abnormalities recognizable noninvasively.

We consider that electrocardiological examination supplemented with analysis of Q wave distribution and of other variables of the depolarization is a powerful clinical tool.

Methods

Subjects

We examined 30 patients with coronary artery disease (CAD, 29 men, one woman, 36-63 years old, average 53.8 years) and with a myocardial infarction history from 2 months to 9 years and who had a normal or non-specific pattern on a 12-lead ECG at the time of examination (no diagnostic Q equal to or more than 0.04 s, or more than 1/3 R, no typical intraventricular conduction defect with QRS equal to or more than 0.11 s). After consulting an independent cardiologist we eliminated cases with distinctly poor R wave progression in leads V1 – V3. The diagnosis of non-Q MI was based upon the history of hospitalization for a coronary lesion and upon the ventriculographic or echocardiographic finding of a single site, resting LV asynergy, or an irreversible perfusion defect, detected by myocardial perfusion scintigraphy at the time of our electrocardiological examination. The data about serum cardiospecific enzyme levels in the acute phase were not available in all individuals. The group did not include cases with another confirmed cardiological disorders. The control group consisted of 29 clinically healthy subjects (25 men, 18-62 years old, average age 34.8 years) with no history of any cardiovascular disease, with normal blood pressure and with a normal 12-lead ECG.

Electrocardiological examination

Input data were obtained by the CARDIAG 128.1 device, manufactured by ZPA Čakovice, which enables simultaneous registration of ECGs from 80 recording points on the chest surface by an unipolar technique with the Wilson central terminal as the reference, registration of signals from Frank's orthogonal network and registration of the standard 12-lead ECG (Stojan 1991). We carried out the examination in sitting patients, in the mid-tidal expiration, sometimes during cyclic tidal volume respiration (Sutherland *et al.* 1983). The device consists of input amplifiers, A-D converters, multiplexers and security circuits and is connected by a special card to a personal computer compatible for IBM-AT. The input data are thus transformed to ECG, VCG and maps of electrical activity, both isopotential and with the possibility of data compression as isointegral, isochrone and isopotential peak maps (Wen *et al.* 1990). The diagram of the time course of potential maxima and minima (Filipová and Cagáň 1986) can also be evaluated. The onsets and offsets of the depolarization complex were determined automatically from the spatial magnitude curve on the VCG with the possibility of an correction. For the analysis we used a single beat, software-filtered, without signal averaging. We were particularly interested in the QRS complex and by using a limited set of parameters suitable for the final statement of sensitivity, specificity and positive predictive value for the diagnosis of non-Q MI, which is one of the possible approaches for evaluating the data obtained (Taccardi 1991).

Left ventriculography (LVG) and 2D-echocardiography

All the non-Q MI patients underwent cardiac catheterization with selective coronarography and routinely evaluated LVG in the right anterior oblique (RAO) projection. LV asynergy was classified visually, in some cases the systolic and diastolic endocardial tracings were delineated to distinguish between hypokinesia, akinesia and dyskinesia. The localization of lesions was reduced to the involvement of the anterior and inferoposterior walls of the left ventricle (Toyama *et al.* 1985). 2D-echocardiography in four standard projections (PSLAX, PSSAX, AP4C, AP2C) was thought to be equal to LVG in cases, where it disclosed asynergy and LVG did not. When both methods displayed a pathological finding, we took into account the more serious one.

Myocardial perfusion scintigraphy.

Those subjects, who had normal left ventricular function, underwent perfusion scintigraphy ^{99m}Tc-SestaMIBI (methoxyisobutylisonitril, Cardiolite) by a method distinguishing reversible and irreversible perfusion defects to recognize the scar. The scan itself concerned a left anterior oblique (LAO) projection of 40 degrees, left lateral and anterior projections by a LFOV gamma camera, and an evaluation of patterns using a microcomputer PDP 11/34. Because of a better choice of projections in scintigraphy and 2D-echocardiography, we added patients with a combinations of lateral or septal and posterior involvement into the group of inferoposterior and any combination with the anterior wall into the group of anterior infarctions.

Statistical analysis

We evaluated qualitative parameters by the percentage appearance in a particular group. Quantitative parameters were compared by the unpaired t-test, with a minimal level of probability of 95 % for significant differences. Maps were quantified as peak-to-peak magnitude, and quantitative evaluation of the activation time was provided only in standard chest leadpoints. Isochrone maps of activation time (ICHVAT) were evaluated visually (Ikeda *et al.* 1985). For the detection of Q wave distribution disturbances we used the departure technique (Bertoni *et al.* 1985, Ikeda *et al.* 1990, Matsushita *et al.* 1990, Wen *et al.* 1990). At each leadpoint we calculated the departure index (DI) determined by the equation DI = QI−QI/SD (Ikeda *et al.* 1990), where QI was an average value of the integral of the Q wave in a given leadpoint, QI was the measured value of this integral and S.D. was the standard deviation in this leadpoint. For each individual such departure index map was calculated, which was represented by a single value ΣDI, as an integral of the number of leadpoints, where DI < −2. Distribution of the Q wave (ΣDI < −20) was considered to be pathological. Fig. 1 shows the mean-group map of integral distribution of the Q wave in the control group as well as an example of the ECG and Q wave isointegral, departure and departure index map in a healthy subject independent of the control group.

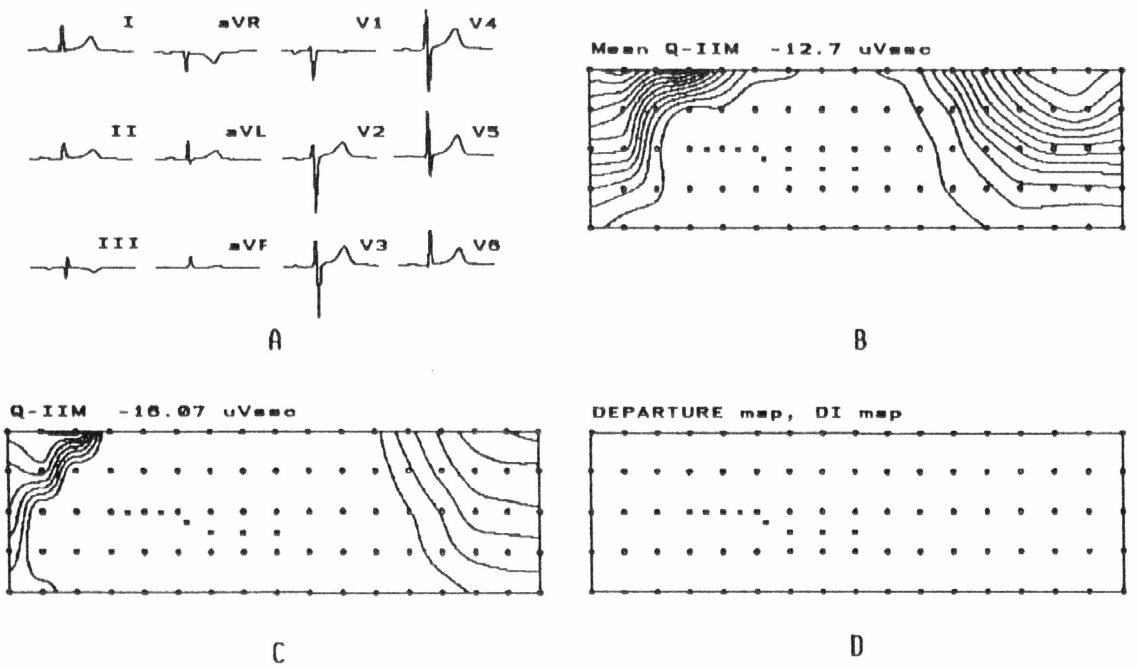

Fig. 1

Normal findings in a subject independent of the control group A – 12-lead ECG, B – mean group isointegral Q-wave distribution map, C – isointegral Q-wave distribution map of the same subject, D – departure map and departure index map is empty because of no significant disturbances in Q wave distribution. Each left half of the map represents the anterior chest whereas the right half represents the back. The left and right edges are the right mid-axillar line. First row is at the level of fossa jugularis and the distance between the rows is about two intercostal spaces.

Results

Morphological and scintigraphic patterns

Out of a total of 30 lesions, 15 were localized anteriorly and 15 inferoposteriorly. Six of them predominantly concerned the basal part and nine rather the apicomesial part of the inferoposterior wall (i.e. inferior wall). The asynergic sites and perfusion defects were not strictly located at the commonly used segments of the left ventricle. In six cases the function of LV was normal without any asynergy, 15 lesions appeared to be hypokinesia, six lesions were classified as akinesia and three as dyskinesia.

Table 1
Review of the parameters with statistically significant differences compared to the control group.

Parameter Unit	Non-Q MI	Anterior	Infero- posterior	Controls	p=
T	12.2±7.3		13.2±7.4	7.0±5.5	0.010
ms		11.2±7.3			0.050
VAT2		32.3±6.0		29.1±4.3	NS
ms	33.2±5.9		34.1±5.8		0.010
VAT3			38.1±5.3	34.8±5.3	NS
ms	38.8±6.3	39.5±7.3			0.050
DIIM	70.1±29.8	78.2±25.6		81.4±28.0	NS
μVs			61.9±32.2		0.030
QRS		102.0±9.2			NS
			105.9±9.5	98.3±9.5	0.017
ms	103.9±9.4				0.027
VEL	206.0±86.6	232.0±85.4		245.9±88.3	NS
μV/s			179.6±82.3		0.021
ΣDI	126.9±238.9	- -	- -	9.9±11.0	0.006
ΣDI %	67 -	73 -	60 -	17 -	(0.05)
STRR %	17 -	- -	- -	0 -	(0.05)

Data are means ±S.D. See text of the Results for abbreviations.

Analysis of ventricular activation

Activation of the ventricles was originally represented by 25 parameters, out of which only seven significantly differed from the control group values. Non-significant differences concerned the occurrence of notches on the depolarization part of the time course of potential maxima, their time relations (i.e. their synchronous appearance in the positive and negative branches of the diagram), peak values on the same diagram, notches on the spatial amplitude curve of VCG, the angle of maximal vector of QRS, and the occurrence of non-characteristic changes of the QRS loop in the transverse plane of the VCG, etc.

The values of the above mentioned seven parameters are given in Table 1.

1. Time shift T (ms) between peak values of the positive and negative branches on the diagram of the time course of the potential maxima and minima with higher values in inferoposterior lesions.

2. Prolonged ventricular activation time VAT (ms) at leads V2 (VAT2) in inferoposterior lesions and V3 (VAT3) in anterior lesions, measured from the onset of the QRS on the spatial amplitude curve of VCG to the peak of the R wave in these leads. Fig. 2 shows diagrammatically the values of VAT in all standard chest leads.

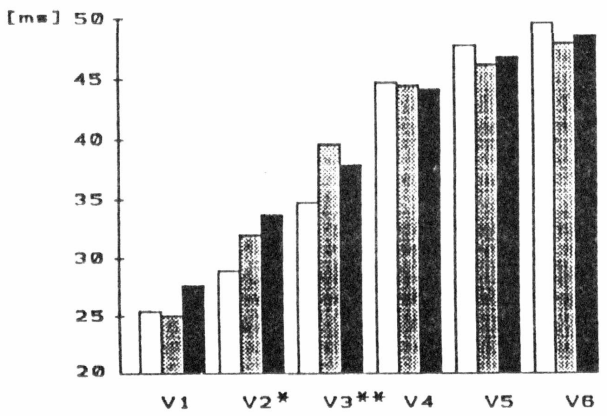

Fig. 2
Activation time in standard chest leads found in control group (empty columns) and in patients with anterior (dashed columns) or inferoposterior (black columns) non-Q myocardial infarction. * – $p < 0.01$, ** – $p < 0.05$

3. A lower peak-to-peak magnitude on the map of depolarization isointegrals DIIM (μVs) in inferoposterior wall lesions.

4. Prolonged interval QRS (ms) again more in inferoposterior lesions.

5. Decreased spatial velocity VEL (μV/s) of the instantaneous QRS vector endpoint in two thirds of QRS duration again in inferoposterior lesions.

6. ΣDI as a function of the Q wave disturbances distribution was significantly higher in absolute values in the studied group than in the control group. The result is, however, is not significant because of the great scatter of values, which is caused by the threshold properties of this variable. It is therefore more advantageous to evaluate its occurrence in percentage of the cases, where the threshold value −20 was reached. This was discovered in 20 out of 30 (67 %) patients of the group studied but only in 5 out of 29 (17 %) controls. Fig. 3 shows the percentage occurrence of Q wave distribution changes dependent on the increasing number of light asynergies and cases with no asynergy. In the subgroup of inferoposterior infarctions only 2 of 6 (33.3 %) patients with affected basal part attained threshold values ΣDI = −20, while in the subgroup of inferior (apicomesial) lesions 7 out of 9 (78 %) fulfilled this criterion.

7. Starr's vectorcardiographic criterion (STRR) for inferior MI (Starr *et al.* 1974) was fulfilled in 5 out of 30 (17 %) patients and none of the controls attained this criterion. However, only one individual had a clear involvement of the inferior wall, but in four cases the lesion of the apex was present.

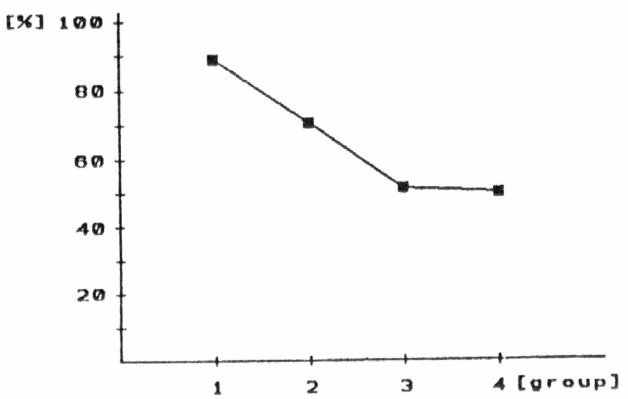

Fig. 3
The dependence of the occurrence of Q wave distribution disturbances on the degree of asynergy. Group 1 – dyskinesis + akinesis, 8 out of 9 cases (89 %); group 2 – dyskinesis + akinesis + hypokinesis, 17 out of 24 (71 %); group 3 – hypokinesis + no asynergy, 11 out of 21 (52 %); group 4 – no asynergy, 3 out of 6 (50 %).

Discussion

As far as left ventricle wall motion abnormalities are concerned, the results confirmed the heterogeneity of the studied group, with a significant appearance of serious types of asynergy. In this paper, however, the extent of LV functional disturbances was defined only by the degree of asynergy, not by the number of involved LV segments and by the ejection fraction. Contradictory results have been reported concerning significant differences between Q and non-Q myocardial infarction. Both the global function of LV and the average level of cardiospecific enzymes in the acute phase were affected as well as the extent of the perfusion defect on myocardial scintigraphy (Wahl *et al.* 1985). The selection algorithm in the above study made it possible to exclude patients with unfavorable haemodynamic parameters. On the other hand, it included cases where the diagnosis was based upon the history of chest pain in combination with electrocardiographic findings without changes in serum levels of cardiospecific enzymes, where the presence of infarction was questionable.

It was also confirmed that the ventricular activation in the non-Q MI was generally impaired. We have observed two types of abnormalities. Firstly, the changes, which are generally considered as the expression of the loss of electrically active tissue elements, i.e. Q wave distribution disturbances (analogous to the diagnostic Q wave in Q myocardial infarction) and the relative reduction of the potential values, namely the peak-to-peak amplitude on the depolarization isointegral map DIIM in the inferoposterior subgroup. None of these variables themselves indicated the transmurality of lesions, because significant differences against control values were found either in light (hypokinesis) or in serious (akinesis, dyskinesis) LV wall motion abnormalities (see Figs 4 and 5). Neither did dyskinesis have to indicate a transmural lesion (Cagáň *et al.* 1987, Ideker *et al.* 1978). Today we know that a pathological Q wave in a standard 12-lead ECG is a sign of subendocardial involvement (Ishikawa *et al.* 1980) and a quite reliable indicator of asynergy itself, irrespective of its extent (Bodenheimer *et al.* 1975). At present, the diagnostic value of ectopic Q wave has been confirmed by mapping techniques in cases of anterior non-Q MIs and it is again known that it reveals asynergy (Yabe *et al.* 1988, Hirai *et al.* 1984). The quantification of Q wave distribution disturbances by departure techniques has turned out to be important, because we often cannot find either qualitative differences in isoline shapes on isointegral maps of Q waves, or quantitative differences between peak values on these maps in patients and control individuals (compare Figs 1 and 4, QIIM).

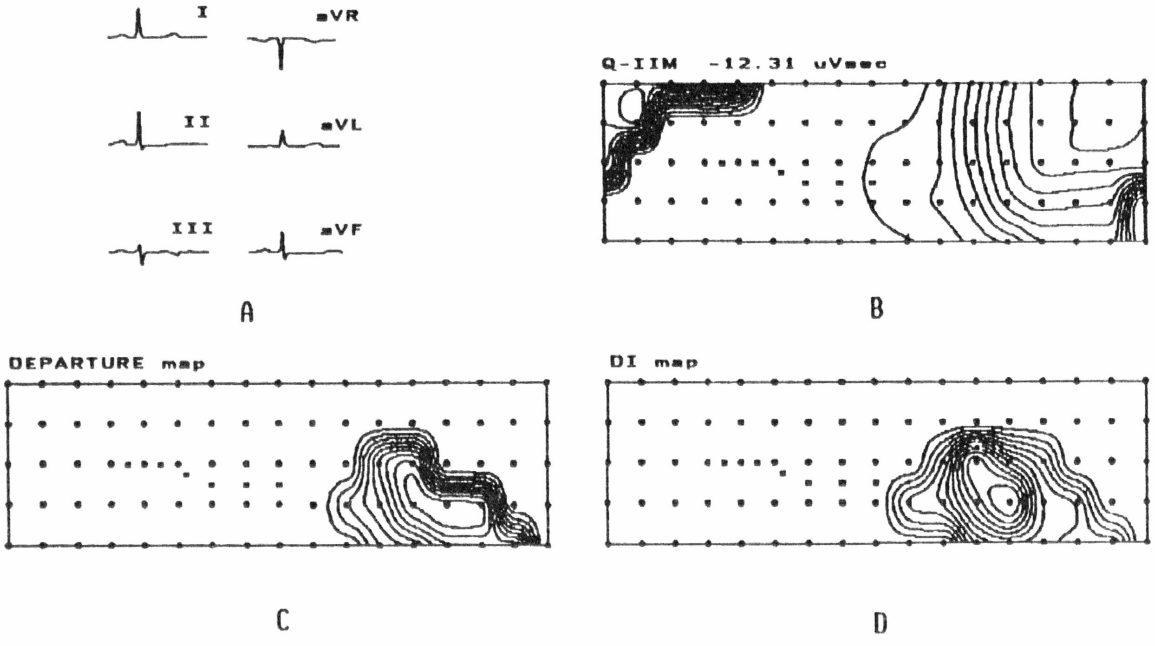

Fig. 4
Records in a subject with akinesis of the inferior wall (apecomesial part). A – ECG (limb leads) without diagnostic Q wave; B – isointegral Q wave map is similar to the normal finding (see Fig. 1) both in the peak value and in the shapes of isolines; C, D – departure map and departure index map with the area of significant values on the lower back ($\Sigma DI = -461$ on the departure index map).

We consider the difference in appearance of the Q wave distribution abnormalities between the subgroups of inferior (apicomesial) and posterior (basal) infarction to be a consequence of the natural way of ventricular activation (Durrer *et al.* 1970), where the basal parts of the posterior wall are activated relatively later (> 20 ms) in the endocardial layer, during the genesis of R wave. For this reason, we are more likely to observe a decrease of positivity production (diminution of DIIM parameter) than Q wave distribution abnormalities.

Prolonged VAT in V2, V3 and a significantly longer QRS interval in inferoposterior lesions together with a slower spatial velocity of the QRS instantaneous vector endpoint represented the second type of activation abnormalities. In the case of leadpoint V3, it is most likely a correlate of a former by-operation with epicardial activation delay in patients with coronary artery disease and the regional wall motion abnormalities (Wiener *et al.* 1982). We observed a markedly prolonged VAT in V3 above the site of anterior hypokinesis and, in accordance with the above mentioned report, it was not the place with the longest VAT on the chest surface (see Fig. 5, the profile). It is not quite clear, to what extent the further myocardial ischaemia in adjacent tissue influences the local activation delay, because we did not observe it in the repolarization phase and could not prove it by any other examination. From the electrophysiological point of view, the genesis of VAT delay in V2 leadpoint will probably differ from that in V3, because statistically the delay was influenced more often by inferoposterior lesions. We suppose therefore that it is rather an expression of vector decompensation for the benefit of the anterior wall in inferoposterior infarctions, than the activation delay in the above mentioned anterior wall involvement. In case that the Q wave distribution abnormalities and the VAT prolongation occur at the same time, and are mutually spatially connected, we can consider the real electrical expression of nontransmural lesions on the basis of computer simulation studies (Ishikawa *et al.* 1980). This information is not provided by the conventional ECG (see Fig. 5).

Prolongation of the QRS interval together with the slow-down of vector end-point spatial velocity in two thirds of the QR duration in inferoposterior myocardial infarctions is certainly a specific demonstration of peripheral intraventricular activation blockade in the posterior wall. This was formerly described in inferior or posterior Q myocardial infarctions as changes in aVF and V1 and was even connected with deteriorating functional parameters of LV (Babbitt *et al.* 1991).

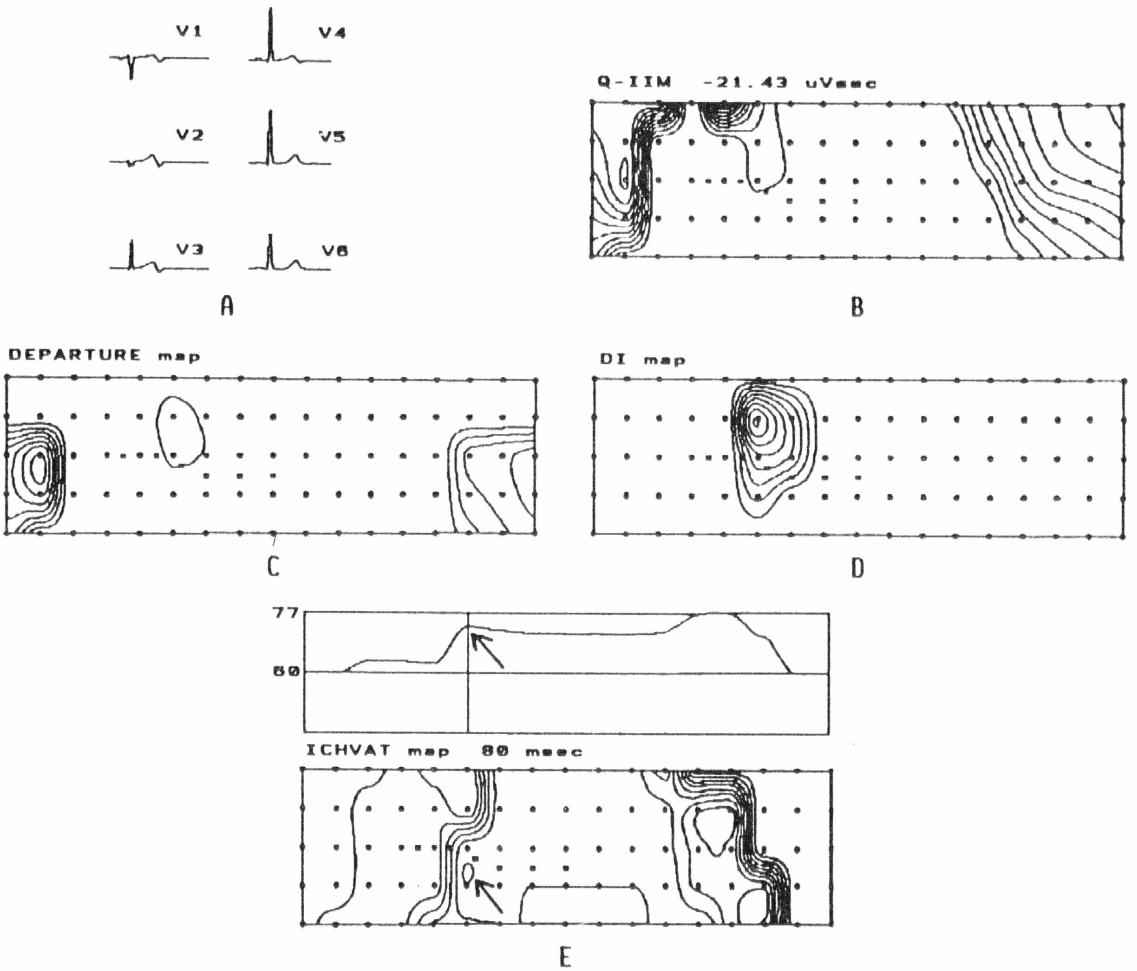

Fig. 5
Records in a subject with anterior wall hypokinesis. A – ECG (chest leads) with non-specific poor R wave progression in V1, V2, (no diagnostic Q or QS pattern is present); B – Q wave isointegral map with outlined area of mild ectopic Q wave neighbouring on V2, V3; C – departure map with markedly more different values in the area of natural occurrence of Q wave than in the area of the ectopic Q; D – departure index map transformed previous pattern with respect S.D. value to point out the ectopic Q; E – isochrone map of VAT shows located delay of activation (arrows) as an evidence for the nontransmurality of the lesion (or as evidence of further ischaemia?). The upper half of this sub-image represents the profile of values approximately in the 4th row.

We have observed a time shift between peak values of the positive and negative branches on the diagram of the time course of potential maxima and minima (concerning depolarization), which was significantly prolongated, more in inferoposterior lesions, and which has not been described in non-Q MIs.

From the VCG parameters we verified the higher sensitivity of Starr's criterion for inferior MI in comparison with standard ECG, which was previously proved (Edenbrandt *et al.* 1990) without influencing the specificity. In our study, however, apical asynergy was rather indicated.

Conclusions and clinical impact

The present data indicate that it is possible to detect localized activation sequence disturbances (T, VAT) in non-Q MIs, similarly as in Q MIs. Inferoposterior lesions tend to prolong the entire activation time (QRS) more, and to cause terminal depolarization phase abnormalities, again similarly as in Q MI, together with a tendency to decrease the voltage production during QRS. Anterior and inferior wall lesions near the apex more often give rise to Q wave distribution disturbances or directly to an ectopic Q wave (so-called "missed Q"). The probability of such abnormalities increases with the degree of asynergy, as

was similarly observed in the acute non-Q MI. Some VCG criteria also increase the sensitivity of electrocardiological examinations, compared with the conventional ECG. Non-Q MIs are a heterogeneous group both from electrophysiological and morphological points of view. The immediate clinical impact is, that we are also able to discover localized ventricular activation abnormalities in cases of non-Q MIs, where the morphological finding is normal, and when it only remains to provide the scintigraphic examination with all its disadvantages.

Study limitations

The limited number of individuals in the studied group makes it impossible to observe the behaviour of each parameter in the subgroups with different localizations of the lesions and implies a division into two basic groups which cannot take into consideration e.g. lateral or septal wall involvement. The routine left ventriculography in the right anterior oblique projection is restricted by the same limitation. The small number of samples does not enable state the diagnostic value of parameters, but it can be used as a training set for further statistical setting of this value. We have only chosen those parameters, where such a statement will be meaningful.

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