CHAPTER 51
Non-pharmacologic therapy of acute heart failure: when drugs alone are not enough

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Summary
Pharmacologic agents remain the mainstay of therapy for acute heart failure syndromes (AHFS). However, at all time during the early diagnostic, aetiological, and therapeutic work-up non-pharmacologic therapy may be indicated. In patients not responding to the standard pharmacologic regimen, with or without non-invasive ventilation, myocardial ischaemia should be excluded.

Management of the complex cardiac patient with advanced heart failure (HF) and/or (potential) haemodynamic compromise has become a special dimension for specialized myocardial intervention centres (MICs) providing 24/7 state-of-the-art facilities for (primary) percutaneous coronary intervention (PCI) and advanced (intensive) cardiac care including mechanical ventilation, ultrafiltration with or without dialysis, and extracorporeal circulatory assist.

Through the understanding of the underlying pathophysiology and approaches to the problems of acute HF, one should be better prepared to understand and treat its many facets.

Introduction
The intent of this chapter is to give a readable, concise clinical overview of the non-pharmacologic treatment of different AHFS, providing a basis for clinical algorithms and the clinical use of evidence-based treatment guidelines.

The framework for the utilization of different treatment modalities will be discussed. There will be a focus on physiologic approaches addressing the balance between oxygen demand and delivery, and the manipulation of cardiopulmonary interactions to optimize ventricular function, including the use
Background

The heart can be conceptually approached as a hydrodynamic input–output system, a haemodynamic compression pump, a muscular pump, or a pluricellular tissue pump [1]. The heart acts, in close relationship to the lungs and circulation, with the ultimate goal to preserve adequate tissue oxygenation and function (see Chapter 35 Heart failure, ESC Textbook of Cardiovascular Medicine, 2nd edition, 2009.) The primary physiological task of the cardiorespiratory system is to deliver adequate oxygen (O\(_2\)) to meet the metabolic demands of the body (VO\(_2\)) (Fig 51.1).

Oxygen delivery (DO\(_2\)) is the amount of oxygen delivered to the peripheral tissue, and is obtained by multiplying the arterial oxygen content (CaO\(_2\)) by the cardiac output (CO):

\[
DO_2 = CaO_2 \times CO \quad \text{Eqn. 1}
\]

The oxygen content of blood is the volume of oxygen carried in 100mL of blood. It is calculated by:

\[
(O_2 \text{ carried by Hb}) + (O_2 \text{ in solution}) = (1.34 \times \text{Hb} \times \text{SaO}_2 \times 0.01) + (0.023 \times \text{PaO}_2) \quad \text{Eqn. 2}
\]

where:
- \(\text{SaO}_2\) = percentage saturation of Hb with oxygen
- \(\text{Hb}\) = haemoglobin concentration in grams per 100mL blood
- \(\text{PaO}_2\) = partial pressure of oxygen (0.0225 = mL of O\(_2\) dissolved per 100mL plasma per kPa, or 0.003mL per mmHg).

AHFS may occur de novo or as a complication of worsening chronic HF at different pathophysiological stages of the disease (Fig. 51.2). In its severest form, AHFS consist of a life-threatening medical emergency that presents with severe circulatory failure and symptoms of impaired organ perfusion and resulting damage. The syndrome is complex and often is the result of multiple contributing diseases. The symptoms of HF may be aggravated by non-cardiovascular comorbidities such as obstructive lung disease or coexisting end-organ disease, especially renal dysfunction. HF might be caused by ischaemia, hypertension, atrial fibrillation (AF), or other non-cardiac conditions (e.g. renal insufficiency, diabetes, sepsis), or untoward drug effects (see Chapter 49–50, Acute heart failure classification and pathophysiology; Chapter 35, Heart failure, ESC Textbook of Cardiovascular Medicine, 2nd edition, 2009) leading to a failure of the heart to meet metabolic demands [2].

The management of AHFS is challenging given the heterogeneity of the patient population in terms of clinical presentation, pathophysiology, prognosis, and therapeutic options. The main goals of the acute management of the patient with AHFS are to:

\[
\text{SvO}_2 = 1 - \frac{\text{VO}_2}{(\text{CaO}_2 \times \text{CO})} \quad \text{Eqn. 3}
\]
CHAPTER 51  NON-PHARMACOLOGIC THERAPY OF ACUTE HEART FAILURE: WHEN DRUGS ALONE ARE NOT ENOUGH

Resuscitate the patient. Stabilize and treat life-threatening conditions and prevent further deterioration (pre-hospital, initial in-hospital phase up to 24h)

Improve haemodynamics, often leading to an amelioration of symptoms (in-hospital phase)

Determine the possible aetiology and provide further risk assessment. Enable early intervention when an acute reversible problem exists (in-hospital phase)

Ensure implementation of evidence-based guidelines (pharmacological, surgical, interventional, and implantable cardiac-defibrillator/cardiac resynchronization therapy [CRT]) to reduce future readmissions. (pre-discharge, early post-discharge phase)

Monitor your patient on an ongoing basis, triage early among alternative levels of hospital care, and allocate right hospital resources whenever needed.

Pharmacologic agents traditionally have been the mainstay of therapy for AHFS; however, at each phase of hospital evaluation and management concomitant non-pharmacologic therapy should be considered (Fig. 51.2). The initial AHFS evaluation should begin with a careful history and physical examination. Early triage and management may be guided by vital signs, physical findings, and urinary output (Table 51.1). A standard 12-lead electrocardiogram might indicate potential myocardial ischaemia, rhythm, or conduction abnormalities warranting an early invasive approach [2]. Conventional echocardiography, supplemented by tissue Doppler imaging (TDI), is a highly valuable non-invasive tool for the assessment of cardiac anatomy and function [3]. In addition to established critical roles in the early diagnostic, aetiological, and therapeutic work-up in AHFS [2,4,5], echocardiography plays an important clinical role in early ‘prognostication’ and triage to more oriented therapy [3,4,6]. (For details see Chapter 20 Echocardiography; chapter 35 heart failure, ESC Textbook of Cardiovascular Medicine, 2nd edition 2009)

A more definitive resuscitation strategy in advanced AHFS not responding to standard pharmacologic therapy, and/or patients (at high risk of) developing circulatory failure, may require invasive monitoring and a goal-oriented manipulation of cardiac preload, afterload, and contractility to achieve a balance between systemic oxygen delivery and oxygen demand (Fig. 51.1a). Endpoints used to confirm the achievement of such a balance include normalization of values for mixed venous oxygen saturation, arterial lactate concentration, base deficit, and pH [9]. Mixed venous oxygen saturation (Fig. 51.1b):

\[
SvO_2 = \frac{SaO_2 - \left[ VO_2/(CO \times [Hb \times 13.9]) \right]}{Eqn. 3}
\]

has been shown to be a surrogate for the cardiac index as a target for haemodynamic therapy. In cases in which the insertion of a pulmonary-artery catheter is impractical, venous oxygen saturation (central venous oxygen saturation, \(SvO_2\)) can be measured in the central circulation [10]. Importantly, trends of haemodynamic measures in the right direction are as important as targeting preset specific numbers. In addition, while inotropic therapy is often used in such situations to improve haemodynamic measures, its use is eclipsed by a higher number of adverse events ([Optimize-HF: the Organized Program to Initiate Lifesaving Treatment in Hospitalized Patients with Heart Failure program [59]).

In the critically ill (cardiac) patient the cardiocirculatory system is mainly challenged by two different conditions. Firstly, a drop in \(DO_2\) which can be induced by anaemia, hypoxia, hypovolaemia, HF, or any combination of these. Secondly, fever, pain, stress, and/or respiratory failure, etc. may further decrease \(SvO_2\) or \(ScvO_2\) by increasing whole-body VO\(_2\). It is of pivotal importance that the physician

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<th>Table 51.1</th>
<th>Vital signs and clinical symptoms to guide early management in AHFS (7,8)</th>
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<tr>
<td>Heart rate &gt; 100bpm</td>
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<tr>
<td>Systolic blood pressure &lt; 100 (~120)mmHg</td>
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<tr>
<td>Proportional pulse pressure ≤ 25 (CI &lt; 2.2L/min/m(^2))</td>
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<td>(If) orthopnoea (PCWP &gt; 22mmHg)</td>
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<td>Killip class II–IV</td>
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<td>Age</td>
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Proportional pulse pressure = systolic blood pressure (BP) – diastolic BP/systolic BP.

CI cardiac index in L/min per square metre (normal: 2.6–4.2).

PCWP: pulmonary capillary wedge pressure.

Orthopnoea: cough, especially during semi-recumbency may be the equivalent of orthopnoea.
dealing with this type of patient understands these basic principles. Three items should be evaluated in the early stabilization period:

1. The need for sedation and ventilatory support. (Ventilation)
2. The need for inotropic/vasopressor support. Does the patient need a central venous access? (Infuse)
3. The need for mechanical circulatory support. (Pump)

In the context of non-pharmacologic therapy of AHFS, the 'I' may become 'Intervention' including cardioversion, revascularization, etc.

**Non-pharmacologic therapy of acute heart failure**

**The pathway from stabilization to intervention**

Patient care in AHFS is a dynamic process, requiring ongoing simultaneous diagnosis and treatment. Four phases for patient evaluation and management are proposed: 1) the initial or early phase (i.e. emergency department); 2) the in-hospital phase; 3) the pre-discharge phase; and 4) the early post-discharge phase (Fig. 51.3) [11].

The goal of treatment in the pre-hospital setting or in the emergency room is to improve tissue oxygenation and optimize haemodynamics in order to improve symptoms, permit transport to a higher-level hospital, and ensure survival. After the initial assessment which should be performed quickly, all patients should be considered for oxygen therapy and non-invasive ventilation (NIV: e.g. continuous positive airway pressure [CPAP] or bilevel positive airway pressure [BiPAP]). Escalation to ETI and an invasive approach may be required in cases of severe sensorial impairment (including coma), (high risk of) circulatory failure, life-threatening arrhythmia, progressive hypoxia, or life-threatening complications like pneumothorax etc. However, the majority of patients respond well to an initial goal-directed pharmacologic therapy with or without NIV and one should not intubate and ventilate only for the comfort of the doctor to work with a sedated patient.

Portable circulatory mechanical assist (intra-aortic balloon pump [IABP]) may be indicated early during the initial resuscitation and stabilization phase, in refractory (within 6–12h) or advanced AHFS, especially when drug-resistant hypotension and pulmonary congestion sustains. However, at all stages myocardial ischaemia should be excluded and treated whenever possible [2,12,13].

Patients with AHFS complicating acute coronary syndromes (ACS) requiring emergent PCI are at considerable risk for haemodynamic compromise or collapse at the time of intervention. Management of the complex cardiac patient with (potential) haemodynamic compromise has become a special dimension for specialized MICs providing state-of-the-art facilities for (primary) PCI, including experienced senior operators available on a 24h/7 days a week basis and critical care physicians experienced in all aspects of
diagnosing and treating complex cardiac patients. If these facilities are not available on site, upgrading and transfer to a specialized MIC should be initiated early (within the first 12h) [14]. These patients may require invasive monitoring and ETI for comfort (both patient and investigator) and clinical reasons. Portable mechanical circulatory assist may provide a valuable safeguard to increase (transport and) procedural safety. Early intervention in response to physiological instability might prevent further deterioration in many patients.

Other comorbidities including (supra-)ventricular arrhythmias, bradycardia, infections, pulmonary diseases, severe anaemia, and renal or hepatic dysfunction must be addressed, and corrected when appropriate, on a ongoing base during the hospital stay (see Fig. 51.3).

**Mechanical ventilation and oxygen transport**

In AHFS, pulmonary and systemic congestion due to elevated ventricular filling pressures with or without a decrease in cardiac output and tissue hypoperfusion is frequent [11]. Lung dysfunction can be evaluated in terms of lung mechanics and gas diffusion (see Chapter 20, respiratory monitoring). It is recommended to administer oxygen as early as possible in hypoxaemic patients to achieve an arterial oxygen saturation ≥ 95% (>90% in patients with chronic obstructive pulmonary disease [COPD]) [69].

Following the formula to calculate DO\(_2\) (see Eqns. 1 and 2), tissue oxygen delivery could be increased just by administering supplemental oxygen to increase the arterial oxygen tension (PaO\(_2\)) provided there is an adequate Hb concentration. However, spontaneous ventilatory efforts require muscular activity, thereby consuming O\(_2\) and producing carbon dioxide (CO\(_2\)), which represents a extra metabolic load on the cardiovascular system. The inspiratory effort used by patients with acute respiratory failure is about four to six times the normal value [15–17]. Increased work of breathing will stress the cardiovascular response to maintain adequate tissue perfusion and will result in increased cardiac output to meet the increased oxygen demand (VO\(_2\)).

Mechanical ventilatory support can directly alter oxygen homeostasis in the human body by its effect on the arterial oxygen content, cardiac output, and VO\(_2\) of the respiratory system. Ventilation affects the circulation primarily by altering the preload and afterload conditions of the heart through changes in intrathoracic pressure (ITP) and lung volume [18].

In other words, ventilation is exercise, since it consumes O\(_2\) and produces CO\(_2\).

**Mechanical ventilation (see also Chapter 24 Procedures in ICCU)**

In patients with severe cardiopulmonary distress for whom the effort of breathing is intolerable or inefficient, mechanical ventilation will artificially replace the action of the respiratory muscles. The objectives of mechanical ventilation are primarily to decrease the work of breathing (VO\(_2\) of the respiratory system) and reverse life-threatening hypoxaemia or acute progressive respiratory acidosis. The need for mechanical ventilation and artificial airways can be reduced by substituting non-invasive ventilatory support when appropriate. However, one has to bear in mind that the mechanically-assisted inspiration will result in increased ITPs and mechanically-assisted expiration in decreased ITPs which is the opposite of normal ventilation.

NIV, delivered through tight-fitting face masks, has been shown to maintain adequate gas exchange, reduce the work of breathing, and to limit the need for ETI in patients with acute respiratory failure due to acute pulmonary oedema, statis asthmaticus, or COPD [19–23]. Although its use has been associated with decreased resource utilization, mortality benefit over oxygen for either continuous or bilevel NIV remains questionable [24,25]. NIV can be safely used, even outside the intensive care unit, by experienced nurses, respiratory therapists, and physicians. However, relative haemodynamic stability, full patient cooperation and the ability of the patient to protect their own airway are important prerequisites when choosing a NIV mode. NIV should never be used when there is a need for emergent intubation. Appropriate initial settings for NIV in AHFS are listed in Fig. 51.4. The response to NIV should be assessed after 60min and thereafter on a continuous basis by trained personnel. Parameters of interest are respiratory rate (>35 beats/min and above the value at admission), blood pressure, pulse rate, pH (pH ≤ 7.20 and above the value at admission on two consecutive arterial blood gas samples), PCO\(_2\) (± PO\(_2\)) and level of consciousness.

Fortunately, endotracheal intubation (ETI) and mechanical ventilation are only required in a minority of AHFS patients. Patients at risk are: non-responders to NIV; those with (profound) circulatory failure; or those at high risk of haemodynamic deterioration during intervention or inter-hospital transport.

Ventilatory mode and settings directly relate to outcome in patients suffering from acute respiratory failure [26,27]. Although there is still a debate as to whether a tidal volume of 6–8mL/kg should be applied to all patients ventilated for
acute lung injury, there is a general agreement that end-
plateau pressures should be kept below 30 cmH₂O when-
ever possible. Cyclic closing and re-opening of alveolar
units during mechanical ventilation should be avoided.

BiPAP/airway pressure release ventilation (APRV) (see
\(\text{E} \) Chapter 24) with superimposed spontaneous breathing
improves gas exchange, often with lower maximal airway
pressures, compared with controlled mechanical ventila-
tion. Therefore, it is an attractive mode of ventilation in
‘hypervolaemic’ HF patients. Positive-pressure ventilation
(PPV), by abolishing the negative swings in ITP, will
selectively decrease left ventricular (LV) afterload, as long
as the increases in lung volume and ITP are small (28).
Spontaneous inspiration and spontaneous inspiratory
efforts will decrease ITP. An increase in ITP compresses the
vena cava and thus decreases venous return. Lung hyper-
inflation may further negatively affect cardiac output by
altering right ventricular (RV) preload and afterload.

Applying this knowledge, ventilator settings may even
prove to be more important than ventilator mode.

Static end-inspiratory pressures of ≤25 cmH₂O are safe,
>30 cmH₂O dangerous. Considering the potential worsen-
ing effect of positive end-expiratory pressure (PEEP) on
venous return and cardiac output, in AHFS only moderate
levels (3–5 cmH₂O) should be applied initially and adjusted
upon haemodynamic tolerance. Respiratory rate will be set
between 25–20/min and adjusted according to the patient’s
rate demand and PCO₂. Patients with acute hypoxaemic res-
piratory failure usually have small lung volumes. Decreases
in lung volume induce alveolar collapse and hypoxia,
stimulating an increased pulmonary vasoconstriction by
the process of hypoxic pulmonary vasoconstriction [29].

Recruitment manoeuvres, PEEP, and CPAP may reverse
hypoxic pulmonary vasoconstriction and reduce pulmo-
lar artery pressure [29].

**Transfusion of red blood cells**

Anaemia is common among patients with HF, probably
occurring in approximately 20% of patients, depend-
ing on the definition of terms and severity of patient ill-
ness [30]. The human tolerance of anaemia is dependent
on the recruitment of physiologic reserve, a major com-
ponent of which is the ability to increase cardiac output.

Patients with chronic HF lack normal physiological reserve
to compensate for decreased haemoglobin and may mani-
fest decreased aerobic capacity in response to mild degrees
of anaemia [31]. The clinical utility of blood transfusion in
anaemic cardiovascular disease populations remains con-
troversial [32–34]. Balancing risks and benefits, transfusion
should only be considered as an acute treatment for severe
anaemia on an individualized basis.

**Revascularization and surgical therapy**

Coronary artery disease (CAD), or its atherothrombotic
complications, has emerged as the dominant underlying
aetiological factor in patients with HF and the presence of
ischaemic and/or stunned/hibernating myocardium may
have a profound impact on the initial, in-hospital, and
post-discharge management and prognosis [35–37]. In
patients with AHFS and evidence of myocardial ischaemia,
the diagnosis or suspicion of obstructive CAD should lead
to the consideration of early myocardial revascularization
[38]. The treatment approaches for AHFS with CAD are
discussed elsewhere (see \(\text{E} \) Chapter 45–46).
With increasing operator experience, refinement of angioplasty hardware and technique, and adjunctive pharmacological treatment, the morphologic and clinical profile of patients acceptable for coronary angioplasty has widened considerably. However, even in the acute setting, the benefits of a specific percutaneous procedure should be weighed against the risks involved, taking into account alternative treatment strategies, including the individual operator’s and overall institution’s (interventional and intensive care team) experience [39]. As indicated in the background section of this chapter, in such circumstances, transfer to a ‘centre of excellence’ that routinely performs complex PCI may be the most effective and efficient course of action. Clinical and angiographic characteristics are proven to be equally important in determining procedural risk with PCI [40 – 42].

Management of the complex cardiac patient with (potential) haemodynamic compromise has become a special dimension for specialized MICs.

Very high-risk procedures are, of course, often inevitable because of the inherent risk and presentation of the underlying disease. Knowledge of very high-risk procedural complications should alert the operator and should trigger measures to reduce the extent of the risk whenever possible. A strategy of early angiography and revascularization, where appropriate, in AHFS must also take into account the potential costs.

Revascularization with either PCI or coronary artery bypass grafting (CABG) can improve the perfusion of viable myocardium but does not restore function in areas of infarction. Surgical procedures to eliminate or exclude areas of infarction (surgical ventricular restoration), repair mitral regurgitation, or support the failing myocardium are discussed in Chapter 52.

**Portable cardiac assist**

Percutaneous mechanical circulatory support (MCS) can be provided by a variety of devices and modalities designed to increase forward blood flow and reduce filling pressures (see Chapter 30). Since IABP is incapable of supporting a patient with complete haemodynamic collapse, immediate triage to more advanced percutaneous (or implantable) circulatory support modalities is warranted when needed. Therefore, treatment options for MCS must be tailored to each patient in order to optimize the benefits and minimize the risk of detrimental effects. Experience with these systems continuous to grow, with leading centres and investigators contributing meaningful information towards the application and development of the latest technologies.

A minimal flow rate of 70mL/kg body weight/min is required to provide adequate organ perfusion. To make percutaneous insertion realistic, the maximum diameter of cannulae should be downsized to a maximum of around 10F. Pre-insertion abdominal and iliofemoral angiography is useful to assess feasibility of insertion and to select the appropriate insertion site, thereby minimizing complications. In case of emergency in patients with severe atherosclerotic disease, percutaneous transluminial angioplasty of the femoral artery or surgical placement may be considered. In order to prevent limb ischae-mia caused by femoral cannulation, distal leg perfusion with a small catheter placed in the distal artery is often used.

MCS can be used to resuscitate patients, as a stabilizing measure for angiography and prompt revascularization, or to buy time until more definite measures can be taken [40]. In this setting, the revival of extracorporeal circuitry and hardware that can provide both respiratory and circulatory support to patients for periods up to several weeks is of particular interest (Fig. 51.6).
In addition there is experimental evidence that ventricular unloading of the left ventricle can significantly reduce infarct size. Mechanical support combines the beneficial effects of the myocardial unloading and an increase in tissue perfusion pressure [43–45].

**Fluid management beyond diuretics: haemofiltration with or without dialysis**

The pathophysiology of the cardiorenal interaction in the setting of advanced decompensated heart failure (ADHF) is poorly understood. Renal impairment is often present at time of admission [46]. Structural renal dysfunction due to diabetes, hypertension, and arteriosclerosis are common. Worsening renal function occurs in 20–30% of patients during hospitalization [47–50]. This worsening during or after discharge may result from further neurohormonal and haemodynamic abnormalities (low cardiac output and/or high venous pressure), which may be aggravated by high-dose loop diuretics [51–54].

Isolated ultrafiltration has recently gained great interest in the non-pharmacological management of congestion in HF [55–57]. The rationale for use of extracorporeal methodologies centres on three aspects: fast removal of fluid; avoidance of maladaptive renal tubular autoregulatory responses induced by diuretics; and higher magnitude of sodium clearance. In the acute setting it may even improve lung mechanics through body fluid content reduction (Fig. 51.7). However, excessively high ultrafiltration rates may lead to decreased effective blood volume, hypotension, renal hypoperfusion, pre-renal azotemia, and possibly acute renal failure necessitating dialysis.

**Sinus rhythm and arrhythmia devices in heart failure**

Arrhythmia may frequently precipitate or aggravate AHFS and should be treated aggressively.

AHFS are often associated with electrical and conduction abnormalities [58]. AF and HF often coexist [59,60]. The prevalence of AF increases with the severity of HF and worsens its course by loss of atrial contraction, poor rate control, and irregular rhythm [61].

Some component of (reversible) tachycardia-induced myopathy is seen in up to 50% of patients with LV dysfunction and AF [62–64]. Furthermore, the detrimental effects of AF on HF also may derive from the negative inotropic effects of antiarrhythmic drugs. Over the last decade the non-pharmacologic armamentarium for HF patients has expanded.

**Direct current cardioversion**

When a rapid ventricular response AF does not respond promptly to pharmacological therapy, or in case of severe haemodynamic compromise, intractable myocardial ischaemia, immediate direct-current cardioversion (biphasic, synchronous, high energy: 200–360J) is recommended. In patients who relapse after successful cardioversion, it can be useful to repeat the procedure following administration of appropriate antiarrhythmic medication.

**Ablation therapy**

When in selected patients the rapid ventricular response is refractory to pharmacological therapy and/or cardioversion, ablation of the atrioventricular (AV) junction by catheter radiofrequency energy with subsequent pacemaker implantation can improve cardiac performance. The choice between single- or dual-chamber pacemaker depends on the probability of restoring sinus rhythm. When in the chronic, pre-discharge phase, curative AF ablation seems to be a better therapeutic option [65,66].

Creating heart block and implanting a permanent pacemaker provides regularization of the ventricular rate, and the use of biventricular pacemakers may prevent an adverse effect of RV pacing on LV function.

**Pacing and cardiac resynchronization therapy**

In case of atropine (Isuprel®)-resistant bradycardia, transvenous pacing may be used as an interim measure. If pacemaker implantation is indicated, CRT should be considered as well. A large body of evidence has emerged recently that underscores the harmful effects of long-term RV pacing. LV dyssynchrony imposed by RV pacing can lead to LV remodelling with further dilatation and decrease in LV ejection fraction.
CRT is an innovative, pacemaker-based approach to the treatment of patients with HF who have a QRS interval of at least 120ms on 12-lead electrocardiography [67,68]. The purpose of resynchronization is to provide electromechanical coordination and improved ventricular synchrony in symptomatic patients who have severe systolic dysfunction and clinically significant intraventricular conduction defects, particularly left bundle branch block. With CRT, pacemaker leads are placed to stimulate both ventricles, thus bypassing the conduction block in the left bundle branch. Pacemaker leads are placed to stimulate both ventricles, thus bypassing the conduction block in the left bundle branch (see Chapter 35 Heart failure, ESC Textbook of Cardiovascular Medicine, 2nd edition 2009). Beneficial effects include reverse remodelling, resulting in decreased heart size and ventricular volumes, improved ejection fraction, and decreased mitral regurgitation.

Most biventricular devices are programmed to sense spontaneous activity (i.e. sinus rhythm) in the atrium and provide pacing in the ventricles. Native (non-paced) atrial contraction is preferred, since atrial pacing induces intra-atrial conduction delay, which can lead to alterations in the optimal AV delay and reduced overall effectiveness of biventricular pacing. Pacing in the atrium will be provided only if the patient has an indication for this, such as sinus-node dysfunction. A short AV pacing delay most often is programmed to ensure consistent pacing of the ventricles. However, it is recommended to assess the optimal AV delay with echocardiography on easy to obtain transmirtal Doppler flow.

Loss of biventricular pacing can also occur with lead disruption or malfunction of the LV lead. This may be evidenced by widening of the QRS complex with or without a significant shift in the QRS axis. However, it is not always possible to recognize loss of LV pacing from the surface electrocardiogram alone. If, after a period of clinical improvement, the patient has a sudden deterioration of symptoms, the function of the device should be reassessed. Arrhythmias or inappropriate device settings should be ruled out. Measuring of the stimulation threshold using the pacemaker programmer is recommended. Fine tuning of the pacing output, AV delay, and interventricular timing (preferably with the help of ultrasound) should be performed.

**Personal perspective**

A variety of non-pharmacologic approaches complement drug therapy of AHFS. In the early phase, patient management should be directed to the primary pathophysiologic problem. The development of targeted ('goal-directed') therapy will require a combination of imaging and therapy, which will permit individualized management decisions and hopefully facilitate better clinical outcomes in AHFS.

A combined cardiorenal approach may yield the greatest advances in the contemporary management of acutely decompensated HF. There is a high volume of ongoing research efforts focused on the early treatment of AHFS, including its non-pharmacologic aspects and mechanical circulatory support.

**Further reading**


For additional multimedia materials please visit the online version of the book (http://www.esciacc.oxfordmedicine.com).

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