MAEDICA – a Journal of Clinical Medicine
2025; 20(4): 667-668
https://doi.org/10.26574/maedica.2025.20.4.667

MÆDICA - a Journal of Clinical Medicine
EDITORIAL POINT OF VIEW

Systolic, Diastolic. Is Anyone Worse?

Mircea CINTEZA, MD, PhD[a, b]

[a]Department of Cardiology, Emergency University Hospital, Bucharest, Romania
[b]"Carol Davila" University of Medicine and Pharmacy, Bucharest, Romania

We are in the field of heart failure (HF), of course. Chronic HF.

Both actual main guidelines (European, American) date from about 2021 (1, 2). And in both, the classification of HF includes the main forms, but with another name, and also an intermediate form. This classification incudes:

- HF with reduced ejection fraction (EF) [40% or less EF of the left ventricle (LV)] (HFrEF);
- HF with preserved ejection fraction (HFpEF) (50% or more EF of the LV); and
- HF with moderate reduced ejection fraction (41-49% EF of the LV) (HFmrEF).

The problem is: which of these forms is worse? Can we make any of them consistently better?

When speaking with my patients, I tell them to begin with the conclusion – which is their actual problem. Allow me, please, to do the same with this medical problem.

Chronic HF, also known as congestive heart failure (CHF), is a syndrome caused by impairment in the heart's ability to fill with and pump blood (Wikipedia 2025).

It is as simple as that: a syndrome, not an illness; due to the heart; which loses the ability to fill and pump blood.

STOP! Here begins the problem. The ability to pump? Or to fill? Or both? In what degree and combination?

Some questions are flying around:

- if it is a syndrome, are there particular pathologies associated with one form or another?
- is therapy different or is some therapy common?
- is the prognosis different in the pure forms?

In HFrEF, the muscle of the heart becomes thin and the cavities are generally enlarged. In HFpEF, the muscle becomes stick and cannot relax.

In HFrEF, affected people are in general younger, smoke more and have more often atherosclerotic heart disease. Etiologies like dilated cardiomyopathies of different origins, congenital heart disease or valvular heart diseases wit regurgitations are more frequent.

Patients with HFpEF are generally older and have more often hypertension, diabetes and obesity.

Here we see already important differences between the two forms.

The syndrome of HF includes two main clinical forms: left-sided HF (LHF) and right- sided HF (RHF).

In LHF, the impaired capacity of the left ventricle to pump the necessary blood produces shortness of breath mainly at effort. In RHF, the dominant signs include weight gain, abdominal bloating, leg or/and ankle swel-

Address for correspondence:
Mircea Cinteza, MD, PhD
Department of Cardiology, Emergency University Hospital, 169 Independentei Avenue, District 5, Bucharest, Romania
Email: mirceacinteza@gmail.com

Article received on the 19th of December 2025 and accepted for publication on the 20th of December 2025

ling. Generally, RHF is a consequence of LHF. So, the symptoms and signs in LHF may be initially isolated, then the symptoms of both forms coexist. Rarely, RHF may develop initially independently, like in chronic cor pulmonale. In this case, symptoms and signs of RHF appear isolated.

Regarding the physiopathology and associated pathologies, the therapy is clearly different in the pure forms.

In HFrEF, the goal is to strengthen the heart muscle and block the neurohormonal pathways, when becoming over developed and become dangerous. For treating this, beta-blockers and angiotensin converting enzymes blockers are widely used. Angiotensin receptor blockers had a similar effect, but only in some cases. Sacubitril-valsartan acts in the same direction, acting by two mechanisms.

Enhancing contractility proved to be worse than beneficial on a long term. Beta mimetics or drugs of similar action are used efficiently only in acute cases. On the long term, they exhaust the myofibrils and produce their premature death. Digoxin proved to be a weak contractility enhancer and is therapeutically used only for antiarrhythmic properties.

The therapy in HFpEF acts in a different way. Diuretic are the main drugs, making decongestion in the lungs and producing better oxygenation of the body and reducing the afterload of the left ventricle. Gliflozins, acting on a specific mechanism in the kidneys, proved also to be very efficient. But for the HFpEF the main goal of therapy is to treat the aggressive associated pathologies, hypertension, diabetes and obesity. Improving longevity is also taken into account.

HFmdEF uses therapies from both categories, according to the clinical status of the patient.

One of the main problems that arises is: which form is worse for prognosis?

Both are. Traditionally, HFrEF is considered worse, because the deterioration of heart muscle is often advanced and more dangerous. However, it responds to some therapies (ACE inhibitors, beta-blockers) better and over a long term. In the same time, HFpEF is always associated with one serious condition, which produces the main mechanism of this form of HF. Hypertension, diabetes, obesity and advanced age by itself badly influence prognosis in HFpEF.

Devices seem to influence both the way to treat efficiently some forms of HF and, for sure, influence prognosis in selected cases. Especially, they may improve prognosis in HFrEF. Cardiac resynchronization, mechanical assist devices and defibrillators are already proved. But this happens in a very small proportion of cases, in contrast with the huge total number of HF patients. Heart transplant may be judged in the same direction.

As a conclusion

Heart failure negatively influences prognosis. A summary done by a group of authors concludes that survival is 80-90% at one year, 50-60% at five years and 30% at 10 years (6). It is much better than a prior ten-year evaluation (Framingham 1971 – 60,5% death at one year and 31,5% at five years).

However, prognosis is absolutely different for each patient due to the particular clinical form and associated comorbidities. ❑

References

1. **Adamo M, Gardner RS, Baumbach A, et al.** 2021 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure" (PDF). *Eur Heart J* 2021;42:3599-3726. doi: 10.1093/eurheartj/ehab368.
2. **Heidenreich PA, Bozkurt B, Aguilar D, Allen LA, Byun JJ, Colvin MM, et al.** 2022 AHA/ACC/HFSA Guideline for the Management of Heart Failure: A Report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines". *J Am Coll Cardiol* 2022;79:e263-e421. doi: 10.1016/j.jacc.2021.12.012.
3. **Ponikowski P, Voors AA, Stefan D, et al.** 2016 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure The Task Force for the diagnosis and treatment of acute and chronic heart failure of the European Society of Cardiology (ESC) Developed with the special contribution of the Heart Failure Association (HFA) of the ESC. *Eur Heart J* 2016;37:2129-2200. doi: 10.1093/eurheartj/ehw128.
4. **Ibanescu R, Mitu DA, Goje ID, et al.** History of Heart Failure Definition. *Card Fail Rev* 2025;11:e07. doi: 10.15420/cfr.2024.22.
5. **Izraiq M, AlBalbissi K, Alawaisheh E, et al.** Comparative Analysis of Heart Failure with Preserved *Vs* Reduced Ejection Fraction: Patient Characteristics, Outcomes, Mortality Prediction, and Machine Learning Model Development in the JoHFR. *Int J Gen Med* 2024;17:3083-3091. doi: 10.2147/IJGM.S465388.
6. **Jones NR, Hobbs FDR, Taylor CJ.** Prognosis following a diagnosis of heart failure and the role of primary care: a review of the literature. *BJGP Open* 2017;1:bjgpopen17X101013. doi: 10.3399/bjgpopen17X101013.