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TEMPORARY ELECTRO-STIMULATION IN THE ACUTE MYOCARDIAL INFARCTION - TWO-YEAR EXPERIENCE

Aleksandar Stojković, Zoran Perišić, Nebojša Krstić, Svetlana Apostolović, Mirko Burazor

Cardiovascular Diseases Clinic, Clinical Center Niš
E-mail: saleks@ni.sbb.co.yu

Summary. *The application of temporary electro-stimulation is necessary in acute myocardial infarction complicated by the appearance of symptomatic bradycardia, AV conduction disturbances and asystolia. During the last two years, 38 of 892 patients (4.2%) with the diagnosis of acute myocardial infraction with ST segment elevation, required temporary electro-stimulation (27 with inferior infractions and 11 with anterior infraction). The third degree AV block was diagnosed in 34 patients (89%). and 4 (11%) patients received prophylactic electro-stimulation due to de novo conduction disturbances (the second degree AV block and block of the left bundle branch- 2 patients, and block of the left bundle branch-1 patient, and block of the right bundle branch-1 patient, and haemiblock-1 patient). The mortality in the group with anterior infarction was significantly higher than the mortality in the group with inferior infarction (8/11 vs. 5/27, p<0.05). In the group with anterior infarction and temporary electro-stimulation, patients with extensive infarcts had bad prognosis while the presence of right ventricular infraction significantly contributed to the unfavorable outcome in the group with inferior infarction and temporary electro-stimulation. In the group with anterior acute myocardial infraction and temporary electro-stimulation, the duration of QRS complex on admission, expressed in milliseconds was significantly longer in the deceased patients than in the survivors.*

Key words: *Acute myocardial infraction, temporary electro-stimulation*

Introduction

The appearance of disturbances in conduction of impulses during the acute myocardial infraction (AMI) is distinctive features of a group of patients with high intra-hospital and subsequent mortality (1-4). This event occurs mostly during the acute myocardial infraction with ST segment elevation (STEMI) while the incidence in the acute myocardial infraction without ST segment elevation is around 1% (5). In spite of clear indication for application of temporary electro-stimulation in AMI there are different opinions concerning its effect on the survival rate, especially in those with intraventricular conduction disturbances (6,7-12). With the introduction of fibrinolytic therapy there was a reduction in the incidence of conduction disturbances during and consequently the reduction in the application of temporary electro-stimulation compared to pre-thrombolytic era (13-15).

The purpose of this study was to examine indications and the incidence of the application of temporary electro-stimulation in AMI in patients treated in the coronary care unit of CVD clinic in CC Ni, under diagnosis of acute myocardial infraction with ST elevation. We also observed the intrahospital prognosis of that group of patients depending on the localization and the size of the infraction and the type of conduction disturbances.

Patients and Methods

During the last two years, in the coronary care unit of CVD clinic of CC Niš, we treated 892 patients diagnosed with acute myocardial infraction with ST (segment) elevation (STEMI). The STEMI diagnosis was given based on WHO criteria: angina-like pain lasting 30 minutes and longer, sustained ST elevation of at least 1mm in two consecutive ECG leads, *de novo* block of the left bundle branch, the elevation of markers of myocardial necrosis (CPKMB and/or troponine) (16). Patients hospitalized within 12 hours from the beginning of angina-like complaints, with initial elevation of ST segment or *de novo* block of the left bundle branch, in absence of contraindications, were treated with fibrinolytic therapy. The patients received streptokinase in dose of 1500 000 units (for 60 minutes) or actilyse in dose of 100 mg (90 minutes), according to the accelerated protocol. Anti-aggregation therapy was initiated on admission by means of sublingual application of 150-300 mg dose, and later 100 mg *per os*, daily. Anticoagulant therapy was continued, or initiated immediately in cases where fibrinolytics had not been administrated, with low molecular heparin dosed per kilogram of patients bodily mass *sc*. at 12 hours. Antiischemic therapy was contained: A) polarized solution (5% glucose with KCI and crystal insulin with nitroglycerine for *iv*. application with dosing depending on blood pressure and on the absence of right ventricular infarction) B) oral mononi-

trate preparations, C) cardio-selective beta blocker metoprolol (*iv or p.o.*) - in case there were no contraindications (pulmonary congestion, systolic TA < 100 mm Hg, SF < 60/min and existence of first degree AV block -PQ > 0.24 sec.). ACE inhibitors *per os* were applied in anterior AMI wherever blood pressure (systolic TA > 100mmHg) allowed it.

Electrocardiogram (ECG) has been performed on three-channel electrocardiogram machine, on admission, after the administration of fibrinolytics, 2 and 4 hours since the beginning of administration of fibrinolytics and then every day up to the discharge patients from hospital. Based on ECG, it was determined whether there was Q or non-Q myocardial infraction, as well as its localization. Infraction of anterior localizations were: anteroseptal, anteroapical, anterior, anterior extensive, anterolateral and high lateral. Infraction of inferior localization included: diaphragmal, posterior, inferoposterior, inferolateral, with or without involving right ventricle. We measured the average duration of native (nQRS) and paced QRS (pQRS) complexes in milliseconds. AV and intraventricular conduction disturbances were divided according to advanced: AV block-AV block II (Mobic II) and III degree as well as right bundle branch block, anterior and posterior left haemiblock and left bundle branch block recommendations (17,18). Conduction disturbances according to the time of development were divided into: new-if not present on previous ECG images, old-if they were present and undetermined- if previous ECG recordings were not available. Prophylactic implanting of temporary pacemaker were done without haemodynamic disturbances where existed the risk for aggravation of AV conduction disturbances, while therapeutic implantation was done in presence of haemodynamicly significant bradycardia or asystolia.

Immediately on admission and every 6 hours during the first 24 hours of hospitalization, blood was taken for analysis for cardio-specific enzymes CPK-MB, or for troponine, 8 hours after the beginning of pain.

Echocardiography examination was performed immediately on admission when hemodynamic status allowed it and after the implantation of temporary pacemaker. The left ventricle function was determined by measuring ejection fraction (area-length method).

Indication for temporary electro-stimulation was given by the assigned physician with observance of recommendation for temporary electro-stimulation in AMI. The procedure itself was performed by means of punction of vena subclavia controlled by fluoroscopy. Temporary electrode of VVI pacemaker was placed in apex of right ventricle, and stimulation threshold was determined as well as sensing of QRS complex. The decision to terminate temporary electro-stimulation was made 48 hours after the loss of indications for temporary pacing.

The received results were subjected to statistical analysis and processed by Student t and χ^2 tests.

Results

In the observed group consisted of 892 patients with confirmed STEMI diagnosis, 450 (51.4%) patients had anterior AMI localization and 433 (48.6%) had inferior AMI localization.

A total of 399 patients (44.7%) received fibrinolytic therapy; streptokinase 384 patients (96.2%) and actylise 15 (3.8%).

Two groups of patients were observed: group I- patients with temporary pacemaker and anterior STEMI (I group) and group II - patients with temporary pacemaker and inferior STEMI.

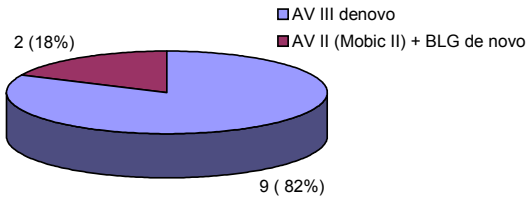
Intra-hospital mortality of the entire group of patients was 12.5% (112 died).

Based on the decision of the assigned doctor and according to ACC/AHA/NASPE recommendations we implanted temporary pacemakers. Significantly greater number of temporary pacemakers were implanted in the II group (Table 1).

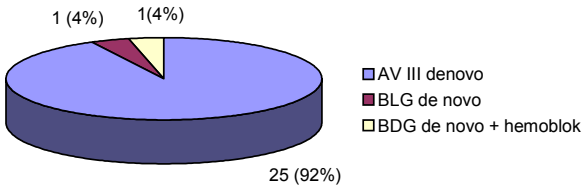
Table 1. Incidence of temporary electro-stimulation in group I and II.

	Group I	Group II	P	Σ
TPM+	11	27	< 0.05	38 (4.2%)
TPM-	448	406		854

Indications for temporary electro-stimulation in the group I and group II are shown in the graph 1 and 2, retrospectively.



Graph 1. Indication for temporary electro-stimulation in group I



Graph 2. Indications for temporary electro-stimulation in group II

There was a statistically significantly higher mortality in group I compared to group II (Table 2).

Table 2. Mortality in group I and II

	Group I	Group II	P
Deceased	8/11	5/27	< 0.05

In the group I there was more frequent occurrence of Killip class >I in the patients on admission, comparing with deceased patients ($p<0.05$) (Table 3).

Table 3. Incidence of Killip class on admission within group I

	Deceased	Survived	p
Killip > I	7	0	< 0.05
Killip I	1	2	

However, there was not a statistically significant difference in occurrence of Killip class >I on admission in the group II (Table 4).

Table 4. Incidence of Killip class on admission within group II

	Deceased	Survived	p
Killip > I	2	2	ns
Killip I	3	20	

There was a significantly higher use of fibrinolytics in survivors in group I (Table 5)

Table 5. Use of fibrinolytics in group I

	Deceased	Survived	p
Fibrinolytic +	1/8	3/3	< 0.05

On the other side, there was not a statistically significant difference in the use of fibrinolytics between survivor patients and deceased patients in the group II (Table 6).

Table 6. Use of fibrinolytics in group II

	Deceased	Survived	p
Fibrinolytic +	2/5	9/22	ns

We also examined the lower ejection fraction (EF) of left ventricle in both group of patients. As shown Table 7 there is statistically significant lower ejection fraction (EF) of left ventricle in deceased patients comparing with survived patients in group I (Table 7). This pattern was not present in the group II (Table 8).

Table 7. EF in group I

	Deceased	Survived	p
EF (%)	24.2 ± 7	38.2 ± 2	<0.05

Table 8. EF in group II

	Deceased	Survived	p
EF (%)	37.4 ± 11.4	38.7 ± 6.2	ns

The deceased patients in the group I had also increased blood level of CPKMB- markers of myocardial necrosis comparing with survived group (table 9) while there was not statistically significant differences in the blood level of CPKMB between deceased and survived patients in the group II (Table 10).

Table 9. Values of markers for myocardial necrosis in group I

	Deceased	Survived	p
CPKMB	965.9 ± 23	431 ± 18	<0.05
Troponin	>22	7.9 ± 0.8	

Table 10. Values of markers for myocardial necrosis in group II

	Deceased	Survived	p
CPKMB	467.9 ± 15	372.4 ± 18	ns
Troponin	11.3 ± 1	8.2 ± 18	ns

In the group II there was statistically significant more frequent infraction of the right ventricle in deceased patients comparing with survived patients.

Table 11. Incidence of right ventricle infraction in group II

	Deceased	Survived	p
IDK+	4	6	<0.05
IDK-	1	16	

On admission, the deceased patients in group I, had statistically significant higher QRS duration compared to the group of the survivors. There was not a statistically significant prolongation of pQRS compared to the nQRS in both subgroups (deceased and survivors) within group I (Table 12).

Table 12. Duration of native (nQRS) and paced (pQRS) in group I on admission

	Deceased	Survived	p
nQRS (msec)	195.8 ± 8	165 ± 7	<0.05
pQRS (msec)	212.3 ± 12	190.2	ns
p	ns	ns	

However we did not found any statistically significant difference between duration of nQRS on admission between group of deceased and survivor patients in the group II. In addition there was not any statistically significant changes between the duration of pQRS compared to the nQRS in both subgroups within group II (Table 13).

Table 13. The duration of native and paced QRS in II on admission

	Deceased	Survived	p
nQRS (msec)	191.7 ± 14	178.4 ± 2	ns
pQRS (msec)	202.2 ± 17	195.1 ± 7	ns
p	ns	ns	

Discussion

Contemporary therapy of the acute myocardial infraction includes prompt diagnosis and therapy aiming at pharmacological (fibrinolytic therapy) or mechanical (primary PTCA) opening of infraction artery and prevention of its re-occlusion. These approaches could limit the size of the infraction and preserve the ejection fraction of both ventricles. All of these leads to a decline the incidence of complications during AMI, such as bradycardial disturbances of the heart rate (13-15) as well as death rate (now around 6.5%).

Several studies indicated that the incidence of temporary application of electro-stimulation during the acute phase of myocardial infraction was from 3.5% (14) to 4.5% (15%). The temporary pacemaker was utilized more in the group of patients with inferior myocardial infarction then in the group with anterior infarction (6.2% vs. 2.4%), (7,9,13,14,15). The reason for that could lay in the facts that patients with anterior infarction and indications for temporary electro-stimulation have extensive necrosis and quite often do not reach health institutions as well had high mortality rate caused by cardiac failure, and cardiogenic shock. On the other side the acute inferior infarctions are usually smaller, often followed by vagus stimulation and transient ischemia of SA and AV nodus. SA nodus and AV nodus are more often fed from lateral branches of the right coronary artery which occlusion caused the inferior infarction. In the ischemic onset, there were a damage of myocites and a subsequently releasing of adenosine. The heightened tonus of the vagus resulted from stimulation of afferent vagal receptors, and the accumulation of adenosine lead to disturbances in formation and conduction of impulses (SA bradycardia and AV block of I, II and III degree) and to hypotension. These features are more frequently seen in the inferoposterior part of the left ventricle than in the anterior or lateral part. However these disturbances can be treated not only by timely and efficient reperfusion but also by means of vagolytic drugs (atropine), adenosine agonists (teofilin). Temporary electro-stimulation is applied in anterior and posterior AMI for protection from asystolia, symptomatic bradycardia and hypotension, both prophylacticly (AV block of I and II degree and/or intraventricular conduction disturbances) and therapeutically (symptomatic bradycardia, complete AV block and asystolia).

There are evidence that the occurrence of complete AV block during AMI can be predicted (19). The presence of (*de novo* or previous) any of the following conduction disturbances presents a risk factor.

– AV block I
– Mobic I AB block II
– Mobic II AV block II
– Front left hemi block
– Posterior left hemi block
– Right branch block
– Left branch block

Each of the risks factors has a value of 1. The total indicates the risk for the occurrence of complete AV block. The value of 0 indicates risk of 1.2 to 6.8%. The value of 1- 7.8 to 10.4%. The value of 2- 25 to 30.1%. The value of 3- risk of 36% or more. Some authors consider that it is not correct that Mobic II should have value of only one point since it carries greater risk. Also, in this scoring system there is no difference between *de novo* and previous branch conduction disturbances.

In our patients, in 89% of cases (34 patients) the indication for implantation of temporary pacemaker was therapeutic (symptomatic AV block of III degree) while only 4 (11%) patients received temporary pacemaker prophylacticly because of *de novo* AV block of II degree and *de novo* block of the right bundle branch and *de novo* haemiblock (1 patient).

Statistically significant higher intra-hospital mortality was noted in the group with anterior AMI and temporary electro-stimulation compared to the group of patients with inferior AMI and temporary electro-stimulation (8/11 vs5/27, $p < 0.05$). Our data are consistent with earlier published (20,21,22) where high mortality was seen in the group of patients with anterior AMI due to the fact that extensive necrosis caused the conductivity disturbances, and that conduction system was irreversibly damaged what lead to insufficient recovery and very rare stable rhythm. The impact of the size of infarction on the mortality within the group with anterior AMI and temporary electro-stimulation is clearly seen from the data concerning significantly more frequent Kilip class > I, on admission (7/0 vs1/2, $p < 0.05$), lower EF (24.2 ± 7 vs 38.2 ± 2, $p < 0.05$), less frequent using the fibrinolytic therapy (1/8 vs. 3/3, $p < 0.05$) and higher values of necrosis markers CPKMB (965.9 ± 23 vs. 431 ± 18, $p < 0.05$) in the group of deceased patients compared to survivors. The influence of the left ventricle function measured by the same parameters on the prognosis in the group with inferior infraction and temporary electro-stimulation cannot be observed because there was not any statistically significant difference in the values between the deceased and the survivors. One explanation could be that a significantly higher incidence of right ventricular infraction occurred in the group of deceased patients with inferior infraction and temporary electro-stimulation compared to the survivors (5/7 vs. 4/20, $p < 0.05$). It is known that the occurrence of right ventricular infraction worsens early prognosis for the patients with acute inferior infraction, especially in the case of bradycardia and/or loss of AV synchronization as in case of occurrence of AV block of III degree (23). Within inferior AMI associated with right ventricular infraction (in 50% of cases), blood flow into left ventricle is diminished and its emptying is aggravated due to ischemic damage, so both ventricles have relatively fixed stroke volume. Minute volume therefore depends on heart frequency and adequate pre-load of both ventricles. The use of VVI temporary pacemaker in our group of patients could not provide AV synchronization and adequate pre-load, meaning that there was

not a positive influence of left and right artrial "kick", which is valuable for infractions of this localization (24).

To the best of our knowledge, this is the first study that showed, the influence of duration of native and paced QRS on intrahospital prognosis of patients with temporary pacemaker in acute myocardial infraction. In the damaged myocardium there is a disturbance of the speed and direction of the propagation of electrical activation, which leads to abnormal ventricular depolarization manifested by QRS prolongation. This leads to non-uniform contraction of the parts of the left ventricle that reduces ejection fraction and reduces systolic function of the left ventricle (25). It is known that the prolongation of duration of QRS complex has an unfavorable influence on the long-term prognosis especially in patients with cardiac failure where the incidence of branch block is from 20% to 35%. The bundle branch block is a strong independent predictor of mortality in this group of patients.

We established an unfavorable influence of QRS duration within group I, meaning that QRS complex was significantly longer on admission in the group of deceased patients compared to survivors (195.8 ± 9 vs. 165 ± 7, $p < 0.05$). This could be an indication of greater damage of the myocardium and sign for a worse intra-hospital prognosis for the patients.

Duration of QRS complex was not significantly different between the deceased and the survivors in group II.

Duration of the paced QRS complex did not significantly influence the prognosis for patients in both groups. The difficulties concerning the interpretation of this influence arise from the inability to uniformly position the electrode of the temporary pacemaker in right ventricle apex leading to difference in electrical vector of the paced QRS complex and thus rendering the comparison impossible.

Conclusion

1. Out of total number of patients with STEMI diagnosis (892) during the last two years, 38 of them received temporary electro-stimulation, which makes 4.2%.
2. Indications were mostly therapeutic (symptomatic AV block of III degree de novo) in 34 patients (89%), while 4 patients (11%) received temporary pacing prophylacticly (de novo AV block of II degree- Mobic II and de novo block of the left chamber and de novo haemiblock).
3. High mortality was reported in the group with temporary electro-stimulation and STEMI of anterior localizations (72.7%) in patients with worse function of the left ventricle (significantly lower EF), that is, with larger infraction of myocardium (significantly higher CPKMB values). The prognosis for this group of patients depended mostly on the timely application of therapy and limiting the size of the infraction, and to a lesser degree on therapeutic or prophylactic application of temporary pacing.
4. Mortality in the group with inferior STEMI and temporary electro-stimulation was 18.5%, which was significantly lower compared to the group with anterior STEMI. The left ventricle function measured by ejection fraction or the size of the infraction measured by CPKMB elevation were not significantly different in the group of the deceased compared to the survivors. The only difference was significantly higher occurrence of the right ventricle infraction in the group of the deceased patients.

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PRIVREMENA ELEKTROSTIMULACIJA KOD AKUTNOG INFARKTA MIOKARDA - DVOGODIŠNJE ISKUSTVO

Aleksandar Stojković, Zoran Perišić, Nebojša Krstić, Svetlana Apostolović, Mirko Burazor

Klinika za kardiovaskularne bolesti, Klinički centar Niš
E-mail: saleks@ni.sbb.co.yu

Kratak sadržaj: *Primena privremene elektrostimulacije neophodna je kod akutnih infarkta miokarda komplikovanih pojavom simtomatske bradikardije, AV smetnjama provodjenja i asistolije. U protekle dve godine od 892 bolesnika sa dijagnozom akutnog infarkta mikarda sa ST elevacijom, njih 38 (4,2%) zahtevalo je privremenu elektrostimulaciju i to 27 sa donjim infarktom i 11 sa prednim infarktom. Indikacija u 34 bolesnika (89%) bila je simtomatski AV blok III stepena. Profilaktičku elektrostimulaciju dobilo je ukupno 4 (11%) bolesnika i to zbog novonastalog AV bloka II stepena i novonastalog bloka leve grane (2 bolesnika) kao i novonastalog bloka leve (1 bolesnik) i novonastalog bloka desne grane i novonastalog hemibloka (1 bolesnik). Smrtnost u grupi sa prednjim infarktom bila je značajno veća nego u grupi sa donjim infraktom (8/11 vs 5/27, p<0,05). Lošu prognozu u grupi sa prednim infarktom i privremenom elektrostimulacijom imali su bolesnici sa velikim infartom dok je prisustvo infrakta desne komore značajno doprinelo lošem ishodu u grupi sa donjim infarktom i privremenom elektrostimulacijom. U grupi sa prednjim akutnim infarktom miokarda i privremenom elektrostimulacijom trajanje QRS kompleksa na prijemu izraženo u milisekundama bilo je značajno duže u egzitiralih bolesnika nego u preživelih.*

Ključne reči: *Akutni infarkt miokarda, privremena elektrostimulacija*