



# ECHOCARDIOGRAPHIC MONITORING OF PATIENTS WITH HEART FAILURE

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## ABSTRACT

Twenty seven patients diagnosed as having systolic heart failure with Ejection Fraction (EF) of less than 40% by echocardiography were monitored over a period of 3 years. The monitored parameters included clinical symptoms, diastolic dysfunction, therapies and survival during three years of treatment.

The results indicate a beneficial effect of treatment with high doses of ACE-inhibitors, spironolactone and beta-blockers in improving clinical symptoms and diastolic function. The survival rate was similar to that in the developed European countries.

KEY WORDS: echocardiogram, systolic dysfunction, diastolic dysfunction, mortality and survival rate

## INTRODUCTION

Heart failure (HF) is a complex clinical syndrome that can result from any structural or functional cardiac disorder that impairs the ability of the ventricle to fill with or eject blood. The cardinal manifestations of HF are dyspnoea and fatigue, which may limit exercise tolerance, and fluid retention that may lead further to pulmonary and peripheral oedema. Both abnormalities may impair the functional capacity and quality of life of the affected individuals, but they do not necessarily have to dominate the clinical picture at the same time (1).

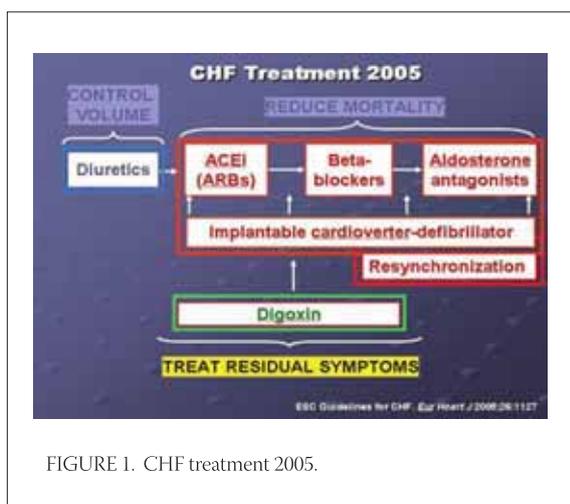


FIGURE 1. CHF treatment 2005.

HF is a growing and increasingly important chronic disease in the Western World, occurring in at least 2% of the adult population and rising to 3% in those aged over 75 years (2). It is characterized by inadequate systemic perfusion due to impairment of the cardiac pump function. Clinical heart failure is progressive condition, typically with high morbidity and mortality rates. It therefore places a significant burden on healthcare resources. One of the key elements in reducing the mortality, morbidity and costs of heart failure is accurate and early diagnosis of left ventricular systolic dysfunction (LVSD) (3). This is essential for successfully addressing underlying diseases or causes and in selection of appropriate therapies (Figure 1). According to the recently released American College of Cardiology (ACC) / American Heart Association (AHA) guidelines for the diagnosis and management of heart failure, transthoracic echocardiography is the single, most useful diagnostic test in the evaluation of patients with heart failure (4). As therapeutic techniques such as cardiac resynchronization therapy (CRT) gain clinical acceptance, new echocardiographic imaging modalities such as

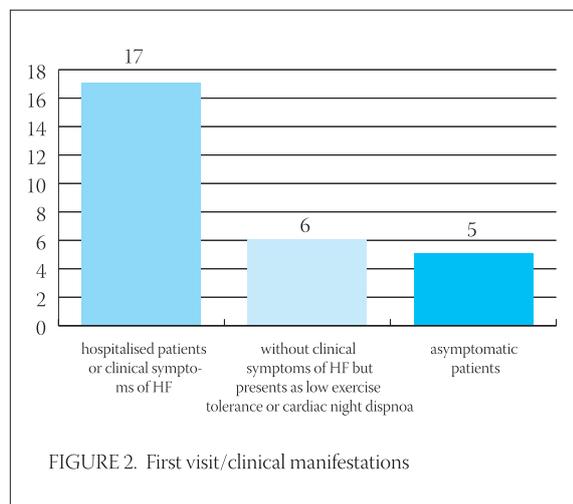
live 3D-imaging and specialized analysis tools for dynamically assessing regional volumes and differences in timing of contraction promise to play an expanding role in the management of heart failure. The common cause of heart failure is left ventricular, systolic dysfunction (approximately 60% of patients). In this category, most cases are the result of end-stage coronary artery disease (CAD) either with a history of myocardial infarction (MI) or chronically underperfused yet viable myocardium or the combination of the two (5). Other disease processes that can lead to heart failure include valvular heart disease, congenital heart disease, diabetes, hypertensive disease and idiopathic and toxic (e.g. alcohol-induced) cardiomyopathies. Less common causes include viral infections of the heart muscle, thyroid disorders, disorders of the heart rhythm, or combinations of those. Heart failure associated with LVSD is characterized by progressive structural change in the left ventricle known as remodeling. While the disease progresses, myocytes hypertrophy and elongation gives rise to left ventricle-dilatation and hypertrophy. In this situation, stroke volume is increased without an actual increase in ejection fraction (EF). This results in increased wall tension, impaired subendocardial myocardial perfusion and may provoke ischemia. While this dilatation progresses, separation of the valve flaps can lead to mitral and tricuspid regurgitation. This may further diminish the cardiac output and increase end-systolic volumes and ventricular wall stress, therefore leading to further dilatation, pulmonary congestion and myocardial dysfunction. Left ventricular volume and ejection fraction are therefore important indicators for morbidity and mortality in heart-failure patients (6). Almost always diastolic dysfunction persists with systolic dysfunction. But, isolated diastolic dysfunction is also possible. Diastolic dysfunction is present when elevated filling pressure is necessary to achieve normal ventricular filling (7). The changes in cardiovascular system that naturally accompany aging tend to affect the diastolic function of the left ventricle rather than the systolic function (8). It is the same with therapy-monitoring of patients with heart failure. Echocardiogram is used to determine systolic function, ventricle size and diastolic filling characteristics (9). Two-dimensional Doppler echocardiography is a reliable and reproducible method for diagnosis and longitudinal follow-up of left ventricular diastolic dysfunction (10). The Nishimura & Tajik grading of diastolic dysfunction is based on clinical and echocardiographic criteria (10) as outlined below:

**Grade 0:** Normal (mitral inflow velocity curve with an E/A ratio greater than 1,0 and a deceleration time of approximately 200 milliseconds);  
**Grade I:** Abnormal relaxation pattern on Doppler echocardiogram; patient develops symptoms on moderate exertion or with onset of atrial fibrillation;  
**Grade II:** Pseudo normalization pattern of the mitral flow velocity and increased filling pressure at rest, producing symptoms with mild to moderate exertion;  
**Grade III:** Restrictive filling pattern on mitral flow velocity curves and severe increase in filling pressures and symptoms at rest or on minimal exertion. Patients respond to diuretic therapy, and their condition improves to grade I or II;  
**Grade IV:** Irreversible grade III changes: patients maintain a severe restrictive pattern despite aggressive diuretic therapy: poor prognosis.

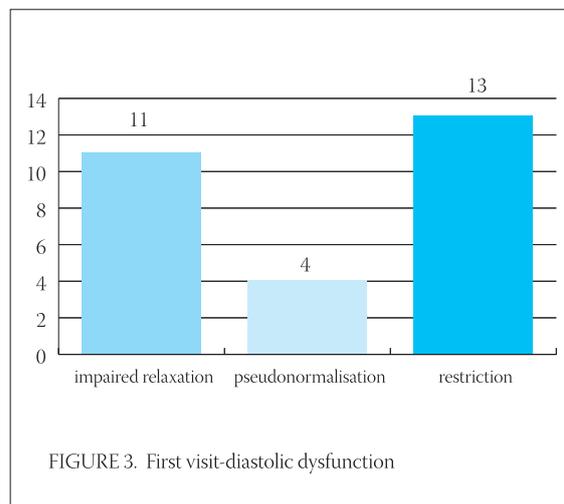
Right and left-sided heart catheterization is the standard method for direct measurement of left ventricular filling pressure and rate of left ventricular relaxation but is not practical for routine use. Typically, the left ventricular end-diastolic filling pressure is elevated in the presence of normal or reduced left ventricular end-diastolic volume (11). Cardiac catheterization is also useful in distinguishing from constrictive pericarditis and in confirming left ventricular diastolic dysfunction. Magnetic resonance imaging is used to exclude pericardial disease (12).

**PATIENTS AND METHODS**

Twenty eight patients were monitored over a three year period. All of them had dilative cardiomyopathy with ejection fraction under 40 % verified by echocardiogram examination. Eleven of them were diabetics and seventeen non-diabetics.

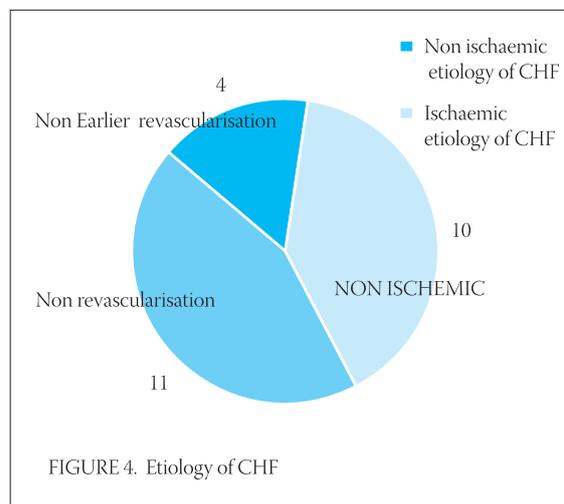


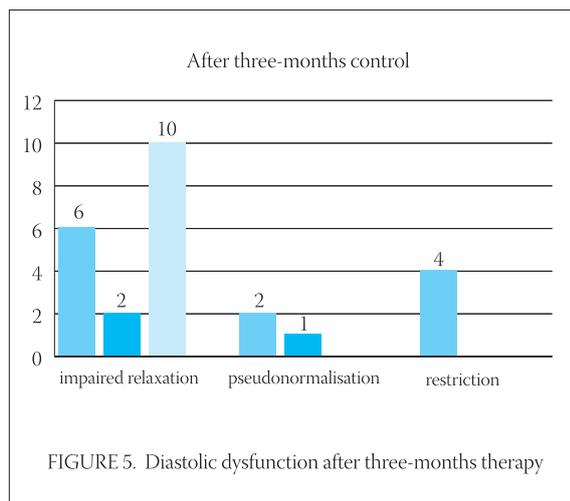
On initial assessment, seventeen patients seen either as outpatients or inpatients had clinical and subjective symptoms of heart decompensation (Figure 2). Further six patients exhibited no stasis in the lungs nor the oedema or hepatomegaly, but showed evidently low exercise tolerance or occasional night dysпноа. Remaining five patients were almost asymptomatic.



All patients had ejection fraction under 40%, diastolic diameter of left ventricle 5,9 - 7,9 centimetres. Diastolic function (Figure 3.) is more important for symptoms and observing patients. In eleven patients mild dysfunction represented as impaired relaxation was found at the first visit. Four of them had moderate diastolic dysfunction - pseudonormalization, and thirteen had severe diastolic dysfunction - restrictive hemodynamics in diastole. During the first period of follow-up three patients were excluded, so, in further findings we monitored twenty five patients.

Our therapeutic goals included following:  
 - In those with acute heart decompensation we ad-



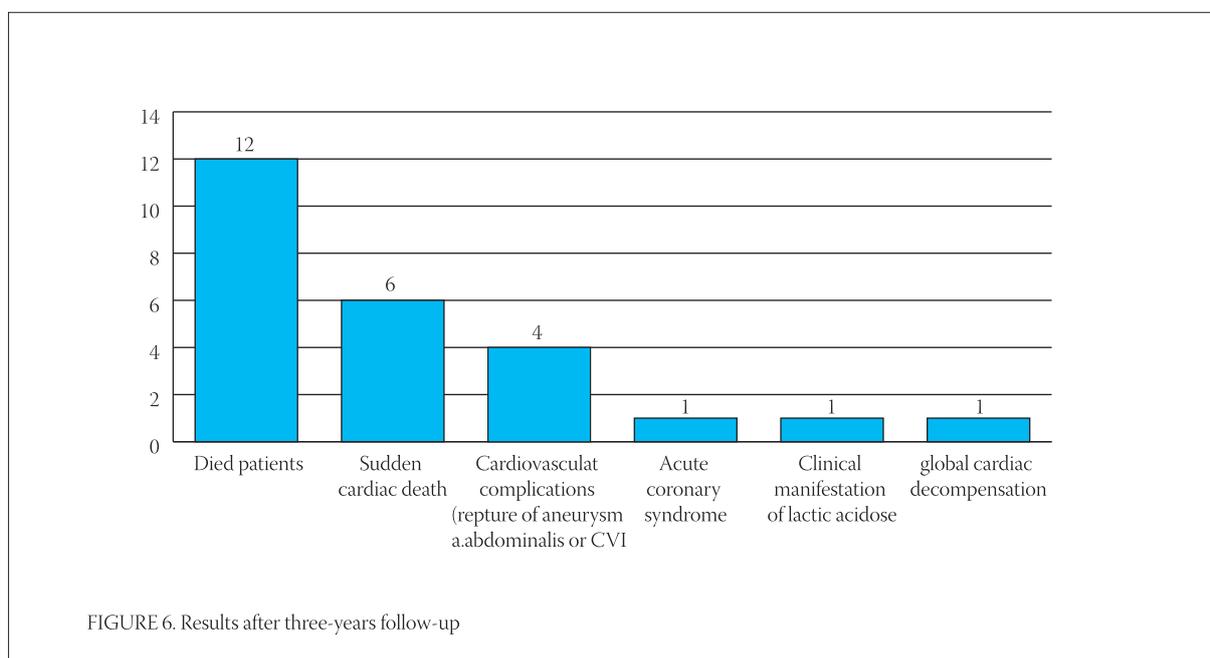


ministrated diuretics to decrease volume-overload (both the intra end extra vascular fluid). After the recompensation, we wanted to check up the etiology of dilatative cardiomyopathy. Exploring etiology of dilatative cardiomyopathy, catheterization and coronarography was done and proved ischemic origin in fifteen patients and in others ishaemia was not proved with accuracy. In the ischemic group, four patients had earlier coronary by-pass revascularization (Figure 4.). - To achieve lower after-load we administrated ACE- inhibitors in maximal tolerated dose. This therapy decreased peripheral resistance and the tissue RAAS-activity was gradually suppressed (target dosage was trandolapril 4 mg, ramipril 10-20 mg, enalapril 40- 60 mg, lisinopril 40 mg, fosinopril 40 mg, cilazapril 5-10 mg). This approach is important to reduce central thirst-sensation that made the later treatment easier. In patients that did not

tolerate ACE-inhibitors (in most patients because of cough), AT 1-receptor inhibitors were necessarily administrated, again with maximal tolerated dosage. - Further, we tried to optimize ventricular frequency (where possible under 75/min and in the sinus- rhythm even in the atrial fibrillation). In the beginning we administrated Amiodarone, which has no negative inotropic effect, for maximum of one month, and after that beta- blockers (or Ca-antagonists). Where that therapy was unsuccessful in lowering frequency, digoxin was added because of the stimulative effect on vagus-activity. Every patient was treated with spironolactone 25 mg, which is, according to the most authors, sufficient cardiac dose, because of final suppression of RAAS and reduction of myocardial fibrosis. The issue of ASA (acetylsalicylic acid) remains opened. Every patient with atrial fibrillation was on anticoagulant therapy with monitoring of INR.

## RESULTS AND DISCUSSION

Results after three months-therapy showed that three patients were rehospitalized because of repeated heart decompensation. They were diabetics and possibly non-cooperative patients, so in later processing only twenty five patients were monitored, (one patient from each group with diastolic dysfunction was excluded). Among twenty five further monitored patients, mild reduction of diastolic LV diameter was observed in twelve patients, ejection was same or minimally better. Considering diastolic dysfunction, monitoring of influence of mitral regurgitation and lung pressure to diastol-



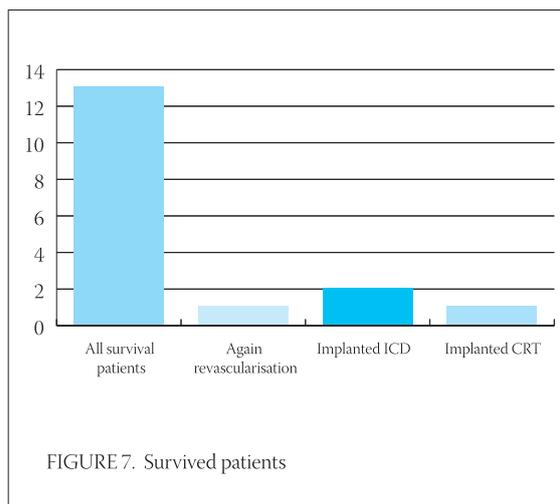
ic function and a course of treatment would be desirable but would also extend beyond the scope of the study.

Diastolic dysfunction after three-month therapy (Figure 5.) showed satisfactory results. In the group with mild diastolic dysfunction we had eighteen patients, among them two patients were earlier in the group of pseudo-normalisation and six patients were earlier presented in the group of severe diastolic restriction. There were 3 patients in the group of pseudonormalisation, two of whom were earlier in the group of restriction and one stayed in the same group. The last four patients were presented continually in severe diastolic restriction (grade IV). We want to emphasize that all were clinically compensated.

Criteria for good prognostic parameters, according to our findings, were as follows:

- higher ejection fraction-over 30%;
- satisfactory turn of diastolic function;
- not registered pairs of VES or short VT on the Holter-monitoring;
- toleration of high doses of ACE-inhibitors as well as beta-blockers;
- preserved renal function;
- non-diabetics.

The after-three-years follow up (Figure 6.) showed that twelve patients died. Five patients probably died due to electrical death (sudden cardiac death), four died due to other cardiovascular complications (the rupture of aortic aneurysm or CVI), one from acute coronary syndrome, one under the state of lactic acidosis (this patient was diabetics, using metformin), only one died with the symptoms of lung oedema. Thirteen surviving patients were clinically compensated (Figure 7.). One of them was revascularised again. Two



patients had ICD, one is waiting for transplantation and one has CRT and he is on the waiting list for a heart transplantation too (it's planned to be realized in Milan). All surviving patients use and well tolerate high doses of ACE-inhibitors. Also, they tolerate well high doses of beta-blockers, spironolactone 25 mg, as low as possible doses of aggressive diuretics of Henley loop (only in cases of significant tricuspid regurgitation). Patients with non-ischemic cardiomyopathy do not use Aspirin. All patients with atrial fibrillation use anticoagulants in therapy with monitoring INR (therapeutic range 2-3) and in cases of clear ischemic CMP with atrial fibrillation, combined with Aspirin. Neither of them, according to the given instructions, consumes more than 1, 3 l of fluid daily. Patients control their weight regularly and every change in body mass that exceeds 3 kg, is an alarm for control examination. Dilative cardiomyopathy is an increasing problem both in our country and the rest of the world. It is obvious that patients with good compliance die unexpectedly due to electrical death (there is no possibility of implanting ICD to all patients with ventricular disturbance of rhythm). Patients with a low compliance develop attacks of dyspnea and lung edema, and die as a result of heart failure literally.

## CONCLUSION

The observation and treatment has to be based on new information in cardiology. Physicians should not be afraid to administer high doses of ACE-inhibitors in low EF, or to increase the dose of beta-blockers to a maximal dose that patient tolerates, either. Nitro drugs are only symptomatic drugs and the first which may be left out, in a long term therapy.

*List of Abbreviations*

EF	-	ejection fraction
ACE	-	angiotensin-converting enzyme
HF	-	heart failure
AHA	-	American Heart Association
ACC	-	American College of Cardiology
MI	-	myocardial infarction
RAAS	-	renin-angiotensin-aldosterone-system
CVI	-	cerebral vascular incident
ICD	-	implantable cardioverter defibrillator
CRT	-	cardiac resynchronisation therapy
LVSD	-	left ventricular systolic dimension

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