

CLINICAL STUDY

The effect of myocardial revascularization on malignant ventricular arrhythmias in coronary artery disease

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Abstract: *Background:* Malignant ventricular arrhythmia in coronary artery disease (CAD) is a severe life-threatening disease and a risk factor for sudden cardiac death. Myocardial revascularization influences the arrhythmogenic substrate of the malignant ventricular arrhythmia in the secondary prevention of sudden cardiac death. Its effectivity remains controversial.

Objectives: The aim of this study is to assess the inducibility of sustained ventricular tachycardia (VT) or ventricular fibrillation (VF) in patients after myocardial revascularization and to compare the effectivity of complete and incomplete revascularization.

Patients: Fifty patients with documented sustained VT or VF and CAD were examined in our department.

Results: Conservatively treated patients were significantly older than revascularized patients (68±8 versus 62±9 years, $p<0.05$). We registered a trend towards a lower inducibility of malignant ventricular arrhythmias in the revascularized group and completely revascularized subgroup, but without statistical significance. Incompletely revascularized patients comprised only of men (100 % versus 66.6 %, $p<0.05$). Fewer ICDs were implanted in the completely revascularized group (55.6 % versus 92.3 %, $p<0.05$).

Conclusion: Myocardial revascularization has little effect on the inducibility of malignant ventricular arrhythmias after myocardial revascularization. Complete revascularization significantly decreases the need of ICD implantation when compared to incomplete one (Tab. 3, Fig. 4, Ref. 24). Full Text (Free, PDF) www.bmj.sk.

Key words: malignant ventricular arrhythmias, coronary artery disease, myocardial revascularization.

Malignant ventricular tachyarrhythmias are considered to be severe life-threatening diseases that influence total cardiovascular morbidity and mortality to a great extent. The most frequent arrhythmogenic substrate affecting their initiation is coronary artery disease (CAD) (1). In connection with the ageing of the population in industrially developed countries and the “epidemic” of heart failure, the number of patients endangered by these arrhythmias is increasing. The rapid growth of revascularization therapy in recent times as well as the improvement of infarct-related coronary artery patency by fibrinolytics or percutaneous coronary intervention has modified the arrhythmogenic substrate, and as a result, the occurrence of malignant ventricular arrhythmias and subsequently the prognosis of the patients (2, 3).

Ventricular arrhythmias are the most frequent cause of sudden cardiac death (SCD). Its yearly incidence in the adult population in industrially developed countries is given in a range from 0.36 up to 1.28 per 1000 inhabitants (4). SCD usually arises in the background of coronary atherosclerosis. Approximately one half of all CAD deaths is sudden and unexpected, occurring just

one hour after the change in the health status. The risk factors of SCD are predominantly the same as the risk factors of coronary atherosclerosis, and so a decrease in CAD prevalence should result in decreased incidence of the SCD (1).

Surgical or interventional revascularization of the myocardium, where coronary flow can be improved and ischaemia limited, has a favourable antiarrhythmic effect (5, 6). This is why it is necessary to assess the presence of CAD and the possibility of revascularization in patients with ventricular arrhythmias. Coronary revascularization is an effective antiischaemic therapy that improves long-term patient survival and decreases the incidence of SCD as well (7, 8). Some authors point out that revascularization is inadequately effective in the prevention of the recurrence of malignant ventricular tachyarrhythmias, especially in the chronic phase of myocardial infarction (MI) (9, 10). Myocardial revascularization probably does not prevent SCD in patients with severely decreased left ventricular function, even though the original arrhythmia was due to transient ischaemia (11). Therefore, it is necessary to assess the need for implantable cardioverter-defibrillator (ICD) therapy in each patient after MI, despite successful revascularization (12).

The aim of this study was to assess the inducibility of sustained ventricular tachycardia or ventricular fibrillation in patients after myocardial revascularization and subsequently to compare complete and incomplete revascularization in the light of the inducibility of these ventricular dysrhythmias.

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Methods

Patients and procedure

In the years 2002 to 2006, we examined 50 adult patients (42 men and 8 women) who had documented sustained ventricular tachycardia (VT) or ventricular fibrillation (VF) and CAD. Mean age was 64 ± 9.5 years (min 39, max 82 years). Each patient gave written informed consent for each invasive procedure. All patients underwent coronary angiography. Patients with normal findings in their coronary arteries were not included in this analysis. Based on a coronarangiogram, revascularization of the myocardium (coronary artery bypass grafting (CABG) or percutaneous coronary intervention (PCI)) was recommended, or not.

We considered the myocardial revascularization to be complete when no significant stenosis (more than 70 %) of any coronary artery was present after the procedure (CABG or PCI). Other cases of revascularization were considered to be incomplete. Stenosis of the left main coronary artery was assessed as significant when this segment had narrowed by 50 % or more.

All patients, after myocardial revascularization and amiodarone loading (in total dosage of 10 g) in combination with a betablocker (unless these were contraindicated), underwent an

electrophysiologic study where the inducibility of ventricular arrhythmias was studied. With patients under local anaesthesia, through the right femoral vein we introduced three multipolar 6F catheters under fluoroscopic control: into high right atrium, His bundle, the right ventricular apex and subsequently the right ventricular outflow tract. We used the external programmable UHS Biotronik stimulator for ventricular stimulation. Superficial and intracardiac electrograms were recorded on a Prucka CardioLab 4.1 and 6.0. In the protocol used we performed programmed ventricular stimulation (PVS) via a series of 8 stimuli at cycle lengths of 400 and 600 eventually 500 ms, followed by one to three or four extrastimuli from two places in the right ventricle – the apex and the outflow tract, with isoprenalin if necessary. The stimulation was performed at twice the stimulation threshold and impulse width of 0.5 ms. The study was evaluated as positive when sustained VT lasting 30 s or more was reproducibly induced, or when, due to haemodynamic instability, a premature termination of the tachycardia by overdrive stimulation or electric cardioversion was necessary (Figs 1 and 2). The result was also assessed as positive in the case of ventricular fibrillation.

We collected retrospectively baseline clinical characteristics, including age, gender, personal history of previous myocardial



Fig. 1. Induction of sustained monomorphic ventricular tachycardia from the apex of the right ventricle during programmed ventricular stimulation. From top to bottom are surface ECG leads I, aVF, V1 and V6, intracardiac electrograms from high right atrium, His bundle and right ventricular apex. After a series of 8 stimuli from the right ventricular apex with a constant coupling interval S1-S1 3 extrastimuli S2, S3, S4 follow. S4 induces ventricular tachycardia with AV dissociation. VT – ventricular tachycardia, HRA d, p – high right atrium, distal and proximal part of catheter, HIS d, m – potential of distal and medium part of His bundle, RVA d, p – right ventricular apex, distal and proximal part of catheter.

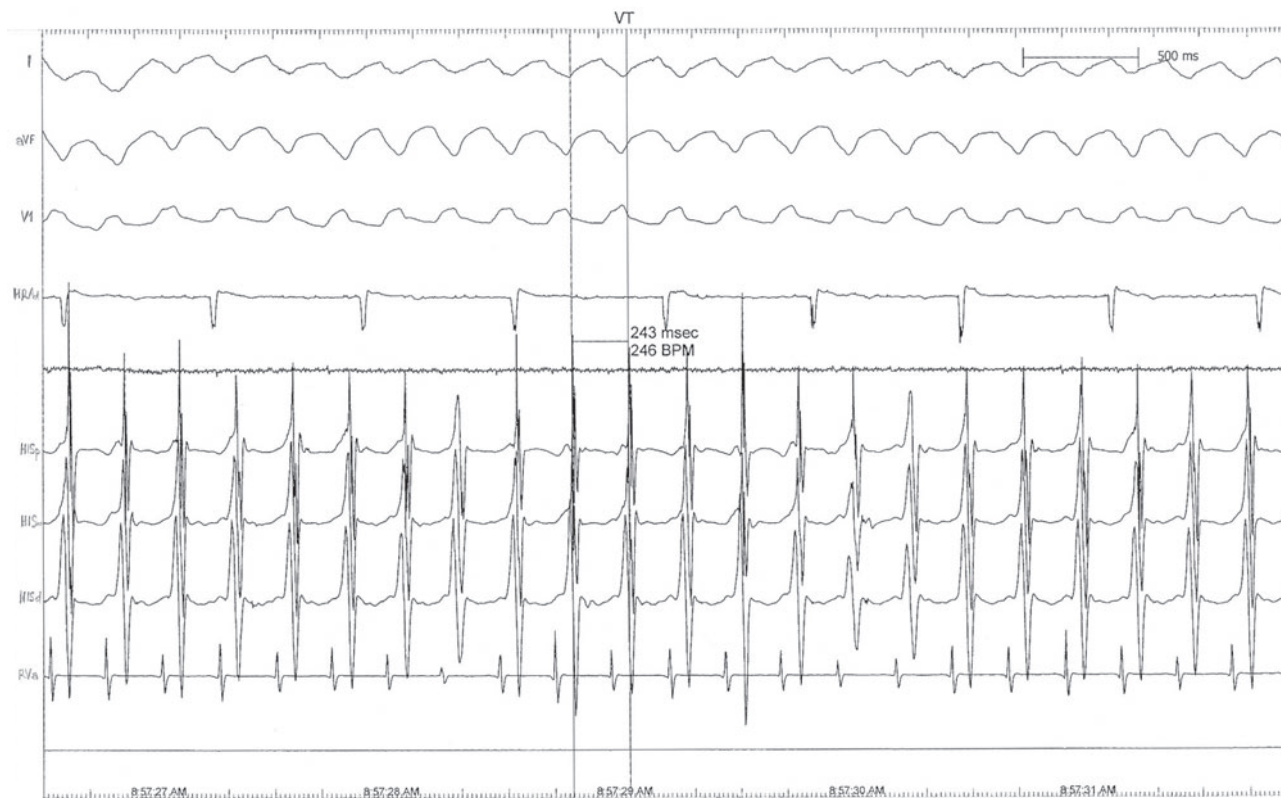


Fig. 2. Sustained monomorphic ventricular tachycardia with cycle length of 243ms induced during programmed ventricular stimulation in a patient after myocardial revascularization. VT – ventricular tachycardia, HRAd – high right atrium, distal part of catheter, HIS p, m, d – potential of proximal, medium and distal part of His bundle, RVA – right ventricular apex.

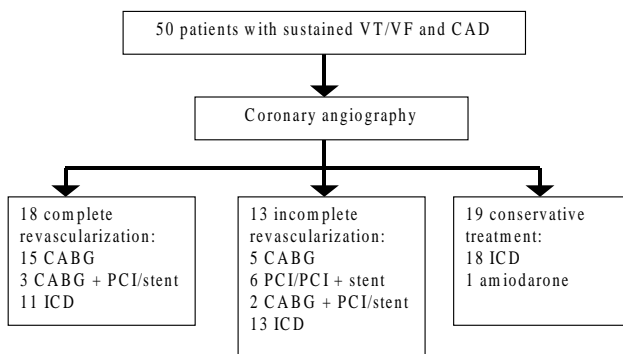


Fig. 3. The type of revascularization in the studied population. CABG – coronary artery bypass grafting, PCI – percutaneous coronary intervention, ICD – implantable cardioverter – defibrillator, VT/VF – ventricular tachycardia/ventricular fibrillation, CAD – coronary artery disease.

infarction, arterial hypertension, diabetes mellitus, dyslipidaemia, drug therapy, the type of revascularization performed, if any (coronary artery bypass grafting (CABG) or percutaneous coronary intervention (PCI)), and the inducibility of malignant ventricular arrhythmias during programmed ventricular stimulation after revascularization. We followed two basic echocardiographic

indicators: global ejection fraction (EF) in percentage and presence of the left ventricular aneurysm as morphologic substrate for initiation of the malignant ventricular arrhythmias.

Statistical analysis

Categorical variables are expressed as numbers or percentages, continuous variables as means and standard deviations. First, we tested the normality distribution using Kolmogorov-Smirnov and Shapiro-Wilks tests. In categorical variables, a chi-square test or a Fisher’s exact test were used for statistical comparisons, as appropriate. The differences between the type of treatment and incomplete/complete revascularization in continuous variables were assessed by a Student’s t-test or a Mann-Whitney U test, as appropriate. A p value $p < 0.05$ was considered to be statistically significant. For statistical analysis we used the Statistical Package for the Social Sciences for Windows version 14.0.

Results

According to their coronary angiogram, 31 patients were indicated for myocardial revascularization, while 19 were left to undergo conservative therapy due to inappropriate anatomical findings or refusal of the patient to give an informed consent for

Tab. 1. Baseline characteristics of patients left with conservative treatment and revascularized patients.

	Conservative treatment (n=19)	Revascularized patients (n=31)	p
Age (years)	68±8	62±9	0.025
Gender (women/men)	2/17	6/25	ns
Previous MI	16 (84.2%)	26 (83.9%)	ns
Arterial hypertension	15 (78.9%)	23 (74.2%)	ns
Diabetes mellitus	7 (36.8%)	8 (25.8%)	ns
Hyperlipoproteinaemia	11 (57.9%)	18 (58.1%)	ns
Ejection fraction (%)	35±9	35±7	ns
Aneurysm of the left ventricle	8 (42.1%)	7 (23.3%)	ns
1 vessel disease	4 (21.1%)	2 (6.5%)	ns
2 vessel disease	5 (26.3%)	10 (32.3%)	ns
3 vessel disease	10 (52.6%)	19 (61.3%)	ns

Abbreviations: p – p value, ns – insignificant, MI – myocardial infarction

Tab. 2. Comparison of drug therapy used in the conservatively treated group and revascularized patients.

	Conservative treatment (n=19)	Revascularized patients (n=31)	p
Amiodarone	19 (100%)	27(90%)	ns
Betablocker	18 (94.7%)	28 (90.3%)	ns
ACE inhibitor	13 (68.4%)	26 (83.9%)	ns
AT1 receptor blocker	4 (21.1%)	1(3.2%)	ns
Acetylosalicylic acid	15 (78.9%)	20(64.5%)	ns
Warfarine	1 (5.3%)	6 (19.4%)	ns
Spirolactone	6 (31.6%)	11 (35.5%)	ns
Statin1	1 (57.9%)	15(48.4%)	ns

Abbreviations: p – p value, ns – insignificant, ACE – angiotensin converting enzyme, AT1 – angiotensin I

the operation. Out of these 19 patients 18 were indicated for ICD implantation, and in one patient amiodarone therapy was found to be effective without inducible VT or VF during a follow-up electrophysiology study.

Eighteen of the revascularised patients underwent complete revascularization and 11 incomplete revascularization. Complete revascularization was mainly performed in CABG – 15 patients, or in a combined procedure consisting of CABG followed by PCI or PCI with stent implantation in three patients. In patients undergoing incomplete revascularization, the method of revascularization was as follows: twice CABG with PCI, 6 times PCI or PCI with stent implantation and 5 times CABG (Fig. 3).

Patients were categorized into two groups depending on the type of treatment – patients left on conservative treatment and revascularized patients. The baseline characteristics and comparison of these two groups of patients are given in Table 1. Patients left on conservative treatment were significantly older than the revascularized patients (68±8 versus 62±9 years, $p<0.05$). Patients did not differ significantly in gender, personal history of previous MI, arterial hypertension, diabetes mellitus and dyslipoproteinaemia.

Tab. 3. Baseline characteristics of patients after complete and incomplete myocardial revascularization and comparison of used drug therapy.

	Complete revascularization (n=18)	Incomplete revascularization (n=13)	p
Age (years)	62±10	62±10	ns
Gender (women/men)	6/12	0/13	0.020
Previous MI	16 (88.9%)	10 (76.9%)	ns
Arterial hypertension	14 (77.8%)	9 (69.2%)	ns
Diabetes mellitus	7 (38.9%)	1 (7.7%)	ns
Hyperlipoproteinaemia	12 (66.7%)	6 (46.2%)	ns
Ejection fraction (%)	36±8	35±9	ns
Aneurysm of the left ventricle	5 (27.8%)	2 (16.7%)	ns
1 vessel disease	1 (5.6%)	1 (7.7%)	ns
2 vessel disease	6 (33.3%)	4 (30.8%)	ns
3 vessel disease	11 (61.1%)	8 (61.5%)	ns
Amiodarone	14 (82.4%)	13 (100%)	ns
Betablocker	16 (88.9%)	12 (92.3%)	ns
ACE inhibitor	17 (94.4%)	9 (69.2%)	ns
AT1 receptor blocker	0	1 (7.7%)	ns
Acetylosalicylic acid	11 (61.1%)	9 (69.2%)	ns
Warfarine	5 (27.8%)	1 (7.7%)	ns
Spirolactone	6 (33.3%)	5 (38.5%)	ns
Statin	9 (50%)	6 (46.2%)	ns

Abbreviations: p – p value, ns – insignificant, MI – myocardial infarction, ACE – angiotensin converting enzyme, AT1 – angiotensin I

We expressed the severity of coronary artery impairment as one-, two- or three-vessel diseases. There was no difference in occurrence of these three between both groups (Tab. 1). When comparing EF and the presence of left ventricular aneurysm we found no statistically significant differences between the two groups of patients. The average EF was low in both groups (35 %).

Results of the comparison of the drug therapy used are included in Table 2. This analysis showed no statistically significant differences in the administration of such groups of drugs that have possible effect on life-threatening arrhythmias (amiodarone, betablockers, ACE inhibitors, AT1 receptor blockers, aspirin, warfarine, spironolactone, statins).

For the inducibility of malignant ventricular arrhythmias during the control electrophysiologic study after revascularization, the p value showed no statistical significance when compared to nonrevascularized patients, even though we did register a trend towards lower inducibility of malignant ventricular arrhythmias in the revascularized group of patients (74.2 % versus 94.7 %, $p=0.127$).

In the next step we divided the group of revascularized patients into two subgroups – patients who had undergone complete revascularization and patients with incomplete revascularization. The baseline characteristics and comparison of these two subgroups of patients are given in Table 3.

The subgroup of incompletely revascularized patients included only men. This represented a significant difference when compared to the subgroup of completely revascularized patients (0 versus 33.3 %, $p<0.05$). Otherwise, the two subgroups did not

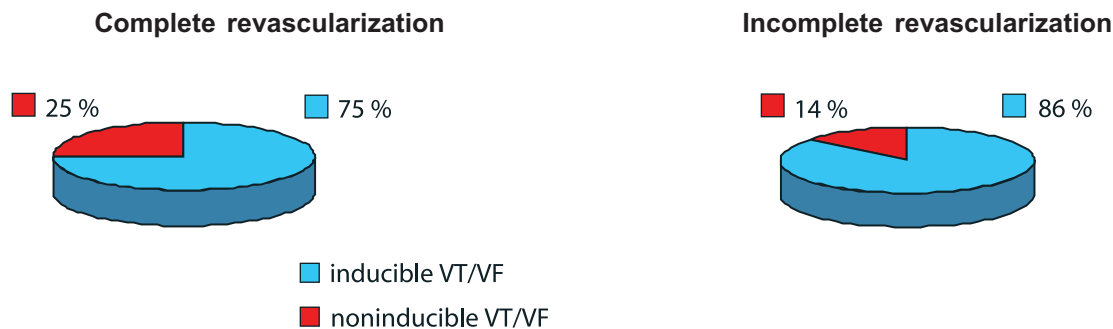


Fig. 4. Inducibility of malignant ventricular arrhythmias after complete and incomplete revascularization. VT/VF – sustained ventricular tachycardia/ventricular fibrillation.

differ in the other parameters studied, including drug therapy (Tab. 3). Inducibility of the malignant ventricular arrhythmias was higher in patients after incomplete revascularization when compared with the patients who had complete revascularization (92.3 % versus 61.1 %, $p=0.095$), yet in this case the p value showed no statistical significance.

Figure 4 shows a clear trend towards the lower inducibility of these arrhythmias in the completely revascularized subgroup of patients.

The above-mentioned data did result in a difference in the number of ICDs implanted: in the completely revascularized group there were significantly fewer cases (11 (55.6 %) versus 13 (92.3 %), $p<0.05$). All of the completely revascularized patients underwent CABG, which was significantly more than in the incompletely revascularized group (100% versus 53.8 %, $p<0.05$).

Discussion

Revascularization of the myocardium during secondary prevention of the SCD influences the arrhythmogenic substrate in CAD. This study focused on a group of patients with chronic forms of CAD and malignant ventricular tachyarrhythmias, with or without previous MI. Our data show a trend towards a decrease in malignant ventricular arrhythmias inducibility after revascularization therapy, even though the level still remains high. Complete revascularization of the myocardium significantly decreases the need for ICD implantation, however.

The greatest influence of myocardial revascularization was shown in the context of acute ischaemia. Reperfusion therapy of acute MI decreases the incidence of spontaneous and/or induced VT in the acute as well as the subacute phase of MI (7). The CAST Trial that demonstrated a higher arrhythmic mortality in thrombolysed patients treated by encainide and flecainide did not register any arrhythmic events in the thrombolysed patients treated by placebo during the first 10 months of follow-up (13).

In the chronic phase of MI, and in present left ventricular dysfunction in particular, the effect of revascularization is controversial. One of the first nonrandomised trials that showed

a benefit (a decrease in the incidence of the SCD) of the surgical myocardial revascularization in patients with CAD without acute MI, was that of CASS (14). Sudden death occurred more frequently in patients treated by drug therapy in comparison to revascularized patients (4.9 % versus 1.6 %) (14). The decrease in the incidence of the SCD was most striking in patients with the three-vessel disease and a history of heart failure. The suppression of ventricular tachycardias mediated by myocardial ischaemia and the subsequent improvement in survival were probably the cause of the insufficient effectivity of implantable cardioverter-defibrillator (ICD) therapy in the CABG Patch trial (2). Kelly et al (15) induced no ventricular arrhythmia in 14 of 30 patients (47 %) patients after CABG after cardiac arrest with preoperatively induced VT. Postoperative inducibility of VT was thus not a predictor of subsequent arrhythmic events (15).

Another work that demonstrated the favorable influence of surgical revascularization on a decrease in the recurrence of cardiac arrest was that of Every et al (16), in which 32% out of 265 resuscitated patients underwent CABG. A 10-year follow-up showed a 52 % decrease in subsequent cardiac arrest risk after surgical revascularization. A group of patients similar to those we studied in our analysis was evaluated by the authors from Leiden (3). In their study, 26 % of 300 patients with ischaemic heart disease who had survived cardiac arrest due to VT or VF underwent myocardial revascularization. They showed, at mean follow-up of 34 months, a decreased risk of recurrences combined with betablocker use and coronary revascularization (3).

These observations represent the notion of a favorable influence of myocardial revascularization on the electrical stability of the myocardium mediated by ischaemia reduction and modulation of the arrhythmogenic substrate (7, 17). It is assumed that prevention of left ventricular remodeling, due to dilation and aneurysm formation, limits the dispersion of refractoriness and suppresses ectopic electrical activity.

On the other hand, some authors point to the unsatisfactory effectivity of myocardial revascularization in the suppression of malignant ventricular arrhythmias. Natale et al (11) analysed a group of 58 patients who, after cardiac arrest caused by documented ventricular fibrillation or polymorphic VT, underwent

CABG together with ICD implantation and programmed ventricular stimulation before and after revascularization. During the follow-up of 4.6 years, 41 patients experienced an adequate shock. Independent predictors for ICD shock were EF lower than 30% and arrhythmia inducibility by one or two extrastimuli. Surgical revascularization of the myocardium itself did not protect the patients from the recurrence of life-threatening arrhythmias (11).

Brockes et al (10) found in a group of 130 patients with CAD, malignant ventricular arrhythmias and implanted ICD, no significant difference in the number of shocks and antitachycardia pacing among revascularized and nonrevascularized patients. Such patients remained at high risk of SCD despite successful surgical or interventional revascularization (10). As study by Brugada et al (9) led to similar results. They demonstrated a high degree of VT inducibility after myocardial revascularization in patients with previous MI and spontaneous ventricular arrhythmias not related to acute ischaemia of the myocardium (no ventricular arrhythmias were induced in only 9 out of 61 patients). He also discovered a high degree of recurrence in these patients during the follow-up (32 out of 61 patients in a mean follow-up of 32 months). Surgical revascularization of the myocardium alone does not lead to prevention of VT/VF recurrences, as Geelen showed in his work (18). Pre- and postoperative programmed ventricular stimulation had no benefit in predicting malignant ventricular tachydysrhythmias recurrences in the population studied.

Conservatively treated patients in our analysis were significantly older. This fact could influence especially the indication for surgical revascularization, which is often abandoned due to old age connected with other severe comorbidity. The analysis of the AVID registry (6) showed a similarly higher age in the group of nonrevascularized patients. In the Leiden study (3), higher age was also combined with significantly higher mortality as well as VT/VF recurrences during long term follow-up.

When comparing complete and incomplete revascularization, we registered a trend towards higher effectivity of complete revascularization on the suppression of inducibility of malignant ventricular arrhythmias. This led to differences in the number of ICD implantations: the need for ICD implantations was significantly lower in the completely revascularized group. Several studies demonstrated worse survival rates and more clinical events in connection with incomplete revascularization (19–21). Many authors recommend a prophylactic ICD implantation in selected group of patients, especially those with low ejection fraction after myocardial revascularization (12, 18, 22).

Chronic hypoperfusion in patients with CAD contributes to systolic dysfunction of the left ventricle. After reperfusion in such an impaired area of still viable myocardium, it is possible to achieve improvement in global ejection fraction and thus improvement regarding heart failure. An adequate complex therapy against heart failure eventually decreases the occurrence of malignant ventricular arrhythmias. Hibernated myocardium by itself contributes to the slowing of the impulse conduction, enlargement of the dispersion refractoriness and increased auto-

maticity. Therefore its revascularization can lead to improvement of electrical stability and better survival (7).

The most effective clinical use of programmed ventricular stimulation was found in chronic CAD. Sensitivity rises with increasing numbers of extrastimuli, but the specificity declines (23). Electrophysiologic study has become an accepted tool for SCD risk assessment. The inducibility of sustained VT in patients after MI with nonsustained VT documented in Holter's ECG identifies a high-risk population of patients indicated for ICD implantation according to MADIT I criteria (24).

Strengths and limitations

Our studied population is a highly selected group of patients with documented VT/VF, often after cardiac arrest. These patients are at high risk of sudden cardiac death and require a complex therapeutic approach to improve their survival.

The greatest limitations of this study are its retrospective character and the limited number of patients that lead to borderline results in statistical analysis of some of the parameters tracked. In a larger group of patients, it would be possible to achieve greater statistical power as well as application of results to the larger population.

The question of the effectivity of ischaemia limitation by myocardial revascularization on electrical stability and the suppression of life-threatening arrhythmias still remains without a clear answer, thus suggesting the need for further investigation in this field.

Conclusion

The inducibility of malignant ventricular arrhythmias after myocardial revascularization remains high, which suggests that it has low effectivity on the arrhythmogenic substrate. Complete revascularization significantly decreases the need for ICD implantation when compared to incomplete revascularization. This can have a positive economic impact on healthcare expenses. It is important to assess the need of ICD implantation for each patient separately.

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