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# Influence of decongestion and heart rate reduction on echocardiographic parameters and dyspnoea during treatment of acute decompensated heart failure

## Wpływ odwadniania oraz redukcji częstości rytmu serca na parametry echokardiograficzne oraz nasilenie duszności podczas leczenia ostrej zdekompensowanej niewydolności serca

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## Abstract

**Introduction.** Decongestion therapy of acute decompensated heart failure (ADHF) leads to significant haemodynamic and heart rate changes. The purpose of the study was to evaluate the impact of decongestion therapy and heart rate reduction on changes in echocardiographic parameters in patients hospitalized due to ADHF.

**Material and methods.** In 34 consecutive adult patients (41.2% females, mean age 70.2 ± 10.8 years) who required hospitalization due to ADHF, an echocardiographic assessment was performed upon admission and at discharge together with clinical and laboratory evaluation.

**Results.** Significant reductions in body weight (84.22 ± 17.46 vs. 78.72 ± 16.95 kg, p < 0,001), heart rate (86.21 ± 15.7 vs. 75.72 ± 11.01 bpm, p < 0.001) and NT-proBNP level (7530.22 ± 5192.52 vs. 3270.32 ± 2947.26 pg/mL, p = 0.001) were found. In the group of patients with a weight reduction of at least 5 kg, a significant decrease in dyspnoea severity was assessed with 0–100 points VAS scale (at admission/discharge ΔVAS 42.14 ± 13.68 vs. 30.00 ± 12.79, p = 0.04), tricuspid regurgitation peak gradient (ΔTRPG 7.53 ± 9.36 vs. 1.91 ± 7.48 mm Hg, p = 0.05) and mitral regurgitation severity (ΔMR VC 1.21 ± 0.65 vs. 0.66 ± 0.77 mm, p = 0.05) were observed. The subgroup of patients with heart rate reduction over 10 bpm exhibited a significant increase in left ventricular (LV) ejection fraction (HR reduction > 10 bpm: at admission 30.56 ± 13.96; at discharge 38.69 ± 13.29 vs. HR reduction < 10 bpm 42.64 ± 14.95; 41.91 ± 13.46 %, p = 0.02), and LV outflow velocity time integral (HR reduction > 10 bpm: at admission 12.61 ± 3.64; at discharge 15.55 ± 4.88 vs. HR reduction <10 bpm: 16.18 ± 5.64; 15.04 ± 4.45 cm, p = 0.04).

**Conclusions.** Treatment of fluid overload in ADHF results mainly in dyspnoea reduction, mitral regurgitation decrease and pulmonary pressure lowering. Heart rate reduction is followed by a significant increase in LV stroke volume and LV ejection fraction. Such echocardiographic changes are expected during successful ADHF treatment. Their absence may direct the attention of the attending physician to the need for more aggressive treatment or a worse prognosis for the patients.

Keywords: acute heart failure, echocardiography, decongestion therapy, heart rate

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## Introduction

Acute decompensated heart failure (ADHF) is defined as a sudden exacerbation of signs and symptoms of heart failure [1]. Among individuals aged 65 years and older, ADHF is a leading cause of hospitalization and is associated with high mortality and rehospitalization rates [2]. Echocardiography is a fundamental diagnostic tool used in the evaluation of patients with worsening heart failure symptoms. As a non-invasive and widely available test, it has broad applications in differential diagnosis, evaluation of specific causes of circulatory system decompensation, and prognosis assessment [3, 4].

During decongestion therapy, with the reduction of volume overload, several echocardiographic parameters may undergo significant changes. Previous studies have demonstrated alterations in the dimensions of the right atrium, right ventricle, inferior vena cava, and mitral inflow parameters [5], as well as the severity of mitral valve regurgitation [6, 7].

Elevated resting heart rate is a well-known unfavourable prognostic factor in patients with chronic heart failure [8]. However, the prognostic significance of heart rate in acute decompensation of the circulatory system is still not fully understood, and the optimal heart rate in ADHF has not been clearly defined [9]. Higher mortality rates have been associated with relatively high heart rates in patients after ADHF treatment [10]. In a model mimicking human ADHF, lowering the heart rate could potentially counteract the worsening of cardiovascular dysfunction associated with decompensation and the onset of pulmonary congestion [11]. As shown in previous studies heart rate reduction can be an important treatment goal with prognostic significance in the ADHF population [12, 13, 14]. On the other hand, tachycardia may serve as a compensatory mechanism to achieve sufficient cardiac output in patients with very low stroke volume. Lowering heart rate may also significantly modify echocardiographic parameters of heart function.

This study assessed the impact of decongestion therapy and heart rate reduction on changes in echocardiographic parameters and subjective dyspnoea severity in patients hospitalized due to signs and symptoms of acute decompensated heart failure.

## Material and methods

### Study population, patient management and evaluation

The study included 34 consecutive patients (41.2% of which were females, mean age 70.2 ± 10.8 years) requiring hospitalization and admitted to the Department of Cardiac and Vascular Diseases and Department of Diagnostics, Jagiellonian University Medical College, John Paul II Hospital in Kraków, Poland, due to exacerbation of heart failure symptoms. Patients were recruited between January 2018 and December 2020. Exclusion criteria were: fever, acute infection, confirmed diagnosis of concomitant acute coronary syndrome, acute heart failure caused by significant cardiac arrhythmia (i.e. atrial fibrillation, supraventricular or ventricular tachycardia) or atrioventricular block (tachycardia/bradycardia), uncontrolled hyperthyroidism/hypothyroidism, pregnancy and postpartum period. None of the patients included in the study were diagnosed with heart failure *de novo*.

In all participants of the study, following the usual procedures for the diagnosis and treatment of acute heart failure, a standard electrocardiogram was performed, as well as an assessment of basic laboratory parameters including N-terminal pro-B-type natriuretic peptide (NT-proBNP) levels (determined by electrochemiluminescence immunoassay Roche Diagnostics, Mannheim, Germany) at baseline and discharge. Basic vital signs were evaluated, including respiratory rate, $SaO_2$ measurement, blood pressure values, and heart rate (patients were at rest in a supine position for at least 5 minutes). Fluid balance and body weight were monitored daily. Body weight was measured in the morning directly after the patient woke up and after voiding urine. A subjective assessment of the degree of dyspnoea was performed using a standardized visual analogue scale (VAS) of 0–100 [15]. Patients were asked to mark the degree of dyspnoea on a linear scale after 15 minutes of rest in bed. Patients enrolled in the study received standard treatment for acute heart failure according to current guidelines [16]. The study was conducted according to all current GCP standards.

### Echocardiographic evaluation

Transthoracic echocardiography was performed in all patients upon admission and at discharge using the Philips iE33 and Philips Epiq ultrasound machines (Philips Healthcare, Eindhoven, Netherlands). Post-processing and study evaluation were conducted using a dedicated workstation. An echocardiographic assessment of heart structure was carried out by current guidelines [17, 18]. The following parameters were analysed: left ventricular end-diastolic dimension (LVEDD), left ventricular ejection fraction (LVEF) calculated using the Simpson biplane method, left atrial dimension (measured in the parasternal long axis view), left atrial and right atrial area (measured in the apical four-chamber view), right ventricular outflow tract (RVOT) diameter, tricuspid annular plane systolic excursion (TAPSE) measured in the apical four-chamber view using M-mode as a marker of right ventricular systolic function, and inferior vena cava diameter.

In the assessment of mitral regurgitation (MR), vena contracta width (VC), proximal isovelocity surface area (PISA) radius, effective regurgitant orifice area (MR ERO), and mitral regurgitant volume (MR vol.) were measured.

A semi-quantitative evaluation of tricuspid regurgitation using VC width was also performed. Tricuspid regurgitation peak gradient (TRPG) was obtained as a marker of right ventricular afterload. Measurements were averaged from 3 consecutive cardiac cycles in patients with sinus rhythm and 3–5 consecutive cardiac cycles in those with atrial fibrillation.

### Statistical analysis

The data were presented as mean ± standard deviation (SD) for continuous variables or as percentages (%) for categorical variables. The distribution of the studied data was assessed using the Kolmogorov–Smirnov test. Statistical analyses were conducted using either the Student's t-test or the Mann-Whitney U test. A p-value less than 0.05 was considered statistically significant. All statistical analyses were conducted using StatSoft STATISTICA 13.1 software (StatSoft Inc., Tulsa, OK, USA).

## Results

A detailed clinical characterization of the study group is presented in Table 1. All patients included in the study initially presented with severe dyspnoea, classified as NYHA class III or IV, with a mean dyspnoea severity score on the Visual Analog Dyspnoea Scale (0–100) of 82.40 ± 8.81. None of the patients had pulmonary oedema. Ischaemic aetiology of heart failure (based on medical history) was observed in 49% of patients. Elevated NT-proBNP level was found in all patients, with a mean level of 7530.22 pg/mL. All patients were chronically treated with beta-blockers, and the majority (70%) received chronic treatment with an angiotensin-converting enzyme inhibitor (ACE-I) and aldosterone receptor antagonists (MRA). None of the patients was receiving angiotensin receptor blockers (ARB) or angiotensin receptor-neprilysin inhibitor (ARNI). During hospitalization, intravenous inotropic/vasopressor medications were required in 7 patients, while no patient required mechanical ventilation. During the observation period, no changes in heart rhythm were recorded (AF or sinus rhythm from admission to discharge). Baseline echocardiographic parameters are presented in Table 2. The study group exhibited a significantly reduced left ventricular ejection fraction (mean LVEF of 36.82 ± 15.48%) with ventricular remodelling (mean LVEDD of 60.62 ± 11.36).

Significant reductions in body weight, dyspnoea severity, heart rate, and NT-proBNP were achieved during hospitalization (Table 3). Table 4 presents changes in echocardiographic parameters based on the weight loss achieved during treatment. Subgroup analysis of patients based on the degree of weight loss (above/below 5 kg

**Table 1.** Baseline characteristics of the study group

| Parameter | Value at baseline |
|---|---|
| Age (years) | 70.2 ± 10.8 |
| Women (%) | 41.2 |
| BMI | 30.1 ± 5.2 |
| Body weight (kg) | 84.22 ± 17.46 |
| Arterial hypertension (%) | 91.1 |
| Diabetes mellitus (%) | 35.2 |
| Atrial fibrillation (%) | 67.6 |
| COPD (%) | 5.8 |
| Coronary artery disease (%) | 49.2 |
| Previous myocardial infarction (%) | 35.2 |
| Previous CABG/PCI (%) | 35.2 |
| QRS duration in ECG (ms) | 116.8 ± 26.4 |
| LBBB in ECG (%) | 11.7 |
| Chronic treatment with ACE-I (%) | 70.5 |
| Chronic treatment with beta-blockers (%) | 100.0 |
| Chronic treatment with MRA (%) | 70.5 |
| Level of dyspnoea (0–100 pts VAS) | 82.40 ± 8.81 |
| Heart rate (bpm) | 86.21 ± 15.7 |
| NT-proBNP (pg/mL) | 7530 ± 5192 |

Data are expressed as the mean value ± standard deviation
ACE-I — angiotensin-converting enzyme inhibitors; BMI — body mass index; bpm — beats per minute; CABG — coronary artery bypass grafting; COPD — chronic obstructive pulmonary disease; MRA — aldosterone receptor antagonists; PCI — percutaneous coronary intervention; VAS — visual analogue scale

**Table 2.** Baseline parameters in echocardiography

| Parameter | Value |
|---|---|
| LA diameter PLAX (mm) | 54.51 ± 6.61 |
| LA area in AP4 (cm2) | 35.56 ± 9.07 |
| RA area in AP4 (cm2) | 32.50 ± 7.70 |
| RV RVOT diameter in PLAX (mm) | 38.90 ± 5.27 |
| LVEDD in PLAX (mm) | 60.62 ± 11.36 |
| LVEF [Simpson Bipl. Method] (%) | 36.82 ± 15.48 |
| TAPSE (mm) | 15.07 ± 4.83 |
| VC MR (mm) | 6.16 ± 1.45 |
| MR EROA (cm2) | 0.35 ± 0.16 |
| MR volume (mL) | 44.85 ± 22.24 |
| TR V max (m/s) | 3.3 ± 0.48 |
| TR VC (mm) | 6.43 ± 2.51 |
| TRPG (mm Hg) | 44.30 ± 11.92 |
| Vena cava inferior diameter (mm) | 27.96 ± 4.42 |

Data are expressed as the mean value ± standard deviation
AP4 — apical four-chamber view; EROA — effective regurgitant orifice area; LA — left atrium; LVEDD — left ventricular end-diastolic diameter; LVEF — left ventricular ejection fraction; MR — mitral regurgitation; PLAX — parasternal long axis view; RV — right ventricle; RVOT — right ventricle outflow tract; TAPSE — tricuspid annular plane systolic excursion; TR — tricuspid regurgitation; VC — vena contracta

**Table 3.** Change in heart failure parameters during the treatment period

| Parameter | Value at baseline | Value at discharge | p-value |
|---|---|---|---|
| Level of dyspnoea (0–100 pts) | 82.40 ± 8.81 | 46 ± 10.88 | < 0.001 |
| Body weight (kg) | 84.22 ± 17.46 | 78.72 ± 16.95 | < 0.001 |
| Heart Rate (bpm) | 86.21 ± 15.7 | 75.72 ± 11,01 | < 0.001 |
| NT-proBNP (pg/mL) | 7530.22 ± 5192.52 | 3270.32 ± 2947.26 | 0.001 |

Data are expressed as the mean value ± standard deviation

during hospitalization) revealed a significant reduction in dyspnoea severity, right ventricular afterload (TRPG), and severity of mitral valve regurgitation in the subgroup of patients who achieved a weight loss of at least 5 kg. However, there was no significant increase in left ventricular ejection fraction or right ventricular systolic function parameters (as measured by TAPSE). Regarding the achievement of a significant reduction in heart rate (> 10 bpm vs. < 10 bpm, see Table 5), the subgroup of patients with a heart rate reduction over 10 bpm exhibited a significant increase in LVEF and left ventricular outflow tract velocity time integral (LVOT VTI). However, heart rate reduction did not affect cardiac chamber dimensions, severity of atrioventricular valve regurgitation, dyspnoea severity, or right ventricular systolic function.

## Discussion

The main findings of the current study are as follows: 1) In patients with acute decompensated heart failure (ADHF), reduction of congestion (expressed by weight loss) is associated with a decrease in dyspnoea levels, reduction of mitral regurgitation severity, and right ventricular afterload as assessed by echocardiography; 2) Reduction of heart rate is associated with an increase in LVEF and LVOT VTI; 3) Reduction of heart rate during ADHF treatment does not affect the severity of mitral and tricuspid regurgitation or the level of dyspnoea.

The reduction of heart rate and weight are important treatment goals in decompensated heart failure. Previous studies have demonstrated that higher admission heart rates are associated with higher in-hospital mortality in patients hospitalized for ADHF [19]. Conversely, a study by Rosa et al. [20] showed that only the discharged heart rate is a significant predictor of long-term prognosis. Persistently high heart rates after ADHF treatment, whether in sinus rhythm or atrial fibrillation, are associated with increased mortality in one-year follow-up, while no such relationship is observed for baseline heart rate or the degree of heart rate reduction during hospitalization. In the EVEREST study [21], patients with sinus rhythm and LVEF < 40% who had sustained heart rates above 70 bpm after hospital discharge had a higher risk of long-term mortality (median 9.9 months). Unlike stable heart failure conditions, a high heart rate in patients with acute decompensated heart failure may indicate reduced systolic function and serve as a compensatory mechanism for low stroke volume. A high heart rate is also associated with increased oxygen consumption and reduced coronary perfusion, which can lead to worsened left ventricular function [22].

The following study found a relationship between heart rate reduction and increased stroke volume, as evidenced by an increase in LVOT VTI and left ventricular ejection fraction measured by echocardiography. LVOT VTI is a relatively simple echocardiographic parameter that reflects left ventricular systolic function and cardiac output. Decreased values of LVOT VTI (< 10 cm) have prognostic significance [23].

Daily weight loss is an indicator commonly used in clinical practice as a marker of decongestion. It is less influenced by interfering factors compared to daily fluid balance assessment and is often used to evaluate the effectiveness of diuretic therapy [24]. In the present study group of patients with ADHF, a relationship was found between weight loss and a reduction in dyspnoea severity. A relationship between the degree of weight loss and the reduction in tricuspid regurgitation peak gradient (TRPG) was also observed. Evaluation of right ventricular systolic pressure (RVSP) through TRPG assessment is important as a prognostic factor in patients with ADHF [25]. TRPG increases during ADHF treatment have been shown to predict kidney function worsening, which is also associated with prognosis [26]. Adjusting ADHF therapy based on weight loss reduction and TRPG reduction measured by echocardiography may improve treatment outcomes.

Functional mitral regurgitation in the ADHF population has a dynamic nature. In the present study, consistent with previous observations on changes in echocardiographic parameters during ADHF treatment, a reduction in the severity of mitral regurgitation was found. Interestingly, found was also a relationship between the degree of reduction in mitral regurgitation severity and the degree of weight loss, which may have implications in daily clinical practice for treatment monitoring and further management, including decision-making on surgical treatment of valvular heart disease.

**Table 4.** Changes in echocardiographic parameters during hospitalization depending on the reduction in body weight

| Parameter | Body weight reduction > 5 kg (n = 18) | | | Body weight reduction < 5 kg (n = 16) | | | p-value |
|---|---|---|---|---|---|---|---|
| | Admission | Discharge | Value change | Admission | Discharge | Value change | |
| Dyspnoea score | 82.85 ± 8.25 | 40.72 ± 12.06 | **42.14 ± 13.68** | 80.00 ± 10.00 | 50.00 ± 7.38 | **30.00 ± 12.79** | **0.04** |
| Heart rate (bpm) | 86.71 ±19.16 | 74.85 ± 9.42 | 11.85 ± 14.52 | 83.38 ± 12.30 | 74.90 ± 13.96 | 6.36 ± 14.20 | 0.95 |
| LA PLAX (mm) | 52.42 ± 3.47 | 49.21 ± 4.04 | 3.21 ± 4.80 | 56.77 ± 8.43 | 52.58 ± 8.05 | 5.00 ± 3.64 | 0.46 |
| LA area AP4 ($cm^2$) | 32.95 ± 5.03 | 29.11 ± 4.42 | 3.84 ± 3.39 | 38.38 ± 11.57 | 36.40 ± 12.05 | 2.85 ± 3.34 | 0.46 |
| RA area AP4 ($cm^2$) | 31.96 ± 5.95 | 27.42 ± 7.96 | 4.54 ± 4.20 | 33.08 ± 9.45 | 31.37 ± 10.21 | 2.54 ± 3.40 | 0.20 |
| LV EDD (mm) | 58.50 ±10.43 | 55.78 ± 10.08 | 2.71 ± 2.70 | 62.92 ± 12.27 | 61.33 ± 11.75 | 2.58 ± 2.64 | 0.90 |
| LVEF (%) | 35.81 ±14.44 | 41.23 ± 12.90 | 6.89 ± 7.19 | 37.91 ± 17.05 | 39.16 ± 13.99 | 4.00 ± 4.62 | 0.24 |
| RV RVOT (mm) | 37.92 ± 5.12 | 35.57 ± 3.41 | 2.35 ± 3.20 | 40.00 ± 5.43 | 38.33 ± 5.49 | 2.00 ± 3.21 | 0.78 |
| TAPSE (mm) | 15.00 ± 4.64 | 16.00 ± 3.13 | 1.53 ± 2.40 | 15.15 ± 5.22 | 15.33 ± 4.35 | 0.58 ± 2.46 | 0.34 |
| MR VC (mm) | 5.88 ± 1.54 | 4.73 ± 1.5 | **1.21 ± 0.65** | 6.46 ± 1.33 | 5.83 ± 1.58 | **0.66 ± 0.77** | **0.05** |
| MR PISA r. (mm) | 6.78 ± 2.51 | 5.53 ± 2.10 | 1.30 ± 1.03 | 8.92 ± 2.25 | 7.25 ± 2.13 | 1.66 ± 1.07 | 0.40 |
| MR ERO ($cm^2$) | 0.29 ± 0.08 | 0.17 ± 0.09 | 0.10 ± 0.06 | 0.40 ± 0.21 | 0.30 ± 0.20 | 0.12 ± 0.09 | 0.89 |
| MR vol. (mL) | 36.35 ± 17.14 | 24.53 ± 12.44 | 12.23 ± 9.73 | 54.00 ± 24.04 | 37.75 ± 22.38 | 16.00 ± 11.93 | 0.39 |
| TRPG (mm Hg) | 43.00 ± 11.44 | 35.38 ± 8.41 | **7.53 ± 9.36** | 45.69 ± 12.72 | 43.42 ± 12.41 | **1.91 ± 7.48** | **0.05** |
| TR V max (m/s) | 3.24 ± 0.46 | 2.94 ± 0.42 | 0.29 ± 0.35 | 3.35 ± 0.50 | 3.26 ± 0.48 | 0.07 ± 0.30 | 0.11 |
| TR VC (mm) | 6.11 ± 2.33 | 5.30 ± 2.09 | 1.05 ± 0.77 | 6.77 ± 2.74 | 6.08 ± 3.02 | 0.83 ± 1.26 | 0.61 |
| Vena cava inf. (mm) | 27.36 ± 4.78 | 23.46 ± 3.64 | 4.61 ± 3.27 | 28.67 ± 4.05 | 25.18 ± 5.25 | 4.00 ± 2.44 | 0.61 |

Data are expressed as the mean value ± standard deviation

AP4 — apical four-chamber view; EROA — effective regurgitant orifice area; LA — left atrium; LVEDD — left ventricular end-diastolic diameter; LVEF — left ventricular ejection fraction; MR — mitral regurgitation; PISA r. — proximal isovelocity surface area radius; PLAX — parasternal long axis view; RV — right ventricle; RVOT — right ventricle outflow tract; TAPSE — tricuspid annular plane systolic excursion; TR — tricuspid regurgitation; VC ± vena contracta

**Table 5.** Changes in echocardiographic parameters during hospitalization depending on heart rate reduction

| Parameter | Heart rate reduction >10 bpm (n = 18) | | | Heart rate reduction <10 bpm (n = 16) | | | p-value |
|---|---|---|---|---|---|---|---|
| | Admission | Discharge | Value change | Admission | Discharge | Value Change | |
| Dyspnoea score (0–100) | 80.77 ±11.15 | 44.61 ± 7.76 | 36.15 ± 14.45 | 82.14 ± 6.99 | 45.38 ± 13.91 | 36.92 ± 14.93 | 0.89 |
| LA PLAX (mm) | 55.38 ± 6.97 | 50.84 ± 6.99 | 4.53 ± 3.59 | 53.71 ± 6.42 | 50.69 ± 5.86 | 3.53 ± 5.04 | 0.56 |
| LA area AP4 ($cm^2$) | 37.69 ± 9.30 | 33.80 ± 11.39 | 3.88 ± 4.00 | 33.59 ± 8.71 | 31.13 ± 7.02 | 2.88 ± 2.58 | 0.46 |
| RA area AP4 ($cm^2$) | 32.88 ± 5.56 | 29.42 ± 8.29 | 3.46 ± 5.12 | 32.14 ± 9.47 | 29.07 ± 9.96 | 3.77 ± 2.37 | 0.91 |
| LV EDD (mm) | 66.23 ± 9.02 | 63.84 ± 8.37 | 2.38 ± 2.56 | 55.42 ± 11.05 | 52.84 ± 10.85 | 2.92 ± 2.75 | 0.61 |
| LVEF (%) | 30.56 ±13.96 | 38.69 ± 13.29 | **8.13 ± 5.19** | 42.64 ± 14.95 | 41.91 ± 13.46 | **2.66 ± 6.00** | **0.02** |
| LVOT VTI (cm) | 12.61 ± 3.64 | 15.55 ± 4.88 | **2.94 ± 3.55** | 16.18 ± 5.64 | 15.04 ± 4.45 | **–0.14 ± 3.38** | **0.04** |
| RV RVOT (mm) | 41.07 ± 5.65 | 38.30 ± 4.97 | 2.76 ± 2.35 | 36.92 ± 4.15 | 35.38 ± 3.88 | 1.61 ± 3.79 | 0.36 |
| TAPSE (mm) | 14.69 ± 4.78 | 16.30 ± 3.72 | 1.08 ± 2.43 | 15.42 ± 5.03 | 15.00 ± 3.71 | 0.50 ± 2.61 | 0.26 |
| MR VC (mm) | 6.56 ± 1.52 | 5.53 ± 1.5 | 0.95 ± 0.75 | 5.78 ± 1.32 | 4.95 ± 1.74 | 0.87 ± 0.74 | 0.64 |
| MR PISA r. (mm) | 8.46 ± 2.33 | 6.61 ± 1.94 | 1.48 ± 1.04 | 7.21 ± 2.75 | 6.08 ± 2.60 | 1.08 ± 0.79 | 0.06 |
| MR ERO ($cm^2$) | 0.40 ± 0.20 | 0.24 ± 0.18 | 0.11 ± 0.07 | 0.30 ± 0.12 | 0.22 ± 0.13 | 0.08 ± 0.04 | 0.09 |
| MR vol. (mL) | 49.00 ±23.87 | 32.46 ± 21.09 | 14.04 ± 10.78 | 41.00 ± 20.72 | 29.17 ± 16.65 | 11.33 ± 8.53 | 0.23 |
| TRPG (mm Hg) | 47.38 ±10.25 | 40.69 ± 9.93 | 4.84 ± 8.82 | 41.42 ± 12.99 | 37.67 ± 12.44 | 2.83 ± 7.51 | 0.28 |
| TR V max (m/s) | 3.43 ± 0.36 | 3.16 ± 0.39 | 0.18 ± 0.34 | 3.17 ± 0.54 | 3.01 ± 0.55 | 0.11 ± 0.34 | 0.27 |
| TR VC (mm) | 6.27 ± 2.19 | 5.31 ± 2.28 | 0.94 ± 1.02 | 6.57 ± 2.84 | 6.08 ± 2.87 | 0.91 ± 1.08 | 1.00 |
| Vena cava inf. (mm) | 28.84 ± 3.43 | 24.00 ± 4.89 | 4.33 ± 2.88 | 27.07 ± 5.21 | 24.54 ± 4.03 | 3.72 ± 3.16 | 0.35 |

Data are expressed as the mean value ± standard deviation

AP4 – apical four-chamber view; EROA – effective regurgitant orifice area; LA – left atrium; LVEDD – left ventricular end-diastolic diameter; LVEF – left ventricular ejection fraction; MR – mitral regurgitation; PISA r. – proximal isovelocity surface area radius; PLAX – parasternal long axis view; RV – right ventricle; RVOT – right ventricle outflow tract; TAPSE – tricuspid annular plane systolic excursion; TR – tricuspid regurgitation; VC ± vena contracta

To conclude, echocardiography may be used not only to diagnose ADHF at admission but also to be a useful non-invasive tool to monitor the course of ADHF treatment. Decrease of fluid overload results mainly in dyspnoea reduction, mitral regurgitation downscaling and pulmonary pressure lowering. The heart rate reduction is followed by a significant increase in left ventricle stroke volume and left ventricle ejection fraction. Such echocardiographic changes are expected during successful ADHF treatment. Their absence may direct the attention of the attending physician to the need for more aggressive treatment or a worse prognosis for the patients.

### Limitations of the study

A limitation of this study is the fact that it is a single-centre analysis with a relatively small study group. This study reflects standard treatment for ADHF in a university hospital, although it may affect the generalizability of the findings to a broader population. The study population was heterogeneous regarding admission left ventricular ejection fraction with a prevalent group of patients (over 70%) with heart failure with reduced ejection fraction (HFrEF). However, a subgroup of patients from heart failure with mildly reduced ejection fractiogroup showed patterns similar to those in the HFrEF group, albeit to a lesser degree.

This study is constrained by its limited follow-up duration, and investigations into the more extended effects and outcomes associated with decongestion therapy and heart rate reduction necessitate larger future studies. Furthermore, the analysis only considered a limited number of potential factors that could influence the observed outcomes. While the authors focused on specific factors relevant to the research question, it's important to recognize that various other variables and influences may exist. This limitation implies that there may be additional unexplored factors that could have had an impact on the results.

Finally, this study is observational in nature, meaning it observes and reports associations rather than demonstrating causation.

## Additional information

### Data availability statement

Available upon request.

### Ethics statement

Before enrolment, all patients provided written informed consent to participate in the study, the protocol of which was approved by the Bioethical Commission of the Jagiellonian University in Krakow (No. 122.6120.294.2015). The study was conducted in accordance with the ethical guidelines of the Declaration of Helsinki of 1975.

### Author contributions

Conceptualization: KB, WP; Writing — original draft preparation: KB; Writing — review and editing: KB, WP, LD; Supervision: WP; Funding acquisition: WP; Statistics supervision: PB.

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None.

### Conflict of interest

All authors declare no conflicts of interest.

### Supplementary material

None.

### Streszczenie

**Wstęp.** Leczenie ostrej niewydolności serca (ADHF) ukierunkowane na redukcję zastoju prowadzi do zmian parametrów hemodynamicznych oraz częstości rytmu serca. Oceniono wpływ leczenia odwadniającego oraz zmniejszenia częstości rytmu serca podczas leczenia ADHF na parametry pracy serca w analizie echokardiograficznej.

**Materiał i metody.** W grupie 34 pacjentów (41,2% kobiet, średni wiek 70,2 ± 10,8 lat) hospitalizowanych z powodu objawów ADHF przeprowadzono ocenę echokardiograficzną przy przyjęciu oraz przy wypisie wraz z oceną kliniczną oraz laboratoryjną.

**Wyniki.** Stwierdzono istotną redukcję masy ciała (84,22 ± 17,46 *vs.* 78,72 ± 16,95 kg; p < 0,001), częstości rytmu serca (86,21 ± 15.7 *vs.* 75,72 ± 11,01 bpm; p < 0,001) oraz poziomu NT-proBNP (7530,22 ± 5192,52 *vs.* 3270,32 ± 2947,26 pg/ml; p = 0,001). W grupie chorych, u których uzyskano redukcję masy ciała o co najmniej 5 kg stwierdzono znamienną redukcję nasilenia duszności ocenionej wg standaryzowanej skali VAS 0–100 pkt (ΔVAS przy przyjęciu/przy wypisie 42,14 ± 13,68 *vs.* 30,00 ± 12,79; p = 0,04), gradientu ciśnienia fali zwrotnej trójdzielnej (ΔTRPG 7,53 ± 9,36 *vs.* 1,91 ± 7,48 mm Hg; p = 0,05) oraz nasilenia niedomykalności mitralnej (ΔMR VC 1,21 ± 0,65 *vs.* 0,66 ± 0,77 mm; p = 0,05). W podgrupie chorych, u których uzyskano redukcję częstości rytmu serca > 10 bpm rejestrowano wzrost frakcji wyrzutowej lewej komory (redukcja HR > 10 bpm: przy przyjęciu 30,56 ± 13,96; przy wypisie 38,69 ± 13,29 *vs.* redukcja HR < 10 bpm 42,64 ± 14,95; 41,91 ± 13,46%; p = 0,02) oraz całki przepływu od czasu w drodze odpływu lewej komory (redukcja HR > 10 bpm: przy przyjęciu 12,61 ± 3,64; przy wypisie 15,55 ± 4,88 *vs.* redukcja HR < 10 bpm: 16,18 ± 5,64; 15,04 ± 4,45 cm; p = 0,04).

**Wnioski.** Leczenie przewodnienia w ADHF skutkuje głównie redukcją nasilenia duszności, stopnia niedomykalności mitralnej oraz ciśnienia w prawej komorze. Przy redukcji częstości rytmu serca obserwowano wzrost frakcji wyrzutowej lewej komory oraz objętości wyrzutowej lewej komory. Powyższych zmian w analizie echokardiograficznej powinniśmy spodziewać się w trakcie skutecznego leczenia ostrej niewydolności serca, podczas gdy ich brak może skłonić ku intensywniejszej terapii, a także informować o gorszym rokowaniu chorych.

Słowa kluczowe: ostra niewydolność serca, echokardiografia, leczenie odwadniające, rytm serca

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