Choosing the Ideal Hemodynamic Therapy in Acute Right and Left Heart Failure

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Learning Objectives

Learning objectives start revising the main symptoms of acute right and left heart failures (► Sect. 32.2), etiologies and differential diagnoses of AHF (► Sect. 32.3), and the current practice of therapy of acute right and left heart failure (► Sect. 32.4).

32.1 Introduction

AHF refers to both new and rapid presentation of acute heart failure or deterioration of known heart failure [1]. Signs of acute heart failure (see ► Sect. 32.2) should raise immediate attention to this potentially life-threatening condition. AHF causes a high burden of mortality, morbidity, as well as repeated hospitalizations. Thereby it also accounts for a multiplier of overall health costs. Multidisciplinary practical guidance and handling are warranted for tailored and focused treatment approaches that can be applied to the various types and different clinical appearances of AHF [2].

32.2 Signs and Symptoms of Acute Heart Failure

Right and left ventricular failure are both life-threatening entities that are not always easy to detect and therefore have to be suspected based on clinical findings. Signs and symptoms of acute right and left heart failure are shown in ◘ Table 32.1.

The mentioned symptoms may dramatically worsen into respiratory failure and hemodynamic compromise. Due to inadequate blood supply, AHF can rapidly affect proper functioning of all vital organs.

Diagnosis of heart failure is confirmed by BNP (brain natriuretic peptide) > 400 pg/ml or NT-proBNP (N-terminal pro-brain natriuretic peptide) >1200 pg/ml.

◘ **Table 32.1** Signs and symptoms of acute right and left heart failure

Acute right ventricular failure [3]	Acute left ventricular failure
Signs of systemic congestion: jugular venous distension, hepatojugular reflux, peripheral edema, pericardial effusion, congestive hepatosplenomegaly, ascites, anasarca[a]	Signs of pulmonary congestion: dyspnea, orthopnea, pink frothy sputum, persistent cough or wheezing
Signs of right ventricular dysfunction: third heart sound, systolic murmur of tricuspid regurgitation, hepatic pulse, signs of concomitant left ventricular dysfunction, paradoxical pulse[b]	Signs of left ventricular dysfunction: new-onset arrhythmia, mitral regurgitation
Signs of low cardiac output state: central nervous system abnormalities, exercise intolerance, weakness or fatigue of acute onset, hypotension, tachycardia, angor, oliguria, cool extremities	
Others: Hypoxemia, Kussmaul sign[c]	Others: hypoxemia

[a]Particularly in acute decompensation of chronic RV failure
[b]Abnormally high decrease in systolic pressure and amplitude of pulse wave during inspiration
[c]Increase in jugular venous pressure on inspiration (e.g., RV infarction, constrictive pericarditis)

Table 32.2 Etiologies and differential diagnoses of right and left ventricular failure

Right ventricular failure [3]	Left ventricular failure
Acute left ventricular failure	Acute right ventricular failure
Right ventricular ischemia/infarction	Left ventricular ischemia/infarction
Sepsis	
Chronic pulmonary hypertension (groups 1–5) [4]	Chronic arterial hypertension
Arrhythmias (supraventricular or ventricular tachycardia)	Arrhythmias (supraventricular or ventricular tachycardia)
Congenital heart disease (e.g., atrial or ventricular septal defect, Ebstein's anomaly)	Congenital heart disease (e.g., bicuspid aortic valves, mitral valve prolapse)
Valvulopathies (e.g., tricuspid valve regurgitation, pulmonary valve stenosis)	Valvulopathies (e.g., mitral valve regurgitation, aortic valve regurgitation or stenosis)
Cardiomyopathies (e.g., arrhythmogenic right ventricular dysplasia, peripartum/postpartum, [5–7] familial, idiopathic)	Cardiomyopathies (e.g., peripartum/postpartum, familial, idiopathic)
Myocarditis or other inflammatory diseases	
Cardiac surgery (e.g., cardiac transplant or left ventricular assist device implantation)	Cardiac surgery (e.g., cardiac transplant, valve replacement) [8]
Hematological disorders (e.g., acute chest syndrome in sickle cell disease, polycythemia)	Hematological disorders (e.g., left ventricular diastolic dysfunction in sickle cell disease, polycythemia)
Acute pulmonary embolism	Tako-tsubo or tako-tsubo-like left ventricular dysfunction [9]
Chronic diseases (e.g., diabetes, HIV, hyperthyroidism, hypothyroidism, hemochromatosis, amyloidosis)	
Exacerbation of chronic lung disease and/or hypoxia	
Acute lung injury or respiratory distress syndrome	
Pericardial disease (tamponade)	

32.3 Causes and Differential Diagnoses of Acute Right and Left Ventricular Failure

Acute right and left ventricular heart failure reveal numerous etiologies that have to be kept in mind during the diagnostic work-up (Table 32.2):

32.4 Therapy [2, 3]

Diagnosis of AHF should be completed in a timely manner to start early therapy and prevent death. A high index of suspicion is warranted and should be based on the following clinical findings:

- Chest discomfort.
- Signs of pulmonary (dyspnea, increased respiratory rate, orthopnea, rales on auscultation) and systemic (jugular vein distension, hepatomegaly, peripheral edema) congestion.
- Signs of hypoperfusion (cold periphery, clammy or mottled skin, cyanosis, confusion, oliguria, hyperlactatemia).

Early recognition of AHF is crucial since the first hours after symptom onset are characterized by a high complication rate which includes death [10]. This is substantiated by the fact that earlier diagnosis, triage, and initiation of specific treatment for AHF are associated with reduced mortality and shorter lengths of hospital stay [11–14]. Lung ultrasound is an easy-to-use, inexpensive, noninvasive, reliable, and reproducible method that should be used already in the emergency department: B-line evaluation identifies extravascular lung water [15]. Studies confirm a better outcome after immediate intravenous administration of nitrates and noninvasive positive-pressure ventilation [13, 16, 17]. Special attention is also warranted on precipitating factors like acute coronary syndrome, pulmonary infection, or atrial fibrillation that need specific therapies. A therapy overview is provided at the end of the chapter (Table 32.4).

32.4.1 Choosing the Ideal Therapy in the Prehospital Setting

Prehospital treatment should be based upon the patient's symptoms and vital signs. Oxygen therapy should be started in case of SpO_2 below 90%. In cases of respiratory distress or pulmonary edema, noninvasive ventilation (NIV) should be initiated immediately as it was shown that immediate application of CPAP alone in the prehospital setting improves physiological variables (e.g., PaO_2) and symptoms (e.g., dyspnea) and reduces incidence of tracheal intubation [15]. When findings of congestion are assessed, intravenous diuretics (i.e., furosemide 0.5 mg/kg or the double home dose of loop diuretic) should be administered. Normal or high blood pressure should be treated with intravenous/sublingual/spray nitrates. A careful fluid challenge (i.e., 4 ml/kg or 250 ml) is recommended when the patient is hypotensive or shows signs of hypoperfusion (see above).

32.4.2 Choosing the Ideal Therapy Within the First 2 h in AHF Without Cardiogenic Shock

When "time is muscle," efficient management is crucial. Diuretics, vasodilators, and administration of oxygen or (non-)invasive ventilation represent the main therapeutics for the first 2 h in the hospital. Early administration of intravenous loop diuretics has been shown to be associated with reduced in-hospital mortality in AHF [18]. Target SpO_2 is recommended between 92% and 95% [19–21]. In a recent investigation, routine use of intravenous morphine in AHF has been questioned and was suggested to correlate with increased 30-day mortality in these patients [22].

A loop diuretic (i.e., furosemide; for dosage, see ▶ 32.4.1) should be administered within 30 min after admission if not already done in the prehospital setting [15]. Furosemide (40 mg i.v.) may have two beneficial effects: immediate venodilation and subsequent diuretic effect. This should be repeated if respiratory distress persists after 2 h. In

case of oliguria, dosage should be increased. In some patients additional administration of thiazide or other classes of diuretics might be the key to success [23, 24]. A missing response can sometimes be attributed to diuretic resistance. The mechanism of which is unknown to date. Other therapeutic strategies (e.g., tolvaptan, a vasopressin v2 receptor antagonist, sequential nephron blockade) [25, 26] might be promising but did not gain sufficient importance to be integrated into the management guidelines of AHF. (Early) ultrafiltration demonstrates no superiority to pharmacotherapy [27] but has not been studied in this specific cohort [23].

When APE appears with abrupt hypertension, systemic volume overload is usually absent, and high-dose diuretics (i.e., >1 mg/kg) in this case are not recommended. In this situation, vasodilators and NIV represent first-line therapies. Overall, vasodilators are used in 30% of AHF [28]. They should be used with respect to close blood pressure monitoring due to their potential to produce severe hypotension. Their safety and benefit on outcome have been investigated in several trials. Vasodilators can be safely administered in systolic blood pressure >100 mmHg [29]. Nitrates serve as venodilators and arteriodilators at the same time and have been evidenced to be safe and effective even in high doses in severe APE [17]. In addition, high-dose treatment with nitroglycerin was shown to diminish rates of endotracheal intubation and ICU admission [30]. Overall, following the European Society of Cardiology (ESC) and American guidelines, the use of nitrates is strongly recommended [29, 31]. Contraindications of vasodilators are state of shock and significant mitral or aortic valve stenosis, and they should be used with caution in predominant right ventricular failure due to the risk of reduced coronary perfusion pressure.

Even though routine use of opiates is not recommended, morphine can be and is often used in anxious patients with acute respiratory distress [29].

Oxygen therapy and mechanical ventilation are indicated in APE which affects roughly 20% of AHF patients [32]. Depending on the severity of APE, conventional oxygen therapy can be sufficient to treat mild hypoxemia. In case of respiratory failure, NIV is indicated. Contraindications to install NIV therapy are a significantly altered mental state, poor cooperation, apnea, hypotension, vomiting, and possible pneumothorax [2]. In case of fatigue, weakness, obtundation, or failure of NIV, the patient should be intubated.

Figure 32.1 discloses the recommended approach to early diagnosis, management, and treatment of AHF for the first 2 h after hospital admission [2].

32.4.3 Choosing the Ideal Therapy in Cardiogenic Shock

The state of cardiogenic shock is the most severe form of appearance of AHF. CS is characterized by hypotension and signs of organ hypoperfusion due to severe circulatory failure of cardiac origin. It accounts for less than 5% of AHF cases in the western world [32] and is caused by acute coronary syndrome in 80% of cases [33]. Other etiologies include severe decompensation of chronic heart failure, valvular disease, myocarditis, and takotsubo syndrome [2].

All shock patients need urgent ECG, troponin essay, and prompt coronary angiogram if the shock state is not explained elsehow (Fig. 32.2). Urgent revascularization has been proven for benefit [34] and is recommended in the guidelines [35]. After revascularization controlled hypothermia is advocated [36]. For hemodynamic monitoring, echocardiography should be performed immediately and repeatedly used for surveillance of the patient's clinical condition. Inotropes and/or vasopressors should be installed only in

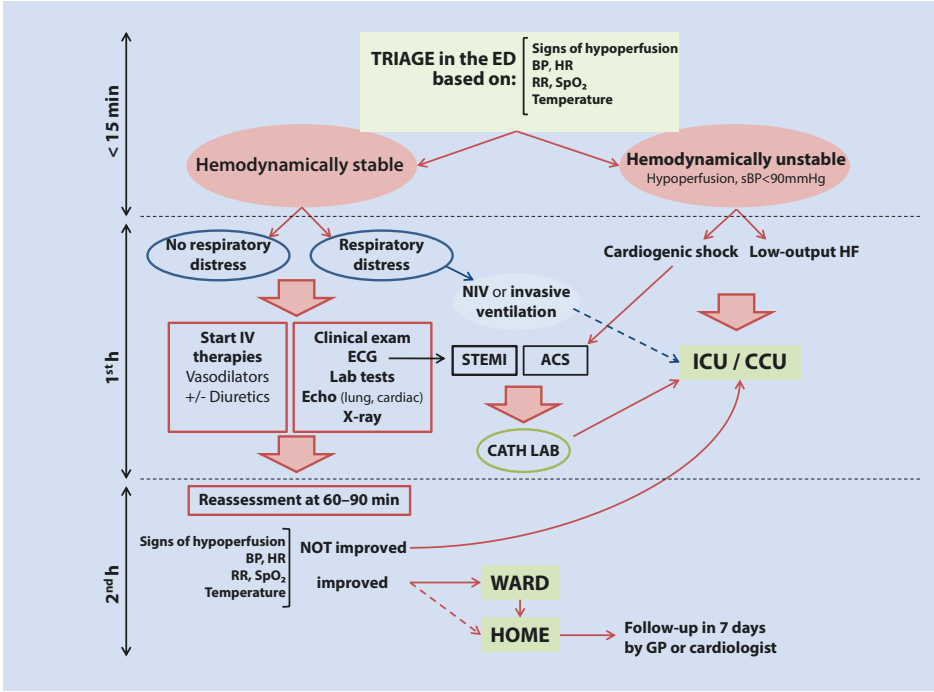

Fig. 32.1 The hospital management of patients with suspected acute heart failure. ED emergency department, BP blood pressure, HR heart rate, RR respiratory rate, SpO2 peripheral capillary oxygen saturation, Temp body temperature, NIV non-invasive ventilation, IV intravenous, ECG electrocardiogram, lab tests laboratory tests, echo ultrasound (lung ± cardiac), ACS acute coronary syndrome, STEMI ST segment elevation myocardial infarction, cath lab cardiac catheterization laboratory, ICU intensive care unit, CCU cardiac care unit, HF heart failure, GP general practitioner. Low output HF systolic <90 mmHg without signs of tissue hypoperfusion, usually in patients with end-stage heart disease

evidence of hypoperfusion. Inotropes and/or vasopressors should be administered in the lowest possible dose and for the shortest possible duration to achieve targeted perfusion pressure to minimize their side effects [37]. They should be applied over a central venous catheter (CVC) preferentially. The insertion of a CVC also allows to analyze $ScvO_2$ for the assessment of the ratio of global oxygen demand and supply, hence response to therapy. Dobutamine or levosimendan composes first-line therapy. Levosimendan might be preferred in the history of chronic heart failure or postinterventional myocardial stunning. Milrinone can be proposed before levosimendan in cases of concomitant pulmonary artery pressure as both reduce cardiac filling pressures, but also pulmonary vascular resistance [38]. Epinephrine has little or no indication due to higher incidence of side effects (e.g., arrhythmia, lactic acidosis) [39] and evidence of independent association with excess mortality in cardiogenic shock [40]. Norepinephrine, added to an inotrope, is the ideal vasopressor in CS [41, 42].

An arterial catheter should be inserted as soon as possible. This allows frequent uncomplicated bloodwork to monitor respiratory and hemodynamic support (e.g., lactate) [43, 44]. If the patient doesn't respond to therapy, the insertion of a pulmonary artery catheter (PAC) should be considered [29]. Early echocardiography is not only helpful in establishing a diagnosis but also in speeding up triage to ideal treatment and estimate prognosis (e.g., ejection fraction, right and left ventricular function). Follow-up echocardiography may evaluate treatment response.

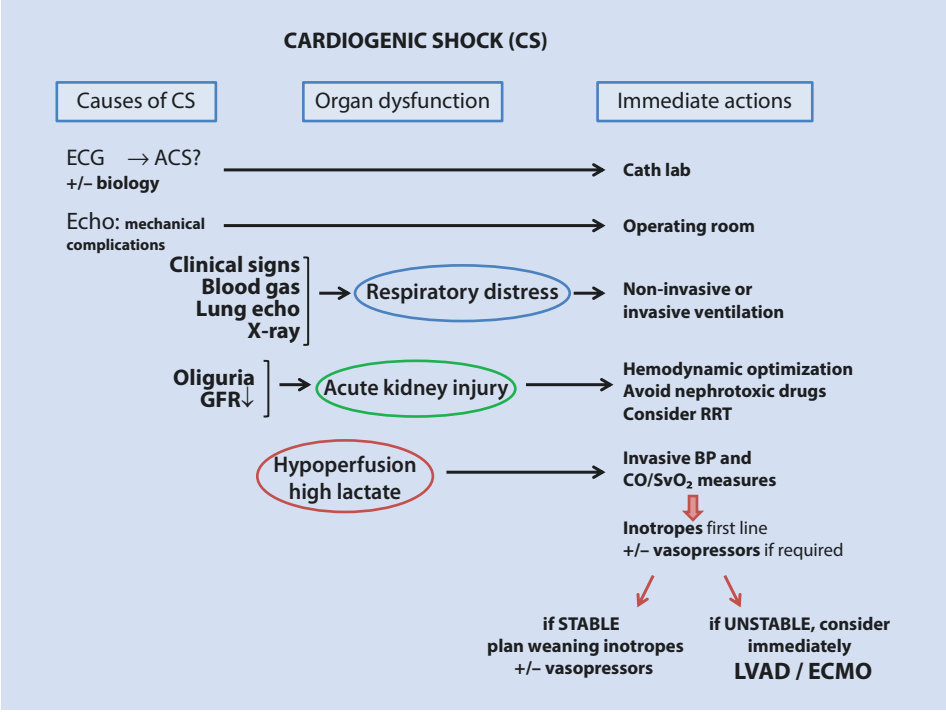

Fig. 32.2 All forms of cardiogenic shock need urgent ECG, troponin essay, and prompt coronary angiogram if the shock etiology is not otherwise explained. Immediate actions can be listed by the specific organ system failure

In selected cases advanced cardiovascular support is needed, such as device therapy. The intra-aortic balloon pump might be indicated for prophylactic reasons (e.g., profound ventricular dysfunction, critical left main disease) or when the patient cannot be weaned from the aforecited medical hemodynamic support [45]. However, no mortality benefit has been shown to date [46, 47]. Guidelines recommend left ventricular assist device (LVAD) or extracorporeal membrane oxygenation (ECMO) without preference in case of required temporary circulatory support [35, 48].

32.4.4 Choosing the Ideal Therapy in AHF with Acute Coronary Syndrome

AHF in this setting is usually a consequence of larger ischemia and myocardial dysfunction, and acute coronary syndrome is one of the main precipitating factors for AHF. The indications of emergency invasive evaluation and revascularization include:

- AHF with STEMI
- AHF with other high-risk ECG signs (e.g., ST-segment elevation in lead aVR, persistent deep precordial T-waves, ST-segment depression)
- ACS associated with persistent chest pain
- ACS with unstable AHF
- ACS with CS

32.4.5 Choosing the Ideal Therapy in Myocarditis

Myocarditis is an inflammatory disease of the myocardium. It often results from viral infections or post-viral immune-mediated responses but shares broad etiology (i.e., infectious including bacterial, fungal, helminthic, protozoal, rickettsial, spirochetal; autoimmune; hypersensitivity reactions to drugs; toxic reactions to drugs; toxic; others including arsenic, copper, iron, radiotherapy, thyrotoxicosis) [3]. Various treatment options have been proposed but are beyond the scope of this review (see Suggested Reading). NSAIDs are being used in uncomplicated myocarditis. However, this treatment is questioned, and aspirin as well as NSAIDs are thought to counteract part of the efficacy of ACE inhibitors in human endocarditis [49].

Patients with fulminant myocarditis or hemodynamically unstable AHF in the context of myocarditis require treatment in an expert center. Treatment is supportive and symptomatic and may include assist devices. Patients refractory to treatment should be evaluated for transplantation.

32.4.6 Choosing the Ideal Therapy in Predominant Right Ventricular Failure

Acute right ventricular failure is rapidly progressing leading to systemic congestion due to either impaired RV filling and reduced RV flow at a later stage. It progresses to dyssynchrony and eventually asynchrony of right and left ventricular performance causing systemic hypotension. This is based on the concept of systolic and diastolic ventricular interdependence where functioning of the left ventricle affects the right ventricle and vice versa.

Four aspects are important in the therapy of right ventricular failure:

1. Volume optimization
2. Vasopressor administration first, and inotrope if needed
3. Mechanical circulatory support
4. Therapy of specific clinical scenarios

32.4.6.1 Volume Optimization

Cautious volume loading based on central venous pressure monitoring is crucial. Even though patients with right ventricular failure can be preload dependent, it is important to not overdistend the right ventricle by volume overloading. Volume overload increases wall tension, decreases contractility, aggravates tricuspid regurgitation, increases ventricular interdependence, impairs left ventricular filling, and ultimately reduces systemic cardiac output.

32.4.6.2 Vasopressor and/or Inotrope Treatment

Norepinephrine is first indicated when hemodynamic instability is present. It is used to restore organ perfusion, especially the coronary and cerebral. A major advantage would be the improvement of systemic hemodynamics without a change in pulmonary vascular resistance.

Inotropes will be administered in case of evidence of low cardiac output. Dobutamine, levosimendan, and phosphodiesterase III inhibitors improve contractility and increase cardiac output. In pulmonary hypertension caused by left heart disease, levosimendan and phosphodiesterase III inhibitors may be preferred over dobutamine.

32.4.6.3 Mechanical Circulatory Support

Mechanical circulatory support may be indicated in RV myocardial infarction, acute PE, following left ventricular assist device implantation, or primary graft failure after heart transplantation.

ECMO and other forms of extracorporeal life support (ECLS) can be chosen for short-term mechanical support. Alternatively, catheter-mounted microaxial pumps can be used (i.e., Impella RP). The problem here is the limited maximum pump capacity. Other right ventricular assist devices such as RVADs can even be used for months but are officially approved for up to 4 weeks. When needed, they can be combined with oxygenators. Rarely, RV function is not restored, and the insertion of an implantable continuous-flow assist device or even heart transplantation has to be considered.

32.4.6.4 Therapeutic Aspects in Specific Clinical Scenarios

Pulmonary Embolism

Acute pulmonary embolism (PE) is one of the most frequent causes of acute right ventricular failure. Early detection and initiation of therapy is pivotal. Choosing the right therapeutic management is mainly based on the patient's condition. The solely, widely accepted indication for systemic thrombolysis is persistent hypotension or shock (i.e., a systolic blood pressure <90 mmHg or a decrease in the systolic blood pressure of ≥40 mmHg from baseline) following acute pulmonary embolism. In hemodynamically stable patients, the hemorrhagic risks of thrombolysis appear to outweigh the clinical benefits. But they should be monitored over the first 2–3 days for consideration of rescue thrombolysis in case of subsequent deterioration.

If thrombolysis is contraindicated or has failed and the patient is hemodynamically unstable, surgical pulmonary embolectomy should be deliberated. Imminent hemodynamic decompensation in combination with anticipated high bleeding risk under thrombolysis is another indication for surgical intervention [50].

Various catheter-directed techniques for the removal of obstructing thrombi from the main pulmonary arteries have been developed over the past years:

- Thrombus fragmentation with pigtail or balloon catheter
- Rheolytic thrombectomy with hydrodynamic catheter devices
- Suction thrombectomy with aspiration catheters
- Rotational thrombectomy

"Purely interventional" options are reserved for patients with absolute contraindications.

Endovascular treatment such as EKOS™ can be used for PE, deep vein thrombosis (DVT), and peripheral arterial occlusion (PAO). EKOS™ represents an ultrasound-enhanced lysis system. The EKOS™ System's targeted ultrasound waves accelerate thrombus dissolution by unwinding the fibrin matrix. Studies reveal lower patient risk and higher procedure predictability when compared to systemic thrombolysis [51, 52].

Pulmonary Hypertension

Right ventricular function is the major determinant of morbidity and mortality in pulmonary hypertension. Looking at the high number of causes of this condition (see Suggested Reading), the diagnostic work-up and the identification of triggers are somewhat time-consuming but also important [53–56]. However, the most frequent cause is infection, and as anticipated, sepsis is known to increase mortality significantly [55]. PH can become

Table 32.3 Most important symptoms of PH and therapies to be considered

Sign/symptom/course	Therapy
Supraventricular arrhythmia	Electrical cardioversion
Anemia	Correction
Hypoxia	Oxygen therapy (arterial oxygen saturation > 90%)
Hypercapnia/acidosis	NIV
Hypothermia	Correction
Venous congestion	Diuretics
Diuretic resistance	RRT
Elevated RV afterload	Intravenous prostacyclin, epoprostenol, inhaled nitric oxide or prostacyclin
Severe PAH but right atrial pressure ≤20 mmHg and arterial oxygen saturation ≥85% on room air	Balloon atrio-septostomy (high-risk technique, not in emergency)
Unresponsive	Bridge to recovery or lung transplant: Impella, ECMO, RVAD

clinically noticeable due to various symptoms. The most important signs and symptoms of PH and therapy considerations are addressed in the following table (Table 32.3).

Concerning correction of anemia, no optimal value for hemoglobin or hematocrit has been determined in patients with RV failure. Focus on correction of hypoxia, hypercapnia, acidosis, and hypothermia is essential as these promote or aggravate pulmonary vasoconstriction. Fluid status should be closely monitored (i.e., cardiac ultrasound, pulmonary artery catheter) due to the aforementioned matters.

Right Ventricular Infarction

Usually, acute inferior wall myocardial infarction (MI) caused by occlusion of the proximal right coronary artery is responsible for right ventricular infarction [57]. The right ventricle tolerates ischemic injury better than the left ventricle due to the following reasons [58, 59]:

- Lower oxygen demand
- Greater oxygen extraction reserve capability during stress
- Dual anatomical supply from the right and the left coronary arteries
- Relatively homogenous transmural perfusion across the cardiac cycle
- Increased propensity to acute collateral development

Treatment includes early myocardial reperfusion, evaluation of inotropic support, correction of bradycardia by atropine or aminophylline, AV sequential pacing in case of high-grade AV block, and reversion of acute atrial fibrillation. In right ventricular infarction as opposed to predominant left ventricular involvement, treatments comprising RV preload such as nitrates or diuretics must be used with caution as they may be deleterious. Treatment of cardiogenic shock has been described above.

Tamponade

Cardiac tamponade mimics acute RV failure and should be considered as differential diagnosis. Urgent percutaneous or surgical pericardial drainage is mandatory and possibly has to be preceded by vasopressor therapy [60].

Acute Right Ventricular Failure in the Intensive Care Setting

Acute RV failure is frequently seen in the ICU [61, 62], and ARDS represents the main cause. Protective ventilation strategy (i.e., plateau pressure < 27cmH_2O, $PaCO_2$ < 8 kPa, adapting PEEP to RV function, considering prone positioning for PaO_2/FiO_2 < 20 kPa/150 mmHg) for prevention or amelioration of its complications is adviced.

Valvular Disease

Acute RV dysfunction develops in both left-sided and right-sided valvular heart diseases. Surgery is recommended in valvular disease with right heart failure, severe tricuspid regurgitation and poor response to diuretics, infective endocarditis difficult to eradicate, large vegetations, and recurrent emboli [63].

Surgery

RV failure can develop after non-cardiac surgery and after cardiac surgery. In non-cardiac surgery, it most often develops secondary to increased right ventricular afterload following acute pulmonary hypertension. In cardiac surgery, it is frequently provoked by volume overload, myocardial ischemia, pre-existing RV dysfunction, or arrhythmia. Therapy should focus on the underlying cause including intolerance of sternal closure after cardiac surgery.

32.5 General Aspects and Long-Term Treatment

When the patient's situation is under control, he or she should be interrogated for cardiac risk factors if not done in the first place. Lifestyle changes (e.g., stop smoking) should now be addressed as a priority. Heart failure medication should be started or adjusted with respect to the installed therapy before acute decompensation. ACE inhibitors are indicated even in asymptomatic patients and should be commenced as soon as possible. Their beneficial effect on outcome including mortality in patients with severe congestive heart failure had been confirmed already in 1987 [64] and was confirmed in studies that were performed after [65, 66]. In 1996, the advantageous effects of beta-blockers (carvedilol) on outcome including mortality in patients with heart failure were confirmed [67]. A recently published paper revealed that the administration of metoprolol can even prevent infarct size [68]. Heart failure medication can be extended to aldosterone blockers (synonym: mineralocorticoid receptor antagonists) and hydralazine or nitrates if hypertensive blood pressure cannot be sufficiently controlled by an ACE inhibitor and a beta-blocker. In case of intolerance of ACE inhibitors, angiotensin 1 receptor antagonists should be considered. Recent findings demonstrate a significant mortality benefit in patients with heart failure with reduced injection fraction when treated with sacubitril plus valsartan (Entresto®), also when compared to an ACE inhibitor alone [18, 19]. More detailed information on heart failure medication is beyond the scope of this chapter but is easily accessible.

Another aspect for the prevention of severe future deterioration of the patient's clinical condition is to explain the nature of this disease to the patient and to emphasize the importance of heart failure medication intake. Comorbidities (e.g., COPD, sleep apnea, anemia, depression, and memory disorders) [2] should not be missed here. The patient should be instructed to see his family doctor every semester initially for documentation of further evolution and continuing adjustment of therapy if indicated.

In summary, the management for prevention of rehospitalization for heart failure includes the following actions [2]:

1. Continuation or initiation of long-lasting therapies of heart failure
2. Optimal management of underlying heart diseases
3. Optimal management of comorbidities
4. Patient education on water and salt restrictions
5. Nutritional support
6. Careful patient education

Conclusion

Acute heart failure is a complex and heterogeneous clinical syndrome. It can be a serious life-threatening condition that in some cases needs urgent and aggressive treatment to achieve patient stabilization [29]. Despite therapeutic advances, the prognosis of AHF remains poor [2, 70]. However, earlier diagnosis, triage, and initiation of specific treatment for AHF are associated with reduced mortality and shorter length of hospital stay.

Take-Home Messages

- Organ congestion is a frequent acute or subacute feature in AHF.
- Early initiation of treatment leads to better outcome.
- Perform intubation and install controlled mechanical ventilation rather early in respiratory deterioration.
- In case of hemodynamic instability, start with inotropes in the left ventricular dysfunction and with vasopressors in the right ventricular dysfunction.
- Ventricular interdependence is an important pathophysiological mechanism in right ventricular failure.
- A multidisciplinary approach is warranted to improve outcome including prevention of rehospitalization.
- AHF management consists of situation-adjusted therapeutical procedures that are summarized in the following ◘ Table 32.4.

Table 32.4 Overview of clinical signs and adequate therapy in AHF

	Clinical signs	Hemodynamic therapy
Prehospital setting[a]	Signs of congestion Signs of right or left ventricular dysfunction Signs of low cardiac output	NIV Diuretics Nitrates Fluid challenge CPR
First 2 h[a]	Warm/wet AHF [29, 69] Dry/cold AHF (CS)	Diuretics Vasodilators Oxygen NIV (APE) Fluid challenge Inotropes Vasopressors
AHF in coronary syndrome	Signs of AHF Angor (may be absent)	Coronary angiogram Supportive therapy based on clinical symptoms Heart failure medication
Myocarditis	Signs of AHF	Supportive therapy based on clinical symptoms
Predominant right ventricular failure[a]	Signs of right ventricular dysfunction, including RV congestion Signs of low cardiac output state Hypoxemia Kussmaul sign	Cautious volume loading Vasopressors and/or inotropes Mechanical circulatory support Etiology-specific treatment
Cardiogenic shock	Hypotension and signs of organ hypoperfusion	Coronary angiogram Inotropes Vasopressors Respiratory support Device therapy

[a]See Table 32.1

To Sum-Up

Key points in acute right and left ventricular heart failure.

Section	Feature	Key points
► 32.2	Main symptoms of acute right heart failure	Jugular venous distension, paradoxical pulse, Kussmaul sign, alteration of mental state, hypotension, tachycardia
	Main symptoms of acute left heart failure	Dyspnea, pink frothy sputum, persistent cough or wheezing, new-onset arrhythmia, alteration of mental state, hypotension, tachycardia
► 32.3	Most common etiologies of right ventricular failure	Acute right ventricular failure, right ventricular ischemia/infarction, sepsis, chronic pulmonary hypertension, arrhythmia

Section	Feature	Key points
	Most common etiologies of left ventricular failure	Acute left ventricular failure, left ventricular ischemia/infarction, sepsis, chronic arterial hypertension, arrhythmia, valvulopathies
▶ 32.4	Current practice of therapy of acute right and left heart failure	Based on etiology (see Table 32.4)
▶ 32.5	Management for prevention of rehospitalization	Continuation or initiation of long-lasting therapies of heart failure, optimal management of underlying heart diseases, optimal management of comorbidities, patient education on water and salt restrictions, nutritional support, careful patient education

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Suggested Reading

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