# Volume assessment in heart failure - a review

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## ABSTRACT

Heart failure represents a significant medical and economic burden and is also an important cause of mortality and morbidity. The advances in treating coronary artery disease mainly pertaining to modern invasive cardiology significantly reduced the mortality from coronary atherosclerosis, but on the other side caused an increase in incidence and prevalence of congestive heart failure. Heart failure is a disease of the elderly. In that population the incidence is 10 in 1000 patients older than 65 years and has a high five-year mortality despite all modern treatment modalities. (1)

*Key words: heart failure, physical examination, congestion*

## INTRODUCTION

Heart failure is a clinical syndrome in which a clinical evaluation of signs and symptoms represents a cornerstone in making the diagnosis, guiding of treatment and estimating a patient's prognosis. Determining left ventricular filling pressures by using non-invasive or invasive techniques is the most important element in choosing the best treatment option. In that aspect, the degree of congestion becomes the key element in the overall clinical picture that decides on the need for hospitalization.

## DISCUSSION

Congestive heart failure is in essence a problem of congestion and volume overload. The large number of patients suffering from this syndrome dictates the need for effective methods of detecting and treating congestion to be found. Heart failure patients are often rehospitalized, mirroring the still insufficient tools for volume status assessment in that specific population. Evaluation of congestion remains a challenge. Every patient is unique in his or her body habitus and comorbidities, which makes the clinical evaluation of congestion difficult and expresses the need for using various methods. (2) The lack of a singular clinical tool should stimulate the emergence of novel methods pertaining to implantable devices which can give an accurate measurement of volume load over time. Today certain types of resynchronization pacemakers can give precise measurement of volume status using transthoracic impedance. It is reasonable to expect that these devices will become the cornerstone of treatment decision making in the future.

Heart failure is mainly a clinical diagnosis and no single diagnostic test can confirm its presence or absence. The most frequent clinical findings are related to decreased exercise tolerance or fluid retention. In that respect the New York Heart Association (NYHA) classification of heart failure is widely used in clinical practice and in clinical studies to quantify the degree of dyspnoea and of heart failure. Dyspnoea, the main symptom of left ventricular failure, may manifest with progressively increasing severity as exertional dyspnoea, orthopnoea, paroxysmal nocturnal dyspnoea, dyspnoea at rest or acute pulmonary oedema. Common extracardiac signs and symptoms of heart failure include anorexia, nausea, weight loss, abdominal distension, fatigue, weakness, oliguria, nocturia, and cerebral symptoms ranging from anxiety to memory impairment and confusion. Orthopnea is an early symptom of heart failure and may be defined as dyspnea that develops in the supine position and is relieved with elevation of upper body. Blood is displaced from the extrathoracic compartment to the thoracic vasculature, and the failing left ventricle, operating on the flat portion of the Frank-Starling curve, cannot accept and pump out the extra volume of blood delivered to it. As a result, pulmonary capillary pressure rises, causing interstitial pulmonary oedema, reduced pulmonary compliance, increased airway resistance, and dyspnoea. In advanced LV failure, orthopnoea may be so severe that the patient cannot lie down and must sleep sitting up. Cough, particularly during supine position, may be an orthopnoea variant. It can be caused by pulmonary congestion and is relieved by the treatment of heart failure. Paroxysmal nocturnal dyspnoea occurs at night and is defined as the sudden awakening with a feeling of severe anxiety and air shortage. The patient must sit up in bed to catch some air. Bronchospasm is often present increasing respiratory work. On lung auscultation, the bronchospasm of heart failure can be difficult to distinguish from an acute asthma exacerbation, although both types of bronchospasm can be present in a single patient. In contrast to orthopnoea, which may be relieved by immediately sitting up in bed, paroxysmal nocturnal dyspnoea may require 30 minutes or longer in this position for relief. Dyspnoea at rest in heart failure is the result of decreased pulmonary function secondary to increased airway resistance, increased pulmonary capillary wedge pressure (PCWP) and respiratory muscle strength decrease. Acute pulmonary oedema is defined as the

sudden increase in PCWP (usually more than 25 mm Hg) as a result of acute and fulminant left ventricular failure. It is a medical emergency with a dramatic clinical presentation. The patient is tachypnoeic, tachycardic, hypoxic, and coughing, with an increased work of breathing and with using accessory muscles for breathing while expectorating foamy, blood tinged sputum.

In addition to previously mentioned symptoms and their correlation to heart failure, the physical examination and evaluation of signs of the disease are a crucial element of assessing the presence and the degree of congestion. Unfortunately it has significant limitations. The assessment of fluid load by physical examination consists of several components: distention of jugular veins, hepatojugular reflux, auscultation of lung fields and peripheral oedema. Jugular venous pressure is estimated from the angle of the sternum to the top of the column of visible distension, with elevations >2 to 3 cm being abnormal. Hepatojugular reflux is appreciated with palpation of the liver and adds to the sensitivity of examination of the jugular veins. When clinically noted, the presence of jugular venous distention is highly sensitive for elevated pulmonary capillary wedge pressure (PCWP). (3) Oedema is an important finding, usually symmetric, beginning with the feet and ascending to the abdomen. Unfortunately it is not specific and depends on gravity, therefore on the position of the body. Many extracardiac diseases like liver, kidneys, or vascular system and obesity may cause the oedema as well. In such cases, using the presence of oedema alone without other signs of volume excess can lead to inaccuracies in patient assessment. Rales upon lung auscultation, when present, are an obvious indicator of hypervolemia. However, they may be absent even in the case of obvious worsening of heart failure. Rales can be absent in the majority of patients with systolic HF because of increased lymphatic drainage and chronic perivascular compensation. In conclusion, symptoms and signs do not answer the question completely and accurately. Large studies dealing with heart failure found that dyspnoea as a symptom was present in approximately 90% of patients, and peripheral oedema in only 2/3 of patients roughly. Some observational studies demonstrated however, that half of the patients were actually asymptomatic. Consequently, the biggest problem was residual congestion at discharge as outlined in EURObservational Research Programme Heart Failure PILOT. Almost one quarter of patients had peripheral and pulmonary congestion at discharge and these patients do die more often according to ACUTE HF trial. Can we do better in assessing and treating congestion?

The simplest way for evaluating fluid status is to monitor weight changes. This can be done in and out of hospital, at home by the patient or by health care professionals. There are also methods of weight monitoring that utilize the remote monitoring system either with implantable or non-implantable monitoring devices. There is substantial evidence to support this method, showing improvements in mortality rates. The European trial (The Trans-European Network Homecare Monitoring Study) demonstrated that patients randomized to home telemonitoring of weight, blood pressure, heart rate and rhythm had lower mortality rates than usual care patients. In contrast, weight measurements can lead to inaccurate fluid assessment due to variations of percentage of body fat. In terminal heart failure patients, cardiac cachexia and oedema impede the evaluation of fluid status because there may be stable weight with worsening symptoms and fluid overload.

Apart from clinical examination, there are other non-invasive and invasive techniques at our disposal. Firstly, there are biomarkers. The laboratory measurement of brain natriuretic peptide (BNP) was found to correlate with the severity of heart failure and there were grand expectations in using BNP to monitor fluid status. In reality, BNP values have been shown to decrease with the use of beta-blockers and no study has shown consistent and statistically significant decreases in wedge pressure with decreased BNP levels. Improved BNP does however correlate with improved NYHA functional status and serial measurements do have a role in chronic heart failure management but, as with other physical examinations and symptoms, it is not a sufficient and stand-alone parameter. Therefore, biomarkers are an adjunct in making the diagnosis, specifically when evaluating dyspnoea and ruling out other differential causes apart from heart failure. Still, caution is advised. There is a grey zone of prognostic uncertainty in the spectrum of laboratory values of BNP between 100 and 400pg/nml. Substantiating that there is the REDHOT study in which physicians who had to assess the degree of heart failure were blinded to the BNP levels. Based solely on clinical assessment, the group of patients who were discharged as having only mild heart failure had in fact 22% higher BNP levels than the group declared more significantly decompensated. One must also bear in mind that BNP levels are proportional to body mass index (BMI), so in patients above 35 BMI, the cut off value of BNP should be doubled. BNP levels on the other hand, correlate with invasively acquired PCWP. Therefore, serial measurements of BNP during hospitalization have a role of assessing the patient's readiness to be discharged. In that spectrum, the pre-discharge value may be the most important as the majority of heart failure patients will eventually be readmitted, and it was found that an elevated pre-discharge BNP value has an increased likelihood of rehospitalization and/or death. (4)

Echocardiography, as a ubiquitous method in cardiology, can provide important information on the fluid status in heart failure. Estimated right ventricular or pulmonary artery pressures may be used to estimate filling pressures and mitral inflow Doppler imaging provides information about diastolic function. The most often used echocardiographic measurements relate to parameters regarding the inferior vena cava diameter and left ventricle diastolic diameter. In that aspect there are static measurements (IVC diameter, central venous pressure and end-diastolic left ventricle diameter) which have no value in heart failure patients whatsoever. The mainstay of assessment remains with dynamic parameters: IVC distensibility index, delta VTI or Vmax in left ventricular outflow tract following passive leg raise maneuver. These parameters may be used to help answer the question of fluid responsiveness or fluid overload, but also have significant limitations regarding spontaneous breathing or mechanical ventilation and the existence of pre-existent cardiac disease. In these circumstances the echo-values acquired will be inaccurate, so the clinician should be aware of those limitations.

Radiographic findings of CHF may be present during worsening episodes. The typical findings of perivascular congestion, Kerley B lines, pleural effusions, and fulminant oedema are definitive in their diagnosis of congestion. Their absence, however, is not a reliable method for assessing volume status. Even in patients with a markedly elevated PCWP, radiographic

evidence of congestion may be absent in almost half of patients.

The pulmonary artery catheter (PAC), introduced in 1970, enabled accurate hemodynamic measurements of the right and left heart beat to beat. Despite that, today right heart catheterisationhas a questionable role in the intensive management of heart failure patients. A metaanalysis of multiple randomized controlled trials did not demonstrate a superiority of PAC regarding mortality. (4) The 2008 European Society of Cardiology guidelines for the diagnosis and treatment of acute and chronic heart failure recommended PAC with a class IIa recommendation (level of evidence C) in haemodynamically unstable patients who are not responding as expected to conventional treatment. PAC placement should be considered in distinguishing cardiogenic from non-cardiogenic pulmonary oedema in complex patients with concurrent cardiac and pulmonary disease, especially when echo-measurements are difficult to obtain or when levels of BNP are inconclusive. (6) The PAC remains the principal means of assessing pulmonary vascular resistance and reactivity in patients undergoing evaluation for cardiac transplantation and occasionally for placement of a left ventricular assist device. In patients with suspected pulmonary hypertension, right-heart catheterisation is required to confirm the presence of PH, establish the specific diagnosis, and determine the severity of PH. In addition, in patients with suspected PH right-heart catheterisation is required to guide the therapy. The insertion of a PAC for the diagnosis of acute heart failure is usually unnecessary. In myocardial infarction complicated by cardiogenic shock or progressive hypotension, right heart catheterisation was considered as a class I indication in previous guidelines. However, in view of the recent data concerning PAC use, such a recommendation is no longer included in the recently published guidelines on myocardial infarction. (7)

## CONCLUSION

HF patients are complex to treat, and a proper assessment of fluid status by physical examination alone can be difficult and misleading, especially in the population of obese patients and patients with chronic lung disease. Physical examination remains the primary tool of assessment in heart failure. Daily weight measurements serve as a useful outpatient management tool. Novel methods for home monitoring of volume status emerge to complement the physical examination and allow us to be aware of the problem before it becomes manifest and therefore prevent hospitalization. The management of volume status in heart failure patients is crucial. Significant congestion at discharge is something we must try to reduce as it represents a poor prognostic factor. The goal in the future lies in embracing novel technologies in order to treat patients in a way that protects the quality of life and extends it in an optimal way.

## REFERENCES


1. Ivanuša M, Kralj V. Epidemiologija zatajivanja srca u Republici Hrvatskoj. Medix. 2014.;112:76-82
2. Stevenson LW, Perloff JK. The limited reli¬ability of physical signs for estimating hemo¬dynamics in chronic heart failure. JAMA. 1989;261:884–888.
3. Butman SM, Ewy GA, Standen JR, et al. Bedside cardiovascular examination in patients with severe chronic heart failure: importance of rest or inducible jugular venous distention. J Am Coll Cardiol. 1993;22:968–974
4. Lee SC, Stevens TL, Sandberg SM, et al. The potential of brain natriuretic peptide as a bio¬marker for New York Heart Association class during the outpatient treatment of heart failure. J Card Fail. 2002;8:149–154.
5. Richard C, Warszawski J, Anguel N. Early use of the pulmonary artery catheter and outcomes in patients with shock and acute respiratory distress syndrome: a randomized controlled trial. JAMA. 2003;290(20):2713–2720
6. Binanay C, Califf RM, Hasselblad V, et al; ESCAPE Investigators and ESCAPE Study Coordinators. Evaluation study of congestive heart failure and pulmonary artery catheteriza¬tion effectiveness: the ESCAPE trial. JAMA. 2005;294:1625–1633.
7. Shah MR, Hasselblad V, Stevenson LW, et al. Impact of the pulmonary artery catheter in criti¬cally ill patients: meta-analysis of randomized clinical trials. JAMA. 2005;294:1664–1670