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Electrocardiogrphic changes in the heart after myocardial infarction under Maharishi Amritt Kalash effect

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In this investigation we have tried to examine Maharishi Amritt Kalash (MAK-4) effect on such important index of heart functional state, as electrical activity, and to reveal quantitative and qua-litative changes of ECG after myocardial infarction [4-6,8,12].

Materials and Methods

The investigations of electrocardiographic changes were conducted on 150 rats of Vistar line with experimental myocardial infarction (EMI), induced by tying the descending branch of the left coronary artery. ECG was recorded on the 2, 7, 15 and 30th days after EMI. 130 animals were operated. 80 of them obtained 6% aqueous solution of Maharishi Amritt Kalash (MAK) Ayurvedic as food supplement, 2–2.5 g for each animal during 30 days, others served as control [4,9,11,13,19].

Following criteria of ECG were used for quantitative estimation of ECG changes: Rind; ind RS-T segment elevation and its dynamics; T tooth. The significance of these indices has been proved by observations of many years in clinical practice and proceeds from analysis of peculiarities of ECG processes in infarcted and periinfarcted zones.

The short substantiation of used ECG indices is given below.

It is widely recognized that pathological Q tooth on ECG is the most typical index of EMI. Not only the debth of Q tooth, but its length also has a certain significance. In this respect, our examinations showed that it is expedient to determine the area of pathological Q tooth. The informational value of the latter index rises considerably, when all areas of pathological Q tooth appearing on ECG and registered in two widely recognized leads are summarized.

The number of leads, where pathological Q teeth are found, is important as well. Thus, Q tooth index is calculated according to the formula.

 $Q_{ind} = \Sigma O_{ar} \times Q_n$ where ΣQ is the sum of pathological

Q teeth areas in examined leads (in our case these are chest leads V1-6); Q_n - the number of leads, where those teeth were found.

The area is determined planimetrically (in *mm/sec*). Only pathological Q teeth are to be taken into account.

Attaching a great importance to the changes of R tooth in diagnostics of acute myocardial infarction for determination of the size of infarcted area volume and the dynamics of the process, R index has been calculated

 $R_{ind} = \Sigma R \times R_n$

where Σ R is the sum of leads amplitude of R teeth (in mm) in the examined leads; R_n - the number of leads, where R tooth is found. This index for all versions of frontal myocardium infarction is determined in I, avl, V 1-6 leads.

We attach great importance also to the RS-T segment elevation, as to the earliest manifestation of myocardial ischemia, only after which the changes of depolarization appear (QRS complex).

Not only the fact of RS-T segment elevation, but also the prevalence of this phenomenon in different leads were taken into consideration as ischemia and infarction volume index, as far as its dynamics after infarcted area reproduction permit to judge about the dynamics of the process. Changes of T tooth, which happen simultaneously with blood circulation improvement in ischemia zone, permit to take this index into account also [3,6,7,14,15,17,20].

Results and Discussion

The RS-T segment elevation is the earliest index of injured myocardium. During our examinations in the process of the experiment this phenomenon was displayed already at the first 10 minutes after tying the descending branch of the left coronary artery. Diastolic, as well as systolic phenomenon should be considered responsible for RS-T segment elevation mechanism. Chronologically, diastolic mechanism starts operating in result of rest po-



Fig. 1. Dynamics of RS-T segment elevation after EMI

tential diminution of ischemized cells, and then systolic mechanism – due to shortening of action potential and diminution of its overshoot.

We observed a typical displacement of RS-T segment in leads, corresponding to the affected zone. These are basically chest leads V1-6, rarely the I,avl lead. The observations of the dynamics of this process exposed differences between two groups of rats: those who obtained MAK-4 preparation and those who did not (fig. 1). It is typical that a considerable reduction of RS-T segment was no-ticed in animals getting MAK-4 on the seventh day of the observations with following expressed positive dynamics by the 15th day. On the 30th day the ECG of some animals showed RS-T segment reduction to the isoelectric line or remained slightly raised.

The ECG of animals that had not obtained MAK-4 showed slower dynamics of RS-T segment and by the end of the observations the elevation of RS-T kept within 1.5 mm limits above the isoelectric line. Appearance of reciprocal changes in this group of the animals was noted: RS-T segment reduction in II,III, avl leads by the second day of the disease observations. This fact confirms the thesis about more serious course of illness in the group of not treated animals. Though in the recent years opinions considering the significance of Q tooth at myocardial infarction have undergone considerable changes is being called in question, we consider the pathological Q tooth to be an indisputable sign of definite section of myocardium assinergy as a result of its cicatrization and in number of cases of aneurismatic expansion.

The size of Q index and its dynamics in experimentally reproduced acute transmural myocardial infarction confirm the high diagnostic value of Oind.

So, unequal dynamics of pathological O tooth in both groups of animals attracts attention (fig. 2). The treated animals had more obvious dynamics of this tooth diminution, while pathological Q tooth was absent by the end of the observations on the 30th day of the disease. It can be stated that the presence of "mute zone" in the myocardium, which pathological Q teeth are often connected with, not always and not completely are determined by myocardial cells necrosis and not always are connected with irreversible injury of myocardium. It is possible that collateral blood circulation improvement on trea-ting background (in circumstances of current observation under MAK-4 preparation effect), can call forth electric activity rise of surrounding the injury zone sections of myocardium. Hypertrophy development, that brings to "compression" of infarcted section of myocardium, is promoting that. This fact is proved by R index dynamics during the observation process.

While the Q index is in direct correlation with the infarcted area value, R index then displays opposite correlation: the less is its size, the more is the affection value, and on the contrary. It is confirmed by studying the arithmetic meaning of amplitude sums of Q and R teeth and their correlation in examined groups of animals (fig. 2, 3).

We noted a considerable reduction of R tooth amplitude already during the experiment after ligation of the coronary artery. QR configuration was registered on the ECG of animals obtaining MAK-4 on the second day



Fig. 2. Q index influence after EMI



Control D before treatment Safter treatment

Fig. 3. R index size after EMI

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of observation. It can be explained by the presence of necrotic tissue, as well as injured but still viable myocardium. R tooth collapse was more expressed in not treated animals, and the QRS complex acquired Q_2 shape, and even QS, which testifies to necrosis of myocardial section. In separate cases such picture could be observed in treated animals too.

Not only the mentioned configuration of QRS complex, but the number of leads where that configuration was registered attracted our attention. After EMI this configuration spreaded all over chest leads, while in treated animals it was applied to registered number of leads (V3,4). This fact, i.e. the opposite correlation of Q and R teeth, and their prevalence among number of leads points indirectly to the expansion of the affected zone.

The results of the observations conducted on treated animals on the 7, 15 and 30th days testify to a natural rise of R tooth amplitude and index, which by the end of the observations come to average arithmetic value of the control group. That witnesses to a considerable improvement of blood supply in myocardium's affected zone, and therefore of its limitation. This dynamics expression is considerably less in animals with EMI, a comparatively small rise of R index is noted, and it remains lowered until the 30th day of observations.

 R_f/R_n index was calculated for additional objectification of the obtained data and estimation of preparation effect, where R_f is the factual amplitude of R tooth in the fixed lead (in *mm*), and R_n is that (in *mm*) of a normal size of R tooth for the fixed lead (fig. 4).



Fig. 4, R_n/R_n correlation in lead 4 by the end of the observations affer EMI

As the presented data show, on the ECG of animals treated with MAK-4 the rise of this index was noted by the end of the observations, even up to one, i.e. the amplitude of R tooth in the fixed lead (we took into consideration V4 lead, as the most optimal reflection of pathological process) is coming to normal state. In comparison with data of the control group Rf/Rn correlation index in the group of animals treated with MAK-4 witnesses to the effecti-veness of the preparation and its positive effect on the infarcted area [10, 16, 24].

The interpretation of T tooth changes is rather difficult. Although it is a sensitive manifestation of ventricular repolarization, it lacks in large specificity due to myocardium ischemia. Usually definite parallelism is observed between the dynamics of RS-T segment



Fig. 5. Dynamics of the T tooth amplitude after EMI

reduction and the T tooth changes. The latter took place in our obser-vations during the experiment as well (fig. 5). Negativeness of T tooth was mostly expressed in treated animals by the 15th day of EMI, with following considerable reduction of T tooth negativeness by the 30th day. In separate cases, an isoelectrical T tooth was observed by that term. The expressed negative T tooth on the 15th day of observations in the group of not treated animals reduced by the 30th day just in a small extent [21-23].

We connect the dynamics of T tooth mentioned above with the reduction of periinfarction zone ischemia and the improvement of blood supply in myocardium in the group of rats treated with MAK-4 preparation, in contrast to the control group of animals.

Summarizing our observations, we have come to the conclusion that Maharishi Amritt Kalash preparation exerts a positive therapeutic effect upon the infarcted area, promoting acceleration of repairing processes and improvement of the injured myocardium blood supply.

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Մրտի Էլեկտրասրտագրական փոփոխությունները սրտամկանի ինֆարկտի ժամանակ Մահարիշի Ամրիտ Կալաշի ազդեցության ժամանակ

Մ.Հ.Վարոսյան, Կ.Գ.Ադամյան

150 առնետների էլեկտրասրտագրական քանակական և որակական փոփոխությունների ուսումնասիրությունը ՄԱԿ-4-ի ազդեցության տակ սրտամկանի ինֆարկտի ժամանակ ի հայտ են բերել դրական տեղաշարժեր 2-, 7-, 15-, 30-րդ օրերին։ Փորձարարական ինֆարկտից հետո 6%-ոց ՄԱԿ-4 լուծույթի կերային հավելումը R, Q, RS-T և T ատամիկների վրա ունեցել է բուժական ազդեցություն՝ արագացնելով ինֆարկտային օջախի ապաքինումը, միաժամանակ լավացնելով խանգարված պսակաձև արյան շրջանառությունը։

Электрокардиографические изменения сердца под влиянием Маариши Амрит Калаша после инфаркта миокарда

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Изучались ЭКГ количественные и качественные изменения после экспериментального инфаркта миокарда (ЭИМ) под воздействием препарата МАК-4.

Исследования ЭКГ изменений проводились у 150 крыс линии Вистар с ЭИМ, полученным путем перевязки нисходящей ветви левой коронарной артерии. ЭКГ записывали на 2, 7, 15 и 30-й дни после ЭИМ. Было оперировано 130 животных, из них 80 получали 6 % раствор МАК-4 в виде пищевой добавки, осталь-

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ные служили контролем. Для оценки ЭКГ изменений нами использовались R_{пок}; Q_{пок}; подъем сегмента RS-T и его динамика; зубец T.

Результаты исследования показали, что МАК-4 в виде пищевой добавки оказывает положительное терапевтическое действие на очаг инфарцирования, способствуя ускорению репаративных процессов и улучшению кровоснабжения поврежденного миокарда.

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