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Short title: Expert opinion on the peri-discharge management of the patients with heart failure

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INTRODUCTION

Despite advances in the treatment of heart failure (HF), the rate of hospitalisation for exacerbations of the disease remains high. One of the underlying reasons is that recommended guidelines for the management of HF are still too rarely followed in daily practice. Disease exacerbation requiring inpatient treatment is always a factor that worsens the prognosis, and thus signals disease progression. This is also a key moment when therapy should be modified for HF exacerbation, or initiated
in the case of newly diagnosed disease. Inpatient treatment and the peri-discharge period is the time when the aetiology and mechanism of HF decompensation should be established. Therapy should be individualised based on aetiology, HF phenotype, and comorbidities; it should take into account the possibilities of modern treatment. According to the recommendations of the European Society of Cardiology (ESC), patients with HF should receive multidisciplinary management. Cooperation between the various members of the multidisciplinary team taking care of patients with HF improves the efficiency and quality of treatment. This document expands and details the information on the peri-discharge management of HF contained in the

2021 ESC guidelines and the 2022 American Heart Association (AHA)/American College of Cardiology (ACC)/Heart Failure Society of America (HFSA) guidelines.

HOSPITALISATIONS FOR HEART FAILURE — A MEDICAL, EPIDEMIOLOGICAL AND PROGNOSTIC PROBLEM

Heart failure is a progressive condition with periods of exacerbations, periodically also requiring intravenous treatment and modification of medical management [1]. Hospitalisations resulting from HF exacerbations significantly worsen the prognosis of patients. In Poland, current data on hospitalisations of HF patients were obtained from an analysis conducted by the Ministry of Health (MoH), covering the entire adult population of Poland (41 532 268 people) from 2013 to 2018, focusing on people with a diagnosis of HF (1 686 861 people). In this group, almost half of the patients (817 432 people; 48.5%) were hospitalised. It was shown that between 2013 and 2018, the number of hospitalisations increased by as much as 33% (2013 — 198 881; 2018 — 264 808). Since 2008, the rate of hospitalisation for HF in Poland has been the highest among the Organization for Economic Cooperation and Development countries. The cost of hospitalisation of HF patients increased by 125% between 2015 and 2020 in Poland. Expenditures of the National Health Fund (NHF) related to HF in Poland were estimated at PLN 6.2 billion in 2018, accounting for as much as 0.3% of GDP [2, 3]. The higher incidence of readmission was seen primarily in women over 65 years of age, with comorbidities [4, 5].

Inpatient stay should be a key time for optimising therapy and changing the existing treatment. However, in daily practice, in most cases, medications prescribed at discharge are based on a pharmacotherapy regimen similar to that before hospitalisation, that is a regimen that has proven ineffective in preventing cardiovascular destabilisation [6]. Moreover, early initiation and intensification of pharmacotherapy does not occur in the peri-discharge period, although numerous studies have shown that this is a safe procedure associated with improved patient prognosis [7–10]. Long-term observations have shown that the post-discharge period, especially the first 30 days, is the time when cardiovascular events, exacerbation of HF and the need for readmission are most common [11]. Haemodynamic destabilisation and readmissions are factors that particularly worsen the prognosis of patients with HF [1, 6, 11]. This is also indicated by Polish data from the MoH, according to which the chance of surviving 720 days from hospital discharge decreases significantly as the number of subsequent hospitalisations increases. With one hospitalisation, the survival rate is 66.4%, and with four or more it is only 43.9%.

BASELINE PHENOTYPE AND RESPONSE TO HOSPITAL TREATMENT AS DETERMINANTS OF POST-DISCHARGE MANAGEMENT

Clinical knowledge shows that in patients with acute heart failure (AHF), the quality of treatment in the period immediately after hospital discharge fundamentally affects short- and long-term morbidity and mortality [12]. Factors to be considered include individualised escalation of therapy, monitoring of its effectiveness and possible side effects of drugs, as well as rehabilitation carried out early after hospitalisation. In practice, the implementation of HF treatment recommendations is not sufficient. The reasons for this may depend on both the patient and the health care system, and may also be conditioned by the social environment and the psychological profile of the patient. The sum of these factors is called the patient's clinical phenotype, whose identification during a given hospitalisation significantly modifies the possibilities of implementing, escalating and sustainably continuing the recommended treatment [13].

According to the individual natural history of HF, the patient's phenotypic features can be grouped according to the chronology of treatment, from the first clinical presentation, contact with the health care system, to the patient's discharge from the hospital. For each hospitalisation, the cycle of events is similar and several groups of such factors can be mentioned:

- the patient's historical data known at the time of admission;
- the clinical presentation of HF, including its aetiology and the cause of decompensation;
- inpatient response to treatment and adverse events;
- individual determinants of patient cooperation after discharge.

The medical records provided, as well as taking a thorough history from the patient and his/her family, are irreplaceable sources of information. An effort should be made to gather as much data as possible, not only on cardiovascular risk factors and comorbidities, but also to establish a chronology of events. Non-medical data, including social, psychological and other issues, are also useful in planning patient care. The information obtained makes it possible to identify factors limiting the implementation, escalation and maintenance of recommended therapy after discharge. Among the most significant factors are [14]:

- HF etiology, if already established;
- the age of the patient, taking into account differences between chronological and biological age;
- number of previous hospitalisations for cardiovascular decompensations;
- duration and complications observed during previous hospitalisations;

- the time from the onset of the first symptoms of concern to the patient's contact with a physician and initiation of treatment (for previous and current hospitalisations);
- the presence of comorbidities, especially atrial fibrillation (AF), type 2 diabetes mellitus (T2DM), chronic obstructive pulmonary disease (COPD), cancer, chronic kidney disease (CKD), liver failure, anaemia, and neurological conditions progressing with dementia;
- changes in "oedema-free" body weight during HF (losses and gains after hospitalisations), with determination of percentage weight loss compared to the pre-HF period;
- frailty syndrome;
- the presence of right ventricular dysfunction in previous hospital stays;
- left ventricular ejection fraction (LVEF) during previous hospitalisations.
- treatment used to date, in particular the type and doses of drugs recommended in the guidelines and the doses of diuretics;
- problems with patient adherence known from previous hospital stays (non-compliance, abandonment of medications, lack of conscious control of fluid supply, diuresis and body weight, etc.);
- mood disorders, depression and other mental illnesses.

A still underestimated factor that determines a patient's subsequent outcome is the delay between the appearance of the first symptoms of an HF exacerbation and medical intervention [15]. Investigations conducted in the first hours of hospitalisation should provide answers to further relevant questions. Among these, not only the aetiology of HF, if already established, but the specific circumstances and factors that may be responsible for the current cardiovascular decompensation are crucial. In addition to conducting an analysis of acute causes of HF according to the CHAMPIT (acute Coronary syndrome/Hypertension emergency/Arrhythmia/acute Mechanical cause/Pulmonary embolism /Infections/Tamponade) algorithm, an effort to elucidate the non-aetiological causes of disease exacerbation is essential [13]. Determining the aetiology in the case of *de novo* HF presentation and searching for the causes of decompensation of previously stable HF can reveal the clinical circumstances – the specific phenotype of the patient – that determine further management. Undertaking treatment appropriate to the identified problem can clearly modify the extent of HF treatment after discharge [13].

Among the most important aetiological factors are:

- acute coronary syndromes (ACS) with the need for invasive treatment (revascularisation);

- valve diseases for which invasive treatment can be used;
- infections, especially those requiring surgical management and long-term antimicrobial treatment (infective endocarditis or lead-related endocarditis, infected bedsores and others);
- dysfunctions of implanted cardiac devices;
- thromboembolism;
- central nervous system ischaemic events:
- arrhythmia;
- discontinuation or inappropriate use of pharmacotherapy, side effects of drugs (especially nephrotoxic or leading to thyroid dysfunction), alcohol and illegal drugs;
- clinically significant bleeding;
- malignant neoplasms and their treatment;

In parallel and independently of aetiologic diagnosis and the causes of cardiovascular decompensation, the clinical presentation of HF itself can also influence post-discharge treatment. Current guidelines distinguish four main phenotypes of AHF: acute pulmonary oedema, decompensated chronic heart failure (CHF), isolated right ventricular HF and cardiogenic shock. However, it is important to note that overlap between these phenotypes is possible in individual patients. The most important phenotypic features identified at the time of admission that may pose serious limitations to the use of recommended therapies after discharge are summarised below [11]:

- class IV according to New York Heart Association (NYHA);
- “cold/wet” and “cold/dry” haemodynamic profiles of AHF;
- low blood pressure (BP);
- high natriuretic peptide levels, elevated troponin levels, hyponatraemia, high urea levels and high urea/creatinine ratio;
- impaired glomerular filtration, especially in those with a documented high percentage loss of “oedema-free” body weight;
- low (<50–70 mEq/l) urinary sodium concentration 3 hours after intravenous loop diuretic administration;
- increased multiorgan congestion, especially with the presence of exudative fluid in body cavities;
- no prior treatment with renin-angiotensin-aldosterone system (RAAS) blockers and beta-blockers.

In addition to aetiological intervention and treatment of the cause of cardiovascular decompensation, elimination of congestion and/or organ hypoperfusion usually requires diuretics, in some phenotypes vasodilators, and in others drugs that increase myocardial contractility and peripheral vascular resistance. Determining the target condition, which is complete resolution of congestion and/or hypoperfusion, and initiation or escalation of therapy recommended in the guidelines, and tracking the clinical response to this treatment (based on daily examination and laboratory test results) allows to define four basic clinical courses:

- steady clinical improvement toward a defined goal;
- initial clinical improvement, followed by stabilisation without reaching the target;
- steady clinical improvement, but with worsening clinical parameters and additional test results (hypotonia, bradycardia, hyponatraemia, greater than expected deterioration of renal function, hyperkalaemia, metabolic alkalosis), individual or in combinations;
- clinical worsening.

With the exception of the first one, all of the above scenarios require management modifications and may affect post-discharge management. Of paramount importance is the effectiveness of eliminating congestion, especially using the current recommendations for diuretic treatment (this factor is critical in maintaining clinical stability) and adequate treatment of comorbidities [16, 17]. Post-discharge treatment tactics and strategies can also be influenced by clinical adverse events observed during therapy. The same factors that are one of the causes of the initial exacerbation of HF can also later be complications of treatment.

The individual determinants of patient cooperation after discharge are among the least appreciated factors determining successful HF therapy. Measures to improve this cooperation are not implemented often enough. The factors that have the greatest impact on the effectiveness of cooperation include:

- the patient’s level of education and occupation;
- place of residence with special attention to the possibility of efficient contact with the various levels of the health care system (primary health care, cardiology outpatient clinic, hospital emergency department/emergency room, hospital ward), laboratory, pharmacy;
- opportunities for the patient and his family to use telemedical technologies during treatment;
- economic status;
- family and neighborhood environment.

METHODS FOR ASSESSING PROGNOSIS IN HEART FAILURE RISK STRATIFICATION OF READMISSIONS AND DEATH AFTER DISCHARGE AND THEIR PRACTICAL UTILITY

There are many factors that are associated with a particularly poor prognosis in patients with HF [18]. These include disease progression expressed as consecutive stages A to D, NYHA classes I to IV, and in the group with severe HF, the INTERMACS scale of 7 to 1. The risk is particularly high in patients after multiple hospitalisations for cardiovascular decompensation, in patients with CKD and other comorbidities [19, 20]. The prognosis is worse with decreasing LVEF, in patients with spherical left ventricular (LV) geometry (sphericity index >0.7) and with concomitant, haemodynamically significant valve diseases (especially mitral and/or tricuspid regurgitation) [21]. There is also an increased risk of decompensation or death in patients with a restrictive LV filling profile, significantly reduced LV longitudinal fibre function (reduced mitral annular velocities and longitudinal strain) [22]. A higher risk is observed in patients with right ventricular enlargement and dysfunction, pulmonary hypertension (tricuspid regurgitation velocity >2.8 m/s, mean pulmonary artery pressure >30 mm Hg) [23]. Patients >65 years of age, males, non-compliant patients, patients with depression, low body weight (cachexia) and nutritional deficiencies (including iron), ongoing infections and high natriuretic peptide levels also have a worse prognosis [24].

Prognosis in HF is unfavourable in terms of both life expectancy and risk of hospitalisation. According to data from the ESC Heart Failure Long-Term Registry, the prognosis is significantly worse in patients who were hospitalised than in outpatients. The annual overall mortality rate in the first group was 23%, while in the second group it was 6.4%; the composite endpoint (overall mortality or HF hospitalisation) in the first group was 35%, in the second 23% [25]. A huge problem are frequent readmissions, especially those in the first 30 days after discharge. According to Spanish data from 2003–2011, the rate of readmissions increased by 1.36% per year, from 17.6% to 22.1% [26]. The majority of hospitalisations had a cardiovascular cause (60%), with HF in first place. However, in recent years, attention has been drawn to the fact that conditions other than HF are responsible for a large proportion of hospitalisations [20]. These data indicate the need for appropriate treatment of comorbidities in patients with HF. It is noteworthy that 1 in 6 patients discharged after cardiovascular decompensation is readmitted urgently to the hospital within 30 days of discharge [27]. The association of repeated hospitalisations due to HF exacerbations with long-term prognosis has also been pointed out in other works. In one of them, 30-day mortality rate was determined to be 7.4%, and one-year mortality rate was 27.3% after hospitalisation [28]. Each subsequent

hospitalisation was associated with a shorter survival. The average survival after the first hospital stay for HF was 2.6 years, 1.8 years after the second, 1.5 years after the third, and only 1.3 years after the fourth hospitalisation. However, the authors point out that this does not prove that reducing the frequency of readmissions would reduce mortality [28]. Further studies are needed to better understand the impact of readmission on HF progression.

There is no single ideal prognostic indicator in HF. Such assessment is always multifactorial, depending, in addition to the above-mentioned determinants, also on the aetiology of HF and the assessment of the reversibility of its cause (e.g., successful revascularisation of the coronary arteries in patients with ischaemic cardiomyopathy, successful treatment of a valve disease). Only a holistic view of these factors allows an experienced clinician to estimate the risk of serious complications and select patients for whom special care should be provided. Such analysis is not entirely accurate – a small number of risk factors and seemingly early disease progression do not mean that a given patient's prognosis is good [24]. In recent years, the MAGGIC scale, constructed from an analysis of data from 39 372 HF patients with preserved (HFpEF) and reduced ejection fraction (HFrEF) from 30 clinical trials, has been increasingly used; the scale includes 13 prognostic parameters [29]. These easily available scored indices include age, male sex, LVEF, NYHA class, creatinine level, not using beta-blockers, not using angiotensin-converting enzyme inhibitors (ACEIs)/angiotensin receptor blockers (ARBs), systolic blood pressure (SBP), body weight, time since HF diagnosis, smoking, presence of T2DM and COPD. In the MAGGIC study, the median score was 23. Low risk, defined as <17 points, was associated with a 3-year risk of death of 10%. In contrast, very high risk (>33 points) was associated with a 3-year risk of death of 70%. A calculator to determine the one-year and 3-year risk of death in HF can be found at www. heartfailurerisk.org. An analysis of data from the above-mentioned ESC Heart Failure Long-Term Registry showed that less than 1% of practicing physicians assessed the prognosis of their HF patients using the available scales [30]. Such patient assessment is not simple, but very useful. The finding of a worse prognosis based on the calculation of a composite index, such as the MAGGIC score, is a signal for more intensive treatment, close, more frequent monitoring of the course of HF, and possibly referring the patient to a transplantation centre or palliative care.

NON-PHARMACOLOGICAL MANAGEMENT IN THE PERI-DISCHARGE PERIOD

A very important aspect of therapy is non-pharmacological management. Current guidelines devote considerable attention to non-pharmacological interventions that should be implemented during hospitalisation (Table 1) [24].

With regard to the tasks, as well as where these goals are pursued during the peri-discharge period, non-pharmacological management consists of three phases: the pre-discharge optimisation phase, the discharge and the early post-discharge phase (Figure 1) [14].

The essential tasks of the team coordinating the treatment of a patient with HF include [31]:

- HF diagnosis and monitoring for disease progression;
- prescribing treatment, optimising and monitoring HF therapy;
- patient and caregiver education about the disease and treatment;
- lifestyle education and recommendations (regarding diet, physical activity and stimulants, among others);
- assessing the need for psychological and social support;
- coordination of care for comorbidities;
- counseling and palliative care at the end of life.

The ESC guidelines support the multidisciplinary team care of patients with HF. In the pre-discharge phase, the team should provide clinical assessment, optimisation of therapy, patient education and a post-discharge care plan. Clinical evaluation of the patient in the hospital should include daily measurements of the following parameters: BP, heart rate (HR) and respiratory rate, body weight and fluid retention levels. Periodically, it is also advisable to measure levels of biomarkers of myocardial overload and damage (B-type natriuretic peptide [BNP]/N-terminal pro-B-type natriuretic peptide [NT-proBNP], troponin), and assess renal function (creatinine/estimated glomerular filtration rate [eGFR], urea, electrolytes) [13].

In the peri-discharge period, patient education is a very important aspect affecting the effectiveness of the interventions provided. This period should be used to comprehensively discuss with the patient issues such as general knowledge of HF and prognosis, monitoring of vital signs, symptoms of fluid overload, and fluid intake. Implementing education and teaching patients to self-manage their symptoms reduces the risk of both HF and all-cause hospitalisation (by 34% and 27%, respectively) [32]. Nurse-led face-to-face education is the most commonly chosen strategy in educating patients with HF.

It should include the following topics discussed in a comprehensible manner with the patient:

- Basic information about the definition, cause and course of HF (including prognosis).
- Basic knowledge of pharmacotherapy (drugs, dosage, side effects, contraindicated drugs).
- Essential information on implantable devices, percutaneous or surgical interventions.
- Information on diet and use of stimulants (alcohol, cigarettes, use of psychoactive substances).

- Knowledge about engaging in sexual activity.
- Information on prophylactic vaccination.
- Information on safe travelling.

Observations have shown that the rate of readmission within 30 days of the last hospital stay was significantly lower in the group of HF patients educated by a nurse (20.4%) compared to the group without education (50.0%) [33, 34]. It is also important to remember the need to educate family members and relatives of HF patients. Education and family support contribute to better adherence to pharmacological and dietary recommendations, and patients show better motivation and self-confidence [35, 36]. Special attention should be given to diuretic treatment with practical education on both diuretic dosage and fluid intake, as well as monitoring for symptoms of fluid overload. Symptom monitoring is an important aspect of patient-physician collaboration and should include assessment of: shortness of breath, fatigue, BP, HR and body weight. These observations should be kept in the form of a diary/passport. It is important to teach the patient which symptoms are cause for concern (e.g. increased shortness of breath and/or oedema, or rapid weight gain of more than 2 kg in 3 days), and how to contact medical staff if there is an increase in symptoms that may indicate incipient cardiovascular decompensation. Cooperating patients can be taught to modify diuretic treatment and potassium supplementation depending on the severity of their complaints, control of renal function (creatinine/eGFR) and electrolyte levels. Patient involvement in self-management of symptoms and modification of diuretic treatment reduces the risk of HF hospitalisation and mortality [13]. The Polish Society of Cardiology has launched an educational portal for patients with HF (www.slabeserce.pl), where patients can improve their knowledge of the disease through accessible and understandable content. This portal can also be used as a supportive tool to help educate patients. The Heart Failure Patient Passport can be downloaded from: https://niewydolnosc-serca.pl/sprawozdanie/paszport-pacjenta_z%20NS.pdf. A certified nursing education program is also available for nurses who would like to expand their competencies regarding the care of HF patients, and become specialised HF educators.

The onset of HF is accompanied by the onset of depressive symptoms, loneliness, anxiety and withdrawal [37, 38], so it is advocated that psychosocial support be provided to patients, their families and/or caregivers. In recent years, cognitive behavioural therapies based on mindfulness techniques, applied in a group of patients with HF, have confirmed the significant effect of this type of intervention on reducing depressive symptoms [39–41].

Patients admitted to the hospital for an exacerbation of HF can be discharged home if [42]:

- they are clinically stable (no signs of cardiovascular decompensation – in extreme HF this condition not always can be met) and haemodynamically stable;
- are in euvolemia, and their renal function parameters have been stable for >24 hrs;
- have been properly educated in the context of both self-monitoring and HF itself.

The patient, when leaving the hospital, should receive [43]:

- a discharge letter with details of his/her hospital stay;
- recommendations for prevention and monitoring of symptoms;
- information specifying the course of rehabilitation;
- recommendations for post-discharge management regarding both the patient and his/her primary care physician (PCP).

It is also advisable to schedule a follow-up visit within 1–2 weeks after discharge from the hospital (Table 2). Such early outpatient follow-up (preferably on day 7) is primarily aimed at assessing signs of fluid overload, tolerability of pharmacotherapy and the possible need to change the treatment, including doses of disease-modifying drugs and diuretics. The introduction of a follow-up visit on day 7 after discharge reduces the rate of 30-day readmission by 30% [13, 44–47]. In the early post-discharge phase, it is extremely important for patients to consciously and responsibly perform self-monitoring with regard to the presence of clinical symptoms, BP, heart rate, body weight, periodic assessment of clinical chemistry parameters (in PCP or cardiology clinic setting), diet and physical activity [48]. It is recommended that patients with HF undergo regular medical checks, whose frequency depends on the stage of treatment and the disease itself in a given patients. When planning care after an exacerbation of HF requiring hospital treatment, follow-up visits should be more frequent and scheduled at the time of patient discharge from the hospital. During post-hospital follow-up, indications for electrotherapy (implantable cardioverter-defibrillator, cardiac resynchronization therapy) should also be verified with >3-month period of optimal pharmacotherapy. In the period between exacerbations, once the patient's condition is stabilised and all planned interventions have been carried out, outpatient check-ups may occur less frequently, but no less than once every 6 months. These visits should take place regardless of the presence/severity of symptoms in order to optimise the pharmacotherapy and detect asymptomatic disease progression early. Patients with a history of HF exacerbation and significant modification of pharmacotherapy should be monitored more frequently, but the guidelines do not specify at what intervals. Recommendations for the frequency of follow-up visits in CHF — according to ESC guidelines — are shown in Table 2 [13].

INDIVIDUALISATION OF THERAPY — AN IMPORTANT ASPECT OF DISCHARGE MANAGEMENT

According to the 2021 ESC guidelines, optimising therapy after hospitalisation for AHF reduces the risk of readmissions, cardiovascular death and improves quality of life. Individualisation of therapy in HF is one of the areas of emphasis in current guidelines and it is based on clinical profiles that take into account the following data [13, 49]:

- BP;
- HR;
- heart rhythm type (especially the presence of AF);
- renal function and/or hyperkalaemia;
- fluid overload.

The individualisation of therapy should also take into account the patient's preferences and abilities. The guidelines place particular emphasis on careful assessment of the features of fluid overload in patients before discharge, and optimisation of oral diuretic treatment. In fact, the presence of features of fluid overload in a patient discharged after an exacerbation of HF is associated with a high risk of death and readmissions [50, 51]. For patients not previously treated with beta-blockers, but who show features of fluid overload, these drugs should not be the first line of therapy, as they may lead to clinical deterioration.

In the pre-discharge period (once acute cardiovascular decompensation is controlled), it is necessary for patients with HFrEF to include oral medications to improve prognosis. This stage is possible in those patients who have achieved haemodynamic stability and have no significant fluid retention. The introduction of these drugs into therapy requires consideration of both the clinical profile and the form of AHF (*de novo*, CHF exacerbation), as highlighted above. Primary medications for HFrEF that modify the course of the disease include beta-blockers, ACEI/ARB/angiotensin receptor neprilysin inhibitors (ARNI), mineralocorticoid receptor antagonists, (MRA) and sodium-glucose co-transporter 2 (SGLT2) inhibitors [13, 52, 53]. The TRANSITION and PIONEER-HF trials confirmed the clinical benefits of ARNI therapy in patients hospitalised for acute manifestation of HFrEF, both *de novo* and as CHF exacerbation [9, 10]. On the other hand, the PERSPECTIVE study – presented during the recent ESC 2022 congress in Barcelona – showed that ARNI does not impair cognitive function compared to valsartan in patients with HFmrEF or HFpEF, although there was a reduction in the deposition of β-amyloid in the brain in patients treated with ARNI requires further research. The results of the studies showed that initiating ARNI therapy in the pre-discharge period is safe and is associated with early and sustained improvements in reducing the risk of major cardiovascular

events and lowering biomarkers (NT-proBNP, troponin). It is noteworthy that patients with *de novo* HF benefited most from ARNI therapy introduced in the pre-discharge period. ARNI treatment can be started if SBP is not <100 mm Hg, eGFR is >30 mL/min/1.73 m^2, and potassium is <5.4 mmol/L. In persons previously receiving ACEI, 36 hours must elapse from the last dose of the drug. Given the current state of knowledge, in the opinion of the experts of the Heart Failure Association of the Polish Society of Cardiology, ARNI (sacubitril/valsartan) should be the preferred drug over ACEI/ARB in patients with HFrEF. This is supported by the recommendations in the latest 2022 AHA/ACC/HFSA guidelines.

The clinical benefits of beta-blocker treatment in HFrEF have been confirmed in a number of studies. Moreover, retrospective analyses have documented that dose reductions of these drugs or their discontinuation in patients hospitalised for HF exacerbation were associated with a worse prognosis [54]. The inclusion or continuation of MRA and SGLT-2 inhibitor therapy, on the other hand, can be safely carried out even in patients with low SBP values (<90 mm Hg), with the exception of those with coexisting chronic coronary syndrome (CCS) for whom SBP >120 mm Hg is recommended [13]. The EMPA-RESPONSE-AHF trial in patients with AHF treated with empagliflozin reported a reduction in the risk of a composite endpoint consisting of worsening HF, readmissions, and cardiovascular death at 60-day follow-up [55]. On the other hand, in the SOLOIST-WHF study in patients with T2DM and HF exacerbation, treatment with sotagliflozin, initiated before or shortly after discharge, resulted in a significantly lower total number of cardiovascular deaths and HF hospitalisations and urgent visits compared to placebo [56]. The latest EMPAG-HF study shows that early inclusion of empagliflozin in standard diuretic therapy increases the effectiveness of diuresis without adversely affecting renal function in patients with AHF. These results somewhat coincide with the EMPULSE study mentioned below, which showed, among others, safety of empagliflozin therapy in stable patients just after an episode of AHF.

It is worth recalling that high HR is an unfavourable prognostic factor at discharge. Reducing HR is an important therapeutic goal in the treatment of HFrEF. This type of strategy is beneficial for patients with sinus rhythm and HR greater than or equal to 70 bpm. The ETHIC-AHF trial and the Optimize Heart Failure Care programme have demonstrated that intensification of treatment before discharge with concomitant administration of beta-blockers and ivabradine to patients stabilised after decompensated HFrEF resulted in both benefits as early as in the first month of therapy (higher percentage of patients with HR <70 bpm) and after one year of follow-up [8, 57, 58]. For patients treated with beta-blockers and ivabradine, improved LVEF, reduced risk of death and readmission for HF, and better quality of life have been reported after

12-month follow-up [57, 58]. Although according to the latest ESC guidelines, it is optimal to use representatives of all four drug groups (beta-blockers, ACEI/ARB/ARNI, MRAs, and SGLT2 inhibitors), even at the expense of possibly not reaching target doses, this is not always possible in daily practice [13]. Table 3 shows the clinical profiles with each drug group. The therapy established before discharge is the starting point for further optimisation in the outpatient setting. The pre-discharge period usually does not allow for achieving optimal doses of the listed HF course-modifying drugs, so after the patient is discharged from the hospital, it is necessary to gradually increase them until the target or maximum drug doses tolerated by the patient are reached. Such information should be included in the hospital discharge letter and in the information to the family doctor.

While for the main pharmacotherapy of HFrEF, the current ESC and AHA/ACC/HFSA guidelines are, except for the positioning of ARNI versus ACEI/ARB, convergent (the use of beta-blockers, ACEI/ARB/ARNI, MRAs and SGLT2 inhibitors has a Class I recommendation), some important differences emerge for patients with LVEF >40%. For patients with heart failure with mildly reduced ejection fraction (HFmrEF), the ESC guidelines recommend (to reduce the risk of HF hospitalisation and death) the use of beta-blockers, ACEI/ARB/ARNI, MRAs and SGLT2 inhibitors in recommendation Class IIb, without specifying (beyond treatment of concomitant diseases and control of risk factors) recommendations for pharmacotherapy to improve prognosis in HFpEF patients. In part, this was due to the fact that the guidelines were published before the results of recent studies on treatment options for HFpEF [13]. More recent AHA/ACC/HFSA guidelines from this year recommend the use of SGLT2 inhibitors as first-line therapy for both HFmrEF and HFpEF (Class IIa), before beta-blockers, ACEI/ARB/ARNI, MRAs (Class IIb) [53]. This is largely due to the results of studies such as EMPEROR-Preserved and DELIVER. The EMPULSE trial evaluated empagliflozin versus placebo in patients hospitalised for AHF regardless of LVEF. For patients receiving empagliflozin during 90 days of follow-up, it was shown that they were 36% more likely to experience a clinical benefit in terms of reduced risk of cardiovascular death, hospitalisation for HF and improved quality of life. The drug was started once clinical stability has been achieved, usually on the 3rd day of hospitalisation [59, 60]. The benefit of treating HF without significantly reduced ejection fraction (LVEF >40%) has also been proven for another SGLT2 inhibitor, dapagliflozin. The DELIVER study confirmed that in patients with HFpEF/HFmrEF (LVEF >40%), dapagliflozin significantly reduces the risk of cardiovascular death or exacerbation of HF [61].

IMPACT OF COMORBIDITIES ON INPATIENT COURSE AND OUTPATIENT CARE PLANNING

Heart failure is often accompanied by other cardiovascular conditions and diseases of other organs and systems. According to the ESC Pilot Survey registry, 74% of patients with HF have at least one non-cardiovascular concomitant disease, which translates into a significant increase in mortality in this patient population [62]. Current guidelines devote a great deal of attention to the treatment of comorbidities as important causes of readmissions when they are not recognised and/or not treated effectively [13]. Particularly noteworthy in the peri-discharge period are:

1. Among cardiovascular conditions: CCS, AF, arterial hypertension (AH).
2. Beyond cardiovascular conditions: iron deficiency (ID), T2DM, CKD.

Chronic coronary syndromes

The most common cause of HF in our population is coronary artery disease, which can lead to significant abnormalities in LV contractility, size and shape. Myocardial ischaemia should therefore be considered whenever patients are hospitalised for AHF, especially if a reduction in LVEF is observed *de novo*. Documenting ischaemia using non-invasive exercise tests can be difficult in patients with HF due to often poor exercise intolerance and chronically elevated LV end-diastolic pressure. Coronary angiotomography or invasive coronary angiography can be performed to determine the presence and severity of CCS, which will be critical in determining possible indications for coronary revascularisation if stenocardial complaints persist despite optimal pharmacotherapy [63]. Beta-blockers, which are one of the main groups of drugs in the treatment of patients with HFrEF, are also recommended in CCS, primarily for their antianginal effects. Ivabradine, on the other hand, should be considered as an alternative to beta-blockers (if contraindicated) or as an additional treatment to reduce ischaemia in patients with HR >70 bpm [63]. Other antianginal drugs (primarily calcium antagonists, nicorandil, ranolazine and nitrates) can also be effective in treating angina symptoms. Moreover, the addition of trimetazidine, which improves LV function and exercise tolerance in patients with HFrEF and CCS already treated with chronic beta-blockers, may be considered. In patients with HF, short-acting nitrates should be used with caution because they can cause hypotension. It is also important to note that diltiazem and verapamil are contraindicated in patients with HFrEF [13].

Atrial fibrillation

Atrial fibrillation is the most common type of arrhythmia in HF patients (15%–30%), especially those >65 years old. The risk of AF is particularly high in patients with HFpEF (40%) and it is an independent factor for a worse prognosis in this group of patients (increased risk of stroke, thromboembolic complications, hospitalisation for HF, and death) [13]. The finding of AF in a patient with HF requires first and foremost:

- identifying and treating the causes and triggers of cardiac arrhythmias;
- treatment of HF;
- prophylaxis of thrombotic complications;
- choosing a strategy for sinus rhythm control or ventricular rate control.

In all patients with HF and paroxysmal, persistent or permanent AF, unless contraindicated, chronic oral anticoagulant treatment is recommended. Non-vitamin K oral anticoagulants are preferred for preventing thromboembolic incidents because they have similar efficacy to vitamin K antagonists, with a lower risk of bleeding [64]. However, this applies to patients with AF without significant mitral valve stenosis or the presence of a mechanical valve prosthesis. In patients with a contraindication to oral anticoagulant therapy, left atrial appendage closure may be considered.

The cornerstone of AF treatment is symptom control through HR control. In cases of significant irreversible myocardial impairment with obviously enlarged cardiac cavities (especially the left atrium), a strategy of ventricular rate control rather than rhythm type control may be recommended. This is due to the low probability of both restoring and maintaining sinus rhythm in this group of patients. Pharmacological control of ventricular rate can be achieved by using primarily beta-blockers and digoxin [64]. The choice of drugs depends on the HF phenotype, symptoms, comorbidities and potential side effects. Dronedarone, diltiazem and verapamil are contraindicated in patients with HFrEF, while amiodarone, due to its numerous side effects, can usually be used only for a short period (<6 months) [13]. The acceptable resting ventricular rate in patients with permanent AF is 110 bpm, although some experts suggest that it should be in the range of 60–100 bpm [65, 66].

Hypertension

Hypertension is one of the main risk factors for the development of HF, and nearly two-thirds of patients with HF have a history of AH. Hypertension causes LV hypertrophy, thereby impairing its diastolic function; it is also a strong predictor of HF development (even with

preserved LVEF), thus playing a special role in the aetiopathogenesis of HFpEF. Treatment of AH significantly reduces the risk of developing HF and hospitalisation for HF, especially in people over >65 years of age. It must be remembered that inadequately controlled AH can lead to episodes of acute cardiovascular decompensation manifesting as pulmonary oedema. The most important recommendations for the treatment of AH in patients with HF are as follows [13, 67]:

1. In patients with HFrEF, ACEI/ARB, beta-blockers, and diuretics and/or MRAs are recommended. With inadequate BP control, treatment with dihydropyridine calcium antagonists (amlodipine or felodipine) can be added to therapy.
2. In patients with HFpEF, treatment is based on: ACEI/ARB, beta-blockers, diuretics and calcium antagonists. BP thresholds for starting treatment and therapeutic goals should be the same as those for patients with HFrEF.

ARNIs are also effective in lowering BP; moreover, they significantly improve the prognosis of patients with HFrEF. Drugs in this group are therefore recommended as an alternative to ACEI/ARB for the treatment of AH in patients with HFrEF. Non-dihydropyridine calcium antagonists (diltiazem, verapamil), alpha-blockers, and centrally acting drugs such as moxonidine are not recommended in patients with HFrEF [13].

Iron deficiency and anaemia

ID is an important comorbidity in HF patients. There is evidence that ID is associated with greater severity of HF symptoms, more frequent HF hospitalisations, and an increased risk of death [68, 69]. Clinical trials have indicated that intravenous iron supplementation (in the form of iron carboxymaltose) has significant benefits in patients with HF [70–72]. It should be emphasised that oral iron supplementation in HF patients is ineffective and not recommended [73]. In the latest ESC recommendations for the diagnosis and treatment of HF, the place of intravenous iron supplementation is as follows [13]:

1. The use of intravenous ferric carboxymaltose should be considered in patients with stable symptomatic HFrEF (LVEF <45%, so also in patients with HFmrEF) and ID to improve the quality of life, exercise capacity and reduce the severity of HF symptoms [70, 71].
2. Intravenous ferric carboxymaltose should be considered in patients with HFrEF and HFmrEF (LVEF <50%) clinically stabilised after an episode of AHF (current or recent hospitalisation) and ID to reduce the risk of subsequent unplanned hospitalisation for HF progression [72].

In view of the aforementioned benefits, all patients with HF, regardless of haemoglobin levels, renal function and LVEF values, should be periodically screened for ID, also during hospitalisation for AHF. Iron deficiency in HF patients is diagnosed on the basis of ferritin concentration <100 μg/L or ferritin concentration 100–299 μg/l (in this case, if accompanied by transferrin saturation <20%). If ID is found during hospitalisation for AHF, the first dose of ferric carboxymaltose should be given while still in the hospital. In addition, intravenous iron supplementation can (and should!) be continued and carried out on an outpatient basis. In the CONFIRM-HF and AFFIRM-AHF studies, patient body weight and haemoglobin levels were taken into account when dosing intravenous ferric carboxymaltose in patients with HF and ID. The drugs are given at baseline and at 6 weeks. A total dose of 0.5–2.0 g of ferric carboxymaltose is given in a regimen of up to 1.0 g at baseline and the remaining dose at 6 weeks [72, 73]. If haemoglobin concentration is >15 g/dl, intravenous iron should not be administered. Abnormal renal function, BP and HR values are not contraindications to the administration of intravenous ferric carboxymaltose. Patients on intravenous iron should be re-evaluated for iron status after 3–6 months and, if required, supplemented again. It should also be mentioned that no allergy tests need to be performed before the first intravenous administration of ferric carboxymaltose.

Type 2 diabetes mellitus

Data from the literature indicate that up to 30% of patients with HF have comorbid T2DM, and as many as two-thirds of the HF patient population have carbohydrate metabolism disorders (diabetes or pre-diabetes) [13]. Type 2 diabetes mellitus significantly increases the risk of developing HF, now becoming one of the leading causes of CHF along with CCS and AH. T2DM patients have a 2–5 times higher risk of developing HF compared to those with normal glucose metabolism. In cases where T2DM and HFrEF are established, it is recommended that SGTL2 inhibitors (empagliflozin or dapagliflozin) be used first and foremost, which, in addition to their hypoglycaemic effects, are, as already mentioned, one of the four groups of drugs included in the fundamental therapy of HFrEF [74, 75]. Metformin is a safe drug in patients with HF; however, it should not be used in patients with eGFR <30 ml/min/1.73 m^2 and in those with liver failure because of the risk of developing lactate acidosis. Glucagon-like peptide-1 (GLP-1) analogues and dipeptidyl peptidase 4 (DPP-4) inhibitors, with the exception of saxagliptin, which increases the risk of hospitalisation in HF, are not currently recommended in patients with HF due to their neutral effects on the risk of death and hospitalisation for HF [13, 76]. The use of sulfonylurea derivatives and thiazolidinediones (glitazones) is associated with

an increased risk of HF and/or hospitalisation for HF, and hence is not indicated for T2DM therapy in patients at risk of HF or those already diagnosed with CHF [13, 76].

For type 1 diabetes mellitus, insulin remains the drug of choice. Its use leads to sodium retention in the body, which can result in increased fluid retention and consequent cardiovascular decompensation in patients with HF. Therefore, initiation of insulin therapy in patients with HF and diabetes requires close monitoring of the patient's condition for early detection of possible fluid retention and incipient exacerbation of HF [13, 76]. It should be emphasised that a patient with diabetes mellitus and HF requires special monitoring (PCP, cardiology, diabetes) in the outpatient setting.

Renal impairment

Heart failure and CKD share common risk factors, such as T2DM and AH. Chronic kidney disease is one of the major independent determinants of increased mortality and morbidity in HF. In the course of CHF, especially when the disease is exacerbated, renal function often deteriorates. One reason for the increase in plasma creatinine levels is the use of diuretics in combination with ACEI/ARB/ARNI, MRAs, SGLT2 inhibitors, and nephrotoxic drugs, which include iodine contrast agents, certain antibiotics (gentamicin, trimethoprim), and non-steroidal anti-inflammatory drugs (NSAIDs). It should also be remembered that patients with impaired renal function may accumulate renally excreted drugs such as digoxin, insulin and low-molecular-weight heparin. It is therefore very important to adjust the dosage of these drugs appropriately according to the degree of kidney damage.

Patients with HF and coexisting CKD are at higher risk for cardiovascular incidents. In the presence of renal impairment or in people over >65 years of age with good baseline renal function after inclusion of RAAS, ARNI or SGLT2 inhibitors, the initial drop in glomerular filtration pressure may lower eGFR and increase serum creatinine. These changes generally resolve during long-term treatment. An increase in serum creatinine by <50% above baseline (as long as it is <266 μmol/l), or a decrease in eGFR by <10% compared to baseline (as long as it is >25 ml/min/1.73 m^2), may be considered acceptable. Transient deterioration of renal function during initiation of therapy should not lead to its discontinuation, as the new drugs recommended for the treatment of HFrEF (ARNIs, SGLT2 inhibitors) show a nephroprotective effect [77, 78]. ARNI, compared to enalapril, has been shown to reduce the rate of renal function deterioration [79]. A similar benefit has been indicated for the use of SGLT2 inhibitors (dapagliflozin, empagliflozin) compared to placebo, both in patients with HFrEF and those with CKD [77, 80].

With regard to diuretic treatment, small and transient increases in serum creatinine levels during treatment of acute HF are also not associated with a worse prognosis. In patients with very low eGFR, the effectiveness of diuretics (thiazide and loop diuretics) may be reduced. Diuretics should therefore be used in properly adjusted doses, as often a similar effect can be achieved with smaller, safer doses.

MONITORING A PATIENT WITH HEART FAILURE — THE ROLE OF TELEMEDICINE

Current ESC guidelines indicate that home telemonitoring of HF patients can be considered to reduce the risk of cardiovascular death and hospitalisation and the risk of HF exacerbation [13]. This form of patient care is associated with a 20% reduction in overall mortality and 37% reduction in HF hospitalisations. Telemonitoring has proven to be a particularly valuable tool during the coronavirus disease 2019 (COVID-19) pandemic. Monitored parameters such as symptoms, body weight, heart rate and BP can be collected and stored in an electronic health record as part of medical record keeping, and used to optimise therapy or provide medical advice remotely on this basis [75]. Teleconsultation is a relatively new tool in patient care in Poland. Teleconsultation was officially introduced into the National Health Fund's catalogue in March 2020 in connection with the COVID-19 pandemic, as procedure No. 89.0099 — medical advice via ICT or communication systems.

The simplest form of teleconsultation is telephone advice, which allows to monitor the patient's condition, remind of the need to take medication, and make sure the patient is using the appropriate dosage. Telephone advice allows to optimise the therapy if the physician knows the patient and has seen him/her recently at the medical facility. During the phone call, the patient should be asked about his/her current well-being as well as any recent changes, the presence of peripheral oedema, body weight changes and modifications in treatment. The patient should also provide values of regular home BP and heart rate measurements, as well as the results of previously ordered laboratory tests. During a telephone advice, the doctor provides the patient with further recommendations, and may also suggest the need to visit a medical facility in person or, in exceptional urgent cases, to go to the hospital.

Ideally, the first follow-up visit after discharge from hospitalisation for HF exacerbation should be a personal visit. However, this is not always possible, especially during the COVID-19 pandemic. If such a visit is to have the form of a telephone advice, then during such consultation the physician should, first of all:

- assess the patient's general condition and the degree of cardiovascular compensation (NYHA class, possible severity of symptoms indicative of decompensation);
- analyze and, if necessary, modify drug treatment;
- continue to educate the patient about HF (including self-management of symptoms) and related lifestyle modification, in which the Heart Failure Patient Passport is a great help;
- define and discuss the essential goals of treatment with the patient again;
- assess the compensation and treatment of comorbidities;
- make a possible qualification for a personal visit at the office or indications for readmission.

Many implanted therapeutic devices can wirelessly and remotely provide information about the device itself (generator and electrode function), rhythm disturbances, or patient's clinical data (heart rate, activity, heart tone volume, bioimpedance). There is strong evidence that monitoring can detect device malfunctions earlier than conventional monitoring, and may be useful for detecting cardiac arrhythmias such as AF. However, there is little evidence that device monitoring reduces HF admissions or mortality.

INPATIENT AND OUTPATIENT CARDIAC REHABILITATION IN PATIENTS WITH HEART FAILURE — THE CHALLENGE OF MODERN TIMES

Numerous clinical studies and meta-analyses classify cardiac rehabilitation with physical training, the importance of which has changed over the years, as one of the most important non-pharmacological management options for patients with HF [81–84]. Physical training is safe and recommended for HF patients, and the benefits of systematic, controlled exercise outweigh the associated risks [85]. However, in patients with advanced HFrEF combined with multimorbidity, a cardiac rehabilitation program based on supervised exercise should be considered [13]. Figure 2 shows a diagram of cardiac rehabilitation dedicated to patients with HF, indicating the various stages of rehabilitation depending on the patient's condition.

TELEREHABILITATION IN HEART FAILURE — OPPORTUNITIES IN THE 21st CENTURY

HF patients diagnosed with COVID-19 or having survived it, i.e. so-called convalescents, are a new challenge in cardiac rehabilitation. The individualised cardiac rehabilitation of these patients depends on both the severity of CHF and the symptoms and short- and long-term health consequences of COVID-19. Such rehabilitation invariably includes education of the patient and his/her family, as well as physical training (breathing, endurance, resistance exercises,

relaxation). It is worth using the modified 10-point Borg dyspnoea scale, especially in more severe clinical cases [86, 87]. Following consultation with a physician and analysis of risk factors, a return to recreational low-to moderate-intensity sports can be considered, in parallel, however, with a structured exercise program under specific supervision of a specialist regarding the type and intensity of exercise [13, 85]. Regular physical activity should always be individualised, well monitored, but also tailored to the patient's current needs and lifestyle, taking into account the factors affecting them [82, 88, 89].

It is emphasised that cardiac rehabilitation during the pandemic period should be carried out with the shortest length of stay in a facility in favour of monitored home rehabilitation, using new technologies and telemonitoring [13, 88–90]. In 2021, a consensus of four prestigious arrhythmology societies, International Society for Holter and Noninvasive Electrocardiology, Heart Rhythm Society, European Heart Rhythm Association and the Asia-Pacific Heart Rhythm Society, was published on ambulatory electrocardiographic telemonitoring, outlining cardiac telerehabilitation as a dedicated procedure for patients with cardiovascular conditions [91]. The COVID-19 pandemic has made telerehabilitation sometimes the only possible intervention, so the European Association of Preventive Cardiology is calling for action to widely implement cardiac telerehabilitation during the COVID-19 pandemic as the optimal way to conduct secondary prevention [92]. Hybrid telerehabilitation is one of the possible forms of implementing cardiac rehabilitation programs funded by the National Health Fund. Published data indicate that it is effective, safe and accepted by patients, resulting in good interactive patient cooperation [93–95]. It also leads to improvement in the quality of life [96]. It may be of particular importance for patients being discharged from the hospital. Telerehabilitation should be conducted by a team of trained specialists including a doctor, physiotherapist, nurse, psychologist and nutritionist. It uses equipment that allows remote monitoring of symptoms, parameters (electrocardiogram, BP, body weight) and control of physical training.

Hybrid telerehabilitation consists of two stages:

- the first preliminary stage is carried out in inpatient or outpatient setting;
- the second basic stage is carried out at home (telemonitored training sessions).

The initial stage is aimed at assessing clinical condition, exercise capacity, education, planning and conducting several training sessions. If it is carried out in an outpatient clinic, it begins with an initial visit, during which, in addition to standard examinations, the patient has an exercise test, based on which training is planned. Over the next 5 days, the patient participates in educational meetings that include: learning how to use the telerehabilitation equipment, exercise techniques, consultations with a nutritionist and psychologist, and lectures on

pro-healthy lifestyles, diet, the benefits of regular physical activity, and first aid. In the case of implementation of the initial stage during hospitalisation, all the procedures described above take place during hospitalisation, and following discharge, the patient implements the second stage of telerehabilitation at home. After the telerehabilitation cycle, a follow-up visit is scheduled with an exercise test and further recommendations are given to the patient [97, 98].

During the pandemic period, in an effort to minimize the exposure of medical personnel and patients, a modification of the hybrid telerehabilitation procedure has been prepared [99]. It was proposed to shorten the initial outpatient stage to 2 days and conduct further training using audio/video communicators with the patient already at home. In addition, when the initial stage takes place during hospitalisation, it has been proposed that it can be carried out by specialised teams (meeting the requirements for hybrid telerehabilitation set forth in the relevant orders of the National Health Fund) in each clinic/department, and not, as is currently the case, only in rehabilitation clinics/departments. In addition, in well-defined cases, the authors propose conducting the final visit using only ICT systems [99].

The increasingly common availability of hybrid telerehabilitation in HF provides an opportunity to cover a much larger number of patients with rehabilitation and to equalise regional disparities. Possible modifications make it the optimal, and in the case of high-risk patients such as those with HF, it sometimes becomes the only possible form of rehabilitation during epidemic periods of infectious diseases.

TASKS AND COMPETENCIES OF THE FAMILY PHYSICIAN IN THE TREATMENT OF PATIENTS WITH HEART FAILURE

A family physician provides medical care to a population of healthy and sick people of all ages who have chosen him or her as a primary care provider. Each family physician cares for an average of 12 to 24 patients with HF [100, 101].

The tasks of the family physician in the care of patients with HF have been described in detail in numerous international and national management recommendations [101–104]. They emphasize teamwork, including collaboration with an environmental/family nurse and a cardiology specialist. Intersectoral cooperation, especially with social welfare institutions, is also important with regard to the care of a portion of the HF patient population. In the period immediately following the discharge of a patient hospitalised for HF, the most important tasks of the family physician include [102]:

- Optimise pharmacotherapy implemented in the hospital setting.
- Monitor relevant clinical parameters and laboratory and imaging results.

- Identify and treat comorbidities [105].
- Educational activities conducted jointly with the environmental/family nurse for both the patient and his/her caregivers and immediate family members.
- Implementing significant preventive measures and, if necessary, referring the patient to hospital treatment.
- Assistance in solving social problems [101]. • Implementation of the immunisation programme, especially against influenza and Pneumococcus. Vaccination against severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) is also of particular importance during the current pandemic. As already emphasised, the first medical consultation should take place within 1–2 weeks (optimally 7 days) after the patient's discharge from the hospital [101]. An indication for readmission of a patient with HF in the peri-discharge period is a significant exacerbation of the disease course.

In terms of pharmacotherapy, it is particularly important to increase the dosage of HF course-modifying drugs (beta-blockers, ACEI/ARB/ARNI, MRAs) to the target or maximum dose tolerated by the patient, and to include new drugs recommended in the guidelines if the patient has not received them before (e.g., SGLT2 inhibitors). Depending on the patient's profile and baseline cardiovascular risk, it is possible to apply different types of interventions to an individual patient with class II drugs (ivabradine, digoxin, ferric carboxymaltose, vericiguat). Family doctor should [104]:

- adjust the selection and dosage of diuretics according to the patient's current clinical condition (assessment of fluid overload, BP);
- periodically monitor renal function (creatinine/eGFR, urea) and electrolyte levels (sodium, potassium) in the HF patient, especially during the period of drug therapy modification;
- decide whether to include other drugs, such as ivabradine and digoxin, in the treatment;
- make decisions about discontinuing/replacing medications that can worsen HF (e.g., glitazones, NSAIDs, calcium antagonists, tricyclic antidepressants) [13].

The decision to reimburse (30% payment) SGLT2 inhibitors (dapagliflozin and empagliflozin) for HF patients as of 1 May 2022 in Poland will certainly increase the availability of this effective treatment. The reimbursement indications include patients with HFrEF (LVEF <40%), regardless of comorbid diabetes, who have persistent symptoms, in NYHA class II–IV, despite therapy based on beta-blockers, ACEI/ARB/ARNI and, if such treatment is indicated, MRA [106]. Patients with diabetes and CKD will additionally benefit from the inclusion of SGLT2

inhibitors. Reimbursed treatment with SGLT2 inhibitors can be introduced by any physician in the system caring for a patient with HF, not just a cardiologist.

One of the most important considerations for making therapeutic decisions in patients after hospitalisation for HF is to monitor body weight, hydration status and signs of circulatory congestion (including increased sensation of fatigue/dyspnoea, lower extremity oedema, ascites and auscultatory features of pulmonary congestion), BP, HR and respiratory rate. These parameters allow not only to optimise pharmacotherapy, but also to decide on the timing of possible readmission of the patient [13, 103]. Laboratory parameters that may need to be monitored include peripheral blood count, iron deficiency markers, thyrotropic hormone, liver aminotransferases, glucose levels (or glycated haemoglobin), and lipid profile. A laboratory test of great utility is the determination of natriuretic peptide (BNP, NT-proBNP) levels. The listed goals of treatment and tasks related to the care of patients with HF within primary health care will certainly improve the coordinated care introduced to the practices of family doctors in Poland. Within the entrusted budget, it is possible to perform an extended panel of diagnostic tests and carry out specialist consultations with the patient, without the need to refer the patient to outpatient specialist care. A patient with HF within the framework of coordinated care in primary health care should be provided with:

• a comprehensive visit with the development of an individual medical care plan (once a year),
• individual follow-up visits (depending on the clinical condition),
• the possibility of consulting a cardiologist - directly (if the patient's condition requires it) or in the form of a medical consultation using telemedicine techniques (a primary care physician - cardiologist),
• educational advice (nursing and dietary),
• selected additional tests.

These tests include primarily: NT-pro-BNP, electrocardiographic stress test, transthoracic echocardiography, continuous Holter ECG monitoring, and continuous ambulatory blood pressure monitoring. These tests not only can, but should be used in the care of patients with HF, depending on the indications and clinical assessment made by the family physician, in selected cases also after consultation with a cardiologist. If it is necessary to deepen the diagnosis or conduct specialist treatment, the patient — as before — should be referred to outpatient specialist care [107].

THE ROLE OF THE NURSE IN CARING FOR PATIENTS WITH HEART FAILURE

Current ESC guidelines invariably point to adherence to self-management as an important element in improving outcomes for patients with HF, reducing mortality and improving quality of life [13]. Therefore, most recommendations for the management of HF place a strong emphasis on promoting self-management behaviours, such as lifestyle modifications and fluid intake restrictions [108].

Nursing care is considered a very important part of the health care system for CHF patients [109, 110]. Nurses should conduct educational activities by identifying access to professional information, promoting patients' health awareness, and thereby empowering them [111, 112].

Many countries have programs in which HF nurses provide continuity of care, working closely with the family physician, cardiologist, and the patient himself and his family/caregivers [113, 114]. The role of the nurse focuses on:

1. Educating the patient about his/her disease (definition, aetiology and risk factors of HF), symptoms that require a doctor's visit, and factors that contribute to HF exacerbation.
2. Taking part in monitoring adherence to therapeutic recommendations (drug dosage, options for flexible supply of diuretics).
3. Providing advice and recommendations on diet, physical activity, fluid intake, recommended vaccinations and more.
4. Education on techniques for measuring heart rate, BP, saturation, respiratory rate and body weight, assessing peripheral oedema and feeling of dyspnoea, as well as monitoring for any adverse effects of the treatment being used, pointing out the possibility of modifying the doses of certain drugs (primarily diuretics and BP-lowering drugs).

These activities aim to prepare the patient for self-management and self-care. Self-care can be assessed using standardised questionnaires [115–121]. This is of particular importance because, as already mentioned, the reasons for the high mortality rate of cardiac patients after hospital discharge are mainly: inappropriate lifestyle, irregular use of medications or interruption of prescribed pharmacotherapy, lack of control of risk factors, insufficient access to specialised cardiac care after hospitalisation, complications and comorbidities [119].

During the COVID-19 pandemic with its emphasis on social distance, the importance of self-care for HF patients is greater than ever. Hospital stays are associated with a higher risk of SARS-CoV-2 infection, and hospitalisations for HF carry a poorer long-term prognosis. Adherence to medication may be a differentiating factor in this regard. Careful attention to symptoms, as well as daily body weight, can alert patients, their family members and health care professionals about the onset of a CHF exacerbation. Introducing appropriate treatment modifications at this early stage of HF deterioration may save some of these patients from

subsequent hospitalisation. Nurses can play a key role in this process, for example by maintaining telephone contact with patients, and thus promoting self-care [120].

HEART FAILURE DURING COVID-19 PANDEMIC

Due to the COVID-19 pandemic, patients with HF face difficulties in receiving scheduled services for primary and secondary care, in both inpatient and outpatient settings [122, 123]. This affects their safety and makes it difficult to exercise proper monitoring. In the vast majority of patients (>80%), SARS-CoV-2 infection is asymptomatic or paucisymptomatic [124–128]. Severe disease develops in about 18% of confirmed cases of SARS-CoV-2 infection [129]. The so-called cytokine storm (3%–4% of patients with viral sepsis) leading to multi-organ failure can be one of the causes of the patient's death [126, 130, 131].

SARS-CoV-2 has a high potential to cause multi-organ damage, including cardiac damage, both *de novo* (without prior heart disease), and as increased damage of the already diseased myocardium. Whether it occurs as an exacerbation of CHF or develops in patients without prior heart disease, AHF is associated with a very high mortality rate of nearly 50% [132, 133].

Both the burden of cardiovascular disease and cardiovascular involvement in COVID-19 are associated with a worse prognosis, especially in patients over >65 years of age [134–136]. The most common burdens include AH (more than half of patients), obesity and T2DM [137–140]. Some of the cardiovascular complications are due to inflammation and/or acute myocardial damage due to SARS-CoV-2 infection [122, 141–146]:

- thromboembolism;
- AHF *de novo* or as exacerbation of CHF;
- Takotsubo syndrome;
- abnormal heart rhythm;
- ACS.

Confirmation of acute myocarditis is often possible with cardiac magnetic resonance imaging [146, 147]. It is noteworthy that among patients with confirmed COVID-19, cases of Takotsubo syndrome have also been reported, mainly affecting women [148, 149]. Cardiac arrhythmias (AF, ventricular tachycardia and ventricular fibrillation) during hospitalisation for COVID-19 have been reported in a varying percentage of patients, from 7% in those who did not require an intensive care unit stay to as many as 44% of patients treated at these units [150–152].

Differentiating symptoms of COVID-19 infection alone from those of HF exacerbation can be problematic, especially since these conditions can co-occur (145). All available clinical data should be considered (Table 4) [132, 146]. Testing for SARS-CoV-2 should be considered in all

HF patients suspected of having COVID-19, even if they have already undergone the infection or have been vaccinated, and for whom a decision will be made on urgent hospitalisation.

SUMMARY — A DECALOGUE OF PRE- AND POST-DISCHARGE RECOMMENDATIONS

The pre- and peri-discharge management of patients with HF and disease exacerbations is a great challenge not only for modern cardiology, but also for the many specialists who provide care to these patients. The following are basic recommendations that, if followed, should help manage patients in the peri-discharge period:

1. Consideration of the inpatient course of AHF or exacerbated CHF in pre-discharge management. Determining the aetiology, phenotype of HF and clinical profile of the patient, enabling the implementation of personalised treatment.
2. Introducing drugs from the four fundamental groups that improve prognosis in HFrEF (beta-blockers, ACEI/ARB/ARNI, MRAs, and SGLT2 inhibitors) into treatment, if possible, before hospital discharge.
3. Careful evaluation of the patient's clinical condition in terms of the level of residual cardiovascular risk and fluid retention (including a decision on the intensity of diuretic treatment) and the introduction of drugs from class II recommendations.
4. Recognizing and properly treating comorbidities (including ID).
5. Including in the discharge letter a treatment plan that includes appointments for follow-up visits to the PCP, cardiologist, and other specialists as needed.
6. Continued therapy escalation in outpatient setting according to guidelines after hospital discharge (primarily increasing to the maximum tolerated doses of the primary medications used in the treatment of HFrEF: beta-blockers, ACEI/ARB/ARNI, MRAs, inclusion of SGLT2 inhibitors if the patient had not previously received them).
7. Considering the role of cardiac rehabilitation in the treatment of CHF, both inpatient, outpatient and hybrid telerehabilitation.
8. Incorporating new effective monitoring methods based on telemedical systems into the care of HF patients.
9. Continuous education of patients and their families about HF, especially symptoms, treatment and self-care.
10. Cooperation and proper division of responsibilities during the care of patients with HF among cardiologists, family physicians, nurses and other specialists.

Modern medicine offers a range of treatment options for patients with HF. Their use in an individual patient should translate into reduced hospitalisations and mortality, and improved quality of life for this growing group of patients.

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Table 1. Recommended non-pharmacological interventions in patients with heart failure (HF) [24]

Recommendations	Class	Level
It is recommended that HF patients are enrolled in multidisciplinary HF management programmes to reduce the risk of HF hospitalisation and mortality.	I	A
Self-management strategies are recommended to reduce the risk of HF hospitalisation and mortality. Outpatient or inpatient care programmes are recommended to reduce the risk of HF hospitalisation and mortality.	I	A
Influenza and pneumococcal vaccinations should be considered to prevent HF and hospitalisations.	I	A
	II	A

Table 2. Follow-up visits in patients with chronic heart failure

Clinical condition	Follow-up visits	Parameters evaluated	Specialist
Stable patient	Every 6 months	Signs of cardiovascular decompensation in patient history and on physical examination, other symptoms, BP, HR, complete blood count, electrolytes (sodium and potassium), creatinine, other*	Cardiologist/PCP
Patients discharged from hospital	Preferably 1–2 weeks after discharge, then as needed	Signs of cardiovascular decompensation in patient history and on physical examination, other symptoms, BP, HR, complete blood count, electrolytes (sodium and potassium), creatinine, other[a]	Cardiologist/PCP
Patients in the course of	As needed (to optimise therapy)	Signs of cardiovascular decompensation in patient history and on physical examination, other symptoms, BP, HR,	Cardiologist/PCP

therapy	complete blood count, electrolytes (sodium
escalation	and potassium), creatinine, other*

[a]Other – ECG once a year to assess the duration and morphology of the QRS complex and identify conduction and rhythm abnormalities (especially atrial fibrillation); echocardiography in case of clinical deterioration and 3–6 months after optimisation of standard therapy for HFrEF to determine indications for possible modification of pharmacotherapy and/or implantation of devices (ICD, CRT)

Abbreviations: BP, blood pressure; CRT, cardiac resynchronisation therapy; ECG, electrocardiogram; HFrEF, heart failure with reduced ejection fraction; HR, heart rate; ICD, implantable cardioverter defibrillator; PCP, primary care physician

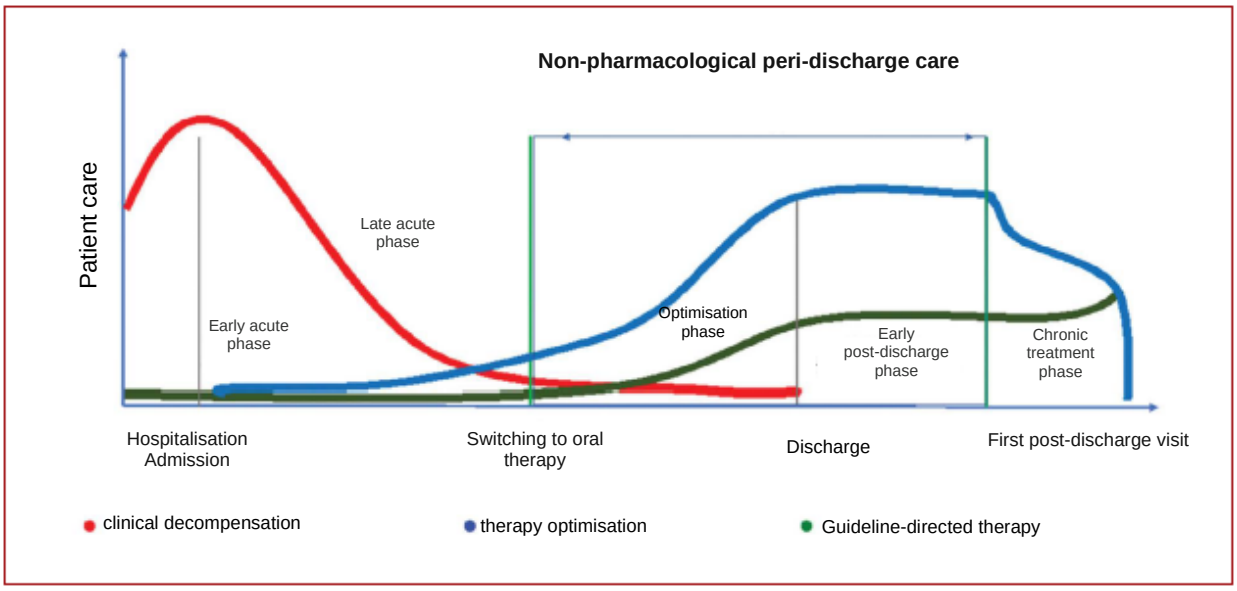

Figure 1. The clinical course of heart failure and the place of non-pharmacological management in the peri-discharge period [14]

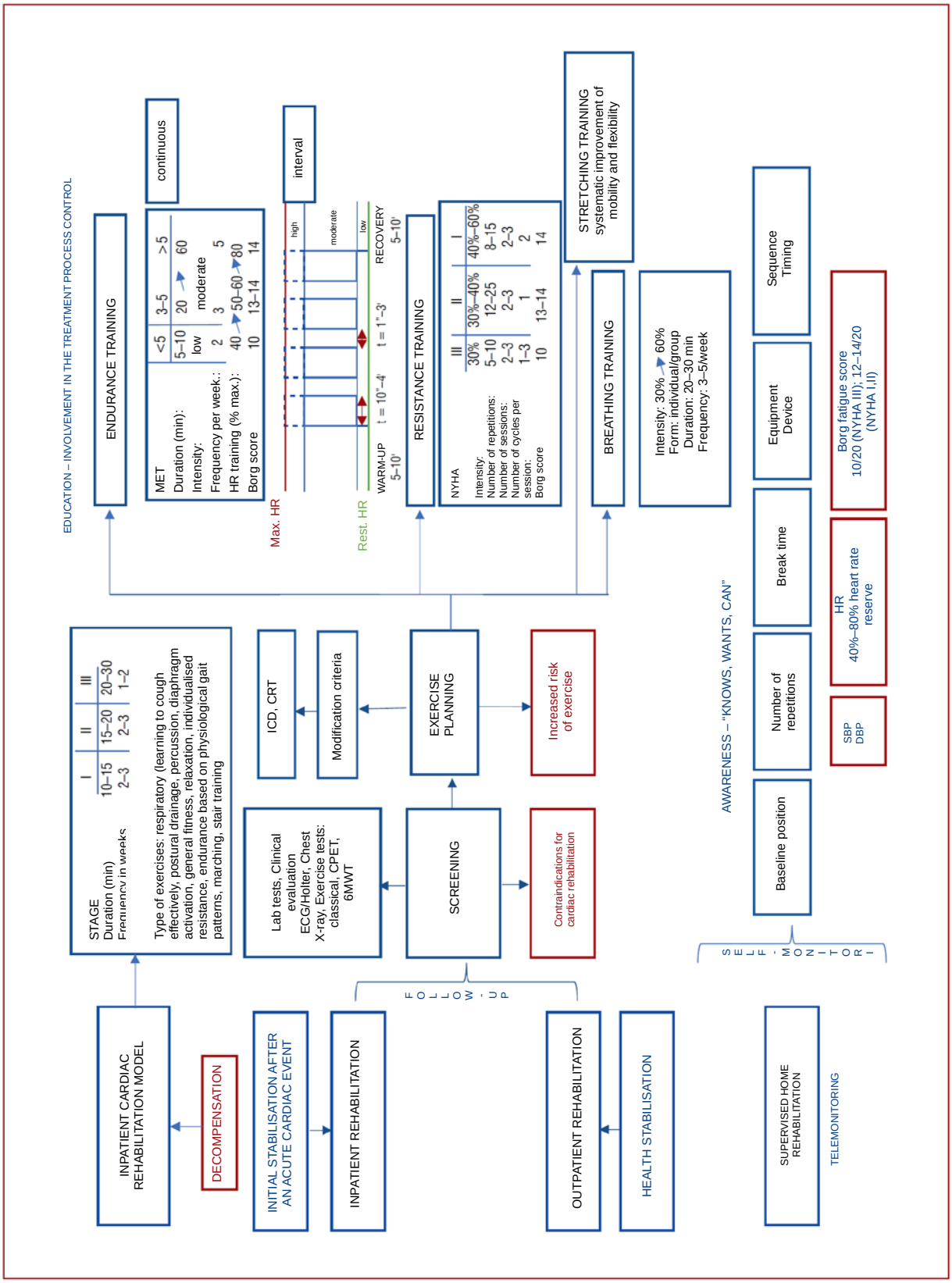

Figure 2. Cardiac rehabilitation scheme in patients with heart failure

Abbreviations: 6MWT, six-minute walk test; CPET, cardiopulmonary exercise test; CRT, cardiac resynchronization therapy; DBP, diastolic blood pressure;

HF, heart failure; HR, heart rate; ICD, implantable cardioverter defibrillator; MET, metabolic equivalent; SBP, systolic blood pressure

Table 3. Pharmacological treatment of heart failure depending on the patient's clinical profile [49]

Patient with low BP (<90/60 mm Hg)	
HR 60–70 bpm	**HR >70 bpm**
MRA	MRA
SGLT2 Inhibitor	SGLT2 inhibitor
↓*beta-blocker*	↓*beta-blocker*
↓*diuretic*	↓*diuretic*
↓*ACEI/ARB/ARNI*	↓*ACEI/ARB/ARNI*
	ivabradine
Patient with high BP (>140/90 mm Hg)	
ACEI/ARB/ARNI	
SGLT2 inhibitor	
beta-blocker	
MRA	
diuretic	
vericiguat	
hydralazine/isosorbide dinitrate	
Patient with low heart rate (<60 bpm)	
BP >90/60 mm Hg	**BP <90/60 mm Hg**
ACEI/ARB/ARNI	SGLT2 inhibitor
SGLT2 inhibitor	MRAs
MRA	↓*beta-blocker*
diuretic	↓*ACEI/ARB/ARNI*
↓*beta-blocker*	↓*diuretic*
vericiguat	
Patient with increased heart rate (>70 bpm)	
ACEI/ARB/ARNI	

SGLT2 inhibitor

beta-blocker

MRA

diuretic

ivabradine

Patient with AF	
QRS complex frequency >60 bpm	**BP <90/60 mm Hg**
beta-blocker	SGLT2 inhibitor
ACEI/ARB/ARNI	ACEI/ARB/ARNI
SGLT2 inhibitor	MRAs
MRAs	↓*beta-blocker*
diuretic	↓*diuretic*
digoxin	**oral anticoagulant (NOAC of choice)**
oral anticoagulant (NOAC of choice)	

Patient with CKD	
eGFR <30 mL/min/1.73 m^2	**eGFR >30 mL/min/1.73 m^2**
sGLT2 inhibitor	sGLT2 inhibitor
beta-blocker	beta-blocker
diuretic	ACEI/ARB/ARNI
vericiguat	MRA
hydralazine/isosorbide dinitrate	diuretic
	vericiguat
	hydralazine/isosorbide diazotate

Patient with hyperkalaemia (K^+ >5.5 mEq/L)

SGLT2 inhibitor

beta-blocker

diuretic

↓*ACEI/ARB/ARNI*

↓*MRA*

potassium-binding products (e.g., polystyrene sulfonate, Resonium A)

vericiguat

↓dose reduction or drug discontinuation

Abbreviations: ACEI, angiotensin-converting enzyme inhibitor; AF, atrial fibrillation; ARB, angiotensin receptor blocker; ARNI, angiotensin receptor neprilysin inhibitor; BP, blood pressure; CKD, chronic kidney disease; eGFR, estimated glomerular filtration rate; HR, heart rate; MRA, mineralocorticoid receptor antagonist;
NOAC, non-vitamin K antagonist oral anticoagulant; SGLT2, sodium-glucose co-transporter 2

Table 4. Selected clinical data to help differentiate SARS-CoV-2 infection and HF exacerbation

	COVID-19	HF exacerbation
History of cardiovascular disease	+/–	+
Fever	+	–
Cough	+	+/–
Myalgia	+	–
Leg oedema	–	+
Leukocyte and CRP levels	Lymphocytopenia and increase in CRP, leukocytosis with secondary bacterial infection	Usually unchanged (unless the cause of the exacerbation is an infection)
Elevated NT-proBNP, BNP	In patients with a severe course of COVID-19	+
Troponin concentration	Elevated only in patients with severe COVID-19 and myocardial damage	Usually stably elevated
ECG	Sinus tachycardia (arrhythmia in severe infection)	Tachyarrhythmias (including AF), non-specific ST segment changes
Echocardiography	Usually normal	Depending on the HF phenotype (reduced global left ventricular contractility, enlarged cardiac cavities, dilated inferior vena cava)
Lung imaging (X-ray, CT)	Subpleural consolidations, “ground glass” opacities, radiographic features of ARDS	Congestive changes, pleural fluid, pulmonary oedema in advanced

	and diffuse consolidations (“white lung”) in COVID-19	exacerbation of left ventricular stage 4. failure

Abbreviations: AF, atrial fibrillation; ARDS, acute respiratory distress syndrome; BNP, B type natriuretic peptide; COVID 19, coronavirus disease 2019; CRP, C reactive protein; CT, computed tomography; ECG, electrocardiogram; HF, heart failure; NT proBNP, N terminal proB type natriuretic peptide; SARS CoV 2, severe acute respiratory syndrome coronavirus 2