Cardiac Surgical Intensive Care

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9.1 Introduction

The chapter will provide an overview of protocols and therapies in perioperative cardiac surgical intensive care. Due to its broad spectrum, cardiac surgical intensive care cannot be discussed in a single chapter. In this context suggested literature provides a more in-depth representation of the topic.

The chapter is written based on the clinical and chronological course of the patient. However, the hectic schedule of cardiac surgical intensive care unit (CSICU) will frequently alter the temporal schedule of a cardiac surgical intensivist. A seasoned intensivist will be able to make the appropriate decision despite limited time in obtaining information and diagnostics. The content of this chapter is aimed at supporting the cardiac surgical intensivist in this critical task.

9.2 Aims of Intensive Care

One of the basic goals of postoperative intensive care is maintain hemodynamic stability, hemostasis, and adequate oxygenation and ventilation leading to prompt extubation in order to move the patient quickly to the step-down unit.

In this process the patient should wake up neurologically intact, have adequate perfusion and hemodynamics, get extubated promptly, tolerate liquids and solid food, and have adequate pain control.

The prerequisite for this process is preoperative normal function of organ systems. Furthermore, it is expected that appropriate anesthesia induction and maintenance, as well as uncomplicated cardiac surgical operation, have been performed.

Here lay two further aims of intensive care: prevention of complications by appropriate preventive intervention and, in case of issues, efficient and quick treatment of complications.

The length of intensive care stay remains a surrogate for «complicated postoperative course» after open-heart operations. It can be assumed that approximately 75 % of the cardiac surgical patients will have an uneventful postoperative course, while 25 % will require a prolonged and complicated CSICU stay.

9.3 General Intensive Care

9.3.1 CSICU Admission

The monitored transport of the patient from the operating room to the CSICU has to be performed by the attending anesthesiologist and a member of cardiac surgical team. The report must be communicated directly to the CSICU physician on duty, and it must contain all relevant information about the operation to estimate the therapeutic milestones for the next 12–24 h.

The report should contain all necessary pre- and intraoperative information. A protocol involving standardized transfer reporting is advisable. Here are suggestions for the content of this report:

- Name
- Age
- Size and preoperative weight
- Medical history (comorbidities, risk factors)
- Preoperative medication (sedative agents, antibiotics, antihypertensive medication)
- Preoperative status (any type of complications, difficulties during intubation, abnormal baseline activated clotting time, and other abnormal laboratory values)
- Preoperative diagnosis and details of the surgery performed
- Anesthesia induction and intraoperative medication
- Ventilator settings and abnormalities in blood gas analysis
- Intraoperative course
- Any complications
- Any incomplete coronary revascularization or remaining valvular regurgitation after repair
- ECG findings (signs of ischemia)
- Bleeding tendency
- Number and location of drains and temporary pacemaker electrodes
- Transfused and any remaining blood products
- Intraoperative volume status and cardiac function after weaning off extracorporeal circulation (heart rate, systolic blood pressure, central venous pressure, cardiac output)
- Course of pharmacologic support, actual medication at transfer
- Intraoperative renal function and positive or negative fluid balance

In times of electronic health records, the CSICU admission report may be performed electronically. However, a complimentary verbal report is highly recommended to clarify any questions, issues, and inconsistencies. The verbal report may specifically enhance alertness to specific existing or upcoming potential problems!

9.3.2 Patient Assessment at CSICU Arrival

9.3.2.1 Physical Examination

The physical examination at the time of admission to the CSICU provides critical information that completes the clinical picture in combination with monitoring tools and laboratory and imaging results.

The physical examination at admission on the CSICU should be expeditious and is grossly performed in the form of inspection, palpation, and auscultation. The clinical exam findings at the time of admission are important indicators of the patients' clinical course.

After connecting the patient to the ventilator, both lungs are carefully auscultated, to ensure adequate and equal ventilation of each lung side. Furthermore the heart auscultation may detect any murmurs and rubs and will help in assessing the heart rhythm at admission. Due to opiate injection, bowel sounds are mostly quiet and of no diagnostic significance at this point. The correct position of the nasogastric tube should be confirmed by air insufflation into the tube and simultaneous auscultation of the epigastrium.

The inspection should include the following points: the light reflex of the pupils and the pupil status, any grimacing of the patient, the location and function of all venous and arterial lines and drains, the position of the endotracheal tube, the position of the extremities, dermal color (normal, cyanotic, or pale), evidence of increased jugular vein pressure, and the adequate perfusion of the visible mucous and extremities.

The palpation should include checking the skin turgor and edema, bilateral thorax excursion, the friction of the abdominal wall, and the pulse status of all extremities. To state the degree of relative hypovolemia, hypothermia, or endogenous and exogenous catecholamine-mediated centralization, the extremities have to be examined equally from both sides from central to periphery, and the results have to be documented. Furthermore the patency of the chest tubes has to be ensured and any intraluminal clots removed.

Finally, the quality and quantity of chest tube drainages have to be analyzed, and the filling state of the pleura vacs on admission has to be documented.

9.3.2.2 Assessment of Anesthesia and Sedation

To appraise the depth of sedation, the intensivist should address the patient first. In case of no response, the patient may be touched on the forehead, for example, or an adequate pain stimulus can be given, as needed. During transportation to CSICU, the patient should preferably have a Ramsay score of 4–5 (◘ Table 9.1). The neurological findings are always correlated with the administered drug levels (Ramsay et al. 1974).

◘ **Table 9.1** The Ramsay Sedation Scale defines three awake levels 1 - 3, and three asleep levels 4 - 6, the latter depending on the patient's response to a light glabellar tap or loud auditory stimulus

Score value	Grade of sedation	Assessment
1	Patient anxious and agitated or restless or both	Mild, inadequate sedation
2	Patient co-operative, oriented, and tranquil	Adequate sedation, pt stable at CSICU
3	Patient responds to commands only	Adequate sedation, pt stable at CSICU
4	a brisk response	Desired level at transport to CSICU
5	a sluggish response	Desired level at transport to CSICU
6	no response	Sedation probably too deep

9.3.2.3 Neurological Status

Glasgow Coma Scale (GCS) is a useful tool in quantifying and monitoring the progress of patient's neurological status (■ Table 9.2). The Glasgow Coma Scale provides a score in the range of 3–15; patients with scores of 3–8 are usually said to be in a coma. The total score is the sum of the scores in three categories. However, the GCS has only a limited use in sedated and intubated patient. Since cerebral ischemia and intracerebral hemorrhage occur in 1–5 % of patients undergoing cardiac surgery (Markewitz and Lante 2006), it is important to monitor the pupillary response, the position of the eyeballs, and the motor response of the extremities on CSICU admission and closely in the postoperative course. Frequent neurological exams are essential in early diagnosis along with prompt diagnostic tools (e.g., cranial computed tomography) to potentially minimize neurological deficit.

■ Table 9.2 Glasgow Coma Scale and Score

Reaction	Action	Score
Eye opening response	Spontaneous—open with blinking at baseline	4
	Opens to verbal command, speech, or shout	3
	Opens to pain, not applied to face	2
	None	1
Verbal response	Oriented	5
	Confused conversation, but able to answer questions	4
	Inappropriate responses, words discernible	3
	Incomprehensible speech	2
	None	1
Best motor response	Obeys commands for movement	6
	Purposeful movement to painful stimulus	5
	Withdraws from pain	4
	Abnormal (spastic) flexion, decorticate posture	3
	Extensor (rigid) response, decerebrate posture	2
	None	1

According to Teasdale and Jennett (1974), Jennett et al. (1977)

9.3.2.4 Volume Status, Centralization, and Body Temperature

The basic principles of postoperative cardiac surgical intensive care are hemodynamic monitoring and the differentiated management of intravascular volume and vasoactive substances, inotropic support, heart rate, and rhythm management. Therefore early on CSICU admission, the intensivist has to estimate patient's intravascular volume status and the degree of centralization or peripheral vasoconstriction. The volume status can be estimated by evaluating core body temperature, central venous pressure (correlated to «positive end-expiratory pressure» on the ventilator), and the undulation of the arterial pressure waveform, the heart rate, and blood pressure.

If there are no signs of volume overload (hypervolemia) at the time of CSICU admission (pulmonary edema, central congestion on the thoracic X-ray, right heart failure, insufficiency of the tricuspid valve), volume substitution should be considered to reduce hypotension due to rewarming and the concomitant loss of peripheral resistance.

9.3.2.5 Heart Rhythm and Heart Rate

To achieve an adequate cardiac output, a heart rate of 60–120 bpm with a cardiac output of >2.0 l/min/m^2 body surface has to be established. Regular atrial and ventricular function in sinus rhythm or under pacemaker sensing/pacing is helpful in maintaining adequate cardiac output. The frequent episodes of bradycardia or tachycardia after cardiac surgery require the continuous heart rate monitoring. Patient's abnormal rhythm should be correlated to invasive arterial blood pressure monitoring and the pulse oximetric curve. In case of discrepancies, and when the electronic measurement is not reliable, the pulse may be palpated.

9.3.2.6 Secure the Pacemaker Function

Depending on the operation and the occurrence of preoperative or intraoperative arrhythmias, the temporary pacemaker wires are attached to the right atrium and the right ventricle and, in special cases, also to the left ventricle. If heart rhythm

disturbances occur in the postoperative course, the cardiac output will be reduced, and this may require external electrical stimulation. Therefore the function of the pacemaker wires has to be tested and documented at the time of CSICU admission: proper capture and sensing is critical!

9.3.3 Basic Monitoring and Diagnostics

To assess all relevant parameters in cardiac surgery patients, various monitoring systems have to be intertwined.

Basic monitoring of CSICU patients includes (Carl et al. 2010):

- Continuous ECG and 12-lead ECG
- Invasive and noninvasive blood pressure measurement
- Measurement of central venous pressure
- Pulse oximetry
- Input and outputs (chest tube drainage, volume input, and urine output)
- Body core temperature measurement

If the postoperative course will be complicated, additional monitoring will be required. After CSICU admission and review of the basic monitoring tools, the diagnostic tools may be supplemented by:

- Mixed venous and arterial blood gas analysis
- Chest X-ray, a - p (lying and in inspiration)
- Routine laboratory diagnostics

9.3.3.1 ECG and 12-Lead ECG

To get a basic monitoring for arrhythmia and ischemia, continuous ECG monitoring is required on CSICU. In lead II the electrical axis is parallel to the sinus node and the AV node. Usually the P wave is clearly detectable in lead II, so supraventricular and ventricular rhythm disturbances can be differentiated. Lead V5 should be added on the monitor in patients with coronary artery disease to recognize any ischemia at the anterior and lateral wall. An ST-segment analysis is recommended for each ECG monitoring. In cardiac surgical patients, a 12-lead ECG should be performed routinely for any patient at CSICU admission and daily on the first three postoperative days. If patients stay longer on CSICU, indication for additional ECG depends on clinical status, especially if any ST-changes occur as seen on the monitor.

9.3.3.2 Invasive Blood Pressure Monitoring

The postoperative measurement of the arterial blood pressure is an essential part of CSICU monitoring. The acquisition of the arterial pressure can be carried out in two ways: a noninvasive method using a blood pressure cuff or an invasive method requiring cannulation of the radial or femoral artery. The noninvasive measurement is error prone and inadequate for a safe blood pressure monitoring. The invasive blood pressure measurement allows close pressure monitoring as well as assessment of the volume status of the patient due to respiratory undulation of the pressure curve. Furthermore during mechanical ventilation and under inotropic and vasopressor administration, arterial blood gas may be drawn from the arterial line for easy blood sample and laboratory analysis.

Possible errors occur if the pressure transducer is not correctly zeroed or if any air bubbles remain in the pressure lines. The air leads to a distortion of the pressure curve due to damping. The mean arterial pressure (MAP) is utilized routinely in CSICU. It can be calculated after measuring the systolic (APsyst) and diastolic (APdiast) blood pressure using the following formula:

$$\mathrm{MAP}\ [\mathrm{mm\,Hg}] = \mathrm{AP\ diast} + 1/3(\mathrm{AP\ syst} - \mathrm{AP\ diast})$$

9.3.3.3 Measurement of Central Venous Pressure

The central venous pressure (CVP) is invasively measured in the upper caval vein in about 1–2 cm distance the right atrium. CVP correlates—in absence of an insufficiency of the tricuspid valve—with the end-diastolic pressure in the right ventricle. The CVP value depends on the intravascular volume, the peripheral vascular resistance, the right ventricular ejection fraction/compliance, the pulmonary artery resistance, and the intrathoracic pressure (PEEP ventilation/intrinsic PEEP).

The CVP is low in relative volume-deficient patients and elevated due to volume overload, right heart failure, pulmonary embolism, pericardial tamponade, tension pneumothorax, and in ventilated patient with high PEEP (CVP real = CVP–PEEP).

The diagnostic value of the CVP is limited due to the high volume compliance of the venous system. However, if followed as a trend, the CVP provides useful information about the volume

status as well as the right ventricular preload and compliance. It is helpful to know the CVP value at the time of weaning from extracorporeal bypass and correlating cardiac function with corresponding right ventricular preload help assist as guideline for postoperative volume management.

9.3.3.4 Pulse Oximetry

The percutaneous spectrophotometric determination of oxygen saturation is a very useful and noninvasive and continuous approach to measure the peripheral arterial oxygen saturation (SaO_2). It is displayed as a pulse synchronous undulating curve. The SaO_2 is defined as percent of oxygenated hemoglobin denominated by the sum of oxygenated and deoxygenated hemoglobin. It can be compared in its diagnostic relevance to the partial pressure of oxygen (paO_2).

The respiratory monitoring allows the assessment of pulmonary oxygen uptake and allows, by knowing the actual hemoglobin concentration, the assessment of the arterial oxygen supply of the tissue. Additionally, the acquisition of the pulse curve shows the mechanical heart function.

Pulse oximetry cannot discriminate among oxygenated hemoglobin, carboxyhemoglobin, and methemoglobin. In these cases, the measured SaO_2 concentration is actually lower than displayed SaO_2 given concomitant dyshemoglobinemia in the bloodstream.

Estimated oxygen saturations (SaO_2) at 40 mmHg carbon dioxide, at a pH 7.4, and physiological body temperature are depicted in ◘ Table 9.3.

9.3.3.5 Fluid Balance

The postoperative fluid balance is close monitoring of fluid input and output (I&O) including urine output and chest tube and nasogastric drainage. In the first 24 h after operation, the hourly documentation of I&O may be useful. However, after the first postoperative day, the interval can be extended to every 4 h. Separate documentation of crystalloid versus colloid volume resuscitation in the postoperative phase has not been clinically useful and has fallen out of favor.

◘ **Table 9.3** Reference values for estimated oxygen partial pressure (paO_2) based on measured oxygen saturation (SaO_2) by pulse oximetry (see text)

Parameter	Values					
PaO_2[mmHg]	26	35	40	60	90	150
SaO_2[%]	50	66	75	90	95	100

9.3.3.6 Chest Tube Output

On admission, the filling state of the pleura vacs has to be documented. The chest tube output is an important indicator for postoperative bleeding in cardiac surgery. However, the chest tubes have to be frequently checked by active manipulation (gentle «milking» motion) for proper drainage. Breathing and pulse synchronous movements of the liquid level are also an indication for patency. In case of a pulmonary parenchymal injury, there will be breath-dependent air bubble leaking from the first fluid chamber.

The total chest tube output should ideally be less than 100 ml per hour. If the drainage is more than 100 ml per hour, it may be necessary to check one or more of these blood coagulation parameters:

- Activated clotting time
- Prothrombin time (PT)
- Partial thromboplastin time (PTT)
- Level of fibrinogen
- Platelet count
- Functional coagulation tests such as thromboelastogram

If the patient is stable without evidence of pericardial tamponade, the abnormal coagulation values should be normalized first. If a high chest tube is combined with a significant hemoglobin drop or a new pericardial effusion leading to atrial or ventricular collapse and circulatory instability, the patient should be taken back for mediastinal exploration and control of hemorrhage (see also ► Section 9.5.3 «Bleeding Complications and Pericardial Tamponade»).

9.3.3.7 Measurement of the Body Temperature

The body temperature should be measured continuously. Among others, it affects the values of the blood gas analysis. Furthermore, hypothermia can exacerbate bleeding tendencies in the early postoperative course. For this reason, the temperature should be collected at least to every arterial blood gas sample taking. Typically, a four-hourly interval is recommended. The temperature can be measured by using a Foley catheter with integrated temperature sensor or transesophageal or intravascular by the Swan-Ganz catheter or by infrared (middle ear temperature) approach.

The interpretation of body temperature is always intertwined with the clinical picture: while in the postoperative rewarming phase and peripheral vasoconstriction, the body temperature

can arise to 102 °F (39 °C) in 2–3 h and decreases to normal values after vasodilatation; a temperature rise combined with peripheral vasodilatation is rather a sign of prolonged systemic inflammatory response (SIRS) or septic event.

9.3.3.8 Blood Sample Analysis

A blood gas analysis should be performed immediately after CSICU admission. Furthermore, blood gas sampling should be repeated if cardiopulmonary instability occurs or if the ventilation settings have been changed (here with a time interval of 30 min after change). At an inspiratory oxygen fraction (FiO_2) of >0.6, a blood gas analysis is recommended every 4–8 h intervals.

To maintain an «oxygen reserve» during the transport from the OR to the CSICU, the patient is generally transferred with a FiO_2 of 1.0. Furthermore, manipulations like disconnection from the ventilator or other problems can be better managed, and the lung function can be estimated on CSICU admission. If a patient under controlled ventilation with an FiO_2 of 1.0 for transport have a body temperature-corrected PaO_2 of <200 mmHg (Horovitz quotient of <200, normal value in pulmonary healthy patients 350–450), a pulmonary problem can be expected (For Horovitz quotient see also ► paragraph Respiratory Failure in Sect. 9.5.4 Lung and Mechanical ventilation, p. 226). Then the need of special attention and close controls is necessary. In patients with opened pleural cavities, an initial PEEP of 8 cm H_2O is recommended to reduce atelectasis and may be weaned down prior to extubation.

Point-of-care laboratory tests using specialized blood gas-analyzing machines, prompt results can be obtained in the CSICU including determination of the acid-base balance, concentrations of electrolytes (especially potassium), and the hemoglobin and blood glucose values.

9.3.3.9 Chest X-Ray

Soon after the patient arrives and is situated in his bed, a chest X-ray is mandatory in order to judge the correct position of any indwelling catheters, endotracheal tube, and chest drains as well as proper lung expansion. The scan should be done in ventilatory inspiration. While acutely the chest X-ray checks for intrathoracic fluid accumulation (e.g., hematothorax) or pneumothorax as well as lung congestion in left heart failure, repeated daily chest X-rays for the first three postoperative days are an ideal follow-up tool to screen cardiopulmonary status. Additional chest X-rays are only ordered for specific questions arising from changes in the clinical status of the patient.

9.3.3.10 Routine Laboratory Investigations

Cardiac surgical operations change important physiological processes: changes to the body temperature, full heparinization and reversal along with heparin rebound, intravascular fluid changes, electrolyte shifts, activation of inflammatory cascades, etc. During surgery only a few laboratory values are tested. Therefore, upon CSICU admission, checking the following parameters may be necessary:

- Blood count
- Activity of lactate dehydrogenase (LDH)
- Urea and creatinine
- PT value
- PTT
- Antithrombin III levels
- Activity of creatine kinase (CK) and CK-MB
- Troponin I levels
- Activity of aspartate transaminase (AST), also known as serum glutamic oxaloacetic transaminase (SGOT)
- Activity of alanine transaminase (ALT), also known as serum glutamic-pyruvic transaminase (SGPT)
- Concentration of C-reactive protein (CRP)
- Lactate
- Magnesium

In an uneventful course, a 12-h lab check interval should be adequate. Additionally, daily laboratory test may include the function of the liver, the lactate, and the total protein and albumin levels. The monitoring of blood glucose levels and electrolyte values is additionally measured with the periodic blood gas analysis. Other laboratory parameters are checked as indicated.

9.4 Objectives and Standard Procedures in CSICU

9.4.1 Objectives

Well-run CSICU should have a clear definition of primary end points of therapeutic regimen. This helps to identify patients with prolonged and complicated course that require an escalation of monitoring and/or an adjustment of the current therapy.

To recognize the optimal time point for adjusting the therapy, the objectives and therapeutic goals and their progress require a close monitoring and documentation (usually once per shift). The following criteria have to be achieved during the first 24 h of an uncomplicated postoperative course:

- Awake patient (Ramsay score of 2) with no evidence of neurological deficit
- Warm extremities without edema with even balance
- No abnormalities in the blood gas analysis compared to patient's baseline
- No evidence of bleeding or clotting disorder
- Hemoglobin concentration of >8 mg/dl
- SaO_2 of >92 % (peripheral)
- MAP of >65 mmHg
- Sinus rhythm with a heart rate of 60–90/min
- Adequate left and right ventricular function on echocardiogram
- CVP of 8–12 mmHg
- Stable urine output >0.5 ml/kg/h
- Normal lactate levels

With increased age and patient's comorbidities, there is increased risk for adverse events associated with complicated operative and postoperative course. The clinical experience shows that errors in the early postoperative period may lead to life-threatening complications, or at least a complicated course, requiring more costly expansion of monitoring, therapeutic arrangements, and longer ICU stay. In the following, we will discuss various therapeutic goals and regimens, some supported by published guidelines.

9.4.2 Sedation and Analgesia

For the patient with an uncomplicated course, a differentiated analgesia and sedation is suggested, aiming for antianxiety, analgesia, and vegetative protection. According to the current guidelines, various combinations of drugs are available for adequate analgesia and sedation. Preferably, the concept has to be adapted to the intraoperative anesthetic management to avoid unnecessary change of medication, possibly causing adverse interactions of various analgesics and sedatives due to different pharmacokinetics. ◘ Table 9.4 provides a list of these medications.

To attenuate autonomic responses during weaning from mechanical ventilation and in association with the extubation, sedation can be complemented with clonidine (IV clonidine is not available in the USA):

- Objective of clonidine use: weaning
- Drug (active ingredient): clonidine
- Drip dosage: 1.5 mg/50 ml
- Concentration: 0.03 mg/ml
- Infusion rate: 1–4 ml/h/70 kg body weight

A deep sedation in cardiac surgery should only be used in special cases:

- Complicated long-term mechanical ventilation
- Abdominal positioning (usually for severe hypoxemia)
- Inadequate oxygen delivery to peripheral organs caused by sepsis or multi-organ dysfunction syndrome

◘ **Table 9.4** Overview of routinely used sedatives and their dosages

Duration of sedation	Drug	Drip dosage [mg/50 ml]	Concentration [mg/ml]	Infusion rate [ml/h/70 kg]
Up to 24 h	Propofol 1 %	500	10	3–10
	Remifentanil	5	0.1	1–12
24–72 h	Midazolam	90	1.8	1–7
	Sufentanil	0.5	0.01	1–10
More than 72 h or for unstable patients	Midazolam	90	1.8	1–7
	Ketamine	1250	25	2–10

Besides sedation and analgesia, peripheral analgesics are administered. For slight or moderate pain, the following nonopioid analgesics can be used:

- Acetaminophen: 1 g q6h per mouth or nasogastric tube (maximum dosage, 4 g/day)
- Ibuprofen: 600 mg q8h per mouth or nasogastric tube (maximum dosage, 2.4 g/day)
- Ketorolac: 30 mg q6h intravenous (maximum dosage, 120 mg/day)
- Tramadol: 50–100 mg q4–6h per mouth or nasogastric tube (maximum dosage, 300 mg/day)

These medications have to be adapted to the patient (allergies, age, renal function) and to the operation (ketorolac may cause increasing bleeding tendency in postoperative course) and should be given orally as soon as possible. Oral or parenteral opioids should be used complementary for severe pain. The goal is to maintain a pain score below 2–3 (0 meaning no pain, 10 being the worst pain) to allow adequate breathing and early ambulation.

9.4.3 Gastrointestinal Ulcer Prophylaxis

Physiologic stress associated with illness and hospitalization is known to result in gastrointestinal ulceration, especially among the critically ill. The complication of this stress-related mucosal disease could be prevented with appropriate application of pharmacologic prophylaxis. Stress-induced ulcer (SU) has decreased significantly in the last 25 years (less than 0.5 %), but remains associated with significant morbidity and mortality. This can be reduced by early enteral nutrition. An ulcer prophylactic medication is generally recommended in CSICU, because cardiac surgical patients often presented with at least one risk factor for stress-induced ulcer. This is generally achieved with proton pump inhibitor (PPI) or H_2-receptor antagonist (H_2RA). PPI medications are preferred by some due to less interaction with other medications and better efficacy. Common drug regimen is esomeprazole 20–40 mg enteral or parenteral every day.

There are, however, no studies available, which have demonstrated a superiority of PPI over H_2RA for SU prophylaxis. Additionally, PPIs have been associated with a risk of community-acquired *C. diff.* (Aseeri et al. 2008; Buendgens et al. 2014), osteoporosis (Yang et al. 2006), pneumonia (Laheij et al. 2004; Sarkar et al. 2008), and interstitial nephritis (Leonard et al. 2012). In centers preferring H_2RA as first-line medication for SU prophylaxis, PPI may still be used in those patients who had suffered from a more recent GI hemorrhage (Alhazzani et al. 2013; MacLaren and Campbell 2014).

9.4.4 Antibiotic Therapy

The importance of prophylactic antibiotics to reduce surgical site infections (SSI) has been clearly demonstrated in a number of placebo-controlled studies. SSIs and particularly sternal and mediastinal infections have implications for significantly increasing both morbidity and mortality. Based on availability and cost, it is reasonable to use cefazolin (a first-generation cephalosporin) for standard cardiac surgical prophylaxis, given the fact that most randomized trials could not discriminate between various cephalosporins.

Based on The Society of Thoracic Surgeons' guidelines, the duration of antibiotic prophylaxis should not be dependent on indwelling catheters such as chest tubes, as practiced in some centers. There is evidence indicating that antibiotic prophylaxis of 48-h duration is effective.

Edwards et al. recommend in a 2006 published guideline to continue the perioperative antibiotic therapy for a maximum of 48 h to avoid the development of antibiotic resistance (Edwards et al. 2006). In order to reduce multiresistance in the CSICU, the duration of antibiotics should be only prolonged for plausible and documented reasons.

In case of the occurrence of fever in combination with septic clinical picture within 48 h postoperatively, the perioperative antibiotic regime should not be continued, rather, changed and adapted.

9.4.5 Administration of Blood and Blood Products

The use of the extracorporeal circulation does significantly alter the primary and secondary

hemostasis and the coagulation system. Additionally, a high percentage of cardiac surgical patients are on dual antiplatelet medications, altering the coagulation profile as well. Therefore, many reference values for the coagulation profile are approximations. ◘ Table 9.5 contains expected values and their development.

Even a significant abnormality in the coagulation profile may not require immediate therapeutic intervention. Before any values are treated, the clinical picture has to be taken into account for decision-making. Only the context of clinical presentation and laboratory extents should lead to a therapy—for example, an elevated blood loss from the chest tubes has to be treated—rather a deranged coagulation profile without any clinical evidence of bleeding. The necessity of blood products has to be checked in any case.

9.4.5.1 Packed Red Blood Cells (PRBC)

The cutoff for transfusion of PRBC has been controversial. There is more evidence supporting the fact that PRBC transfusion increases morbidity and mortality, along with increased local and systemic infectious complications (Engoren et al. 2002; Whitson et al. 2007). However, it is not clear whether the transfusion itself or the comorbidities of the patients requiring transfusions are the ultimate culprit.

STS guidelines from 2011 suggests three important preoperative risk factors are linked to bleeding and blood transfusion: (1) advanced age (age >70 years), (2) low RBC volume either from preoperative anemia or from small body size or from both, and (3) urgent or complex operations usually associated with prolonged CPB time and non-CABG procedures.

The transfusion of PRBC in patients with a hemoglobin level less than 7 g/dL is reasonable, but it is based on good evidence (class IIb, level of evidence C), and the transfusion in patients with a hemoglobin more than 10 g/dL, trying to improve the oxygen transport, is not recommended (class III, level of evidence C) (Ferraris et al. 2011).

In clinical practice the transfusion of PRBC correlates to the patient's condition. Patients in life-threatening situation should receive blood even if the hemoglobin level is not under 7 g/dL. If a surgical problem (e.g., surgical site suture bleeding) is suspected, a resternotomy has to be performed promptly.

Of particular concern are the side effects of blood and blood products:

- Hemolytic transfusion reaction of immediate type
- Hemolytic transfusion reaction of the delayed type
- Febrile, nonhemolytic transfusion reaction
- Allergic transfusion reaction
- Transfusion-related acute lung injury (TRALI)
- Transfusion reactions due to bacterial contamination
- Transfusion-associated infections

Transfusion-associated respiratory insufficiency may exacerbate the clinical picture in a multi-morbid patient, complicating the postoperative differential diagnosis and therapeutic approach.

◘ **Table 9.5** Reference values for the coagulation profile on admission on the CSICU and their postoperative changes

Extent	On admission	Postoperative course
Activated clotting time (ACT)	Basic value	No change
Thrombocyte count	>50.000/µl	Increase
Prothrombin (PT) value	>50 %	Increase
Activated partial thromboplastin time (aPTT)	<40 s	Decrease
Antithrombin III level	>60 %	Increase

9.4.5.2 Platelet Concentrates

The use of platelet concentrates in cardiac surgery is necessary due to platelet dysfunction after CPB. A platelet transfusion is necessary for unstable patients with active bleeding if the platelet count is <50/nl (<50.000/µ1). In hemodynamically stable patients, even without bleeding, the threshold for transfusion is a platelet count of <10/nl (<10.000/µ1). After transfusion of one unit of platelet concentrate, the expected raise of the platelet count will be approximately 30/nl (30.000/µ1).

Furthermore, according to STS guidelines, the use of intraoperative platelet plasmapheresis is reasonable to assist with blood conservation

strategies as part of a multimodality program in high-risk patients if adequate platelets yield can be reliably obtained (Ferraris et al. 2011).

9.4.5.3 Fresh Frozen Plasma (FFP)

FFP contains all coagulation factors and their inactivators. Considered indications for transfusion of FFPs are the emergency treatment of a clinically relevant bleeding and an acute bleeding due to a complex coagulation disorder, expected after prolonged CPB, and the loss and/or a dilutional coagulopathy in patients with severe blood loss and extensive transfusions. In these cases and as rule of thumb, a unit of FFP is transfused for each four PRBC transfusions.

FFPs should not be given to expand volume. Here are further recommendations from STS guidelines for blood conservation (Ferraris et al. 2011):

1. Plasma transfusion is reasonable in patients with serious bleeding in context of multiple or single coagulation factor deficiencies when safer fractionated products are not available (class IIa, level of evidence B).
2. For urgent warfarin reversal, administration of prothrombin complex concentrate is preferred, but plasma transfusion is reasonable when adequate levels of factor VII are not present in prothrombin complex concentrate (class IIa, level of evidence B).
3. Transfusion of plasma may be considered as part of a massive transfusion algorithm in bleeding patients requiring substantial amounts of red blood cells (class IIb, level of evidence B).

9.4.5.4 Prothrombin Complex Concentrate

Prothrombin complex concentrates (PCCs) are hemostatic blood products containing four vitamin K-dependent clotting factors (II, VII, IX, and X), as well as the anticoagulant inhibitor proteins C, S, and Z (Ansell et al. 2004). They are a useful, reliable, and fast alternative to fresh frozen plasma for the reversal of the effects of oral anticoagulant treatments (vitamin K antagonists). The use of PCC rather than FFP is recommended for Coumadin reversal in patients with major bleeding (Baglin et al. 2006; Baglin et al. 2007). The use of PCC is not the first choice in bleeding complications, because PCC stems from a large pool of donors and puts the recipients at increased risk of anaphylactic shock, viral transmission, etc.

Recommended PCC dose is 25–30 IU/kg as initial bolus.

9.4.5.5 Recombinant Factor VIIa (rFVIIa)

The use of rFVIIa has a limited indication in high-risk patients as last resort. Recommended dose ranges vary from 11 to 200 μg/kg body weight. We use 90 μg/kg body weight to be administered within 2–5 min, preferably immediately after FFP transfusion. If the bleeding does not stop, a second infusion may be considered after 20–30 min. There are varying results in literature concerning thromboembolic side effects from 5 % (Warren et al. 2007) to no increased risk (Kluger et al. 2007; Chapman et al. 2011). According to STS guidelines, rFVIIa concentrate may be considered for the management of intractable nonsurgical bleeding that is unresponsive to routine hemostatic therapy after cardiac procedures using CPB (class IIb, level of evidence B).

9.4.5.6 Antithrombin III (AT III)

Antithrombin III concentrates has a class I recommendation according to STS guidelines for blood conservation: AT III is indicated to reduce plasma transfusion in patients with antithrombin-mediated heparin resistance immediately before cardiopulmonary bypass (level of evidence A). In case of heparin resistance, and instead of FFP transfusion, it is strongly suggested to administer AT III to improve activated clotting time for the CPB.

Various dosing algorithm exists based on baseline antithrombin activity levels. If such levels are not available in the operating room, 1,000 IU is given parenterally, and the activated clotting time is remeasured.

9.4.6 Nutrition

Poor nutritional status can adversely affect thoracopulmonary function in spontaneously breathing and mechanically ventilated patients with respiratory disease by impairment of respiratory muscle function, ventilatory drive, and pulmonary defense mechanisms. The questions of whether, when, and how intensive care patients must be fed are essential in planning the nutrition plan at the CSICU. In critically ill patients, including cardiac surgical patients, an underfeeding is related with a poor outcome (Preiser et al. 2015). It is recommended to consider enteral feeding in patients who will not

have oral intake within the next 5 days. An enteral nutrition is preferable, unless the patient is on high-dose inotropes, making adequate gut perfusion unlikely, or there is an enteral passage malfunction due to injury or surgical operations. Enteral feeding is started after the position of jejunal nutrition tube has been confirmed on X-ray and with elevation of the upper part of the body. For patients with ventilator failure, it is recommended to avoid overfeeding, causing nutritionally associated hypercapnia. This may delay timely extubation.

It is important to provide patients with an estimated prolonged intubation time with additional calories, to be started within the first 24 h. The required amount of calories is 20–25 kcal/kg body weight/day (Kreymann et al. 2006). Controlling subsequent hyperglycemia, therefore reducing the risk of infection using insulin drip protocols, remains critical aspect in ICU nutrition (van den Berghe et al. 2001; Bhamidipati et al. 2011; Haga et al. 2011).

Optimal support would establish neutral or positive nitrogen balance, depending on the need for protein repletion. In the critically ill patient with renal function impairment, this can be accomplished by giving 1–3 g of protein/kg daily. Generally, this amounts to approximately 20 % of total calories being administered as protein. While recommendations for an appropriate substrate mix of carbohydrates and fats vary, generally 60–70 % carbohydrates are given with 20–30 % fats.

9.4.7 Discharge from ICU

The decision whether the patient is ready to be transferred from CSICU should only focus on medical criteria. Most cardiovascular patients will be transferred to an EKG-monitored step-down unit for further recovery. Patients with ongoing organ support, hemodynamically unstable patients, patients with complex heart rhythm disturbances, or patients with a Ramsay score >3 have to stay in the CSICU.

It is common practice that the daily need for ICU capacities deforms the clinical picture of the patient to a better one. Unfortunately this may lead to higher readmission rate to the CSICU. If the readmission rate exceeds 5 %, discharge criteria should be revisited.

9.5 Special CSICU Arrangements

9.5.1 Introduction

9.5.1.1 The Importance of Communication

Especially in critically ill patients, the importance of communication with the patient himself and his family, but also with the medical and nonmedical staff on the CSICU, is of great importance. The clinical situation of the patient as well as the medical course for the next 24 h has to be evaluated and discussed in this «family-and-CSICU-staff team.» It is important to create a mutual trust, especially if serious and final decisions have to be made. Additionally clerical or psychological support can be an important part of the family-and-staff team and should be consulted early, especially in a complicated course. The bulk of problems can be avoided using open communication and care, whereas poor communication skills and a lack of understanding may lead to complaints from patient, family members, or health-care providers.

9.5.1.2 Incidence of Organ Malfunction and Failure

To predict the incidence of organ malfunction and failure of CSICU patients, different scores can be used which take preoperative data into account. ◘ Table 9.6 is a general overview of prevalence and mortality rates with multiple organ failure according to our own experience (Markewitz and Lante 2006).

One-fourth of patients will have a CSICU stay of more than 48 h. This is closely related to the

◘ **Table 9.6** Prevalence and mortality of multi-organ failure after cardiac surgery

Parameter	Organ complications/failure				
	Circulatory system	Lungs	Kidney	Intestine	Central nerve system
Prevalence (%)	4–7	3–9	1–5	1–3	1–5
Mortality (%)	38	20–25	40–80	10–100	20–25

From Markewitz and Lante (2006)

preoperative risk and an increased use of resources (Giakoumidakis et al. 2011). It is obvious that failure of one or more organ system will influence the physiological function of the other organ systems (Sealy and Christou 2000). Clinical practice shows that the right therapeutic strategy leads to a recovery of the malfunction organ, but the interactions between the different systems are still undiscovered. This lack of knowledge is one reason for the high mortality in organ failure. As the knowledge of these interactions grows, it might be possible to influence them, to decrease mortality. The mission of the intensivist is to define the therapeutic course as clearly as possible and adjust it, as needed.

9.5.2 Circulatory System

9.5.2.1 Postoperative Physiology and the Incidence of Its Failure

The postoperative cardiac physiology is modified depending preoperative cardiac anatomy and physiology and the type of index operation. For the postoperative CSICU therapy, the awareness and knowledge of prevalent postoperative temporarily impaired cardiac function are important. A reduced myocardial compliance leads to a stiffer ventricle with higher intracardiac filling pressures. Preoperative risk factors, such as advanced age, hypertension, decreased left ventricular ejection fraction (<30 %), a left main stem stenosis, diabetes in combination with its vascular transformations, renal insufficiency, or pulmonary disease, as well as intraoperative complications, increase the risk of postoperative heart failure (St. André and DelRossi 2005). The treatment strategy should include preload optimization and administration of intravenous inotropes and vasopressors. High dosages of parenteral drugs are necessary in one-third of the patients (Vargas-Hein et al. 2006) and mechanical circulatory support in 4–5 % (Vargas-Hein et al. 2006).

9.5.2.2 Goals of Postoperative Cardiovascular Therapy

The goal of postoperative cardiovascular therapy is defined to maintain a sufficient tissue perfusion and a normalization of the oxidative metabolism (Carl et al. 2010). The required blood oxygen saturation is directly associated with lung function, cardiac output, and intravascular volume.

Optimal ranges for postoperative cardiovascular parameter are:

- Mean arterial pressure (MAP) of >65 mmHg
- Central venous pressure (CVP) of 8–12 mmHg (in dependence on the ventilation)
- Diuresis of >0.5 ml/kg/h
- Lactate concentration of <3 mmol/L
- Central venous oxygen saturation ($ScvO_2$) of >70 % and mixed venous oxygen saturation (SvO_2) >65 %
- Cardiac index of >2.0 L/min/m^2
- Pulmonary capillary wedge pressure (PCWP) of 12–15 mmHg
- Left ventricular end-diastolic area index (LVEDAI) of 6–9 cm^2/m^2
- Intrathoracic blood volume index (ITBVI) of 850–1,000 ml/m^2
- Global end-diastolic volume index (GEDVI) of 640–800 ml/m^2

The MAP as tissue perfusion pressure and the CVP as index for right ventricular preload are recommended as basic ICU pressure monitoring (Carl et al. 2010). Furthermore the hourly produced urine as a marker for a sufficient renal glomerular perfusion, the serum lactate level as a marker for a sufficient end-organ tissue perfusion and oxygenation, and the central venous oxygen saturation—as approximate mixed venous oxygen saturation—should be also considered as basic postoperative monitoring.

If the cardiac function is impaired, more cardiac parameters are required to refine the therapeutic course (extended monitoring). A Swan-Ganz catheter should be placed to monitor the mixed venous oxygen saturation and the pulmonary capillary wedge pressure. The left ventricular end-diastolic area index (an approximate for left ventricular preload) should be measured using transesophageal echocardiography. The intrathoracic blood volume index and the global end-diastolic volume index are also used to determine the left ventricular preload and are obtained analyzing the pulse curve contour.

9.5.2.3 Diagnostic Approaches in Postoperative Cardiovascular Therapy

The following findings are consistent with low cardiac output syndrome (LCOS), requiring prompt attention and therapeutics:

- MAP of <60 mmHg
- Urine excretion of <0.5 ml/kg/h for more than 1 h
- SvO_2 of <60 % in a patient with SaO_2 of 98 %
- Lactate concentration of >2.0 mmol/L
- Peripheral vasoconstriction with cold extremities as a sign of centralization

The immediate initiation of therapy for low cardiac output syndrome is critical and directly associated with outcome, similar to the septic patients (Polonen et al. 2000; Rivers et al. 2001).

9.5.2.4 Pathophysiology, Clinical Presentations, and Differential Diagnosis of the Postoperative Low Cardiac Output Syndrome (LCOS)

Usually the immediate therapy will solve the problem. Therefore the correct choice of the therapy is primarily dependent on the cause.

The most common causes of postoperative LCOS are:

- Volume deficiency (especially bleeding)
- Rhythm disturbances
- Pericardial tamponade
- Myocardial infarction
- Left heart failure
- Right heart failure
- Vasoplegic syndrome
- Maximum variant: cardiovascular arrest

A tension pneumothorax and a circulatory compromise due to unfavorable mechanical ventilation should be excluded as part of the differential diagnosis. In the first step, easily correctable problems should be eliminated, for example, lack of volume and arrhythmias.

▪▪ Lack of volume

A lack of volume will present as a low CVP and a respiratory undulation of the blood pressure curve. The main cause of a lack of volume is the postoperative blood loss, consistent with increased chest tube output, if the drains are not clogged.

The causal therapy is to optimize the preload by fluid administration. There are two different fluid solutions available: crystalloid solutions that serve as full electrolyte solutions and colloids, such as hydroxyethyl starch (HES, nonionic volume expander), succinylated gelatin solution, or human albumin. There is no reason not to use HES, and there is no new scientific literature on this issue.

From total crystalloid volume infusion, at least 25 % stay in the intravascular space. In comparison to colloids, more crystalloids have to be administered to have an equivalent preload-increasing effect. Therefore colloid transfusion especially human albumin in 5 or 25 % concentration is preferred in the CSICU. However, it is not clear whether any choice of fluid solutions will improve patient's survival.

The side effect of synthetic colloids is anaphylactic reaction especially after administration of gelatin products, whereas the succinylated forms are less reactive. Increased bleeding complications and renal function impairment after HES infusion have been documented, in particular in the high-molecular, high-substituted solutions. The complication rate of currently used low-molecular, low-substituted solutions is very low, keeping the daily dose limit of 50 ml/kg in mind. Albumin has anti-inflammatory effects, very helpful in patients with poor nutritional status or liver disease, and—in concentrated form and with increased osmolarity—may help reduce peripheral edema. However, there is a very small risk of infection, and therefore the use of albumin should be wisely considered. Last but not least, it is important to stress that blood and blood products are not appropriate for volume replacement therapy (Ferraris et al. 2011). Unless hemoglobin is low anyways, or blood is the volume missing, like in bleeding states.

▪▪ Rhythm disturbances

While evaluating the volume status, the heart rhythm should be analyzed, and obvious hemodynamically compromising rhythm disturbances have to be treated immediately. Details will be given in the following. If there is any evidence of LCOS after correction of preload and dysrhythmias, the extended monitoring has to be implemented to find the cause of the circulatory impairment.

▪▪ Pericardial tamponade

Pericardial tamponade is associated with elevated CVP, decrease of the urine production, and cold extremities, and it is a clinical diagnosis. The X-ray of the chest may show an enlarged mediastinum with a «tent sign.» A transesophageal echocardiography (TEE) should confirm the clinical diagnosis, with compressed atria or right ventricle as well as respiratory variation of

mitral valve inflow; however, a TEE comes rather late during this often rather rapid process. It is commonly associated with decreasing chest tube output after significant output just before, consistent with clogged drains. A sterile suction of chest tube may be attempted; however, if the drains cannot be reopened and bleeding is thought to be due to «hypoprolenemia» (slang for missing sutures), a rethoracotomy has to be performed promptly. If the patient is in an unstable condition, the tamponade has to be treated immediately at the CSICU. Having sternotomy carts, established protocols, and trained CSICU nurses can make this procedure more efficient and successful.

Myocardial infarction

The occurrence of a perioperative myocardial infarction may be recognized in the ECG with typical ST elevation or echocardiogram findings of regional wall abnormalities. It is accompanied by rhythm disturbances, elevated myocardial markers in the serum such as troponin, CK, and CK-MB (understanding that these markers are frequently elevated without myocardial infarction), up to hemodynamically unstable patients. An upward trend in CK and CK-MB serum levels in sequential analyses is more disturbing than an isolated high value. If the patient is stable enough to be transported to the cath lab, an immediate coronary angiography should be performed. The cath result may mandate an interventional angioplasty or another cardiac operation. If the patient's condition is too critical or if there is no further intervention possible, the acute circulatory failure has to be treated medically using nitroglycerin, aspirin, ß-blockers, an adequate analgesia, and heparin, or more advanced therapies such as balloon pump and ventricular assist and extracorporeal membrane oxygenation (Antman et al. 2004).

If myocardial infarction is excluded, it is important to take the following differential diagnosis into account: left heart failure, right heart failure, biventricular failure, or vasoplegic syndrome. To gain information the extended monitoring will be necessary.

Left heart failure

The failure of the left ventricle is one of the major complications after cardiac surgery. Predisposing factors that cannot be influenced are:

- Advanced age
- The extent and severity of underlying heart disease, especially preexisting reduction of left ventricular ejection fraction
- Previous cardiac surgery
- Presence of peripheral vascular disease
- Urgency of index operation

Factors that can be influenced are the intraoperative factors, such as the cross clamp time, the quality of myocardial protection, and the degree of success in the operative approach. The main cause of the left heart failure is an excessive myocardial pressure and/or volume overload. Special forms are the «myocardial stunning» and the «hibernating myocardium.»

Usually the myocardial stunning is a completely reversible, mostly diastolic, prolonged myocardial dysfunction after a short period of myocardial ischemia. If appropriate and effective revascularization is performed, no permanent cell defects will remain; however, the contractility may be impaired over a prolonged period of time. The basic research is ongoing to explore the pathways of the «stunning.» It can occur after a regional ischemia, such as myocardial infarction, or after a global ischemia, triggered by a cardiac arrest.

In contrast, the «hibernating» myocardium is defined as an adaption of the myocardium to the reduced coronary perfusion. After a proper restoration of the perfusion, the contractility will get back to normal function, although on occasion a «stunned» situation can appear.

Right heart failure

Diagnosis and treatment of right ventricular failure is challenging. Pulmonary hypertension and long-standing valvular disease are predisposing factors. Insufficient cardioplegia or intravascular volume overload may contribute to the disease. Additionally left heart failure, anaphylactic reaction to protamine administration, or an inflammatory response to the operation can acutely increase right ventricular afterload that can lead to myocardial damage. In echocardiography the right ventricle is enlarged and hypo- or akinetic with potentially normal left ventricular function. Often, new onset tricuspid regurgitation is remarkable.

Vasoplegic syndrome

Reduced peripheral vascular resistance occurs in at least 20 % of the patients after cardiac

surgery. The standard therapy is the administration of volume and norepinephrine (Carrel et al. 2000). In some patients there is a norepinephrine-resistant reduced peripheral vessel resistance (Levin et al. 2004). This is called a vasoplegic syndrome. A possible correlation to preoperatively administered ACE inhibitor therapy is discussed. Besides ruling out hypoadrenal syndrome, initiation of intravenous vasopressin therapy or methylene blue is recommended (Egi et al. 2007).

▪▪ Cardiac arrest

The worst case of a LCOS is the cardiac arrest that appears in 1–2 % of cardiac surgery patients (European Resuscitation Council, Nolana et al. 2010). The most common causes of postoperative cardiac arrest are (modified after European Resuscitation Council, Truhlár et al. 2015):

- Myocardial ischemia
- Tension pneumothorax
- Pericardial tamponade
- Massive bleeding with hypovolemic shock
- Pacemaker dysfunction in patients with little or no intrinsic rhythm
- Electrolyte disturbances, especially in low or high levels of potassium

The cardiac arrest can be directly monitored by the minimal or missing peaks in the blood pressure curve. Ongoing ventricular fibrillation or cardiac arrest is displayed by the ECG. Therapy has to be started immediately. Obvious reasons can be treated easily: If a tension pneumothorax is present, the situation can be handled by deployment of a thoracic drain. If the pacemaker is not active or the connection is defective, it must be reconnected or replaced or a transvenous or transcutaneous pacer be placed. If the electrolyte situation is unbalanced, it has to be corrected.

A myocardial ischemia leading to ventricular fibrillation can be treated using a biphasic electric defibrillation shock with maximum energy. Additionally the ischemia has to be treated with a cardiopulmonary resuscitation. The resuscitation has to be performed according to ACLS protocol. If classical ACLS protocol does not resolve the cardiac arrest in CSICU, immediate opening of the chest is recommended, as last resort. This will alleviate tamponade and pneumothorax, will help diagnose significant bleeding, and will allow internal cardiac compression and defibrillation (◘ Fig. 9.1). Differences in Guidelines from the European Resuscitation Council and the American Heart Association are featured in a Critical Care Nurse article more recently (Ley 2015).

The survival rate of these patients is better than expected: overall 33 % of patients in whom a rethoracotomy due to cardiac and circulatory arrest on the CSICU was performed will survive (Mackay et al. 2002), compared to 48 % survival rate if the rethoracotomy was performed within the first 10 min after the arrest (Mackay et al. 2002).

9.5.2.5 Therapy of LCOS

▪▪ Pharmacological circulatory support

The therapy of LCOS is the continuous intravenous application of circulatory supportive medication that will be described in the following.

▪▪ Catecholamines

Dopamine acts directly and indirectly on α-adrenergic receptors as well as on β-adrenergic receptors, in addition to its affinity to dopamine receptors. The affinity to receptors is dose dependent: 0.5–3 μg/kg/min leads to a vasodilatation of renal and abdominal vessels via dopamine receptors, 3–10 μg/kg/min raises the heart rate and the cardiac output with increasing the arterial and pulmonary artery pressure via stimulation of the ß-receptors, and a dosage of more than 10 μg/kg/min increases the peripheral vascular resistance due to stimulation of the α-adrenergic receptors with additional release of norepinephrine. An increase in mean pulmonary artery pressure and the wedge pressure can be corrected using pulmonary vasodilators. Important side effects are suppression of the pituitary gland hormone, ischemia of gastrointestinal mucosa, and, as with all catecholamines, an increase of myocardial oxygen consumption. Important to mention that low-dose dopamine has not been shown to prevent renal failure (Lassnigg et al. 2000).

Epinephrine activates ß1-, ß2-, and α-adrenergic receptors. The receptor response is dose dependent: 0.02–0.05 μg/kg/min increases the inotropic effect predominantly through the ß1-receptors, 0.05–0.2 μg/kg/min increases the inotropic effect and the peripheral vascular resistance by the ß- and the α-receptors, and a dosage >0.2 μg/kg/min will increase the peripheral vascular resistance by increasing effects on the

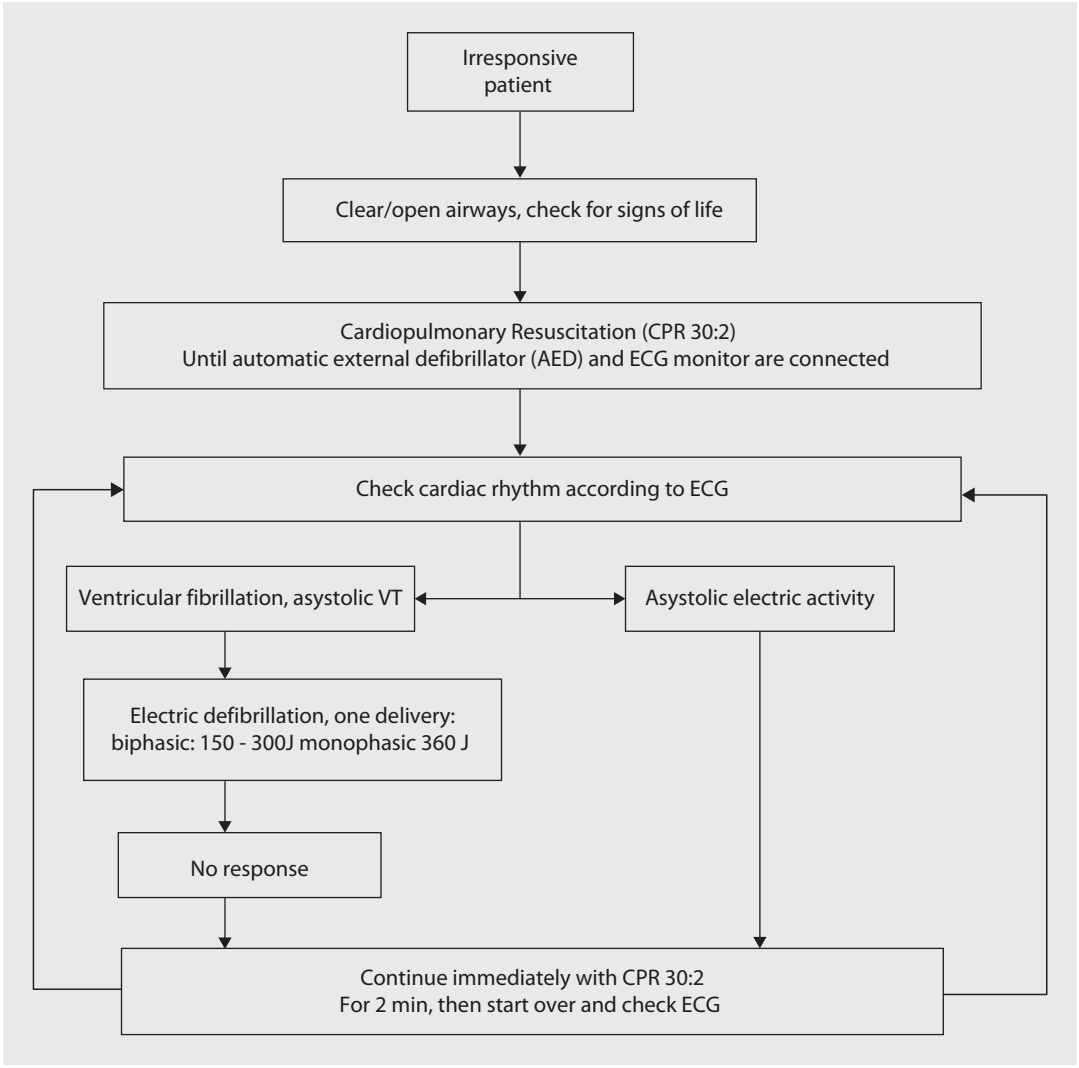


Fig. 9.1 Therapeutic algorithm for cardio-circulatory arrest (Modified after European Resuscitation Council (2010)). *VT* ventricular tachycardia, *ECG* electrocardiogram

α-receptors. Important side effects are tachycardia, increase of the mean pulmonary artery pressure and the wedge pressure, ischemia of gastrointestinal mucosa, and an increase of myocardial oxygen consumption.

Norepinephrine activates α-adrenergic receptors, but also ß1- and ß2-receptors. Although there is increase in contractility, the cardiac output overall is not increased due to higher peripheral vascular resistance. The arterial vasoconstriction improves the perfusion pressure to all organs. Important side effects are tachycardia (less than epinephrine) and increase of mean pulmonary artery and pulmonary artery wedge pressure.

Norepinephrine is the first choice as vasoconstrictor and is recommended in two situations:

- In patients with low blood pressure due to low systemic vascular resistance, if this cannot be effectively treated by administration of volume or positive inotropic agents
- To compensate the initial blood pressure drop after starting a phosphodiesterase-III-inhibitor therapy such as milrinone

Dobutamine activates the ß1-receptors and significantly ß2- and α-adrenergic receptors. Dobutamine is a positive inotropic and lusitropic agent (improves myocardial relaxation in the diastole) and has also vasodilatory effect. This

improves the cardiac output. Most common relevant side effects are tachycardia and increased myocardial oxygen consumption.

Due to lack in increase of the mean pulmonary artery pressure and the wedge pressure and maintenance of perfusion of the visceral arteries, in our practice the use of dobutamine is the first choice in increasing cardiac output.

■■ Vasodilators

Phosphodiesterase-III inhibitors such as milrinone: In contrast to catecholamines, milrinone is an adrenoreceptor-independent inotrope with vasodilatory characteristics. The administration leads to an increase of intracellular cAMP levels due to a blockade of cAMP depletion. This triggers the calcium influx into the cell and an increased release of calcium from the sarcoplasmic reticulum. Smooth muscle cells with an increased level of cAMP dilate. In addition milrinone may increase the heart rate by affecting the sinus node and increase the atrioventricular conduction transmission. In contrast to catecholamines, the myocardial oxygen consumption is not significantly augmented due to a simultaneous reduction of the pre- and afterload. In summary, milrinone has a positive inotropic effect with an increased cardiac output along with a drop of cardiac filling pressures and the pulmonary vascular resistance. Side effects are long half-life time with difficult dose adjustments, increased pulmonary shunting, and hypotension requiring vasoconstrictors. In clinical practice, milrinone is used when inotropic effect of dobutamine is reduced due to ß-adrenergic receptor overregulation.

■■ Levosimendan

Levosimendan is used to prevent and to treat LCOS and acts as a calcium sensitizer. It is increasingly being used in Germany. Levosimendan is currently not available in the USA for clinical use. For further information see ► Chapter «Critical Care in Pediatric Cardiac Surgery», Sect. 10.3.2.4.

■■ Nitroglycerin

The administration of nitroglycerin leads to a dilatation of vessels, especially the venous and the coronary vessels and, in smaller amount, the arterial system. The primary use is the prophylaxis and the therapy of a myocardial ischemia. Furthermore it can be used to reduce an elevated pulmonary artery pressure and in right heart failure. Important side effect is a hemodynamically relevant tachycardia with hypotension. Further, the inhibition of hypoxemia-triggered pulmonary vasoconstriction and an increased intrapulmonary right-left shunt can lead to decreased oxygenation. Finally, a high dosage can result in severe headaches, and prolonged applications will cause tolerance that mitigates the effect. Details of pharmacotherapy are discussed under treatment of arterial hypertension (► Sect. 9.5.2.6).

■■ Nitroprusside

Sodium nitroprusside decreases the afterload and the preload with consequent increase of cardiac output. Therefore, the myocardial oxygen consumption is decreased.

In severe LCOS the use of sodium nitroprusside is recommended to reduce systemic vascular resistance. Similar to nitroglycerine, the side effects are decreased oxygenation, and in our Los Angeles experience, we do not recommend it for aortic dissection, as there is evidence that nitroprusside may increase shear stress on the aortic wall. Some references, however, suggest to employ it combined with beta-blockers (Erbel et al. 2001; Tsai et al. 2005). The most dangerous side effect is cyanide intoxication. In order to avoid this side effect, nitroprusside medication should be accompanied by IV sodium thiosulfate infusion.

Details of pharmacotherapy of sodium nitroprusside are discussed under treatment of arterial hypertension (► Sect. 9.5.2.6).

■■ Inhalative Vasodilators

Inhaled vasodilators, such as nitric oxide (NO) are often used to treat a pulmonary hypertension. The mode of NO action is the activation of guanylate cyclase (cGMP) with a specific dilation of pulmonary vessels. The drop of the pulmonary resistance and the shift of blood flow through ventilated lung areas reduce the pulmonary pressure and improve the arterial oxygenation. In contact with hemoglobin, the NO is been deactivated. Therefore, the NO does not enter the systemic circulation. Important side effects are the methemoglobinemia due to prolonged use and an increased bleeding tendency, but the incidence is low by using the recommended dosage of <20 ppm.

An alternative for pulmonary vasodilatory effect is the phosphodiesterase-V inhibitor sildenafil. Sildenafil is available orally with the similar effects such as NO. There is a potential side effect of systemic hypotension with sildenafil.

Vasopressors

Vasopressors are primarily used to increase the arterial perfusion pressure, in cases where the optimized volume status and positive inotropic response is inadequate for peripheral perfusion. Vasopressin as well as methylene blue belongs to the vasopressors.

Vasopressin

Vasopressin activates the vasopressin-1 receptor with an increase of the intracellular calcium level. Vasopressin is very effective especially in the vasoplegic syndrome, unresponsive to maximal norepinephrine dosage. The side effect of vasopressin even in low dose is significant decrease of microcirculation. Therefore the use of vasopressin should be considered as second or third choice and preferably should be used in low dosage.

Methylene blue

The effects, side effects, and indications for use of methylene blue are similar to vasopressin. Although there are promising reports, overall there is little data supporting routine use of methylene blue. Further details are given in ◘ Table 9.7.

Basic rules in pharmacological circulatory support

A LCOS has to be treated immediately according to the underlying reason. Therefore an algorithmic approach to pharmacological circulatory support is critical. The results of Swan-Ganz catheter and transthoracic echocardiography are frequently not available immediately, allowing for algorithmic approach recommended in ◘ Figs. 9.2 and 9.3.

However, the basic circulatory monitoring will provide important information, allowing for following rules of thumb:

- In a patient with MAP <60 mmHg and a central venous pressure of >12 mmHg, treatment with positive inotropic agents should be considered.
- The worse the left ventricular function is in the patient, the earlier should these drugs be initiated.
- The worse the left ventricular function is in the patient, the more sensitive the heart may respond to volume administration, especially in an already preoperatively increased left and right ventricular preload.
- Initial drug of choice is dobutamine in combination with norepinephrine and/or epinephrine given the aforementioned advantages and side effects. Dobutamine reduces systemic and pulmonary vascular resistance along with inotropic effect. If the vasodilatory effects are too strong, norepinephrine or epinephrine may be used.

◘ **Table 9.7** Dosage of inotropes and vasopressors

Drug	Bolus	Dosage
Dopamine	None	<3 μg/kg/min: renal effect
		3–10 μg/kg/min: β-adrenergic effect
		>10 μg/kg/min: β- and α-adrenergic effect
Dobutamine	None	2–20 μg/kg/min: β-adrenergic effect
Adrenalin	During ACLS protocol	0.05–1.0 μg/kg/min
Noradrenalin	None	0.05–1.0 μg/kg/min
Milrinone	25–75 μg/kg over 20 min	0.25–0.75 μg/kg/min
Enoximone	0.25 bis 0.75 μg /kgKG	1.25 bis 7.5 μg/kg/min
Levosimendan [not available in the USA]	12 bis 24 μg/kgKG[a]	0.1 μg/kgKG/min (0.05 bis 0.2 μg/kgKG/min)

[a]Should not be given as bolus in patients with hypotension

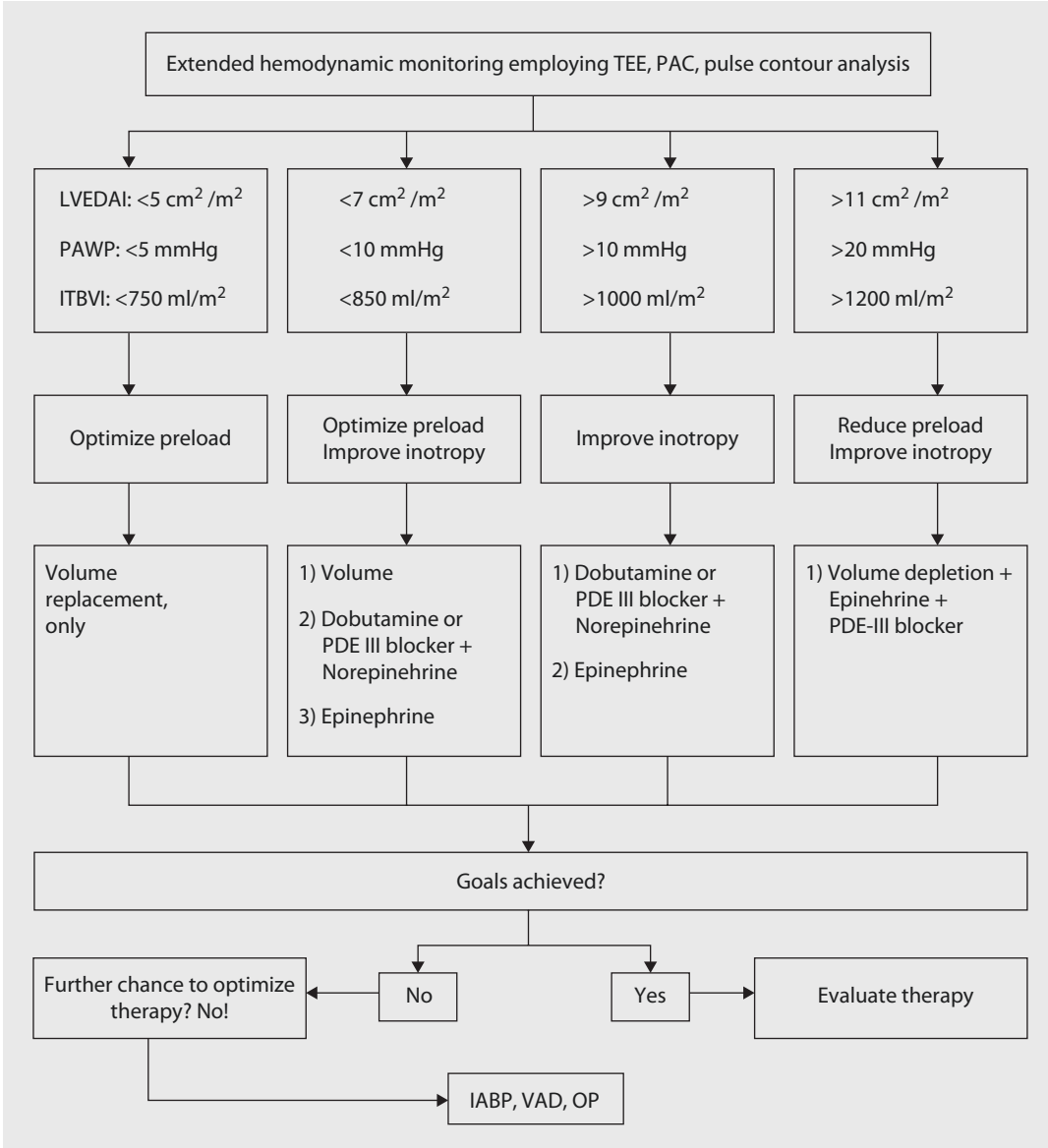


Fig. 9.2 Therapeutic algorithm for left heart failure according to results of extended monitoring. *IABP* intra-aortic balloon pump, *ITBVI* intrathoracic blood volume index, *LVEDAI* left ventricular end-diastolic area index, *OP* redo surgery, *PAC* pulmonary artery catheter, *PAWP* pulmonary artery wedge pressure, *PDE III* phosphodiesterase III, *TEE* transesophageal echocardiography, *VAD* ventricular assist device (Modified after Carl et al. (2010). This is an open-access article distributed under the terms of the Creative Commons Attribution License (►http://creativecommons.org/licenses/by-nc-nd/3.0/deed.en))

Alternatively milrinone/vasopressin combination is used especially in patients with high propensity of going into atrial fibrillation, causing further drop in cardiac output. Both drugs do not possess any adrenergic receptor response.

In case of intravascular volume overload or increase of the CVP without augmentation of MAP, the intravascular volume has to be reduced. This can be assured using diuretics or renal replacement therapy. Furthermore, in the presence of right heart failure, right ventricular afterload reduction using nitroglycerine, milrinone, and inhaled NO may be necessary.

If hemodynamic stabilization cannot be achieved with pharmacotherapy, a prompt decision for mechanical circulatory support is

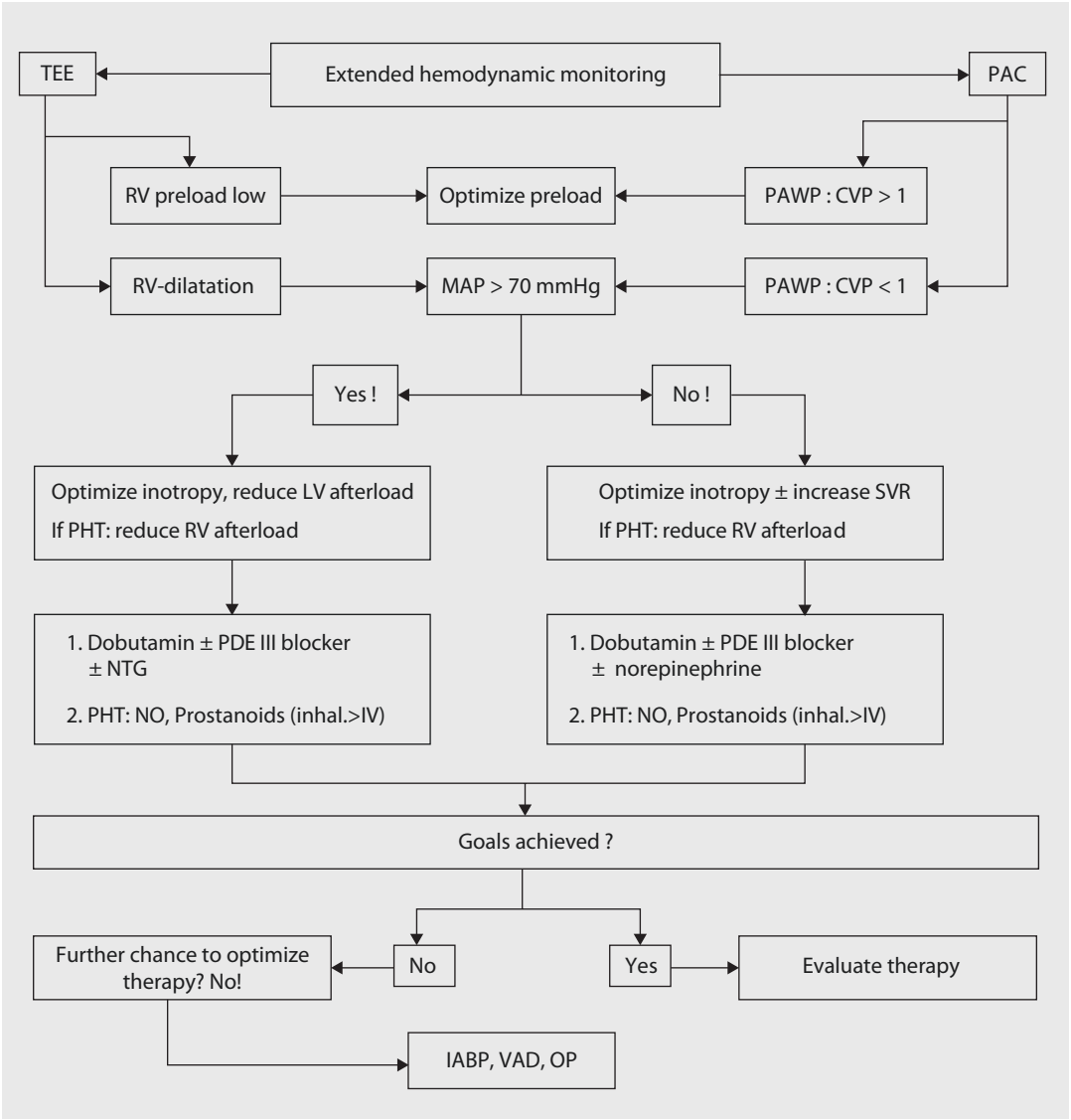


Fig. 9.3 Therapeutic algorithm for right heart failure according to data from extended monitoring. *IABP* intra-aortic balloon pump, *OP* redo surgery, *PAC* pulmonary artery catheter, *PAWP* pulmonary artery wedge pressure, *PDE III* phosphodiesterase III, *TEE* transesophageal echocardiography, *VAD* ventricular assist device. *CVP* central venous pressure, *LV* left ventricular, *MAP* mean arterial pressure, *NO* nitric oxide, *NTG* nitroglycerin, *PHT* pulmonary hypertension, *RV* right ventricular, *SVR* systemic vascular resistance

required. Besides pharmacological or mechanical circulatory support, adequate therapy for the other organ systems must be provided, including:

- Adequate oxygenation
- Balanced acid-base balance, especially due to a poor response of adrenergic receptors to catecholamines in an acidic pH range
- Adequate blood sugar levels with a target of <180 mg/dl

Mechanical Circulatory Support: Intra-aortic Balloon Pump

The intra-aortic balloon pump (IABP) is one of the mostly used mechanical circulatory support devices in cardiac surgery due to ease of transfemoral implantation into the descending aorta with relatively low risk and side effects (Robicsek et al. 2003). The main goal is the augmentation of the diastolic blood pressure by balloon inflation

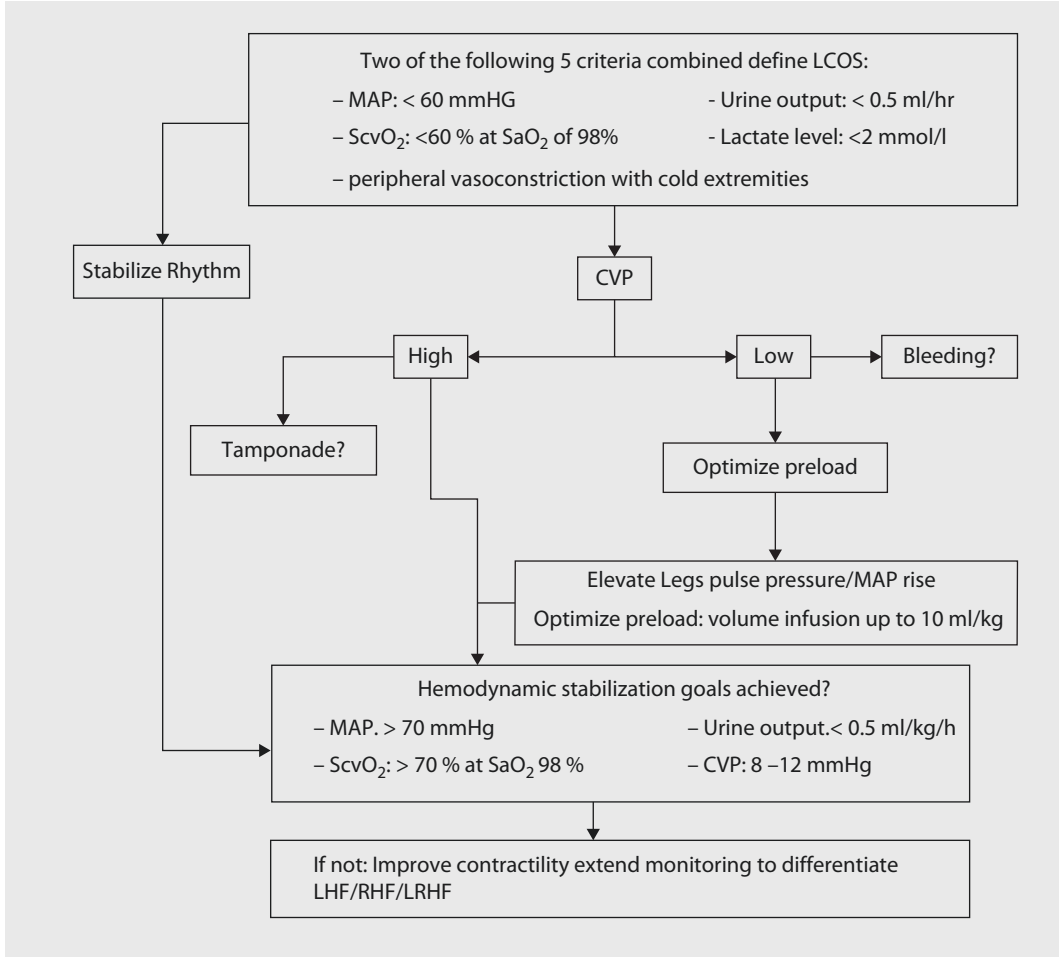


Fig. 9.4 Staged approach to therapy of postoperative low cardiac output syndrome (LCOS), stage I: *CVP* central venous pressure, *LHF* left heart failure, *LRHF* left and right heart failure, *MAP* mean arterial pressure, *RHF* right heart failure, SaO_2 oxygen saturation, $ScvO_2$ central venous oxygen saturation

and a decrease of the left ventricular afterload by well-timed balloon deflation. This leads to an enhancement of coronary perfusion and a relief of cardiac work with the result of an improved oxygen supply-demand ratio and a somewhat increased cardiac output.

The appropriate time of initiation of the IABP is as soon as possible (Baskett et al. 2002; Christenson et al. 2002; Ramnarine et al. 2005). It is frequently helpful for the treatment of LCOS and especially helpful for coronary patients during the weaning process from extracorporeal circulation. The exceed threshold of an epinephrine dose level of >0.2 μg/kg/min or a dobutamine dose level of >10 μg/kg/min should be the indication for IABP use. Preoperatively, IAPB will be very useful in patients with an impaired left ventricular ejection fraction, left main stem stenosis, unstable angina, or the need of a coronary reoperation to improve outcomes (Dunning and Prendergast 2003).

The useful effects of the IABP use can be immediately seen at the pressure curve after IABP is initiated: due to the reduced cardiac output, diminished first peak in the arterial pressure curve is frequently followed by a second higher peak as function of the preload reduction. Recovery of the left ventricular function leads usually to a conversion of the peaks—the first one overgrow the second one. This is an indication for removal of IABP, especially in face of significantly reduced pharmacological circulatory support. The IABP should be weaned from a 1:1

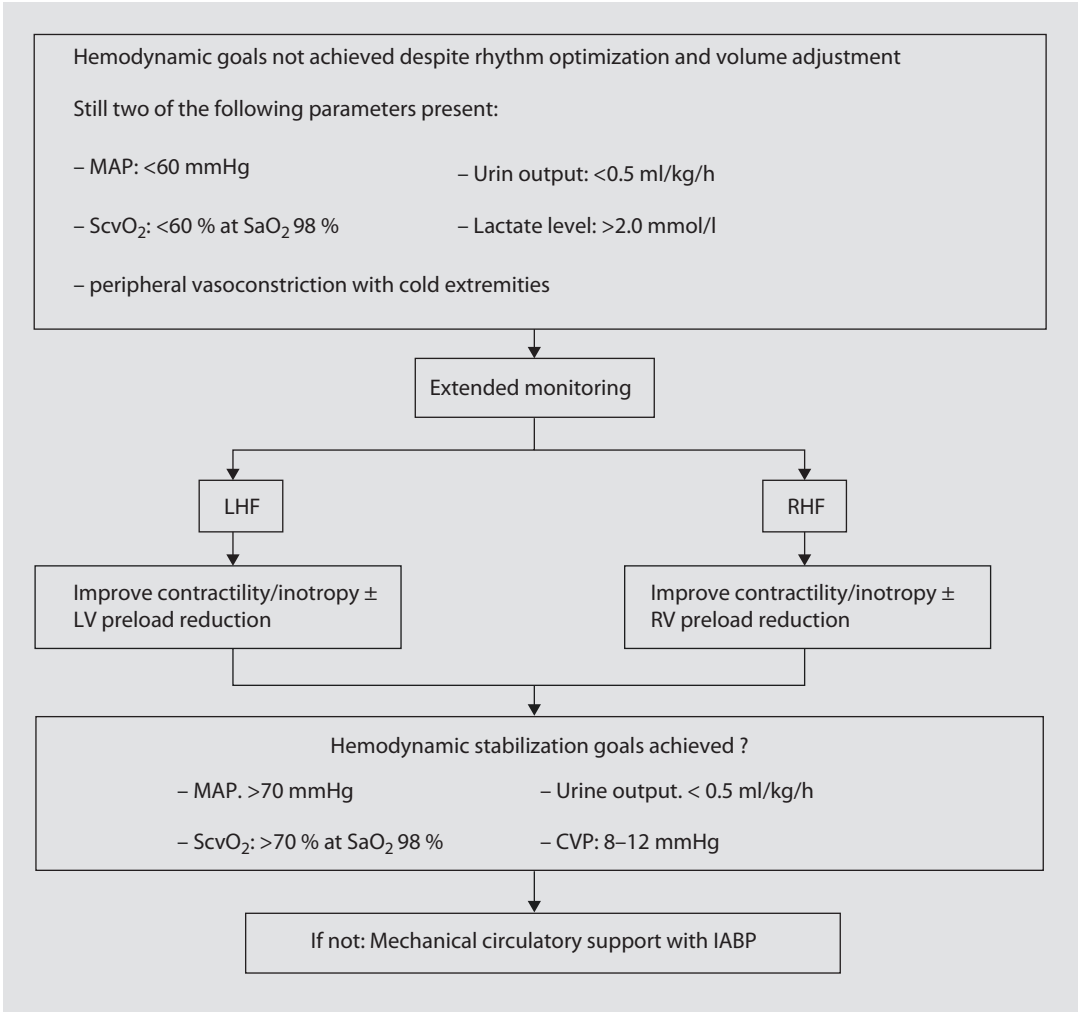


Fig. 9.5 Staged approach to therapy of postoperative low cardiac output syndrome (LCOS), stage II: *CVP* central venous pressure, *IABP* intra-aortic balloon pump, *LHF* left heart failure, *LRHF* left and right heart failure, *LV* left ventricular, *MAP* mean arterial pressure, *RHF* right heart failure, *RV* right ventricular, SaO_2 arterial oxygen saturation, $ScvO_2$ central venous oxygen saturation

augmentation to 1:2, finally to 1:3, understanding that thrombus may form on the balloon surface at this rate. The complication rate should be below 10 %, mostly related to vascular problems such as site bleeding, thromboembolic events, and ischemia of lower extremities.

The therapy success relates to the basic problem and its reversibility. While stunned myocardium should return to near-normal function, the scarred myocardium will not improve contractility despite IABP support. In patients with failed IABP support, mechanical circulatory support may have to be escalated to the next level using ventricular assist devices.

Mechanical Circulatory Support: Ventricular Assist Devices (VADs)

There is a variety of ventricular assist devices available, some combined with an oxygenator, to treat both cardiac and lung failure in the short, mid, or long term postoperatively. The ventricular assist devices may be pulsatile or axial, uni- (LVAD or RVAD) or biventricular (BiVAD), and intra-, para-, or extracorporeal. There is also FDA-approved total artificial heart for intracorporeal biventricular assistance. Finally, the VADs may be considered as bridge to recovery, sole therapy, or bridge to heart transplant depending on patient's characteristics and medical history. Having an

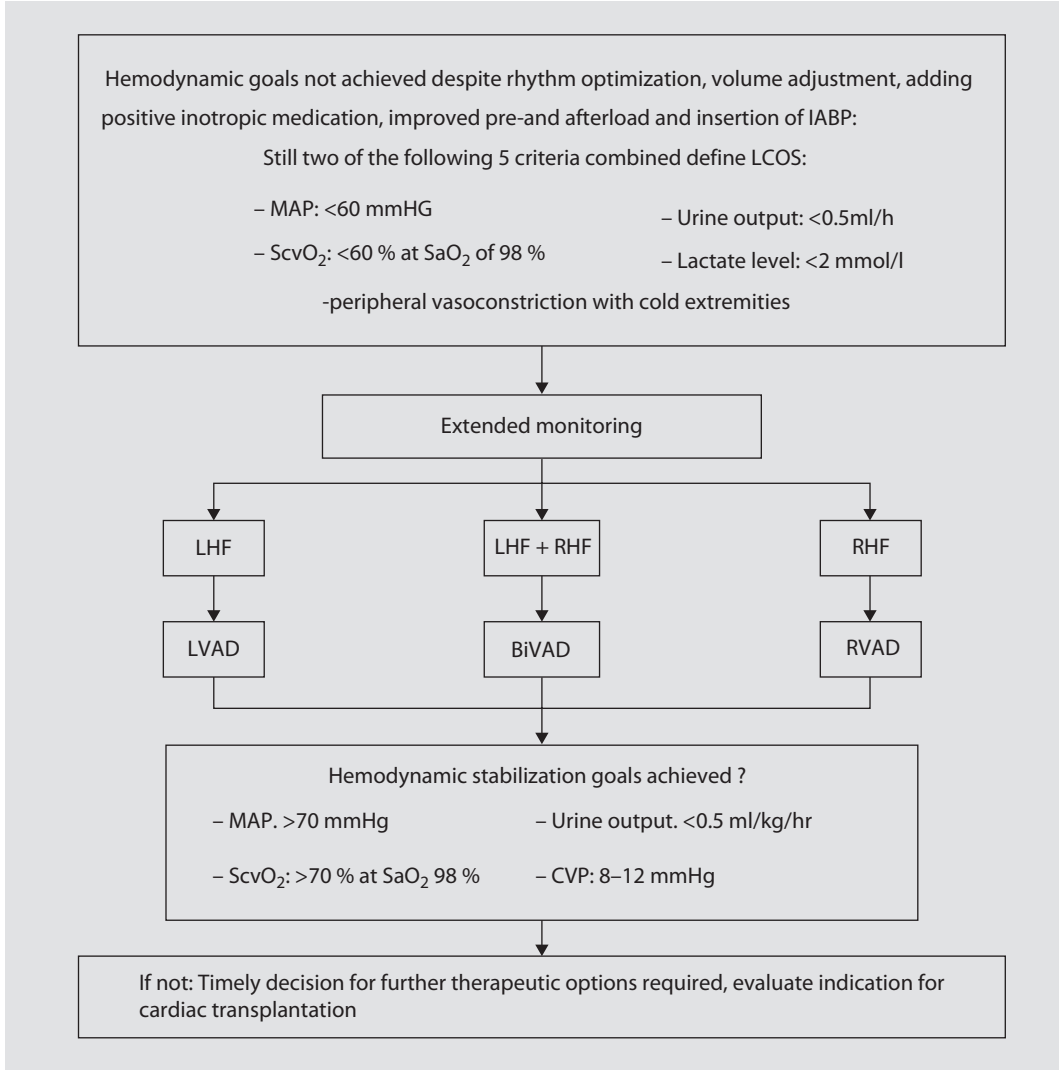


Fig. 9.6 Staged approach to therapy of postoperative low cardiac output syndrome (LCOS), stage III: *BiVAD* biventricular assist device, *IABP* intra-aortic balloon pump, *LHF* left heart failure, *LVAD* left ventricular assist device, *MAP* mean arterial pressure, *RHF* right heart failure, *RVAD* right ventricular assist device, SaO_2 arterial oxygen saturation, $ScvO_2$ central venous oxygen saturation

on-site heart transplantation program can be very complementary for the latter group of patients.

Overall, the management of VAD patients consumes a lot of time, money, and resources. The adjudication and indications for use will be limited in the future to smaller cohort with best possible outcomes. This is important in today's increasing health-care cost environment (See also ► Chapter «Cardiac Assist Devices and Total Artificial Heart», Sects. 38.4–38.7). Figs 9.4, 9.5, and 9.6 summarize graphically the key points of LCOS therapy.

9.5.2.6 Arterial Hypertension

The early postoperative blood pressure in cardiac surgical patients is more often rather too low than too high. The occasional presence of early postoperative arterial hypertension may become problematic especially in patients with fragile cardiac, aortic, and arterial tissue. An extreme case is perioperative care of patients with acute aortic syndrome such as aortic dissection (Khoynezhad and Plestis 2006). After pain has to be excluded as cause, the pressure has to be lowered using the following drugs—alone or in combination (Khoynezhad 2007).

Table 9.8 Review of approach to patients with acute aortic syndrome presenting with hypertension to the CSICU

1. Physician has a high index of suspicion for acute aortic dissection
(a) History:
(i) Severe, sudden onset, sharp or tearing back pain, chest pain, shoulder pain, or abdominal pain
(ii) 50 years or older, history of hypertension, aortic dissection or aortic aneurysm (of family history of such), previous cardiac surgery, connective tissue disorder (bicuspid aortic valve, Marfan syndrome, Ehlers-Danlos syndrome, Loeys-Dietz syndrome), or peripartum
(b) Physical examination:
1. Blood pressure differential in various extremities (pulse deficit)
2. Acute stroke, paraplegia, or paraparesis
3. Abdominal pain, flank pain
2. General measures:
(a) Establish two large bore (18 gauge or bigger) IVs
(b) Supplemental oxygen by nasal cannula or mask
(c) Cardiac monitor
(d) Get an EKG, portable chest X-ray, place a Foley catheter
(e) Obtain CBC, chemistry panel, coagulation panel, UA, CK, Troponin, d-dimer
(f) Type and cross ten units packed red blood cells (PRBCs)
(g) Set up an arterial line
3. Diagnostic imaging:
(a) Computed tomography angiogram (CTA)
(b) Transesophageal echocardiogram
(c) Magnetic resonance angiogram (MRA)
(d) Intravascular ultrasound
4. Prompt cardiothoracic surgical consultation
5. Blood pressure, heart rate, and pain management
(a) First line: β-blockers
(i) Labetalol, bolus (15 mg) ± a drip (5 mg/h)
(b) If hypertension persists, add:
(i) Nicardipine drip (starting dose: 5 mg/h)
(c) If tachycardia persists, add:
(i) Esmolol (loading 0.5 mg/kg over 2–5 min, followed by a drip of 10–20 μg/kg/min
(ii) Diltiazem drip (loading 0.25 mg/kg over 2–5 min, followed by a drip of 5 mg/h)
(d) Goals:
(i) Heart rate 60–80 beats/min
(ii) Systolic blood pressure 100 mmHg
(e) Morphine (for pain relief)
6. Hemodynamically unstable patients:
(a) Endotracheal intubation and mechanical ventilation
(b) Blood pressure support with crystalloid and colloid (PRBCs if rupture is suspected)
(c) TEE at bedside in the emergency department or in the OR
(d) Pericardiocentesis is not recommended (class III)

(From Tran and Khoynezhad (2009); used with permission)

Nitroprusside

Nitroprusside is a potent direct arterial and venous dilator, acting through release of nitric oxide. It has a rapid onset of action, with a half-life of seconds to <2 min and thus is given in the form of continuous infusion. Dose is 0.3 μg/Kg/min IV infusion; with titration q2 min until desired response with maximum dose 10 μg/Kg/min. The hypotensive effects of nitroprusside can be unpredictable because it simultaneously causes potent venodilatation and peripheral arterial vasodilation. This is especially the case for patients with severe left ventricular hypertrophy and preload-dependent diastolic dysfunction. It has been shown to cause coronary steal; it can cause a significant reflex tachycardia, causing increase in aortic shear stress (dp/dt). Therefore, especially when given in aortic dissection patients, it should be combined with beta-blocker therapy.

It is photosensitive, so it requires special handling. Its most serious adverse effect is in the form of cyanide toxicity, which occurs due to accumulation of its metabolites thiocyanate/cyanide, and its clinical presentation may vary leading to difficulty in diagnosis. Thus, it is recommended that this drug be used only when other intravenous antihypertensive agents are not available.

Nitroglycerin

Nitroglycerin acts by release of nitric oxide causing vasodilation, especially of the coronary arteries. It is primarily a venodilator; however, at higher doses it also causes arterial vasodilation. It does not cause coronary steal. Initial dose is typically 5 μg/min IV infusion with increments by 5 μg q3–5 min until desired response, with a maximum of 200 μg/min. A drawback is that it cannot be used for a prolonged duration as patients rapidly develop tolerance to it. Being predominantly a venodilator, it is subject to same hemodynamic issues such as nitroprusside. It may predispose to severe hypotension in patients with left ventricular hypertrophy and preload-dependent diastolic dysfunction.

Nicardipine

Nicardipine is a short-acting calcium channel blocker belonging to the dihydropyridine class. It causes cerebral and coronary vasodilation with only minimal negative inotropic effect, has minimal effects on atrioventricular nodal conduction, and has little effect on cardiac output or pulmonary artery wedge pressure. In contrast to nitroprusside, the pharmacodynamic properties of nicardipine are favorable. Nicardipine has a very long half-life; the ß-half-life of nicardipine is approximately 40 min, whereas its γ-half-life is approximately 13 h. Because about 14 % of the drug is eliminated during the γ-phase, the hypotensive effect can be prolonged. In a study comparing the effect of nicardipine to nitroprusside in patients with severe postoperative hypertension, the two drugs had equivalent efficacy, but only nicardipine reduced both cardiac and cerebral ischemia. Nicardipine is administered through a continuous infusion with starting dose at 5 mg/h infusion. It is titrated by 2.5 mg/h q5 mins with maximum of 15 mg/h. In cardiac surgical patients, nicardipine has been shown to decrease arterial BP acutely with no effects on ventricular preload or cardiac output, suggesting that it has a minimal negative inotropic action. With nicardipine, oxygen delivery to the cells is usually well maintained, and oxygen requirements are unchanged. Because nicardipine is metabolized primarily by the liver, it can be used in patients with renal insufficiency. However, there have been concerns with the use of calcium channel blockers in patients with coronary artery disease, possibly due to sympathetic activation, bleeding caused by inhibition of platelet aggregation, and pro-arrhythmic effects. There remains considerable debate about the role of calcium channel blockers as first-line therapy.

ß-blockers

In perioperative cardiac surgery, especially in caring of patients with aortic dissection and aortic aneurysm, integrity of arterial system depends not only on the absolute blood pressure but also on the velocity of left ventricular contraction (dp/dt). A vasodilator alone, instead of decreasing the heart rate, may even cause reflex tachycardia, thus causing propagation of the aortic dissection. Therefore, the optimum treatment involves a combination of a parenteral ß-blocker and an arterial vasodilator, with heart rate targeted around 55–65 beats/min. The ß-blocker of choice in this situation is generally esmolol and, alternatively, labetalol or metoprolol.

Esmolol is a ß1-antagonist, while labetalol is a combined α1-, ß1-, and ß2-antagonist with an

alpha to beta-blocking ratio of 1:7. Both of them, by slowing down the heart rate, also reduce the myocardial oxygen demand. Esmolol reduces blood pressure by reducing cardiac output and inhibiting renin release, while labetalol decreases afterload directly and also inhibits renin release. Their disadvantage is in the form of their negative inotropic effect and possible reaction in patients with reactive airway disease. Half-life of esmolol is around 9 min, while that of labetalol is 5.5 h.

The hypotensive effect of labetalol begins within 2–5 min after its intravenous administration, reaching a peak at 5–15 min following administration and lasting for about 2–4 h. Labetalol may be administered as a loading dose of 20 mg, followed by repeated incremental doses of 20–80 mg at 10-min intervals until the desired BP is achieved. Alternatively, after the initial loading dose, an infusion commencing at 1–2 mg/min and titrated up to until the desired hypotensive effect is achieved is particularly effective. Bolus injections of 1–2 mg/kg have been reported to produce precipitous falls in BP and should therefore be avoided.

The onset of action of esmolol is within 60 s, with duration of action of 10–20 min. However, because it is metabolized by red blood cell (RBC) esterases, any condition that precipitates anemia will prolong its «short half-life.» The metabolism of esmolol is via rapid hydrolysis of ester linkages by RBC esterases and is not dependent on renal or hepatic function. Typically, the drug is administered as a 500–1,000 μg/kg loading dose over 1 min, followed by an infusion starting at 50 μg/kg/min and increasing up to 300 μg/kg/min as necessary.

Table 9.8 reviews approach to patients with acute aortic syndrome presenting with hypertension to the CSICU (Tran and Khoynezhad 2009).

9.5.2.7 Rhythm Disturbances

Rhythm disturbances can be categorized by their heart rate such as tachycardia and bradycardia, by hemodynamic situation, such as stable or unstable, and they also can be anatomically classified into being of supraventricular or ventricular origin.

▪▪ Bradycardia

Bradycardia after cardiac operations is often seen. The operated heart profits from an elevated heart rate for the first postoperative hours to days, to ensure a sufficient cardiac output in combination with an increased diastolic myocardial perfusion.

Most cardiac surgical patients are pretreated with ß-blockers due to their cardiac disease. This results in a sinus bradycardia after operation. For a sufficient weaning from extracorporeal circulation, a transient pacing of the heart is frequently necessary. In patients after valve operations, the anatomical relation to the atrioventricular conduction system has to be considered for possible irritation or damage with a resulting dysfunction. These rhythm disturbances require short-term external pacing via temporary pacing wires on the right atrium and the right ventricle.

Most patients with normal ventricular function do well with transient ventricular pacing. In patients with decreased left ventricular function, a more physiologic pacing, such as AV sequential pacing, is recommended—especially for hypertrophic and dilated hearts. The pacing should be at 80–100 bpm with an AV interval of 150 ± 25 ms. The power output should be at least double the lowest energy to stimulate the heart (stimulation threshold). If the patient does not have an adequate baseline rhythm, the output should be three times higher as the threshold. In the operating room, the sensing will be turned off due to interferences with the electrocautery. Before closing the chest, the pacemaker has to be recalibrated to detect any own rhythm and to work proper in the inhibited mode (Spotnitz 2005).

The role of temporary biventricular pacing in the perioperative setting is not well defined yet, although some data suggest a benefit in patients with poor left ventricular function. If it is tried, the position of the pacemaker wires on the left heart is important. The recommended site for the left ventricular wire is the posterior lateral position, near the first marginal artery. An anterior position is not recommended (Flynn et al. 2005).

▪▪ Supraventricular tachycardia

Supraventricular tachycardia occurs with an incidence of 50 % after cardiac surgery. In general a low potassium and magnesium blood level is a frequent reason for supraventricular tachycardia, mostly an atrial fibrillation. Supraventricular extrasystole is harmless and can be treated by balancing the electrolyte blood levels. A postoperative atrial fibrillation is associated with an increased mortality. It is also associated with major adverse events, such as ventricular tachycardia, stroke, prolonged hospital stay, and

Table 9.9 Pharmacological therapy in atrial fibrillation

Drug	Rhythm control	Rate control	Prophylaxis
Flecainide	(+)	(+)	(+)
Propafenone	(+)	(+)	(+)
ß-blocker	+	++	+++
Amiodarone	++	+	++
Sotalol	++	+	++
Verapamil	+	++	+
Diltiazem	+	++	+
Digoxin	+	++	+
Magnesium	–	–	++
Electric cardioversion	+++	+++	–

– no therapeutic effect, (+) therapeutic effect controversially discussed, + therapeutic effect possible, ++ therapy effective, +++ therapy very effective

increased hospitalization costs (Dunning et al. 2006; European Resuscitation Council 2005; McKeown and Gutterman 2005).

The most important aspect of supraventricular tachycardia for the cardiac surgical patient is the hemodynamic relevance. If the patient's hemodynamics are unstable, the heart rate has to be immediately decreased to 80–100 bpm, usually in the form of electric cardioversion.

If the patient is hemodynamically stable, one has to decide whether heart rate control or rhythm control, namely, conversion into sinus rhythm, is the ultimate goal. In patients with history of atrial fibrillation or with high pretest probability for atrial fibrillation, such as large atrium or mitral valve operations, conversions may not be persistent. Table 9.9 gives an overview about the drugs used in atrial fibrillation as well as their effects.

If atrial fibrillation is present, the following aspects have to be addressed additionally: pain-induced stress must be treated adequately, the potassium blood level should be more than 4.5 mmol/L, the oxygen saturation should be more than 92 %, and the volume overload with atrial stretch should be avoided.

In the presence of atrial fibrillation over 24 h, a systemic anticoagulation with a PTT prolongation of 50–60 s is recommended. In patients with transient atrial fibrillation, it can be stopped after 24 h if stable sinus rhythm prevails.

If the diagnosis of tachycardia is unclear, administration of 6–12 mg adenosine may be diagnostic and—on rare occasion—therapeutic. Adenosine allows for cardiac pause due to bradycardia and complete AV block in patients with supraventricular tachycardia, in particular in AV-reentry pathology. In patients with ventricular tachycardia, adenosine remains without any response. In the following, less common supraventricular tachycardia forms will be presented.

Sinus tachycardia mostly appears if the patient suffers stress, such as pain or fear due to inadequate sedation. In some cases, ß-blockers, digoxin, or verapamil will be recommended.

Atrial flutter: this rhythm disturbance shows up with a sawlike P wave configuration in the ECG. The goal is to overdrive the flutter with the pacemaker or to perform synchronized electric conversion. In some cases, vagus stimulation or pharmacological AV delay (i.e., with verapamil) will reduce the heart rate, but the rate of conversion is very low.

AV-reentry tachycardia can be influenced using adenosine or verapamil. ß-blockers, flecainide, or propafenone are not very effective.

■■ Ventricular tachycardia

Intermittent ventricular extrasystoles are common after cardiac surgery and do not have to be treated in the majority of cases. The common causes are hypoxemia and unbalanced electrolytes. These have to be treated first. Sometimes the pharmacological effect of ß-blocker or amiodarone can be helpful. If intermittent monomorphic extrasystoles occur, such as nonsustained ventricular tachycardia, the CSICU physician has to be cautioned, because these can lead to an ongoing ventricular tachycardia (VT).

A persisting ventricular tachycardia is, in most cases, hemodynamically compromising, especially with ventricular flutter or ventricular fibrillation. An immediate therapy is required: an electric r-peak triggered cardioversion (biphasic; 100 J → 200 J) in ventricular flutter and an asynchronous defibrillation in the presence of ventricular fibrillation. In general, resuscitation due a relevant drop of the arterial pressure is required, too. A slow VT can be treated medically

Table 9.10 Survey of commonly used antiarrhythmic drugs

Class of drugs	Drug	Dosage	Indication
1 Sodium channel blocker			
1 A	Disopyramide	50–100 mg po	Atrial and ventricular tachycardia
1 B	Lidocaine	100–150 mg IV	Ventricular tachycardia
1 C	Flecainide	100–150 mg po	Prophylaxis of atrial fibrillation
	Propafenone	100–150 mg po	
2 (ß-blocker)	Metoprolol	5–20 mg IV	Supraventricular and ventricular extrasystole, frequency control of atrial fibrillation
		25–100 mg po BID	
	Esmolol	25–50 mg IV	
3 (potassium channel blocker)	Amiodarone	5 mg/BW within 20 min IV	Ventricular tachycardia, atrial fibrillation
	Sotalol	20–100 mg IV	Atrial fibrillation
4 (calcium channel blocker)	Verapamil	5–10 mg IV	Atrial tachycardia, frequency control of atrial fibrillation
	Diltiazem	20–30 mg IV	
Others	Adenosine	6–18 mg	All kinds of atrial tachycardia (except atrial flutter), differential diagnosis for rhythm disturbances
	Digoxin	0.4–0.6 mg	Frequency control of atrial fibrillation

with lidocaine (100–150 mg) or amiodarone (300 mg) or with an overdrive pacing.

While Table 9.10 gives an overview about commonly used antiarrhythmic drugs, Fig. 9.7 shows our treatment algorithm for tachycardias.

According to their mode of action, the commonly used antiarrhythmic drugs can be divided into four classes and others. The four classes are:

1. Sodium channel blockers
2. β-blockers
3. Potassium channel blockers
4. Calcium channel blockers

9.5.3 Bleeding Complications and Pericardial Tamponade

9.5.3.1 Incidence, Cause and Diagnostic

Bleeding complications occur after cardiac surgery with an incidence of 1–3 % (Mehta et al. 2009). The following table indicates general guidelines for tolerable chest tube outputs, which, however, do not apply to all patients (Table 9.11). The amount of chest tube output and its assumed cause are important for therapeutic decision-making. If the diagnosis is unsecure, a hematocrit check of chest tube drainage will establish the diagnosis.

After cardiac surgery employing extracorporeal circulation, function of the coagulation system is severely impaired. Reasons are full heparinization, reduced platelet function (preoperative antiplatelet therapy, perioperative hemodilution, hypothermia, and mechanical irritation), and lack of fibrinogen in combination with a hyperfibrinolysis (in close correlation to the duration of the extracorporeal circulation). Initially increased chest tube output is not unusual but has to be watched carefully.

On ICU admission, the surgeon will report any intraoperative bleeding complication and if there may be a surgical reason for the bleeding. If the deranged coagulation system is the presumed underlying cause, appropriate diagnostics should be run to guide therapeutics:

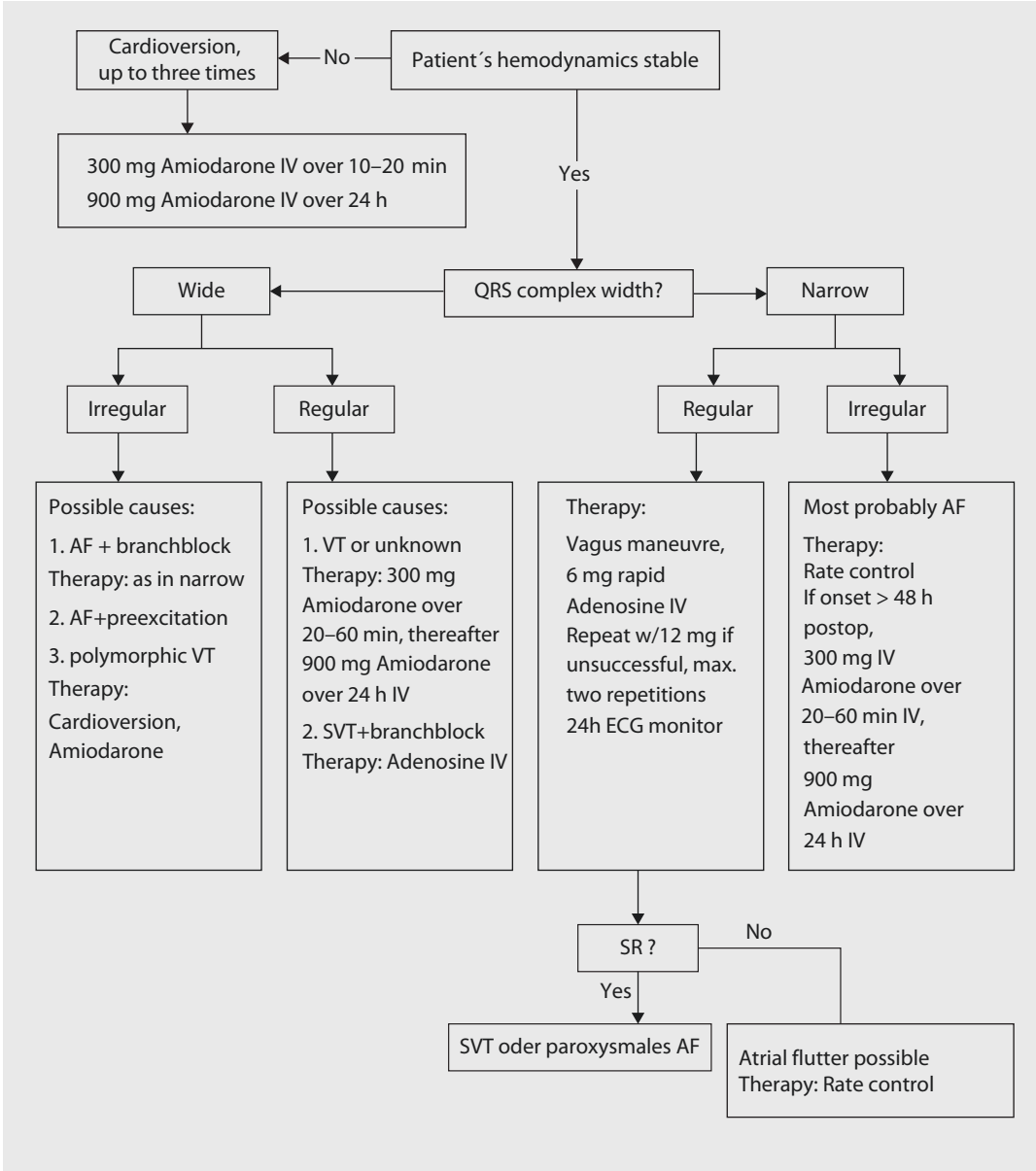


Fig. 9.7 Our management algorithm for tachycardia. *AF* atrial fibrillation, *SR* sinus rhythm, *SVT* supraventricular tachycardia, *VT* ventricular tachycardia

- Protamine administration to correct prolonged activated clotting time
- Administration of platelets in patients with platelet dysfunction or thrombopenic patients
- Administration of fresh frozen plasma or cryoprecipitate due to a lack of coagulation factors

The routine blood gas analysis will provide additionally the actual hemoglobin concentration to detect any hemoglobin drop. Furthermore, hemoglobin sample from chest tubes, chest X-ray, and echocardiography can assist with diagnosis of bleeding and cardiac tamponade.

9.5.3.2 Prevention

The most important way to prevent bleeding complications is a careful chest closure. A famous quote in cardiac surgery puts it well: most common cause of significant postoperative bleeding is

Table 9.11 Limits for the postoperative blood drain loss in adult patients

Time after operation	Maximum amount of tolerable blood loss (ml)
First hour	400
Second hour	300
Third hour	250
Fourth hour and later	200

«hypoprolinemia,» insufficient use of suture material to avoid surgical site bleeding. Furthermore, antifibrinolytics such as ε-aminocaproic acid or tranexamic acid have shown to reduce postoperative bleeding and take backs for hemorrhagic complications.

The potential value of intraoperative monitoring of fibrinolysis, residual heparin content, or platelet function is not unequivocally established, yet.

9.5.3.3 Therapy

Disorders of the coagulation system have to be treated as already described. If there are bleedings from the sternal closure wires out of the bone, a temporarily increased PEEP to 10 mmHg may stop the bleeding. In case of a true blood loss above the limits given in Table 9.11, a prompt reexploration should be considered. The timing for take back is poor, if the patient requires new onset and/or increasing inotropic support. If the diagnosis is in doubt, a more aggressive approach toward operative exploration is warranted. Furthermore, sternal exploration in CSICU should be promptly done in acute hemodynamic compromise: simple bleeders can be treated immediately, and in more complex situations, the hemodynamics can be stabilized, allowing safe transfer of the patient to the OR. Principles of explorations in CSICU should be exercised as «dry run» with the nursing and allied health-care providers, allowing for smoother run in true emergencies. A resternotomy kit should be fabricated, along with adequate lightening tools, improving visualization of surgical site in CSICU.

9.5.4 Lung and Mechanical Ventilation

The lungs in cardiac surgical patients are compromised due to various reasons. Therefore a short period of mechanical ventilation is common. However, if other circulatory and pulmonary reasons are lacking, weaning from ventilator should be attempted at or before 4 h after operation. A long-term ventilation (more than 72 h) is only required in 5 % of the patients after cardiac surgery (Murthy et al. 2007).

Generally, two different ventilation mechanisms are used: mandatory or assisted ventilation. The mandatory ventilation can be volume- or pressure-controlled, the latter being the mode of choice. The goal of mandatory ventilation mode is to improve the gas exchange by improving the ventilation-perfusion ratio, whereas the assisted ventilation aims toward reducing the work of breathing for the patient (Table 9.12).

9.5.4.1 Ventilation Parameters and Settings of the Ventilatory Machine

A series of parameters may be adjusted and regulated on the ventilator. In the following, important ventilation parameters are discussed briefly:

- The *inspiratory oxygen concentration* can be adjusted between 21 % and 100 %, corresponding to FiO_2 of 0.21–1.0.
- The *respiratory rate* is the adjustable frequency of breathing cycles on the ventilator and is usually between 8 and 14 per minute.
- The *tidal volume* is defined as the amount of air introduced during one breathing cycle. The physiological value is between 5 and 8 ml/kg BW.
- The *minute volume* is the product of respiratory rate and tidal volume. The physiological value is between 5 and 8 l/min.
- The *inspiratory flow* is the amount of airflow during inspiration compared to the time. The inspiration flow can be constant, decelerating, or accelerating. Generally the inspiration flow is chosen as short as possible and as high as necessary, mostly in decelerating fashion.
- The *maximum inspiratory pressure* is the guiding parameter in pressure-controlled ventilation. If the inspiratory pressure is not regulated, especially in volume-controlled ventilation, pulmonary complications may occur due to pulmonary barotrauma. Therefore the pressure-controlled ventilation has become the preferred mode of ventilation in the last years.
- The *relation of inspiration and expiration* should be 1:2 to ensure an almost complete

Table 9.12 Summary of ventilation modes

Controlled ventilation	Assisted ventilation	Controlled and assisted ventilation
IPPV, intermittent positive pressure ventilation CPPV, continuous positive pressure ventilation VCV, volume controlled ventilation PCV, pressure controlled ventilation	SIMV, synchronized intermittent mandatory ventilation MMV, mandatory minute volume PSV, pressure support Ventilation ASB, assisted spontaneous breathing CPAP, continuous positive airway pressure	BIPAP, biphasic positive airway pressure

airway gas exchange. The *end-expiratory pressure* is the alveoli pressure at end of exhalation, *and it is in the spontaneous breathing* human ca. 4–5 cm H_2O. Ventilation with a positive end-expiratory pressure (PEEP) ensures patency of the alveoli, by reducing the alveolar collapse, which decreases the ventilation/perfusion interface.

To set the ventilator, size, weight, and clinical condition of the patient have to be considered, and there are certain limits that should not be exceeded. For example, an oxygen concentration of more than 60 % for a prolonged time can cause lung trauma. Furthermore, high maximum inspiratory pressure as well as tidal volume of more than 12 ml/kg BW is associated with a poor outcome (Malhotra 2007; Wheeler and Bernard 2007). Table 9.13 has suggested values for various ventilatory parameters. The settings of the ventilator are adjusted and corrected by blood gas analysis, pulse oximetry, end-tidal CO_2 monitoring, and intrapulmonary pressure curves on the ventilator.

Table 9.13 Proposed ventilator settings

Extent	Setting
Inspiratory oxygen concentration, FiO_2	<0.6
Respiratory rate	8–12/min
Inspiration-expiration relation	1:2
Positive end-expiratory pressure (PEEP)	5–8 cm H_2O
Maximum inspiration pressure	<30 mmHg
Tidal volume	5–8 ml/kg BW[a]

[a]Predicted body weight (female, 45.5 + 0.91 × [height in cm—152.4]; male, 50 + 0.91 × [height in cm—152.4])

9.5.4.2 Weaning and Extubation

Generally, the patient arrives on the CSICU in mandatory ventilation mode until the cardiopulmonary situation is stable and the patient is normothermic. Weaning from ventilator should be performed in stepwise fashion. The mandatory ventilation mode may then be switched to assisted mode, if the acid-base balance and the oxygen level are stable at a FiO_2 0.6 (pH 7.4 ± 0.5; arterial CO_2 partial pressure, 40–55 mmHg; HCO_3^- concentration, 20–28 mmol/L; base excess, 1.5 to 1.5). Controlled reduction of the ventilation pressure support to 3–5 mmHg over PEEP is the next step. Figure 9.8 summarizes our approach for weaning patients from ventilation.

Extubation can be possible if the following extents are achieved:

- Adequate oxygenation under spontaneous breathing with a paO_2 >60–80 mmHg with a FiO_2 ≤0.5 and a peripheral SaO_2 ≥92 %
- PEEP ≤8 cm H_2O
- Pressure support 3–5 mmHg over PEEP
- Tidal volume ≥5 ml/kg BW
- Breathing minute volume <20 L/min
- Respiratory rate 8–18 per min
- pH level 7.3–7.45 with a CO_2 arterial partial pressure of 40–55 mmHg

The clinical presentation of the patient before the extubation is important:

- The patient is awake, alert, and responds adequately.
- The patient can cough on command.
- The cardiac function is stable (heart rate 50–140 bpm, systolic blood pressure 90–180 mmHg, low-dose catecholamines, isovolemia, no pathological ECG changes to the preoperative ECG).
- Body temperature ≥96.8 °F (36 °C).
- Drainage loss ≤50–100 ml/h.
- Intact physiological reflexes.

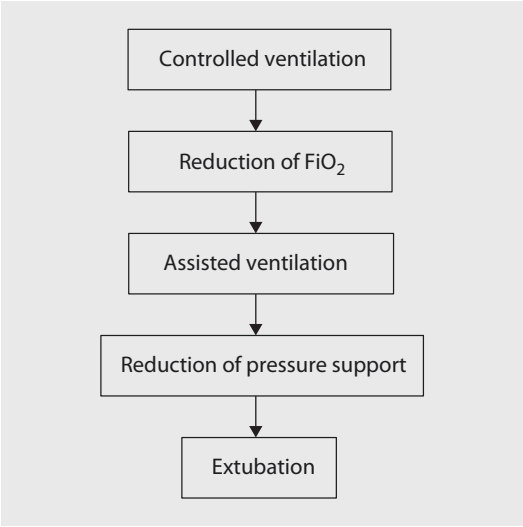


Fig. 9.8 Stepwise weaning from postoperative ventilation

An impaired renal function should not be a reason for prolonged ventilation. Some patients present with agitation and stress during the extubation process (respiratory rate >35 per min, increased blood pressure, and sweating). They should receive light anxiolytics and after ongoing ventilation a second trial of extubation. Only a small percentage of these patients will require reintubation.

9.5.4.3 Respiratory Failure

Acute respiratory failure is the most important pulmonary issue in critical care medicine. The following risk factors are known to increase the risk for an acute respiratory failure:

1. Preoperative risks
   - Emergency operation
   - Cardiac reoperation
   - Age >75 years
   - High level of urea
   - Low hematocrit level
   - Body mass index >30 kg/cm^2
   - Pulmonary hypertension
   - Left ventricular ejection fraction <35 %
   - Cardiogenic shock
   - Chronic obstructive pulmonary disease

2. Intraoperative risks
   - More than 120 min of extracorporeal circulation
   - Transfusion more than six units of PRBCs
   - Cardiac arrest

3. Postoperative risks
   - Serum albumin level <4 g/dl
   - Low cardiac output requiring catecholamines
   - Resternotomy for bleeding

Before an acute respiratory failure can be diagnosed, other causes of hypoxemia have to be corrected first:

- Occluded breathing tube
- Leaking ventilation system
- Improper ventilation mode
- Occluded bronchus with mucus or foreign bodies
- Hemothorax, pneumothorax
- Diaphragm malfunction due to phrenic injury
- Pleural effusion
- Atelectasis
- Significant right-left shunt
- Lung edema of cardiac origin

■■ Symptoms, findings, pathophysiology, causes, and diagnostic of respiratory failure

The most common symptom of respiratory insufficiency is dyspnea, often associated with tachypnea, along with cyanosis. It may have an acute onset. The X-ray displays diffuse, bilateral interstitial infiltrates that are not of cardiac origin. However, the differential diagnosis in cardiac surgical patients may be difficult, and per definition wedge pressure should be less than 18 mmHg.

In respiratory insufficiency oxygen saturation is less than 90 % on pulse oximetry, and the blood gas analysis is consistent with hypoxemia. If the paO_2/FiO_2 ratio (Horovitz quotient) is less than 300, a mild acute respiratory distress syndrome (ARDS) is present. If the quotient is less than 200 (e.g., paO_2 <100 mmHg at FiO_2 0.5), it is defined as moderate ARDS. If it is below 100, the ARDS is classified as severe (The ARDS Definition Task Force 2012).

The causes for respiratory insufficiency after cardiac surgery are:

- Pneumonia
- Aspiration
- TRALI (Transfusion-related acute lung injury)

- Reperfusion injury after long-term extracorporeal circulation
- Pulmonary parenchymal hemorrhage
- Secondary involvement due to multi-organ failure

Based on the causes, the required diagnostics are:

- Bronchoscopy, combined with sampling for microbiological analysis
- Critical analysis of the pulmonary and perioperative history
- Chest X-ray
- CT scan

The pathophysiology of the acute respiratory failure is topic of many research endeavors and is discussed below in brief:

In the beginning of acute respiratory failure, the capillary endothelium is damaged by toxin that leads to hyperpermeability of the endothelium followed by an interstitial pulmonary edema. This increases the gas diffusion distance and causes collapse or narrowing of the alveoli. This process is enhanced by the loss of surfactant factor. Ventilation-perfusion mismatch occurs: the not ventilated lung parts are well perfused, whereas the ventilated lung parts are only insufficiently or not perfused. The thrombotic occlusion of pulmonary capillaries, hypoxemia-triggered vasoconstriction, the release of proinflammatory mediators, and the ventilation-perfusion mismatch worsens the pulmonary function. The ensuing hypoxemia deteriorates the function of other organ systems. Adequate oxygenation needs to be maintained, and the aggressive ventilation mode (FiO_2 1.0, high ventilation pressure) necessary for adequate oxygenation may enhance the structural damage of the alveoli and the lungs.

Therapy

Treatment of acute respiratory failure consists of two basic principles: a causal and a symptomatic. The cause of ARDS must be treated promptly. The symptomatic treatment entails ensuring proper oxygenation of the patient. This includes adequate mechanical ventilation and other supportive care, such as special positioning of the patient. Noninvasive ventilation in patients developing acute respiratiory failure after primary extubation has been advocated (Hill at al. 2007) and gained some interest more recently in cardiac patients, also (Guarracino and Ambrosino 2011; Cabrini et al. 2015), but it remains controversial.

Ventilation in respiratory failure

Since only a portion of alveoli are sufficiently ventilated and perfused, the ventilation of patient with ARDS is challenging: The interstitial edema decreases the compliance of the lungs. Therefore the ventilation pressure is increased to maintain the effective minute volume, imposing additional stress to the alveoli and causing their further destruction. To reduce these deleterious effects, reduced tidal volume to 4–6 ml/kg has been shown to be associated with improved outcomes (The Acute Respiratory Distress Syndrome Network 2000). A possible side effect of this alveoli-protective strategy is an increased CO_2 partial pressure that can be tolerated upon 80 mmHg (permissive hypercapnia), unless the associated metabolic acidosis would be harming the patient.

In acute severe lung failure, high PEEP levels have to be used. The PEEP setting on the ventilator is usually 12–15 cm H_2O but may be increased to maximal 25 cm H_2O. To calculate the approximate required PEEP, the formula PEEP ≤ FiO_2/0.05 can be useful. The exacter method is to adjust the PEEP to the pressure-volume curve, where the PEEP value has to be chosen right above the lower inflection point. The lower inflection point indicates the pressure limit that is necessary to open the alveolus. A PEEP right above this point ensure open alveolus (Hemmila and Napolitano 2006; Malhotra 2007; Wheeler and Bernard 2007). Adequate PEEP will prevent alveoli trauma caused by collapse and reopening. The effect of high PEEP to the circulatory system occurs only in patients with hypovolemia, but higher risk of pneumothorax has to be considered in all patients with sudden respiratory deterioration. It is important to mention that there is no data supporting improved outcome with high PEEP ventilation in respiratory failure. The strategy of a low tidal volume, permissive hypercapnia, and high PEEP is called the open lung concept.

In addition to high PEEP ventilation, recruitment maneuver is helpful in maintaining alveoli open: this includes brief gradual increase of

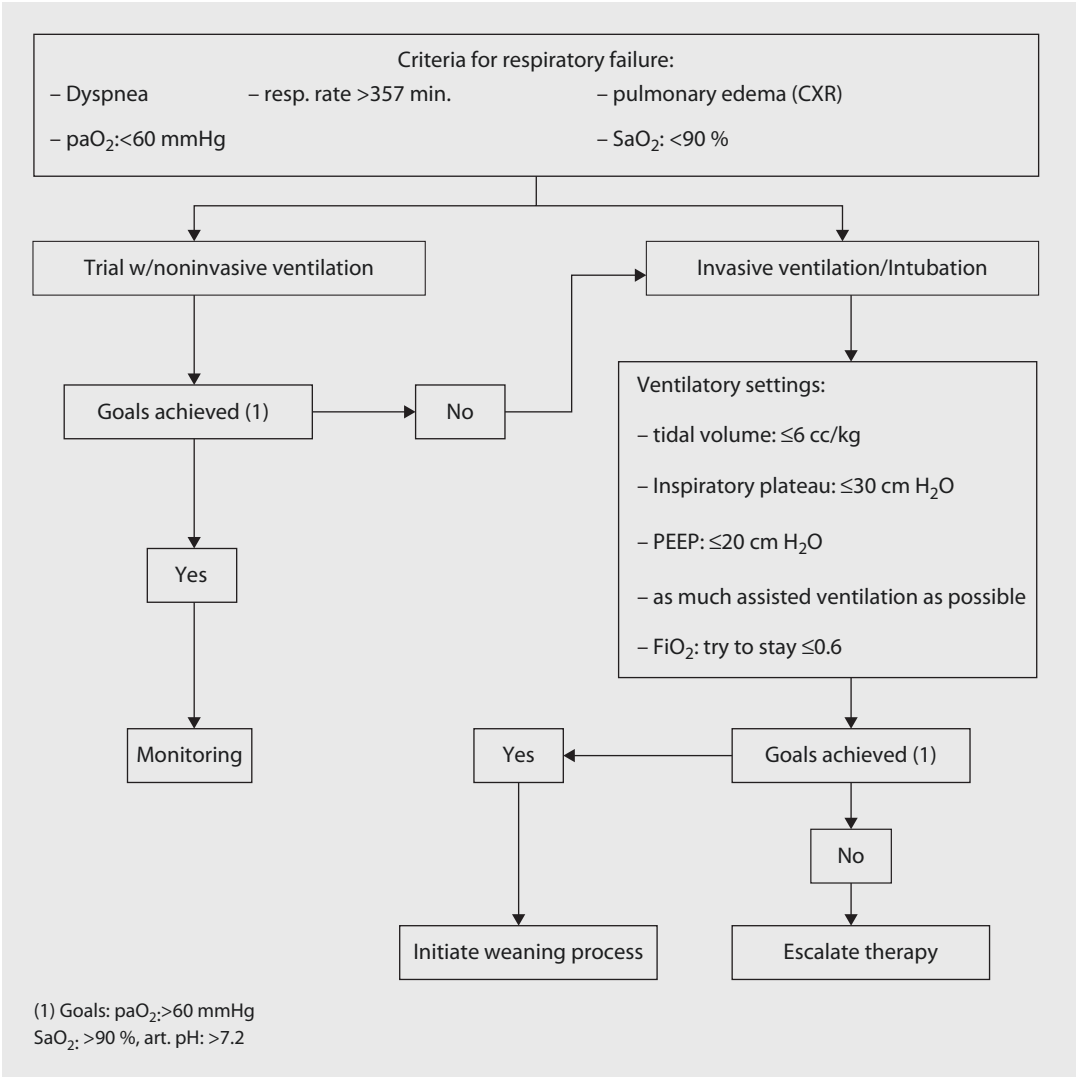


Fig. 9.9 Our initial therapeutic approach in acute respiratory failure/acute pulmonary failure. *FiO_2* inspired oxygen fraction, *paO_2* arterial oxygen partial pressure, *PEEP* positive end-expiratory pressure, *CXR* chest X-ray, *SaO_2* arterial oxygen saturation

maximal ventilation pressure up to 40 cm H_2O to «recruit» (reopen) collapsed alveoli. After recruitment maneuver is finished, the maximal ventilation pressure is reduced slowly to lowest possible PEEP that keeps the alveolus open based on lower inflection point.

If the open lung concept with a low tidal volume and a high PEEP do not sufficiently improve oxygenation, inversed ratio ventilation may be tried: the inspiration-expiration ratio has to be changed to a prolonged inspiration time (2:1 up to 4:1), and the FiO_2 has to be maximally increased to 1.0. This is an effective approach in hypoxemic patients, but usually requires paralyzing medication.

Yet another mode of ventilation that can be useful in patients with acute respiratory failure and maximal reduced lung compliance is the high-frequency oscillatory ventilation mode. A high ventilation frequency between 300 and 600/min reduces the tidal volume to 1–4 ml/kg and subsequently reduces stress to the alveoli (Hemmila and Napolitano 2006). The expiration is actively carried out by a vibrating membrane, in contrary to other forms of high-frequency ventilation (i.e., jet ventilation).

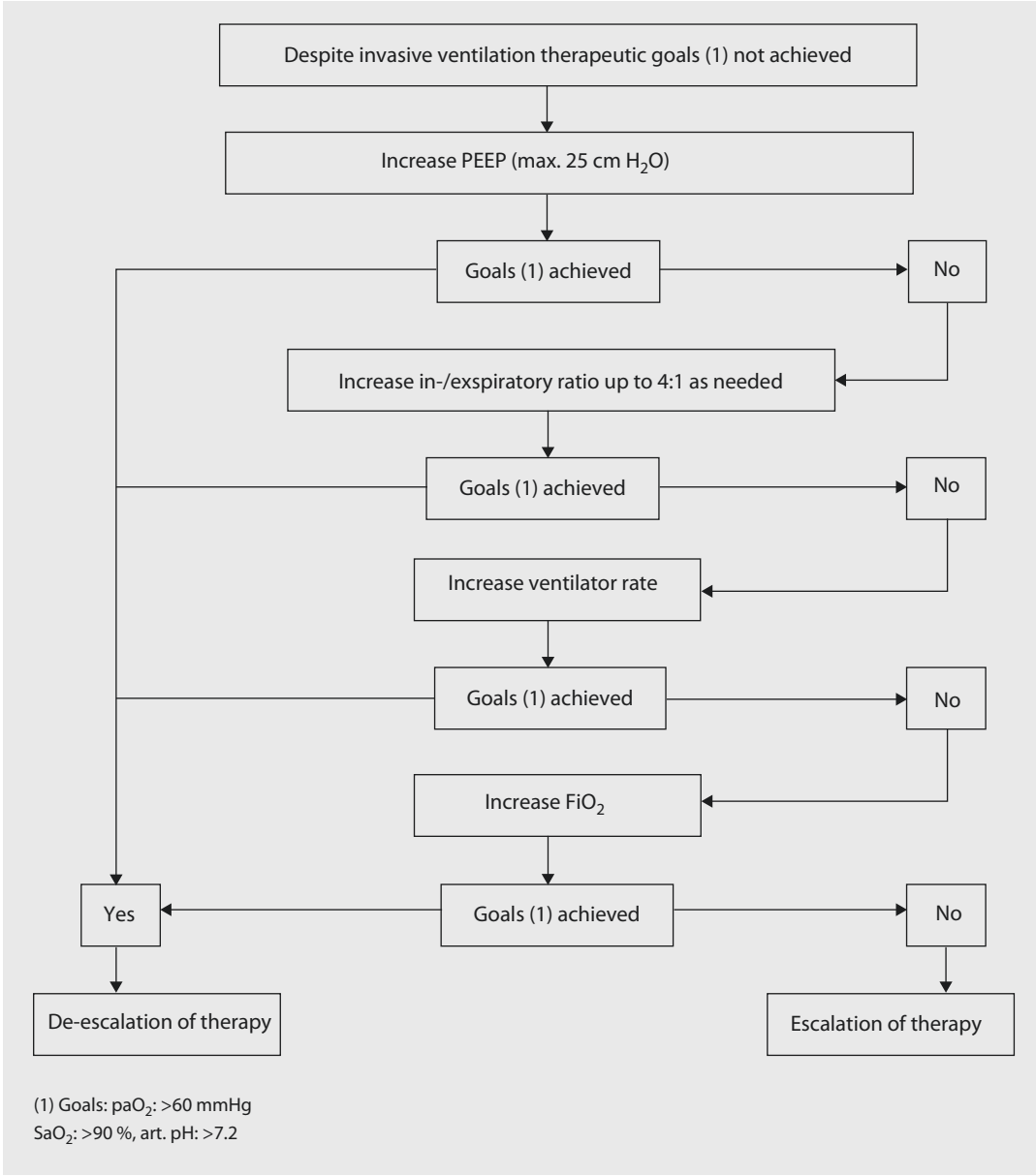


Fig. 9.10 Our approach for therapeutic escalation in acute respiratory distress syndrome: *FiO_2* inspired oxygen fraction, *paO_2* arterial oxygen partial pressure, *PEEP* positive end-expiratory pressure, *SaO_2* arterial oxygen saturation

Other supportive Measures

The proper position of the patient can improve the oxygenation. Elevated upper body position (30–45°) supports the patient's breathing, by reducing pressure from the diaphragm caused by abdominal organs, and reduces silent aspiration. The most effective strategy for oxygenation is ventral positioning of the patient. This has a very positive influence on oxygenation due to improved ventilation-perfusion ratio and homogenized ventilation. The frequency and the duration of this positioning are not strictly defined, but it should be adapted to the patient's clinical condition. The positioning maneuver requires increased logistic and personnel workforce, but the effects on the respiratory system are remarkable.

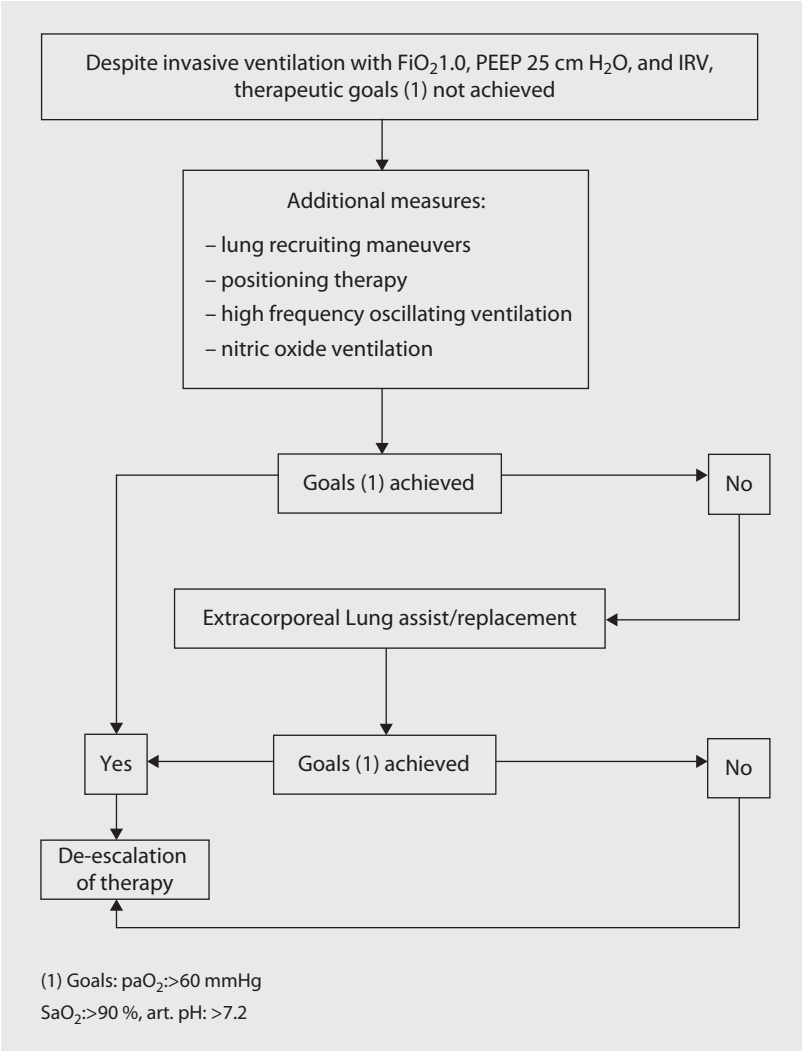


Fig. 9.11 Our approach for further therapeutic escalation in acute respiratory distress syndrome: irresponsive to first stage escalation of therapy: *FiO_2* inspired oxygen fraction, *IVR* inversed ratio ventilation, *NO* nitrogen oxide, *paO_2* arterial oxygen partial pressure, *PEEP* positive end-expiratory pressure, *SaO_2* arterial oxygen saturation

The 135° positioning and special rotational beds are less effective than the true 180° positioning but certainly very helpful. To perform these maneuvers, the patient's hemodynamics has to be stable. This may not be the case early after open-heart cases.

The fluid management of the respiratory-impaired patient has to be restrictive to avoid any volume overload that results in an increase of the existing lung edema, worsened by cardiogenic pulmonary edema. However, intravascular hypervolemia has to be avoided given the high PEEP ventilation.

Furthermore, «sedation holiday» is an effective method in shortening ventilation time and the CSICU stay. This entails daily brief disruption of the sedatives, until the patient is awake and more alert, before reinitiating the sedation.

The early initiation of enteral nutrition has shown a positive effect in reducing ventilator period in patients with mechanical ventilation more than 36 h, whereas the endotracheal surfactant therapy and the inhaled medications, such as prostacyclin or iloprost, have not proven to be efficacious.

Steroids improve blood pressure, the pulmonary oxygenation, and the duration of mechanical ventilation in ARDS but do not reduce the 60- or 180-day mortality.

If, despite maximal aforementioned efforts, adequate oxygenation is not secured, more invasive methods may be considered in appropriate patient cohort, understanding the significant cost involved: there are pump-driven systems, such as extracorporeal membrane oxygenation (ECMO) for heart and lung support, or pumpless systems that work due to the artery-venous pressure gradient. In both systems the blood oxygenation occurs with assistance of a gas exchange membrane oxygenator. The oxygenation is more effective using ECMO, but the pumpless systems are more often used in hypercapnic patients with hypoxemia responding to conventional measures.

Due to its pump drive, the ECMO is not dependent on the circulatory situation, whereas the pumpless systems require a stable or stabilized cardiocirculatory function. ECMO consumes significantly more CSICU resources (Bein et al. 2007).

Figures 9.9, 9.10, and 9.11 show treatment algorithms and therapeutic escalation in respiratory distress situations.

Tracheotomy

Tracheotomy is a supportive measure providing the following advantages: secured airway, easier oral care, more comfort for the patient, and no larynx trauma. Weaning from mechanical ventilation is faster and less complicated in patients having tracheotomy (De Leyn et al. 2007). Reduced dead space ventilation by tracheotomy reduces the work of breathing for the patient. The procedure can be performed surgically or using percutaneous Seldinger approach in CSICU-dilatational tracheotomy—that can be done on bedside with bronchoscopy (Ciaglia et al. 1985).

The optimal timing for a tracheotomy is unclear yet (Young at al. 2013), but the positive effect of this treatment should encourage the intensivist to perform it earlier if prolonged mechanical ventilation is expected.

Weaning from ventilation and extubation

Patients with more than 48 h of mechanical ventilation will have a more difficult course of extubation. The weaning actually starts already preoperatively with breath-exercising respiratory physiotherapy. The weaning process once intubated and ventilated starts by reducing the sedation so far that the patient is stress-free and hemodynamically stable under assisted ventilation. Then the patient controls his breath rate, but the tidal volume is assisted by the ventilator. Subsequently the ventilation pressure is reduced stepwise, allowing the patient do most of the work of breathing. The blood gas analysis and the clinical situation should be stable within 30–60 min without evidence of respiratory fatigue.

Signs of respiratory fatigue are:

- Agitation
- Anxiety
- Sweating
- Tachycardia
- Blood pressure drop or rising
- Centralization (peripheral vasoconstriction)
- Inadequate ventilation time volume
- Inadequate tidal volume
- Breath rate of >35 per minute
- Peripheral oxygen saturation of <92 %
- Arterial oxygen partial pressure <60 mmHg

If the patient displays these symptoms, the extubation should be delayed for another 24 h, before a second extubation is attempted.

If the patient is stable and calm, the sedation should be ended. Just before extubation the patient should be totally awake, should push the hand on command, and should raise the head off the pillow. If a gastric tube is in place, it should be completely suctioned and removed before extubation.

After extubation, the patients are positioned in sitting or with upper body elevated 30–45° to facilitate the work of breathing and reduce aspiration risk. The possibility of reintubation has to be considered in elderly, frail, malnutritioned patients, or those with poor mental status and prolonged ventilator support. Figure 9.12 summarizes our approach to wean patients from ventilation after a complicated course.

Side Effects and complications of mechanical ventilation

Possible side effects of the mechanical ventilation are:

- Increased intrathoracic pressure with a reduction of the cardiac filling

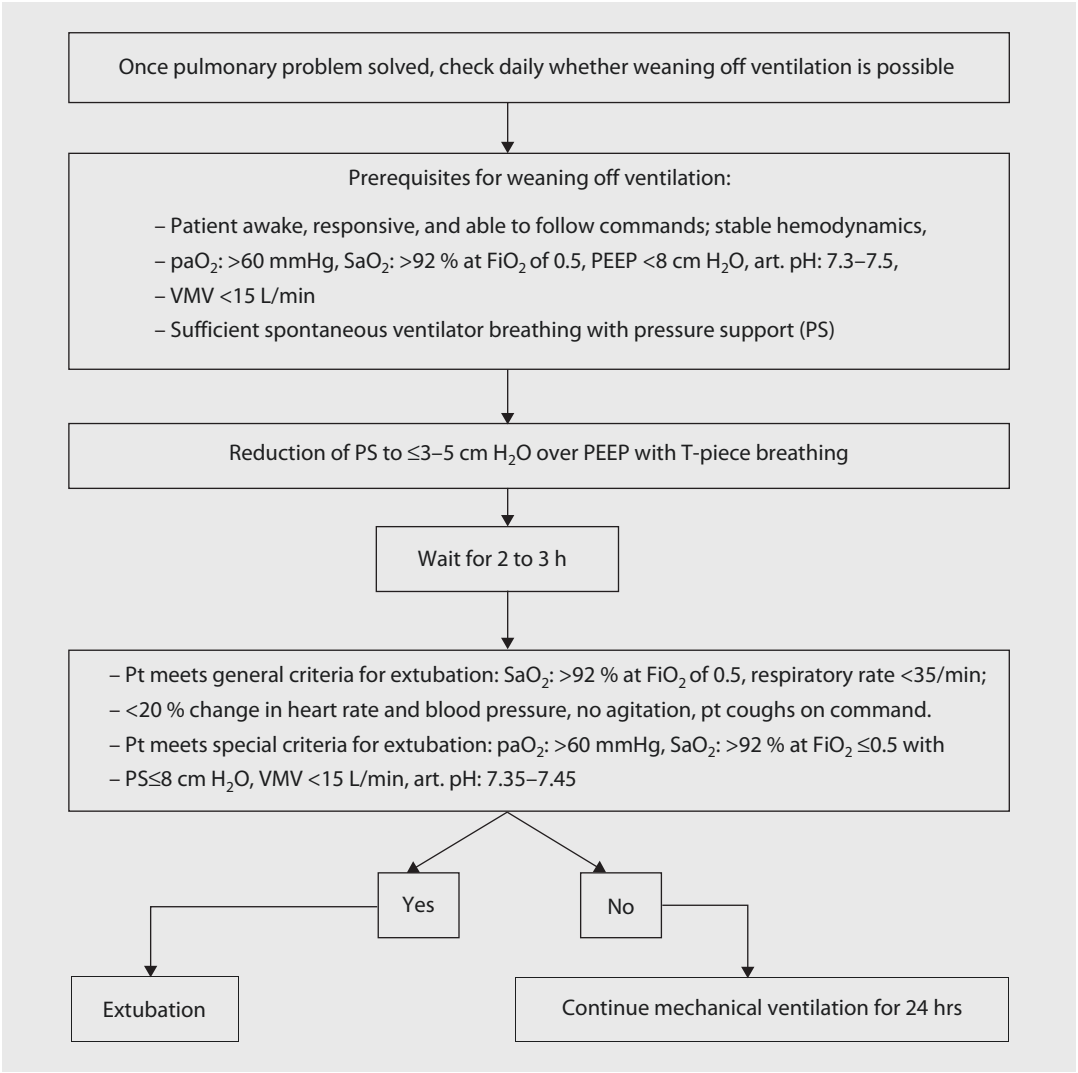


Fig. 9.12 Our approach for weaning off mechanical ventilation. *FiO_2* inspired oxygen fraction, *paO_2* arterial oxygen partial pressure, *PEEP* positive end-expiratory pressure, *PS* pressure support, *SaO_2* arterial oxygen saturation, *VMV* ventilator minute volume

- Decrease of renal blood flow with activation of the renin-angiotensin system
- Reduction of airway mucus clearance
- Damage to the surfactant monolayer by positive airway pressure-mediated shear stress

The following complications should be mentioned:

- Trauma to or edema of larynx, trachea, and bronchi
- Ventilator-associated pneumonia
- Pulmonary trauma due to a high FiO
- Damages of the alveoli due to high tidal volume or a high airway pressure
- Barotrauma of the lungs with the risk of a pneumothorax
- Decubitus and thromboembolic complications due to immobilization

While most side effects and complications may be self-limiting, the most deleterious effect on a patient with acute respiratory failure is from ventilator-associated pneumonia (VAP). VAP

develops 48 h or longer after mechanical ventilation is given by means of an endotracheal tube or tracheotomy and is associated with substantial mortality. VAP results from the invasion of the lower respiratory tract and lung parenchyma by microorganisms. Intubation compromises the integrity of the oropharynx and trachea and allows oral and gastric secretions to enter the lower airways. The prevention of any triggers is most important: sterile exchange of ventilator components, sterile endotracheal suction, positioning of the patient with aspiration precautions, and weaning according to established protocols.

9.5.5 Kidney

9.5.5.1 Incidence, Cause, and Diagnostic of Renal Complications

The perioperative acute renal failure occurs with an incidence 1.2–5.1 % after cardiac surgery and is associated with 40–80 % increased mortality (Markewitz and Lante 2006; Mehta et al. 2006).

The definition of renal failure has been somewhat empiric and vague in cardiac surgical literature; increased creatinine by certain value or percentage, reduced urine output, and other factors had been used in the past, making reliable comparison of literature impossible. The RIFLE classification (◘ Table 9.14) as well as the more recent AKIN classification (Acute Kidney Injury Network) (◘ Tables 9.15 and 9.16) can be used to quantify and qualify renal failure (Bellomo et al. 2004; Hoste et al. 2006; Kuitunen et al. 2006; Mehta et al. 2007). RIFLE, for example, classifies severity and outcomes, it proposes criteria for three grades of increasing severity: risk of acute renal dysfunction (*R*), injury to the kidney (*I*), and failure of kidney function (*F*), and two outcome classes: loss of kidney function (*L*) and end-stage kidney disease (*E*). The RIFLE classification based on estimated glomerular filtration rate (eGFR) and serum creatinine. eGFR is calculated with the abbreviated «Modification of Diet in Renal Disease formula», MDRD (Jin et al. 2008). Calculation of estimated glomerular filtration rate (eGFR) with the abbreviated «Modification of Diet in Renal Disease» formula: Chronic kidney

◘ **Table 9.14** The RIFLE criteria according to Bellomo et al. (2004)

Class	GFR criteria
*R*isk	Plasma Cr increase 1.5 × baseline or GFR decline >25 %
*I*njury	Plasma Cr increase 2 × baseline or GFR decline >50 %
*F*ailure	Plasma Cr increase 3 × baseline or GFR decline >75 % or acute plasma Cr> 4 mg/dL
*L*oss	Persistent ARF = complete loss of kidney function requiring dialysis for >4 weeks but <3 months
*E*nd stage	End-stage kidney disease requiring dialysis for >3 months

◘ **Table 9.15** National Kidney Foundation classification of stages for chronic kidney disease

Stage	Description	GFR
1	Kidney damage with normal or ↑ GFR	>90 (with CKD risk factors)
2	Kidney damage with mild or ↓ GFR	60–89
3	Moderate ↓ GFR	30–59
4	Severe ↓ GFR	15–29
5	Kidney failure	<15 (or dialysis)

◘ **Table 9.16** Grading for acute renal failure according to the consensus conference of the Acute Kidney Injury Network (AKIN)

Grade	Serum creatinine level	Urine output
1	Creatinine ↑ by ≥0.3 mg/dL or ↑ by >150–200 %	<0.5 cc/kg/h for 6 h
2	Creatinine ↑ by >200–300 %	<0.5 cc/kg/h for 12 h
3	Creatinine ↑ by >300 % or by 4 mg/dL or by ≥0.5 mg/dL acutely	<0.3 cc/kg/h for 24 h or anuria (<100 cc in 12 h)
3	Kidney replacement therapy necessary	

Mehta et al. (2007)

$$\left(\text{Estimated eGFR}\left[\text{ml/minute/1.73 m}^2\right] = 186 \times [\text{serum creatinine}]^{-1.154} \times [\text{age}]^{-0.203} \\ : [0.742 \text{ if female}] \times [1.210 \text{ if African} - \text{American}]\right)$$

disease (CKD) is defined as a baseline eGFR ≤60 ml/min/1.73 m^2, while chronic renal failure (CRF) is defined as eGFR ≤30 ml/min/1.73 m^2 (stage III versus IV and V according to the Kidney Disease Outcome Quality Initiative) (National Kidney Foundation 2002).

In clinical practice, many cardiac surgical patients can be classified into RIFLE criteria «risk,» and some may correspond to the level «injury.» However, the recovery is the norm in majority of these patients.

Starting with the level «failure,» the treatment of the patients is going to be more complex and resource intensive. CSICU capacities are limited, and patients that require hemodialysis may occupy them for a prolonged period of time. The preoperative risk stratification of renal failure is not easy to perform, but the Society of Thoracic Surgeons (STS) provides a risk score for postoperative renal failure (Online STS Risk Calculator) (◘ Table 9.17). ◘ Figure 9.13 shows how the results of the STS Risk Calculator translate into a probability for renal replacement therapy.

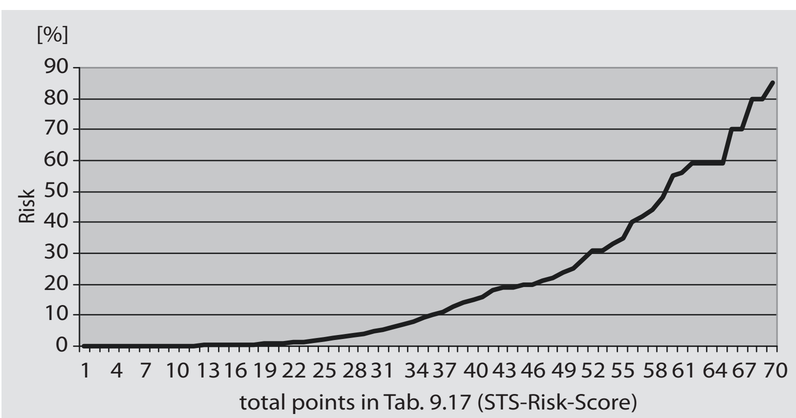
9.5.5.2 Prevention of Renal Complications

Prevention is the best therapy of renal failure. It is important to ensure an adequate hydration, oxygenation, and adequate mean arterial pressure. The perioperative administration of aspirin has proven to have a positive effect (Mangano et al. 2002); this not being the case with N-acetylcysteine or «renal-dose» dopamine. Avoiding any nephrotoxic function, such as aminoglycosides, and all prostaglandin-synthesizing blockers is important but not always possible.

9.5.5.3 Therapy of Renal Complications

During the first hours after cardiac operation, the urine production decreases in nearly all patients due to various causes. Once in a while, the urinary catheter may be clogged or kinked, or the mean arterial pressure is too low. These problems can be solved easily, and they have to be considered early in the evaluation of the patient. Generally diuretics, such as furosemide or torasemide or mannitol, are given to improve urine output, understanding that urine output per se does not imply extend of kidney function. Also, the maximum dose per day of diuretics has to be taken into account.

If urine output cannot be reactivated with the aforementioned methods, the decision for renal replacement therapy should be made within 24 h or less of oliguria or anuria. Options for renal replacement therapy such as intermittent or a continuous filtration mode does not influence the patient's outcome (Garwood 2004). Diuretic administration, however, should to be stopped due to its contra-productive effect (Mehta et al. 2002), as up to 25 % of these patients will require long-term renal replacement therapy.



◘ **Fig. 9.13** Probability to require renal replacement therapy after cardiac surgery in relation to preoperative risk factors (STS risk score acc. to Table. 9.17) Mehta et al. (2006)

Table 9.17 Parameters and corresponding points for risk assessment, if perioperative renal replacement therapy will be required

STS risk score for perioperative renal replacement therapy												Points
Age	<55	55–59	60–64	65–69	70–74	75–79	80–84	85–89	90–04	95–99	>100	
Points	0	1	2	3	4	5	6	7	8	9	10	
Creatinine	0.5	1.0	1.5	2.0	2.5	3.0	3.5	>4.0				
Points	5	10	15	20	25	30	35	40				
Type of operation	CABG only		AV only		CABG + AV			MV only		CABG + MV		
Points	0		2		5			4		7		
Myocardial infarction	No		<3 weeks									
Points	0		3									
Color	White		Nonwhite									
Points	0		2									
Chronic lung disease	No		Yes									
Points	0		3									
Reoperation am Herzen	No		Yes									
Points	0		3									
NYHA IV	No		Yes									
Points	0		3									
Cardiogenic shock	No		Yes									
Points	0		7									
										Sum		

9.5.6 Gastrointestinal Tract

Severe complications of the gastrointestinal tract, such as bleeding, ulcer, prolonged ileus, pancreatitis, intestinal ischemia, or mesenteric artery thrombosis occur in about 3 % of the patients undergoing open-heart surgery.

The outcome of patients with severe gastrointestinal complications is associated with high mortality (Markewitz and Lante 2006). Most common problems involved gastrointestinal bleeding, amenable to endoscopic treatment options, and associated with much better outcome. Ischemic bowel complications are associated with a mortality of 70–100 %, and high index of suspicion is critical to promptly achieve the right diagnosis. The symptoms are usually unspecific, causing late diagnosis. Furthermore, there is no specific monitoring available, but increasing lactate and phosphate levels and persisting therapy refractory metabolic acidosis should increase index of suspicion, prompting diagnostic testing. The metabolic acidosis is usually accompanied by paralytic ileus and leukocytosis. The next step has to be taken very quickly to confirm the diagnosis: mesenteric arteriogram, but if there is no possibility for angiography, immediate laparotomy is requested to diagnose and possibly treat the issue

with superior mesenteric or celiac artery bypass operation. If a nonocclusive ischemia is present, the administration of local vasodilatory drugs such as papaverine is possible. Even with rapid decision taking, the mortality remains at 50 %. When symptoms are present for more then 12 h survival is improbable.

A subtype of patients with acute type A or B aortic dissection may have or develop malperfusion to celiac or mesenteric artery during the index hospitalization (complicated aortic dissection). The malperfusion is usually due to dynamic (dissection flap occluding the orifice of the aortic branch vessels) or anatomic (dissection flap and hematoma in the orifice for the branch vessels). These patients have a treatment paradigm that has significantly changed in recent years, requiring further details here (Khoynezhad et al. 2009).

Prompt diagnosis and anti-impulsive treatment of patients with suspected complicated aortic dissection (CAD) is essential for improved outcome. Originally, the minor only available option used to be surgical fenestration of the membrane separating true and false lumen in order to perfuse both true and false lumen and reduce the pressure gradient that is the cause for the dynamic malperfusion. For anatomic obstructions, mesenteric bypass operation would be added. The endovascular treatment is, however, now the new standard to promptly restore intestinal blood flow. Diagnosis is confirmed with intravascular ultrasound and/or angiogram in the hybrid suite (See also ► Chapter «Endovascular Surgical Therapy of Thoracic and Thoracoabdominal Disease of the Aorta», Sect. 28.7.2).

9.5.7 Central and Peripheral Nervous System

9.5.7.1 Incidence of Neurological Complications

Depending on definitions and the diagnosing physician, incidence of neurological complications may vary. As minor as it may appear to be, it can significantly reduce quality of life longterm and does increase early mortality. Neurological complications are classified into the following categories:

- Ischemic stroke
- ICU psychosis
- Critical illness polyneuropathy
- Other neurological complications such as neurocognitive deficits, neuropsychological complications, and peripheral nerve injuries

9.5.7.2 Ischemic Stroke

The incidence of an ischemic stroke after cardiac surgery is 2–10 %, being between 1.4 % and 3.8 % in patients after coronary artery bypass grafting and up to 10 % with multiple valve operations or total arch replacement (Selim 2007). The patient typically has hemiparesis and aphasia depending on the troubled brain hemisphere. CT scan will be able to show the infarcted area 2 h after stroke. The usual causes for an ischemic stroke are ruptured atherosclerotic plaques from the aorta, or calcific aortic valve, thrombotic material from the left atrium or the left ventricle, acute occlusion of a high-grade-stenosed internal carotid artery. Next to CT and duplex carotid, transesophageal echocardiography is ordered to exclude emboli source from the heart.

Therapeutic approach to thromboembolic ischemic stroke is parenteral lysis with recombinant tissue-type plasminogen activator. However, this therapeutics cannot be used for most cardiac surgical patients given the recent operation. In certain circumstances, super-selective intra-arterial lysis may be performed; however this has to be performed within 6 h of symptoms. In most cases the stroke occurs in patients under sedation, making any therapeutic intervention within that «window» impossible. Therefore the therapy of a stroke in cardiac surgical patients is mostly symptomatic:

- Increase of the blood pressure (160–180 mmHg systolic) as autoregulation in the stroke area may not be present any more
- Adequate oxygenation
- Blood glucose level <150 mg/dl
- Body temperature less than 99.5 °F (37.5 °C)

While systemic heparinization is not recommended, administration of aspirin (50–325 mg) daily as secondary prophylaxis is established.

The most feared complication is brain edema with increase of intracranial pressure. Therefore the monitoring has to be extended to check for signs of increased intracranial pressure and decreased consciousness. Recommended positioning is 30° elevated upper body, diuresis with mannitol, or administration of short-acting barbiturates, such as thiopental. While steroids are no longer recommended, the use of hyperventilation is controversial and may be used as short-term therapy until other measures are initiated. The last option in reducing intracranial pressure is surgical decompression with a craniotomy.

Furthermore, treatment of accompanying seizures may be necessary, along with all accompanying problems of immobilized and unconscious patients, such as aspiration pneumonia, thromboembolic complications, and decubitus ulcer.

The prognosis of survivors with stroke after cardiac surgery is rather grim; only 47 % survive the first 5 years, half of them with permanent deficits requiring external support for daily living activities (Salazar et al. 2001). Early rehabilitation will help to restore the muscular coordination and improve functional outcome.

9.5.7.3 ICU Psychosis

ICU psychosis is a disorder in which hospitalized patients, especially in ICU, may experience anxiety, paranoia, hallucinations, disorientation, agitation, and even violence against nurses and doctors. The condition has been formally defined as «acute brain syndrome involving impaired intellectual functioning which occurs in ICU patients.» ICU psychosis is a form of delirium or acute brain failure, and some organic factors including dehydration, hypoxia, low cardiac output, infections, and drugs may contribute to its development. The postoperative delirium occurs relatively often in elderly and remains clinically challenging for many physicians, nursing staff, and the patient's family.

Mild form of delirium does not require therapy, especially in elderly, or in case of significant fluid loss (fever, sweating, diarrhea) or high ambient temperatures, making adequate volume resuscitation necessary. In the individual case, especially in violent patients, administration of 2–10 mg haloperidol or clonidine IV (clonidine as an IV drug is not available in the USA) as well as the newer generation of atypical antipsychotic drugs may be considered. Only in extreme situations, endangering the patient and the CSICU staff, the sedation with propofol is recommended.

To further differentiate and quantify psychosis, the Richmond Agitation-Sedation Scale (Sessler et al. 2002), in conjunction with the Confusion Assessment Method, may be employed (Ely et al. 2003, 2004; Sessler et al. 2002). The physical fixation of the patient should only be performed in extreme situations; on the one hand, it is restricted by law and needs clear documentation, and on the other hand the symptoms can be exacerbated by physical fixation.

It is not uncommon that patients with ICU psychosis develop an instable sternum requiring surgical reintervention.

9.5.7.4 Critical Illness Polyneuropathy

The cause of this rare complication is not well understood, allowing for no treatment or prophylaxis. This polyneuropathy affects mainly the motor axons, occurring most commonly after sepsis and polytrauma. A correlation with systemic inflammatory reaction syndrome (SIRS), multi-organ failure, and various drugs is highly suggested. The main problem is to wean the patient from ventilator, as breathing muscles are commonly affected by critical illness polyneuropathy. After repeatedly failed extubation attempts, the critical illness polyneuropathy has to be taken into account. Early mortality is about 30 %, another 50 % of the patients recover between 6 weeks and a year, while 20 % will have residual neuropathies (Kane and Dasta 2002).

9.5.7.5 Other Neurological Complications

Other complications, such as neurocognitive deficits, neuropsychological complications, and peripheral nerve lesions, are not specific to cardiac surgical patients. Peripheral nerve injuries are potentially preventable complications during anesthesia, requiring extra attention while positioning the patient.

9.5.8 Acid-Base Balance and Electrolytes

The laboratory values and the common causes for disorders of the acid-base balance are displayed in ◘ Tables 9.18 and 9.19.

Metabolic acidosis is the most relevant acid-base disorder in the CSICU, as the underlying causes can be potentially fatal disorders. An insufficient oxygen supply, caused by LCOS or mesenteric ischemia, initiates a change from aerobe to anaerobe metabolism, releasing more acids in the bloodstream. Main goal is to treat the underlying cause of the acidosis, as sole treatment of the metabolic acidosis may not be enough. Clinical implications are reduced myocardial contractility and decreased effectiveness of catecholamines, clinically relevant in patients with pH below 7.2. The amount of $NaHCO_3$ to balance the acidosis can be calculated with the following formula:

$$\text{Negative base excess} \times 0.3 \times \text{body weight (kg)} = \text{mval } NaHCO_3\ (8.4\%)$$

Administrating large amounts of $NaHCO_3$ results in iatrogenic hypernatremia. If more bicarbonate buffer is needed in case of a sodium level more than 145 mmol/L, Tris (hydroxymethyl) aminomethane (THAM) may be used:

$$\text{Negative base excess} \times 0.1 \times \text{body weight (kg)} = \text{ml tris buffer solution}$$

A metabolic alkalosis is often the result of an overtreated metabolic acidosis. A metabolic alkalosis can also cause a reduction of myocardial contractility and heart rhythm disturbances and will worsen the oxygen tissue supply by shifting the oxygen dissociation curve.

Electrolyte abnormalities will influence the heart function, especially the potassium level have the most clinical effect. The typical potassium level in the blood serum is 3.5–5 mmol/L. A low level of potassium associated with metabolic alkalosis is accompanied by extrasystoles, tachycardia, and ST interval decrease or a prolonged QT interval and will be treated by parenteral administration of KCl. If hyperkalemia is present, high peaks of the T waves can be seen as well as enlargement of QRS complex, branch blocks, arrhythmias, and cardiac arrest. To lower the serum potassium level, the administration of diuretics, $NaHCO_3$, calcium, or glucose with insulin can be helpful. Diuretics will eliminate the potassium, but the other attempts only provide a potassium shift from extracellular to intracellular. Another effective method to reduce potassium levels is Sodium

◘ **Table 9.18** Laboratory values in disorders of the acid-base balance

Disorder	pH level	CO_2 partial pressure mmHg	Bicarbonate concentration mmol/L	Base excess mmol/L
Normal values	7.36–7.44	35–45	22–26	–2 to + 2
Respiratory acidosis	<7.36	>45	Normal	Normal
Respiratory alkalosis	>7.44	<35	Normal	Normal
Metabolic acidosis	<7.36	Normal	<22	Negative
Metabolic alkalosis	>7.44	Normal	>26	Positive

◘ **Table 9.19** Common causes for disorders of the acid-base balance

Disorder	Cause	Therapy
Respiratory acidosis	Hypoventilation	Increase respiratory rate or tidal volume
Respiratory alkalosis	Hyperventilation	Decrease respiratory rate or tidal volume
Metabolic acidosis	Anaerobe metabolism	Bicarbonate and correct the underlying cause
Metabolic alkalosis	Overcorrected acidosis	Acetazolamide and correct the underlying cause

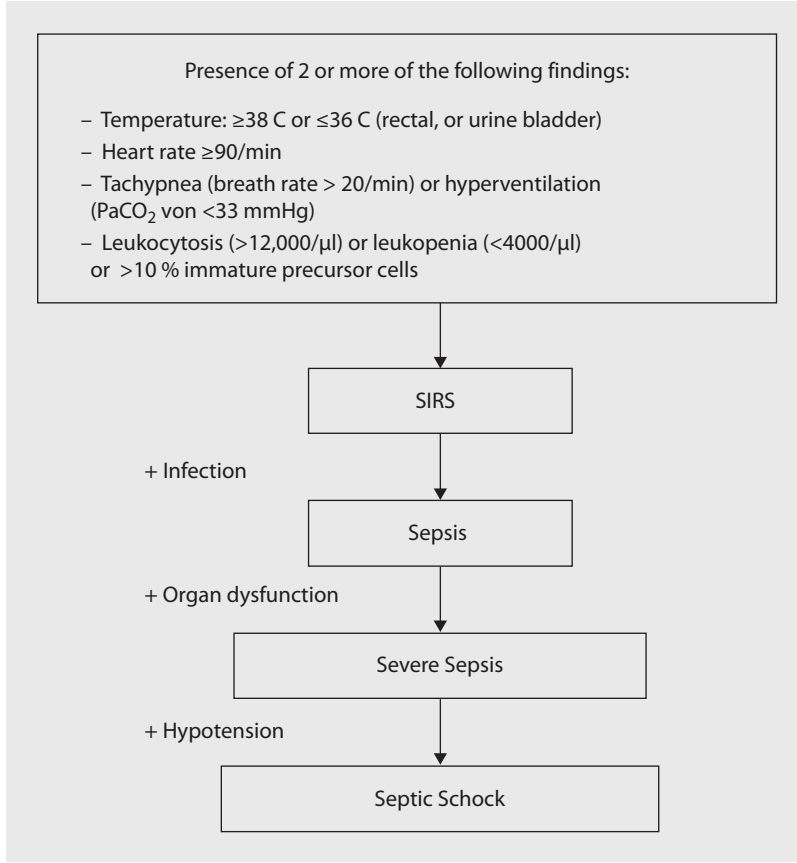


Fig. 9.14 Differential diagnosis and grading of severity of inflammatory reactions without and with infectious cause

Polystyrene Sulfonate Suspension, a cation-exchange resin adminstered orally or as an enema.

If the potassium level exceeds 6 mmol/L or patient is having active symptoms, extracorporeal filtration of potassium using renal replacement therapy may be indicated.

If high levels of sodium are present, the sodium intake has to be stopped immediately to prevent a hyperosmotic coma. All transfusion fluids should be sodium-free, such as glucose 5 %. A hypernatremia due to fluid overload or sodium loss through the kidney or the gut (diarrhea) can be treated by administration of sodium-containing fluids.

9.5.9 Fever and Infection

The rise of the body temperature after cardiac surgical operations is not uncommon and does not require a treatment in the absence of any signs of infection. This is especially true in the first few days after index operation, commonly associated with body's systemic inflammatory response syndrome or early postoperative atelectasis. The temperature course should be followed and does not require normalization if patient is not tachycardic or otherwise symptomatic. Exceptions to this rule are operations for infective endocarditis, or sternal wound infections. The challenge for the intensivist is to differentiate between physiological fever and brewing infection. Figure 9.14 depicts and describes sequence and grading of differential diagnosis in inflammatory processes.

Infection is a clinical diagnosis or positive result in microbiology examination.

Organ dysfunction include:

- Acute encephalopathy with impaired vigilance, disorientation, restlessness, delirium
- Relative or absolute thrombocytopenia with a platelet drop >30 % within 24 h or a platelet count <100,000/µl in the absence of acute bleeding
- Arterial hypoxemia with paO_2 <75 mmHg in room air, Horovitz quotient <250 (see ► Sect. 9.5.4.3, p. 229)

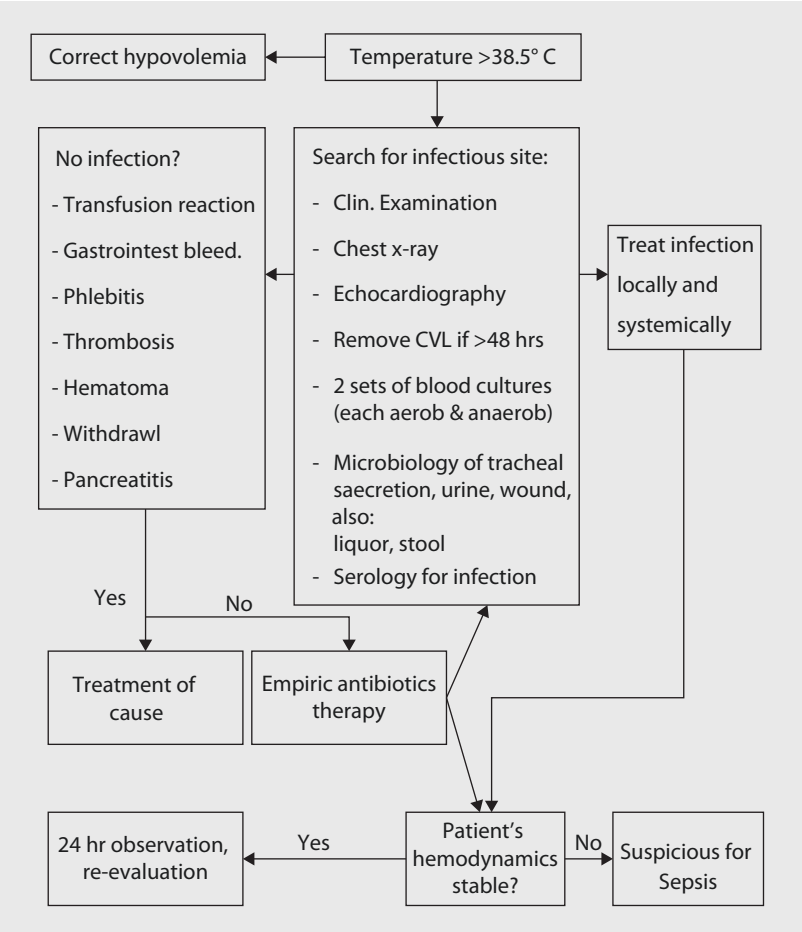


Fig. 9.15 Our algorithm for diagnosis and therapy of postoperative fever. *CXR* chest X-ray examination, *CVL* central venous line

- Renal dysfunction, diuresis <0.5 cc/kg/h for more than 2 h despite adequate volume load or serum creatinine climbed to twice normal
- Metabolic acidosis, negative base excess exceeds −5 mmol/L or lactate increased more than 1.5 normal
- Hypotension with systolic arterial pressure <90 mmHg or mean arterial pressure <70 mmHg for at least 1 h despite volume replacement and IV vasopressor therapy

■■ Practical steps

Fever is defined as a body temperature with more than 100 °F (in Europe 38.3 °C = 100.9 °F). Initially hypovolemia should be corrected that may cause fever by itself. If the temperature is not dropping or another fever is coming up, a high index of suspicion for brewing infection should be maintained. The next step is the search for the infection focus.

Since postoperative cardiac surgical infections are associated with significantly increased mortality and cost, any signs of infection have to be evaluated carefully and promptly. If an infection is present, an appropriate and immediate treatment is recommended. This is especially true for patients with high pretest probability of infection such as immune suppressed, HIV-infected, or poorly controlled diabetic and obese patients

The search for infection after cardiac surgery should focus on:

- Pneumonia
- Urinary catheter infections
- Central vein catheter and peripheral line infections
- Surgical site infections

The first three types of infection occur usually after the fifth postoperative day, whereas surgical site infections appear mostly after more than

1–2 weeks depending on involved pathogen. These wound infections should be treated according to general surgical principles and accompanied by systemic antibiotics.

If bedside investigations, such as chest X-ray, urine, and respiratory cultures, remain negative for an infection, an antibiotic therapy should be started even without knowing the infectious organism. The typical organism described in the literature may be helpful, but it is more useful to know the hospital common organisms and their resistances. Furthermore, the initial antibiotic should be wide spectrum, reducing to more narrow antibiotics once sensitivities are back. If an infection focus is identified, removal of the focus with or without surgical excision should be performed immediately.

Figure 9.15 illustrates our algorithm to evaluate postoperative fever.

If the infection progresses to a full-blown sepsis, the prognosis will be significantly worse. These four strategies have shown to improve severe sepsis or septic shock outcomes:

- Early support for normalized hemodynamic function (Rivers et al. 2001)
- Intensified insulin therapy (van den Berghe et al. 2001)
- Administration of human recombinant activated protein C (APC) (Bernard et al. 2001).
- Widening of antibiotic therapy including antifungal or antiviral in specific patient cohort

In the meantime, target levels for tight glycemic control have been changed (Haga et al. 2011). Activated protein C (APC) has been removed from the market due to questionable benefit (Marti-Carvajal et al. 2012). This topic is rather comprehensive and complex and cannot be sufficiently handled in this chapter. Therefore, further detailed reading is suggested (Dellinger et al. 2013).

9.5.10 Decubitus

A decubitus is tissue damage caused by prolonged compression (Maklebust 2005; Thompson 2005). In clinical practice, decubitus is the short form for the historical term decubitus ulcer. As the word decubitus in Latin means lying down, although these damages can be caused in other positions also, the more general term to describe these tissue damages would be pressure sore. This compression of the tissue reduces the blood supply, and the ensuing hypoxia may damage the nerves in this area within a few hours. If the patient cannot change the position, a permanent skin damage will occur.

Besides the hypoxia, compromised venous drainage is also important in accumulation of acid metabolism products in the skin. Furthermore, the acidic environment initiates vasodilatation with hyperemia, with subsequent exudate and transudate with possible tissue edema and blisters, and microthrombotic vascular changes. This acid environment stimulates usually the ambulatory human to move a little bit, preventing thereby any tissue damage in healthy individuals.

Frequently the decubitus is infected, prolonging the healing process. Body parts without a proper muscle or fat tissue are most vulnerable to decubitus.

The decubitus can be categorized in different depths and sizes and is commonly classified as follows:

Stage I is the most superficial and recoverable, characterized by nonblanchable redness that does not subside after finger pressure is relieved. This stage is visually similar to reactive hyperemia seen in the skin after prolonged application of pressure. Stage I pressure ulcers can be distinguished from reactive hyperemia in two ways: (a) reactive hyperemia resolves itself within three-fourth of the time pressure was applied and (b) reactive hyperemia blanches when pressure is applied, whereas a Stage I pressure ulcer does not. The skin may be hotter or cooler than normal, have an odd texture, or be painful. Although easy to identify on a light-skinned patient, ulcers on darker-skinned individuals may show up as shades of purple or blue in comparison to lighter skin tone.

Stage II is damage to the epidermis extending into, but no deeper than, the dermis. In this stage, the ulcer may be referred to as a blister or abrasion that can be infected.

Stage III involves the full-thickness damage of the skin and may extend into the subcutaneous tissue layer. This layer has a relatively poor blood supply and can be more difficult to heal. At this

stage, there may be undermining tissue damage that makes the decubitus much larger than it may seem on the skin level.

Stage IV is the deepest, extending into the muscle, tendon, or even bone.

The decubitus prophylaxis is the best therapy. The treatment of a fulminant decubitus is very complex and requires plenty of resources. For prophylaxis, the following aspects are very important:

- Tissue-protective patient exercise, positioning, and transfer
- Routine use of softgel or air-filled mattresses
- Continuous performance of prophylactic maneuvers

Local therapy of decubitus contains:

- Debridement
- Treatment of infection
- Moist wound treatment
- Wound conditioning
- Hyperbaric oxygen therapy and VAC wound treatment

The causal wound therapy is:

- Complete decompression of the damaged tissue
- Improvement of nutrition
- Adequate pain therapy
- Improving the general functional condition

Stage II decubitus ulcer or higher is a sign of poor nursing and patient care. It should be investigated if there was omission or human error associated with it. The incidence for decubitus on any ICU should be less than 1 % per year.

9.5.11 Disturbances of the Coagulation System

Most common problems with coagulation system after cardiac surgery have been discussed (paragraph ► Sect. 9.5.3). In this section, heparin-induced thrombocytopenia (HIT) will be briefly described (Napolitano et al. 2006; Selleng et al. 2007). The incidence of HIT complications is 1–2 % after cardiac surgery, being associated with an increased mortality (Kerendi et al. 2007).

HIT is a prothrombotic disease triggered by an immune reaction against the platelet factor 4 heparin complex. The clinical signs are drop of the platelet count more than 50 % and clinical thrombosis. Actual HIT tests are sensitive but less specific. Nearly half of cardiac surgery patients develop antibodies to heparin, whereas only 1–2 % will have a clinical relevant HIT. Functional HIT tests, such as serotonin-released assays, are more accurate by measuring the platelet activation caused by heparin antibodies.

The prompt therapy is to stop any exposure to heparin and to change the anticoagulation to alternatives, such as lepirudin. Additionally, platelet infusions may have to be administered to remove the existing antibodies. ◘ Figure 9.16 summarizes the approach to HIT.

9.5.12 Endocrine System

After intensified insulin therapy to achieve blood glucose levels less than 110 mg/dl had shown to decrease mortality and the incidence of postoperative infections, intensified insulin therapy was on protocols of most CSICUs (van den Berghe et al. 2001). Meanwhile, it could be demonstrated that moderate glycemic control with blood glucose levels less than 180 mg/dl is more advantageous in patients after cardiac surgery (Haga et al. 2011; Bhamidipati et al. 2011). With both protocols, the incidence of hypoglycemia is a concern, being increased up to sixfold. Furthermore, low potassium level can occur, contributing to dysrhythmias. A close monitoring of blood glucose level and potassium should be ensured through close monitoring. In critically sick patients, especially those on high-dose catecholamines, adequate blood glucose levels may be much harder to achieve.

T3 levels in patients after open-heart surgery are reduced. However, T3 supplement has failed to demonstrate any improved outcome (Ronald and Dunning 2006). Same principle applies to postoperative cortisol levels, making supplemental steroid administration useless and possibly deleterious due to effects on postoperative wound healing. In patients requiring perioperative steroids, such as adrenal insufficiency, vitamin A administration may counteract steroid effects on wound healing.

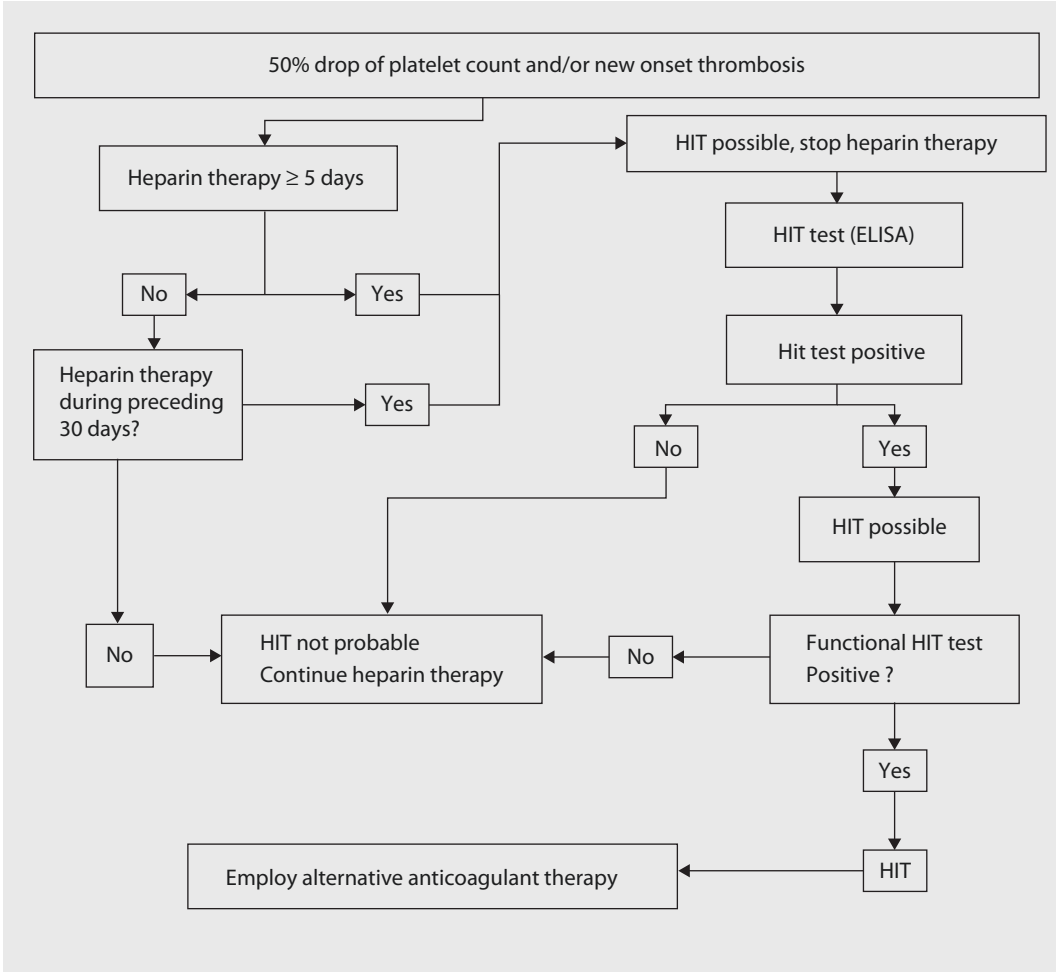


Fig. 9.16 Diagnostic algorithm for heparin-induced thrombocytopenia (HIT). *ELISA* enzyme-linked immunosorbent assay (Modified according to Selleng et al. (2007))

9.6 Scores and Quality Assurance

Both topics are subject for numerous publications of all kind. Their significance for clinical intensive care treatment in cardiac surgery has yet to be shown. There are, however, in various versions a vast number of intensive care medicine *scores* available (see also ► Chap. 2, «Risk Scores in Cardiac Surgery»), which all have been developed for different purposes, for example, APACHE (*A*cute *P*hysiology *a*nd *C*hronic *H*ealth *E*valuation), SAPS (*S*implified *A*cute *P*hysiology *S*core), or SOFA (*S*equential *O*rgan *F*ailure *A*ssessment). To assess the economic aspects of ICU treatment, scores like TISS 28 (*T*herapeutic *I*ntervention *S*coring *S*ystem 28) have been developed, also. Further scores are available in the internet (Société Française d'Anesthésie et de Réanimation 2008). In Germany, currently a combination of a modified SAPS II score and TISS 28 score is employed to evaluate the expenditure of an individual treatment course to be used for reimbursement—an example of how a potentially useful clinical instrument is used for purposes other than originally intended.

Quality assurance in intensive care medicine currently is only done on a voluntary basis. However, any intensive care unit should check its own quality of treatment at least by a minimal data set. An example for some parameters is given in ◘ Table 9.20.

Table 9.20 General guidelines for quality improvement in ICU with goals and thresholds, which should not be surpassed

Parameter	Goal	Threshold
% of patients requiring early readmission to ICU	<3	5
% of patients with de novo decubitus (stage II and higher)	<1	2
% post-op extubation within 6 h	>75	50
% reintubation	<3	5

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