Clinical Update

Hemodynamic Assessment in Heart Failure: Role of Physical Examination and Noninvasive Methods

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Abstract

Among the cardiovascular diseases, heart failure (HF) has a high rate of hospitalization, morbidity and mortality, consuming vast resources of the public health system in Brazil and other countries. The correct determination of the filling pressures of the left ventricle by noninvasive or invasive assessment is critical to the proper treatment of patients with decompensated chronic HF, considering that congestion is the main determinant of symptoms and hospitalization. Physical examination has shown to be inadequate to predict the hemodynamic pattern. Several studies have suggested that agreement on physical findings by different physicians is small and that, ultimately, adaptive physiological alterations in chronic HF mask important aspects of the physical examination. As the clinical assessment fails to predict hemodynamic aspects and because the use of Swan-Ganz catheter is not routinely recommended for this purpose in patients with HF, noninvasive hemodynamic assessment methods, such as BNP, echocardiography and cardiographic bioimpedance, are being increasingly used. The present study intends to carry out, for the clinician, a review of the role of each of these tools when defining the hemodynamic status of patients with decompensated heart failure, aiming at a more rational and individualized treatment.

Introduction

Heart failure (HF) is a clinical syndrome of which signs and symptoms are classically used to define diagnosis, guide treatment and assess prognosis. The correct determination of the left ventricular filling pressures by noninvasive or invasive evaluation is crucial to the proper treatment of patients with chronic heart failure, as congestion is the determining factor of symptoms and hospitalization. Figure 1 shows the possible moments for the identification and treatment of elevated ventricular filling pressures (VFP)1.

Invasive hemodynamic assessment is also very important for the assessment and management of these patients, having been used for decades, initially by direct left ventricular puncture2 and currently, by Swan-Ganz catheter1. However, in recent years, the use of invasive hemodynamic monitoring has been decreasing, especially due to the growing evidence of no benefit with this method3.

Thus, clinical and noninvasive hemodynamic evaluation became prevalent. Hence, diagnostic criteria, such as Boston’s or Framingham’s criteria, have been widely used in clinical trials and guidelines to define HF as they are easy to perform, are low-cost and have good specificity for the diagnosis. Furthermore, clinical evaluation also shows good prognostic correlation. The functional classification of the New York Heart Association (NYHA) and, more recently, Stevenson’s clinical-hemodynamic classification in four hemodynamic profiles, according with the physical examination findings of congestion and peripheral perfusion, constitute well-documented prognostic markers2. Natriuretic peptides (BNP and NT-proBNP) have shown to be useful in the diagnosis of decompensated HF in the emergency room, by confirming or ruling out the diagnosis in patients with dyspnea and giving prognostic value in this population, especially at hospital discharge, to predict future events.

It can be observed however, that the history, physical examination and natriuretic peptides are greatly questioned concerning the power to evaluate the hemodynamic condition, primarily of congestion or low cardiac output in patients with decompensated HF. Physical examination limitations increasingly start with physicians’ lack of interest in performing a good-quality physical examination, supported by the wide availability of complementary examinations that have been used, often replacing the physical examination. Moreover, the reduced consultation time and poor training in many medical schools may be factors associated with the deterioration in the quality of the physical examination.

Several studies have suggested that the agreement in physical findings by different physicians is small. The gold standard for hemodynamic evaluation in heart failure is the pulmonary artery catheter (Swan-Ganz), and several studies have compared the invasive hemodynamic assessment with the physical examination, showing the limitation of this assessment in defining congestion or low output.

Natriuretic peptides were first used in clinical practice at the beginning of this decade, with the promise of increasing the diagnostic accuracy of HF and diagnose VFP elevation,
defining congestion, as they increase in response to ventricular distension. However, with the advancement of knowledge in this area, several limitations were also observed regarding the use of natriuretic peptides in the identification of congestion, and there is no currently defined cutoff for that.

As the clinical evaluation, the natriuretic peptides also fail to predict hemodynamics, as the routine use of the Swan-Ganz catheter is not recommended for this purpose in patients with HF. Thus, the use of noninvasive methods for hemodynamic assessment, such as echocardiography and bioimpedance cardiography, has been increasing.

The present study aims to carry out for the clinician, a review of the role of each of these tools, when defining the hemodynamic condition of patients with decompensated heart failure, aiming at a more rational and individualized treatment.

Clinical Evaluation: history, physical examination, chest x-ray and weight monitoring

History and physical examination

Among the several symptoms of HF, orthopnea (dyspnea that starts with the orthostatic position and is relieved by decubitus elevation or in the sitting position) stands out as the symptom that most correlates with the elevation of ventricular filling pressures.

In a study with outpatients from a HF clinic in Brazil, the presence of orthopnea was the most sensitive marker of elevated filling pressures, both right and left. In this study, the diagnostic performance of different physical examination findings alone was suboptimal in predicting the hemodynamic pattern. After evaluating a clinical score for congestion (containing the variables of pulmonary crackles, pathological jugular venous distention, peripheral edema and third heart sound), the best negative predictive value for congestion was the absence of these associated clinical signs (95% predictive value for left atrial pressure < 20 mmHg).

Persistent orthopnea with treatment also has a prognostic association. Patients who maintain orthopnea complaints throughout one year of treatment for HF have a rehospitalization rate that is four times higher than those free of orthopnea and, moreover, do not show left ventricular function improvement at the end of this period (11 ± 13% vs. -1 ± 6%, p < 0.001).

In the physical examination substudy of the ESCAPE trial, orthopnea was the only symptom that correlated with the elevation in the VFP. When patients had orthopnea, in spite of the use of two or more pillows, the chance of having a pulmonary artery occlusion pressure (PAOP) ≥ 30 mm Hg was 3.6 times higher (OR 3.6, p < 0.05).

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Regarding the physical examination, several studies have evaluated the capacity to estimate the hemodynamic status of patients with HF. Of all findings, the jugular venous pressure (JVP) seems to be more accurate in detecting the elevation of left ventricular filling pressures. Since the introduction of JVP assessment in clinical practice in 1930 by Lewis and later, the standardization of its assessment by Borst and Molhuysen, in 1952, the JVP has been the object of discussion. The
presence of pathological jugular distention (PJD) reflects the increase in filling pressure of the right chambers, which in turn frequently correlates with left ventricular filling pressures. A retrospective analysis of the SOLVD study showed that the presence of PJD may be a marker of poor prognosis, considering that its presence was a marker of re-hospitalization, hospitalization and death from heart failure.11

However, the results of the PJD assessment have not been universally reproducible, and there are questions about its real significance: the capacity to detect medical elevations in the JVP at physical examination, considering the great technological evolution and the availability of complementary diagnostic methods. The diversity of the methodology in assessing the JVP is also an important factor in the studies, whereas there is no universally accepted standardization. In the classic study by Stevenson and Perloff,12 the presence of PJD was defined as the appearance of the internal jugular vein above the clavicle, with the patient elevated between 30º and 45º. Although widely used, this definition may miss slighter filling pressure elevations. Patients could have improved diagnostic accuracy if they were assessed for the presence of hepatojugular reflux.13,14

The several studies of patients with HF, even if decompensated, show a low prevalence of PJD,15-17, from 11% to 14%. Finally, it is estimated that the accuracy of JVP to detect elevated RAP is less than 75% and the concordance of left and right pressures is approximately 75% in patients with HF.18

Another important finding at the physical examination is the presence of third heart sound (S3). Since the first description of a gallop rhythm by Potain19 in 1880, the S3 has been studied. Its presence is highly specific for the detection of ventricular dysfunction and elevated filling pressures, estimated at 93%.20 Additionally, it is pointed out as an independent prognostic marker in patients with HF.21 On the other hand, its sensitivity is low (between 13% and 52%) and it displays a lot of interobserver variability, depending on their experience.22

In a study that specifically evaluated the physical examination findings at the hemodynamic evaluation of patients with advanced HF, the presence of S3 did not add any relevant information to the presence of PAOP > 22 mmHg.6 In the study by Stevenson and Perloff,5 mentioned above, 50 patients with advanced HF awaiting cardiac transplantation were evaluated by physical examination and invasive hemodynamics. Of the total, 48 had S3, which is not indicated, therefore, to discriminate patients with and without elevated filling pressures.

**Chest X-ray**

The chest x-ray is a widely available and inexpensive complementary test, which traditionally helps in the diagnosis of heart failure. In patients with HF, the presence of signs of congestion, for instance, cephalization of the pulmonary vascular network, interstitial edema and alveolar edema, has high specificity for decompensation (above 96%), but has low sensitivity.

Twenty percent (20%) of patients with cardiomegaly at the echocardiogram did not have that diagnosis in the chest x-ray, and patients with elevated ventricular filling pressures may not show any sign of congestion. Pleural effusion, if present, has a high specificity for decompensation (92%) but low sensitivity (25%). It is estimated, therefore, that one in five patients with symptoms of decompensated heart failure who seek the emergency room have a chest x-ray that shows no signs of congestion, in spite of having elevated ventricular filling pressures.22

**Weight monitoring**

Weight monitoring in patients with HF is recommended according to the III Brazilian Guidelines on Chronic Heart Failure, in order to check the volemic status.23 Changes in weight, especially over short periods of time, can be good indicators of volemic worsening. Many studies, however, are controversial on this subject, indicating that little or no weight gain is observed before an episode of decompensation or that a modest weight loss is observed after clinical compensation of an acute HF episode. In many cases, decompensation may occur not due to the build up of fluid, but by water redistribution from the periphery to the lungs by neurohumoral and inflammatory acute activation, leading to cardiac and vascular alterations that promote reduced venous capacitance and increased peripheral arterial resistance.24

**Natriuretic peptides**

There are three types of natriuretic peptides (NP): atrial natriuretic peptide (ANP), B-type natriuretic peptide (BNP) and C-type natriuretic peptide (CNP). ANP and BNP are produced primarily by the heart, and CNP by endothelial cells.

BNP is a hormone produced by cardiomyocytes in response to stretch, secondary to increased ventricular filling pressure or volume overload. Initially, cardiomyocytes produce pre-proBNP, which is converted to proBNP, and finally, in the active metabolite BNP, which promotes vasodilation and natriuresis. Both proBNP and BNP have been long used in clinical practice for diagnosis, to assess volemic status and define prognosis in patients with HF. Several studies have shown a positive association between levels of these NP and the degree of ventricular dysfunction and functional class. Others have shown that there is positive correlation between BNP levels and PAOP ($r = 0.72$) in patients with acute HF25 and that intensive treatment determines a decrease in the levels of PAOP and NP.

Despite this evidence, some authors have not found a good correlation between BNP and proBNP levels with the VFP. In a study of critically ill patients hospitalized for different medical conditions and who had received a Swan-Ganz catheter as part of treatment, it was observed that the NP levels showed no association with pulmonary artery occlusion pressure measurement, therefore, not being a good noninvasive marker of VFP for this population.26 Furthermore, after normalization of the filling pressures, measured invasively, and with therapy for decompensated HF, it was observed that BNP levels, although decreased, still remained significantly elevated.27
Extreme values of BNP, < 100 pg/ml or > 400 pg/ml, have better correlation with normal or elevated filling pressures, respectively. However, the range between these values is called the “gray zone”, characterized by not showing good correlation.

It is noteworthy that many conditions affect the production and clearance of BNP, such as age, weight, renal failure, noncardiac disease, among others, which also limits its use in some of these patients.

**Echocardiogram**

The echocardiography is the most useful complementary examination when evaluating patients with HF. It provides important information regarding heart morphology; quantifies the systolic and diastolic functions and helps define the etiology and prognostic parameters in response to different therapeutic interventions.

In recent years, two new and fundamental echocardiographic evaluations were incorporated into daily practice: the evaluation of ventricular dyssynchrony and hemodynamic evaluation. The latter has been very important to understand the hemodynamic profile of patients with HF, especially decompensated ones or those difficult to manage. The so-called “hemodynamic echocardiogram” refers to the echocardiographic assessment of hemodynamic parameters that reflect the hemodynamic data obtained by invasive monitoring. Chart 1 shows the main echocardiographic parameters that must be used to estimate the hemodynamic status of patients with decompensated HF.

The use of tissue Doppler imaging (TDI) technique to assess the mitral annular motion was incorporated into the routine echocardiographic evaluation, enabling the estimation of left atrial pressure (LAP). This measurement is performed using the apical 4-chamber view of the septal wall or lateral of the mitral annulus, obtaining the early diastolic velocity of the tissue Doppler (E’). With the conventional Doppler measurement of transmural flow, the peak diastolic flow velocity is obtained (E).

The E / E’ ratio is calculated, and its value shows good correlation in the literature with the invasive measurement of left ventricular end-diastolic pressure (LVEDP). Using the formula $1.24 \times (E / E’) + 1.9$, the LAP is calculated.

Ommen et al. showed, in the beginning of the last decade, that an E / E’ > 15 mmHg has a good correlation with LVEDP > 12 mm Hg (86% specificity). But when the E/E’is < 8 mmHg, the correlation is good for a normal LVEDP (97% negative predictive value). In comparison with the BNP, the E / E’ ratio shows a better performance to detect congestion, even in patients with preserved LV function.

Similarly to the BNP, the E/E’ ratio also has a “gray area”. E / E’ values < 8 mmHg correlate well with normal LVEDP whereas values > 15 mmHg showed a good correlation with the elevation in filling pressures. There can be great variation among these values, so other parameters must be evaluated to try to define the presence of congestion. A schematic hemodynamic echocardiogram is shown in Figure 2.

**Transthoracic bioimpedance**

The transthoracic bioimpedance (TTB) or by thoracic impedance cardiography is a noninvasive diagnostic method for hemodynamic assessment, which provides the following parameters: cardiac output and stroke volume, systemic vascular resistance, ventricular contractility parameters and volume standard (chest fluid content). Voltage and chest electrical impedance alterations, mainly due to the variation of blood flow in large vessels (the blood is an excellent current conductor), are translated into hemodynamic parameters.

**Chart 1 - Main echocardiographic parameters in hemodynamic assessment**

<table>
<thead>
<tr>
<th>Hemodynamic assessment</th>
<th>Echocardiographic Parameter</th>
<th>Technique used</th>
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</thead>
<tbody>
<tr>
<td>Perfusion assessment (heat or cold)</td>
<td>Cardiac output (CO)</td>
<td>HR x SV</td>
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<tr>
<td></td>
<td>Systolic volume (SV)</td>
<td>Product of the area of LV outflow tract and time-velocity integral of the LV outflow tract obtained by pulsed Doppler</td>
</tr>
<tr>
<td>Volemic evaluation (dry or congested)</td>
<td>E/E’</td>
<td>Peak velocity of transmitral diastolic flow (E) / early diastolic velocity of tissue Doppler (E’).</td>
</tr>
<tr>
<td></td>
<td>LAP</td>
<td>$1.24 \times (E/E’) + 1.9$</td>
</tr>
<tr>
<td>Size and variation of IVC</td>
<td>RAP</td>
<td>Calculated by the inspiratory collapse of the inferior vena cava</td>
</tr>
<tr>
<td></td>
<td>PASP</td>
<td>Sum of the tricuspid gradient and estimated RAP</td>
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<tr>
<td></td>
<td>PAOP</td>
<td>Pulmonary regurgitation velocity at the end of diastole</td>
</tr>
<tr>
<td></td>
<td>MPAP</td>
<td>Maximum pulmonary regurgitation velocity</td>
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HR – heart rate; RAP – right atrium pressure; PASP - pulmonary artery systolic pressure; PAOP - pulmonary artery diastolic pressure; MPAP - mean pulmonary artery pressure; IVC - inferior vena cava; LAP - left atrium pressure; E/E’ – peak velocity of transmitral diastolic flow (E) / early diastolic velocity of tissue Doppler (E’).
The cardiac output (CO) measurement by TTB correlates well with the invasive measurement of CO, with a correlation coefficient ranging between 0.76 and 0.89. In a study evaluating the cause of dyspnea in elderly patients admitted to the emergency department, the assessment by bioimpedance was able to alter the initial clinical diagnosis in 13% of patients and had an impact in changing the treatment in 39%. This method has the advantage of being performed at the bedside in real time by monitoring the response to therapeutic interventions.

The hemodynamic evaluation by TTB, primarily in the definition of blood volume, has been controversial. The BIG study, the largest randomized trial on TTB in patients with HF, published recently, showed only modest correlation between the cardiac outputs measured by the TTB and the invasive monitoring (r = 0.4-0.6), indicating that the thoracic fluid content did not correlate with the measurement of the PAOP. When comparing the hemodynamic profiles of systemic perfusion and congestion, the TTB did not show concordance with the pattern observed in invasive monitoring, not being able to give accurate information about the LV filling pressures. Moreover, this method is not widely available and has a number of limitations to its use, e.g., pleural effusion, obesity, aortic regurgitation, heart rate extremes, among others.

**Conclusion**

The individualized physical examination integrated to the several methods of noninvasive hemodynamic evaluation for the presence of congestion appears to be the best way to estimate ventricular filling pressures, both to adjust therapy in outpatients and in those with decompensated HF, aiming to identify the early increased filling pressures and prevent clinical decompensation.

It is important to recognize the limitations of traditional signs and symptoms of heart failure, especially edema, crackling rales and the third heart sound, to guide treatment and estimate the hemodynamics of patients with chronic heart failure. Orthopnea and pathological jugular distention are the best markers of elevated filling pressures in this context. As a result, many physicians use BNP to assist in detecting congestion. Very low values of BNP (<100 pg/ml) are good predictors of the absence of congestion, but its elevation is not necessarily associated with elevated filling pressures. The use of BNP to guide treatment has yet to be defined in prospective clinical trials.

The hemodynamic echocardiogram is the best method available to further assist the physician in detecting congestion. Several studies have shown that its parameters correlate significantly with the same parameters obtained by invasive monitoring. E/E’ values < 8 mmHg should be carefully considered, as they have good correlation with normal LVEDP and E/E’ > 15 mmHg, as they correlate positively with increased LVEDP.

The transthoracic bioimpedance and other new technologies that have been tested have yet to demonstrate benefits in future clinical trials to be incorporated into practice on a routine basis. Therefore, the integrated use of careful clinical examination and these complementary

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**Fig. 2 – Hemodynamic echocardiogram. Image reproduced with permission of Elsevier Publishers.**

A - IVCCI – Inferior Vena Cava Collapsibility Index; RAP – right atrial pressure calculation. B - TR Vel. – velocity of tricuspid regurgitation: systolic gradient between right ventricle and atrium– calculation of the systolic pressure of the pulmonary artery. C - PR Vel. – velocity of pulmonary regurgitation, used to calculate the mean and diastolic pressures of the pulmonary artery. D - early mitral flow (E) / mitral annulus movement at the start of diastole (E’) and calculation of the pulmonary artery occlusion pressure (PCWP).
methods allows a more comprehensive and individualized approach in order to reduce morbidity and improve the prognosis of patients with decompensated heart failure.

Potential Conflict of Interest

No potential conflict of interest relevant to this article was reported.

References


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