Interview

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I'm Leonid Makarov, representative of ISHNE in Russia. Currently, the ISHNE is carrying out the web International Symposium on Heart Failure. The President of the Symposium, Prof. Arthur J. Moss (USA) prepared 10 questions about the current problems regarding heart failure. These questions have been arranged to be posed to the most important specialists in the world.

At present, I'm at the office of Dr. Viacheslav Mareev, an important Russian specialist in heart failure, and I would like to ask him these questions.

LM: Dr. Mareev, how important is the problem of heart failure in Russia?

VM: According to the data from the epidemiological study ЭПОХА, carried out in Russia in year 2001, and the diagnostic criteria from the Framingham study, 5.5% of the Russian population suffers from clinical heart failure. This corresponds to 7.5 millions of people, and what is even more significant, the Framingham criteria do not contemplate sublinical HF. That is to say, if we add to these 7.5 millions of patients, those with LV dysfunction too, the number duplicates. Anyway, 7.5 millions is a very important number that generates a great problem. Notably, the number of patients is increasing. Since currently, the major causes of decompensation of HF are not properly controlled high blood pressure and diabetes, this problem becomes interdisciplinary, not merely cardiological, and finding a solution is very important.

LM: I have an additional question: what are the specifics of the problem in Russia, in comparison to developed countries?

VM: According to the official data from the European Society of Cardiology, obtained in the EuroHeartSurvey study (Russia also participated in this study), the incidence of HF in Europe is 2%, and in Russia is 5.5%. We thought that the incidence of HF in Russia would be greater than this figure, since very few cardiovascular surgeries are performed, such as valve replacements and coronary bypasses. Moreover, a proper control is not achieved regarding the diseases that cause HF, such as high blood pressure and coronary heart disease. The ЭПОХА study showed that a proper control of BP is only achieved in 7.2% of unhealthy people. That is to say, only 1 each 14 patients with HBP received the appropriate medical treatment, and this causes an important impact on the incidence of HF. Taking into consideration that in USA there are 5 millions of people with HF, we, before the ЭПОХА study, hypothesized very cautiously, that this number in Russia would be very similar or a little greater, considering the Russian population is smaller. Our prognosis was true: in Russia 5.5% of the general population or 7.5 millions of people have HF.

LM: Then, is this the most important problem of medicine?

VM: Yes, of course.

LM: Second question: which, among HF classifications, do you think is the most appropriate one?

VM: The Russian classification, accepted officially in year 2002, brings together 2 classifications. Its basis is the NYHA classification, which includes 4 functional classes and facilitates the current evaluation of the state of the patient. The advantage of this classification is that it enables us to assess the current state of the patient and its dynamics. The drawback of such classification is that it does not assess the stage of the disease. For instance, a patient with a minimal impairment at cardiac level, who receives an inappropriate treatment, may barely rise from the bed and belong to FC IV; however, after the optimization of the treatment, this patient may be for a long time in FC I-II. On the other hand, a patient with a significant cardiac impairment, who receives a proper treatment, may remain in FC II. For this reason, in USA they attempted to make the classification more complex with stages A, B, C, D, according to the heart disease.

The Russian classification, implemented in 1935, with some modifications, has accompanied us to this date. In the HF classification, which is divided into 4 stages: 1, 2A, 2B and 3, the current state of the patient is assessed and also the degree of the damage at cardiac level. For example, stage 2A implies pulmonary edema or isolated right HF, 2B – disorders at the level of the more pulmonary systemic circulation, stage 3 – irreversible damage of target organs. In the current Russian classification, 2 aspects of severity are assessed: the degree of advancement of the disease and the current functional state of the patient. These 2 aspects are traditional for Russian medicine. On the other hand, the attempt by American cardiologists (in 2001 and then 2005) of graduating all cardiovascular diseases in stages A, B, C, D, did not find any support in Russia, since according to this classification, any patient with a heart disease may be catalogued as in HF. Stage A, for instance, implies some heart disease with no signs of HF. In Russia, we consider that this approach is too comprehensive, because true HF starts with stage C, or at least B, if we add asymptomatic HF.

LM: So, the main approach is clinics?

VM: Yes, of course, doubtless. An approach on clinics and the severity of the cardiac impairment.

LM: According to your opinion, is there no universal classification that would satisfy Russian physicians and that would correspond to international criteria?

VM: No, there is not. For that reason, the functional classification doubtlessly, must remain. Nevertheless, it should be supplemented by the criteria of cardiovascular impairment, since the clinical evolution of the disease influences more on the prognosis of the patient than functional class. It is known that a patient that requires frequent hospitalizations, has a much worse prognosis than a patient in FC IV, who has not been hospitalized.

LM: Thank you. Next question, which are the methods to prevent HF?

VM: The question is very wide; we could discuss this topic for a couple of days. But, as the ЭПОХА study revealed, the most frequent causes are 3: high blood pressure, coronary heart disease and diabetes.

Of course, a proper treatment of these 3 problems is the foundation to prevent HF. Nowadays, we can talk about drugs that prevent the development of HF. The best known group is the ACEI. The first studies conducted with captopril (SAVE) and enalapril (SOLVD) showed us the possibility of prophylaxis of HF in patients with LV dysfunction. Then, 2 studies were conducted (isolated from the others), the HOPE with ramipril and the EUROPA with perindopril, which showed the possibility of HF prevention in patients with cardiovascular risk and preserved left ventricular (LV) function. Undoubtedly, the ACEIs are the most important drugs for HF prophylaxis.

Regrettably, in Russia the angiotensin II receptors antagonists (ARA) are seldom used. There are 2 main causes for this phenomenon. First, the results of the studies are not very conclusive, mostly, in comparison to the ACEI. Often times studies with different designs are mixed, and this makes drawing conclusions difficult. The ELITE, ELITE-2, OPTIMAAL, VALIANT studies showed that captopril resulted equal or even more effective than ARA, and it seems that there is no need to indicate new drugs, since they are more expensive and less effective. Attention must be paid too, to the VALIANT and OPTIMAAL studies, which were conducted with post-AMI patients: in the ELITE 1 and 2 studies (which included patients with HF), there were no differences with the end points observed. The results from the CHARM with candesartan, showed significant advantages from ARA, in comparison to placebo and similar with ACEI. This fact is not widely known in Russia. Currently, here the prevalent opinion is that the ARA are very effective drugs that may be used to treat HF, just as with ACEI. One of the evidences of the prophylactic effect of ARA is the RENAAL study, in which patients were treated with diabetic nephropathy. A 32% reduction was shown regarding the risk of new cases of heart failure with losartan. Similar figures were verified with ACEI. On the basis of these data, I think that the second important group of drugs for HF is the ARA. Moreover, these medications regarding their security, are comparable to placebo and superior to other groups of medications, even ACEIs.

Regrettably, other drugs used to treat HF, did not show preventive power. These drugs (for instance, diuretics and digitalis) are not used as prevention. The topic of beta-blockers is complex. Studies on prevention with BB were not performed. The only work to take into account is the CAPRICORN study, which included post-AMI patients, and part of them had LV dysfunction or HF. About end points (which varied during the study), the study was negative, since the objectives were not reached. However, analyzing the general results, such as reinfarction and mortality, a minimal significant difference was achieved (near the limit), which let us use carvedilol to prevent HF. Also, analyzing the SENIORS study with nebivolol, we may conclude that among elderly

patients (older than 70 years old), with diastolic dysfunction, BB reduce hospitalization (in the limit of significance) without decreasing mortality (considering that hospitalizations are due to HF). In spite of all this, there are no solid data about HF prevention with BB. For this reason, from the point of view of evidence-based medicine, the BB are indicated to treat clinical HF.

LM: My next question is, how to differentiate if AF is the cause or the consequence of HF?

VM: This is a difficult question. In the case of a healthy person who “suddenly” starts with AF, it is very likely that among the complications, HF may appear. But, in a patient with clinical HF and paroxysmal AF, the answer is more difficult. If we could maintain safely the sinus rhythm in all these patients, we should do it. Regrettably, the last studies showed that, even with the most effective drugs, the rate of sinus rhythm cannot be maintained over a 60% preservation rate. We should think too, that in another 40% of the patients (almost half of them). The results from studies show that by controlling heart rate in these patients, the prognosis is no worse than in the group where rhythm control is carried out. Furthermore, the drugs used to control HR are the same in general, that are used to treat HF, such as for instance, BB. If one indicates amiodarone or propafenone (even worse) to control rhythm, it may cause a negative impact on the myocardium. In such situation, sotalol is preferable, since with its BB properties is more appropriate to be used in HF, in spite of its proarrhythmic effects. In the case of a young patient, with no associated pathology, preservation of sinus rhythm should be attempted, since AF in the long run may cause myocardial damage. In the case of a patient with chronic AF associated to heart disease, and increase of left atrium (LA) size, HR control would be better. Of course, if we had a “miraculous” drug that would enable us to control sinus rhythm in 100% of the patients, we would attempt it in all patients. Sotalol, in comparison with amiodarone, is not as effective to preserve sinus rhythm (40% against 60%); but in the case of acute AF, it helps to better control HR, which better prevents the development of HF. I think that sotalol, as a class III antiarrhythmic agent with BB properties, is an appropriate drug to treat HF associated with AF. The studies made in Russia showed that bisoprolol to maintain sinus rhythm is as effective as sotalol. However, the problem is much deeper, since currently there are no studies about AF prophylaxis in HF. Some retrospective analyses reveal that the ACEI and the ARA, through cardiac remodeling, may decrease AF incidence in a 20-23%. These are not antiarrhythmic drugs, but this approach seems to be very important. For this reason, the basis of the treatment of HF with AF, is the RAA system block jointly with use of BB. This treatment helps to prevent AF, but the remaining 40% of the patients with chronic AF, besides the RAA system block, also need low doses of digoxin to control HR. In American guidelines, digoxin holds one of the last places, as an additional medication. In Russian recommendations, this medication is very important, mostly in the combination with BB, for a

proper control of HR. According to the retrospective analysis of the CIBIS-II, the decompensated patients benefit from the combination of BB with digitalis.

LM: Digoxin should be combined with ACEI, too?

VM: Yes, of course! Our study on 737 patients with clinical HF and AF with a 6-year follow up showed that, with the ACEI treatment plus digoxin to control HR, the prognosis for these patients did not differentiate from patients with sinus rhythm. When we published this studio for the first time in 1998, nobody believed us, but later these data were confirmed.

I want to say once more, that in young patients without HF, we should attempt to maintain sinus rhythm.

Another important problem is anticoagulation. After a recent study, the ACTIVE-W, the subject of warfarin seems to be defined. It cannot be replaced with aspirin or any other drug. From my point of view and that of my colleagues, warfarin should be recommended to all the patients with HF and AF. In fact, we have to recommend it to all the patients with a high risk of thromboembolism: older than 65 years old, history of thromboembolism, ventricular thrombi, etc. We also think that a bad hemodynamics (EF<35%) is an indication for oral anticoagulation.

LM: Another important question, if you had just 3 drugs to treat HF, which would you choose?

VM: Captopril, bisoprolol and furosemide, or in general terms: ACEI, BB and diuretics. If it was only 1, I would choose captopril; if there were 2 – captopril and furosemide; if there were 3 – captopril, furosemide and bisoprolol.

LM: And if there were 4?

VM: With 4 I would add an antagonist of aldosterone; with 5 – digoxin.

LM: Between Russian physicians, the use of cardiotrophic drugs, vitamins, etc., is fairly common. What is your opinion about these drugs, and the doubtful effects on the myocardial metabolism?

VM: Well, in this regard Russia is a special country. And their physicians are fans of this idea and they cannot drop it. In their time, there was a boom of ATP, then cocarboxylase, then neoton, kreatinphosphate.

LM: Yes, this management remained.

VM: Of course, to this moment, trimetazidine is used, and another sort of “holy water” is being promoted: mildronate. What can I say? Since this treatment has some theoretical basis, we cannot rule it our completely. There are no evidences of the effectiveness of these drugs, except some small studies with no placebo group, which showed some doubtful beneficial effects. Among the drugs of this group, the use of trimetazidine is the most justified one. Perhaps in the future some effective drug will appear. Until now, there were large studies (for example with L-carnitine), but no effectiveness was shown.

LM: However, L-carnitine is very popular.

VM: Yes, there are many popular drugs, as for instance midronate, produced in Latvia, but there is no scientific evidence for their use. In the Russian guidelines for HF management from year 2006 (which will be discussed over the next 6 months), there will be no “metabolic” drugs, although they will be mentioned very briefly, with no specific names. It is worth adding that trimetazidine is the official indication in coronary heart disease, but not in HF.

LM: You mean that the use of “metabolic” drugs is the responsibility of each one of the professionals.

VM: Well, I think that these medications are not harmful, and if the patient can afford them (besides the main drugs), let him/her receive them. However, the main drugs cannot be replaced.

LM: This is a very valuable opinion. Now, which of the non-cardiological drugs may cause HF?

VM: It is one of the most important questions currently. For instance, adriamicine, doxorubicin and other oncological drugs may cause heart diseases. But, the magnitude of the problem is not so important.

LM: In this situation, the benefit of the oncological treatment exceeds the damage produced by the toxicity of such drugs.

VM: Of course, during the treatment of oncological patients, myocardial biopsies should be conducted to detect in time toxic heart diseases. However, the number of these patients is not so important; the great current problem is the wide use of NSAIs. When we speak of the cardiological effects of the medication, we think of HBP or AMI. But, if we assess the rate of AMI produced by NSAIs, is not so important and it increases only with a much extended use of these drugs (months). Making a comparison with gastrointestinal complications, which occur in 40% with a prolonged use, the risk of AMI is much lower (around 2%). The VIGOR and APPROVE studies revealed that after 18 months of continuing treatment with NSAIs, a decompensation of the HF may occur. Sometimes, just an injection of NSAIs is enough to cause fluid retention (and we described some of these cases). For this reason, the use of NSAIs is not recommended in the period of HF decompensation, at the time of fluid retention and the risk of APE. In the case analgesia is needed, other drug groups are preferred, such as for instance morphine, which has beneficial hemodynamic effects during a non-extended use. If the patient with chronic HF needs NSAIs, the dose of ACEIs and aldosterone antagonists should be increased, since the NSAIs block the synthesis of prostacyclin and the effectiveness of the drugs that act through the kallikrein-quinine system, decreases. It is important to control the potential complications, and an increase of the dose of diuretics, BB, and of course ACEIs and aldosterone antagonists, is advisable. In spite of the discussions regarding COX-2 inhibitors, I think in HF they are the drugs of choice (for example, celecoxib). I understand that currently there is a lot of discussion about the thrombogenic effect of selective and non-selective NSAIs, but this is not the topic of our conversation. Lately,

there is much discussion about the benefits from aspirin in HF. And yet, there is no definitive answer. We should consider that low aspirin doses may inhibit the effects of ACEIs, some BB, diuretics, aldosterone antagonists and accelerate HF progression. Undoubtedly, the aspirin is indicated in acute coronary heart diseases, but I don't agree much with a prolonged use in chronic coronary heart disease.

LM: There is a popular opinion that after turning 40 years, one should ingest aspirin in low doses.

VM: Yes, but this is not for patients with HF. Only the patients with a clear indication for this should take aspirin. However, forbidding currently the use of aspirin in all the patients with HF of ischemic etiology would be impossible.

LM: There are no arguments for this prohibition either.

VM: I honestly think that nowadays, there is no clear evidence for the use of aspirin in HF. Moreover, all the studies with aspirin in the patients with chronic coronary heart disease were performed a long time ago, when modern drugs were not available. For example, the ISIS-2 revealed the brilliant effect of aspirin after AMI, but the comparison was "aspirin vs. nothing." Back then, there was no thrombolysis, no ACEI, and BB were just starting. For this reason, currently there are no studies that compare a group with the complete treatment vs. another group with the complete treatment plus aspirin. We cannot use as basis 30-year-old studies, although we have to take them into account.

The last study about the use of aspirin was the WATCH, conducted in USA, Canada and Great Britain. Regrettably, this study did not include an enough number of patients. Nevertheless, the WATCH and the pilot study WASH showed that the use of aspirin increases in a 31% the hospitalizations by HF acute exacerbation.

LM: Perhaps this is the answer. My next question, what is the significance of sudden death (SD) in HF?

VM: Approximately, half of the patients with HF die suddenly (those with FC IV – 30%, FC II – 70%). The question is, how do we define sudden death? Usually, SD is considered as the cause for an arrhythmia or cardiac arrest, with no prior worsening of the state of the patient. And we should not forget that the patients may die suddenly from an acute coronary syndrome or acute PTE.

LM: It is almost impossible to conclude if death was just from an arrhythmic cause.

VM: Of course, many times it is impossible to differentiate. I participated many times with the group of experts, of the end-point committees of the large studies, in the analysis of the causes of death, for instance in Russia. Frequently, half of the experts consider the cause of death is SD, and the other half does not support this opinion. Of course, when the patient dies of progressive HF or if he/she presents 50 TV salvoes in Holter, and then is found dead in bed, the causes are evident.

On the contrary, if the patient dies without clear prior worsening and with no witnesses, it is much more difficult to define the cause. In general, in the patients with HF, SD is not a synonym of arrhythmic death.

LM: SD is a very important topic and its prevention may influence in a significant way the survival of patients with HF.

VM: We will analyze the CIBIS-III study, which I think is very interesting regarding this issue. In this study, for 6 months patients were treated only with BB, others with ACEI, and then all the patients were treated with a combination of these 2 drugs. Of course, the combination was superior in comparison to each isolated drug. The analysis of the end points, which were death plus hospitalizations by acute exacerbation of HF, did not show a difference between the groups. Nevertheless, at the beginning of the treatment with BB, fewer patients died. On the other hand, the clinical state of the patients and HF evolution were better in the ACEI group.

Taking into account the antifibrillatory properties of the BB in SD prophylaxis, we may assume that BB have been effective to prevent SD; for this reason, a greater number of sick people reached the time of the combination of ACEIs with BB. Beware, this may lead to the conclusion that it would be better to start the treatment for HF with BB, but the investigators from the CIBIS-III did not have as their objective to show the abovementioned hypothesis; for this reason, we cannot draw this conclusion. The purpose of CIBIS-III was to show that the treatment of HF may not necessarily be started with ACEI, but rather with BB, and then combine these drugs. And after a 6-month treatment, the mortality curves split in favor of BB and the clinical state curves in favor of ACEI. That is to say, we may observe a discrepancy between the evolution of the disease, functional class and risk of death. And considering that the patients treated with BB, were hospitalized more frequently and the evolution of their disease was worse, it is not very likely that BB may prevent deaths by HF decompensation.

LM: Of course it is a dilemma: when quality of life worsens, survival improves.

VM: Yes, I completely agree.

LM: And the next question, when is an ICD implantation indicated in patients with HF?

VM: Well, this is a tough question, both for Russia and the patients. Without taking into consideration the economic causes, all the patients with EF lower than 30% are candidates for ICD implantation, regardless of their presenting ventricular arrhythmias or not in Holter. Many years age, we already studied this topic and I'm sure that if we put these patients in Holter monitoring for 2, 3 or 4 days, we will find complex ventricular arrhythmias in 100% of the cases. For this reason, all the patients with EF lower than 30% should have an ICD implanted. The other question is the realization of this indication in real life.

LM: Perhaps we will deal with this topic. Now, the question with a pretty evident answer, may the ICD implantation be associated with resynchronization therapy?

VM: This is the most important question. When we implant an ICD, we save our patient from the risk of arrhythmic death. Nevertheless, it is known that right ventricular pacing may worsen HF evolution. This is the same dilemma we discussed in relation to the pharmacological treatment of HF: the risk of SD is decreased at the expense of a worse evolution of HF. In my opinion, right ventricular pacing is a “good” chance to worsen any patient with HF. In the case of a patient without HF, one may place the wire in the right ventricle without any fear. On the contrary, the patient with HF, nearly for certain will have hemodynamic decompensation within 6 months after having implanted the wire in the right ventricle.

LM: You just mentioned a very important topic. Not so long ago, we discussed some cases of children with pacemakers, who developed HF in some years due to unclear causes (in spite of having the prior ventricular function maintained). I think that the cause of HF in these cases is pretty clear.

VM: Yes, it is clear. When we place the electrode in the right ventricle, we cause a left bundle branch block, which alters interventricular synchronization and results in HF decompensation. On the other hand, LBBB with desynchronization of cardiac activity is the indication for resynchronization therapy. The conclusion is that if the patient with HF requires a pacemaker, we have to put the resynchronization system. For this reason, in USA there is a term, “upgrade,” which means to implant an additional electrode (for resynchronization) in a patient with symptomatic HF.

LM: So a young patient with pacemaker indication is a potential candidate for resynchronization, since the common pacemaker operates as a desynchronizer.

VM: Of course, and the CARE-HF study revealed a decrease in mortality with resynchronization. On the other hand, in the MADIT-II and SCD-HeFT, where the patients received only ICD, SD risk decreased, but risk of progressive HF did not decrease. For this reason, I don’t think of these two devices as different things. If one decided to place a triple chamber device of resynchronization to treat HF, it would be ridiculous not to supplement it with an ICD to prevent SD. And implanting just an ICD is stimulating the development of HF, which requires resynchronization. Because of this, the CRT-D device (resynchronizer plus defibrillator) solves the two aspects of the problem, what was proven in the COMPANION study.

LM: Eventually, the patient with HF and pacemaker arrives anyway to the indication of such device.

VM: Exactly. And in order not to face later the dilemma “better clinically but with risk of SD,” undoubtedly the two devices should be implanted. Currently, this topic is not even discussed any more.

LM: This is an important moment, since the opinions of arrhythmologists in this regard are different.

VM: I would like to emphasize that the indication of resynchronization and ICD should be made by different groups of specialists. These should be electrophysiologists, arrhythmologists and clinical cardiologists. Last year, we spoke about this topic in the society of cardiology, where we discussed with specialists in arrhythmias. They were even offended, saying that I'm outside my field, and that I argue about a non-pharmacological treatment of HF. This year we could reach an agreement and decided to consider the opinions of different specialists. Also, we could convince electrophysiologists that the ICD and the resynchronization are not two different methods. I have no doubt about this, and I can discuss it with anyone.

LM: And the last question, in which cases heart transplantation is indicated in HF?

VM: Personally, I would indicate transplantation in young patients with dilated cardiomyopathy (it was previously called Abramov-Fidler myocarditis with the transition into dilated cardiomyopathy). Young patients with rapid dilatation and EF lower than 20% are usually resistant to medical treatment. What can be done in these cases? We determined in these patients, a dosage of antibodies for beta-receptors, and if it results to be high, we try to perform immunopheresis, which helps only in a few cases. Jointly with the Institute of Cardiovascular Surgery, we place a cardiac net (ACCORN) to prevent remodeling. This is a chance, most of all, if the impairment is not too advanced. The heart is maintained with dilatation and with low EF, but HF does not decompensate, and irreversible changes do not occur in target organs. This method is effective in some cases. The attempt of plastic surgery of the mitral valve with ring reduction, in general, is merely palliative and does not yield good results. The artificial heart is another method to compensate the patient, and it serves as a bridge for heart transplantation. I think that a young patient should be in the waiting list and, meanwhile everything possible should be done. The patients with pulmonary hypertension are candidates for cardiopulmonary block transplantation, which is a much more complex method. And we should not forget that with the transplantation the problem is not completely solved. High costs of immunosuppressive treatment arise, as well as coronary heart disease of the transplanted heart, which requires repetitive cineangiographies and coronary angioplasties, etc.

The problems of the transplanted heart are another subject. In Russia, in general, very few transplantations are made. Devices such the Jarvic or HeartMate artificial hearts should be developed, which may be the future solution for patients with terminal HF.

I have been working in a specialized center in heart failure for 30 years, and during these years the number of patients that I would send for transplantation has decreased. In 1976, when the main drugs were diuretics, digitalis and nitrates, we managed to compensate a much lower number of patients. In our first publications about refractory HF in 1978, we already mentioned BB, ultrafiltration and then ACEIs. With the more effective treatments of AMI, valvular heart diseases,

HBP, the number of patients for heart transplantation has significantly decreased. I repeat, the single problem that could not be solved is young people with rapidly progressive dilated cardiomyopathy. For them, transplantation is the only solution.

LM: Thank you very much, Dr. Mareev. We have discussed the scheduled questions. Maybe you consider there are some other current topics worth mentioning today.

VM: I consider we have already mentioned the main topics. I would like to add the subject of the multidisciplinary approach on HF. According to statistical data, annual mortality of patients with HF treated by family doctors, clinicians, and cardiologists was 12, 11 and 8% respectively. If we analyzed the results between patients treated by a specialist in HF, mortality would be much lower. This year the IIIAHC study ended, which was conducted in 30 Russian cities. The 745 patients included were receiving optimal medical treatment (94% ACEI, 70% BB, 60% aldosterone antagonists and 45% digitalis). The patients were split into 2 groups. The patients of the first group, after having achieved compensation, were given the usual advice for treatment. With the patients of the second group, 4 talks were organized, where symptoms, and principles and objectives of the treatment were discussed, self-control methods were explained, and phone communication was installed between the physician and the patient weekly during the first month and later, once each 2 weeks for a year. The patient made the phone call or he/she received it. Thus, in the study the teaching and the control of the patient were combined regularly. The results within a year showed the decrease of hospitalizations in 47%, and mortality decreased too (8% of the active follow-up group vs. 13% in the control group). The absolute decrease of mortality was significant, 5% ($p=0.044$). Such strategy turned out to be economically very convenient, since hospitalization costs decreased at the expense of 1 cardiologist every 30 patients. In USA this follow-up method is already being implemented, which results economically very profitable. In my opinion, all the patients with HF that receive modern treatments should be under the control of a specialist. And in each clinic there should be a specialist in HF and also, nursing staff and a cardiologist trained in HF. The IIIAHC study revealed the importance of follow up and the education of the patient with HF. This year in Moscow, specialized courses are being organized for clinicians, where the topic of practical control of HF patients will be dealt with. The Web page of our association contains all the necessary information for patients with HF: how to control body weight, diuresis, how to take medications, how to travel, go on holidays, live their sexual life, and so on.

LM: Thank you very much Dr. Mareev. I'm certain that this interview will be very useful for our physicians.