3 Cardiac Patients and Noncardiac Surgery: Pathophysiological Basis for Clinical Management

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3.1 Ischemic Heart Disease

In developed countries about 5–10% of patients eligible for surgical procedures have some degree of ischemic heart disease. They have an increased risk of perioperative acute myocardial infarction (AMI) and 30% of hospital mortality [1].

Usually it is a consequence of a "silent" myocardial ischemia (ischemia without typical symptoms of angina).

The strong association between postoperative silent ischemia and other cardiac adverse events defines the crucial role of anesthesia techniques used to minimize ischemia onset.

Ischemic heart disease is characterized by atherosclerosis plaques of the coronary arteries, resulting in narrowing of vessels and decreased coronary blood flow. Imbalance between myocardial oxygen supply and demand may occur during exercise, leading to precordial pain due to stress-related stable angina.

Unstable angina (pain at rest), silent ischemia, and myocardial infarction are due to plaque rupture, ulceration, or erosion with thrombus and coronary spasm [2].

Several factors in the perioperative period can result in perioperative myocardial infarction (PMI):

- High level of circulating epinephrine and other catecholamine after surgery, with tachycardia, coronary constriction, and increased platelet viscosity [3]
- High blood coagulation with increased risk of coronary thrombosis [1]

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D. Chiumello (ed.), *Practical Trends in Anesthesia and Intensive Care 2018*,
https://doi.org/10.1007/978-3-319-94189-9_3

The risk of perioperative complications depends on predictive factors (patient's condition before surgery, comorbidities, etc.), functional capacity, and surgical risk [4].

Recent myocardial infarction, unstable angina, untreated heart failure, severe arrhythmias, and severe valvular disease are predictors of high perioperative risk.

Intermediate-risk values are stable angina, history of AMI, heart failure under treatment, and diabetes. Low-risk predictors are old age, ECG abnormalities, cerebral ischemic stroke, and uncontrolled hypertension.

Determination of functional capacity is a pivotal step in preoperative cardiac risk assessment, and it should be performed with cardiopulmonary test. Without testing, functional capacity is estimated in metabolic equivalents (METs). One MET is metabolic demand at rest. High-risk patients have poor functional capacity (e.g., inability to climb two flights of stairs or run a short distance, <4 METs) and increased incidence of postoperative cardiac events [4].

The Lee index score [5] predicts the risk of postoperative myocardial infarction and mortality, according to six variables (type of surgery, history of IHD, history of heart failure, history of cerebrovascular disease, preoperative treatment with insulin, and preoperative creatinine >2 mg/dL).

Surgical factors that influence cardiac risk are related to the urgency, invasiveness, type, and duration of the procedure. With regard to cardiac risk, surgical interventions (open or endovascular procedures) can be broadly divided into low-risk, intermediate-risk, and high-risk groups, with estimated 30-day cardiac event rates (cardiac death and myocardial infarction) of <1%, 1–5%, and >5%, respectively.

3.1.1 Anesthetic Management

Anesthetic challenge is to guarantee an adequate balance between oxygen delivery and demand to myocardial tissue. Increased heart rate, afterload, or preload with a consequent increasing in cardiac work is associated with high myocardial oxygen consumption, and it must be avoided [6] (Table 3.1). Several factors can reduce oxygen delivery, low hematocrit value, preoperative anemia, low oxygen saturation, coronary thrombosis, and vasoconstriction. Every surgical operation elicits a stress

Table 3.1 Factors determining the oxygen supply/demand

Oxygen supply	Oxygen demand
Heart rate – *Diastolic period*	Heart rate
Coronary perfusion pressure – *Arterial diastolic aortic pressure* – *End-diastolic ventricular pressure*	Transmural ventricular pressure – *Preload* – *Afterload*
Arterial oxygen content – *Partial arterial pressure of oxygen* – *Hemoglobin concentration*	Contractility

response and may cause an oxygen mismatch, with myocardial ischemia, even without severe coronary lesions. Fluid shift, acute anemia, changes in preload and afterload and heart rate, and activation of neuroendocrine and inflammatory systems caused by surgical stress increase oxygen consumption [6]. In this setting it's crucial to gain an adequate level of anesthesia, avoiding hemodynamic instability or low systemic arterial pressure (which will lead to low coronary perfusion pressure). Anyway, the majority of ischemic events during surgical operation are not related to hemodynamic variations. Other causes are vasoconstriction and coronary thrombosis.

In high-risk patients, intra- and postoperative hemodynamic monitoring is necessary, with increasing level of invasiveness (transesophageal echocardiography (TEE) or Swan-Ganz catheter) [7].

There is conflicting evidence, over whether a specific anesthetic technique is superior to another one, in reducing perioperative mortality in patients with cardiac disease.

Clinical trials comparing outcomes among regional and general anesthetic techniques have shown some evidence of improved outcome and reduced postoperative morbidity with regional anesthesia. Benefits of regional anesthesia are best neuroendocrine stress response control, reduced incidence of postoperative thromboembolic events, and cardiac (coronary thrombosis) and pulmonary complications (respiratory failure).

Other clinical trials have shown that neuroaxial and regional techniques reduce sympathetic tone, leading to reduction in afterload (reduced myocardial oxygen consumption) and in venous return due to increased compliance of the venous system, vasodilatation, and finally decrease in blood pressure (reduced coronary perfusion). So in patients with ischemic heart disease and good left ventricular function, performing regional or neuroaxial anesthesia may have benefic effects [6].

Patients with left ventricular dysfunction may not tolerate hemodynamic changes due to sympathetic blockade, and they may be advantaged by general anesthesia with selected drugs (less impact with cardiac function). Balanced anesthesia with opioids seems to be the best choice in these patients. Volatile agents used for general anesthesia have myocardial protective effects, and they may prevent perioperative myocardial infarction.

According to guidelines, perioperative continuation of beta-blockers is recommended, and preoperative initiation of beta-blockers may be considered in selected patients [4].

3.2 Valvular Heart Disease

Patients with valvular heart disease (VHD) are increasing in number, considering the aging population with more than 13% of mild/severe valvulopathies in elderly [8].

Hemodynamic instability is common in these patients, and hemodynamic management is crucial and really complex (Table 3.2).

Table 3.2 Management of hemodynamics in valvulopathies

	MVS	MVI	AVS	AVR
Preload	↑↓	↑	↑↓	↑
Afterload	=	↓	=	↓
HR	60–70 bpm	90–100 bpm	60–70 bpm	90 bpm
SVR	Keep	Reduce	Keep	Reduce
PVR	Reduce	Reduce	=	=
Rhythm	Sinus, absolutely	AF tolerated	Sinus, absolutely	AF tolerated
Inotropes	Inodilators	Inodilators	Norepinephrine	Inodilators

MVS mitral valve stenosis, *MVI* mitral valve insufficiency, *AVS* aortic valve stenosis, *AVR* aortic valve insufficiency, *HR* heart rate, *SVR* systemic vascular resistance, *PVR* pulmonary vascular resistance

Beyond general indications to guarantee a hemodynamic stability, it is essential to understand the pathophysiology of every single valvular disease and to consider that different valvular dysfunction may be present in the same patient.

Echocardiography should be performed on any patient with known or suspected VHD scheduled for noncardiac surgery, in order to assess the severity of the valvular defect and its hemodynamic consequences, during preoperative evaluation.

3.2.1 Aortic Valve Stenosis

Aortic valve stenosis (AVS) is the most common VHD in Europe. Acquired aortic stenosis is due to an idiopathic calcific degeneration of the aortic valve. The major risk factors are atherosclerosis complicated by inflammatory phenomena. The incidence of AVS increases with age, due to mechanical stress factor and other comorbidities like hypertension, diabetes, smoke, and hypercholesterolemia.

The incidence is 2–4% in the older population (>65 age) but is probably underestimated [9].

Bicuspid aortic valve is the most common congenital cardiac abnormality, affecting approximately 1–2% of the general population. Patients are asymptomatic till the valve opening guarantees an adequate blood flow; subsequently the continuous and repeated abnormal opening and closing of the aortic cusps leads to valve degeneration and leaflets calcification. So elderly patients may present with valve stenosis and or insufficiency (aortic valve regurgitation or AVR) [10].

In developing countries especially in the last decades, as a consequence of migration phenomenon, also in Europe, there are new cases of rheumatic valve degeneration which cause AVS and AVR.

Severe aortic stenosis constitutes a well-established risk factor for sudden irreversible death despite cardiopulmonary resuscitation.

The suspicion of aortic stenosis from symptoms and physical examination, has to be confirmed by echocardiography evaluation.

Aortic valve stenosis signs and symptoms generally develop when narrowing of the valve is severe. Some patients with aortic valve stenosis may not experience

symptoms for many years. Signs and symptoms of aortic valve stenosis may include heart murmur (systolic murmur extended to the neck vessels), angina, and tightness or shortness of breath, especially during activity. The heart-weakening effects of aortic valve stenosis may lead to heart failure and syncope. Echocardiography evaluation is crucial for diagnosis and stenosis quantification. It allows to measure the valve area (normal value 3–4 cm^2) [11]. Severe aortic stenosis is defined according to an integrative approach taking into account valve area (<1.0 cm^2 or 0.6 cm^2/m^2 body surface area, except in obese patients) and flow-dependent indices (maximum jet velocity 4 m/s and mean aortic pressure gradient ≥40 mmHg). Pathophysiology of aortic stenosis is characterized by left systolic ventricular flow obstruction with increased wall stress, which chronically leads to pressure overload and concentric ventricular hypertrophy. Left ventricle produces a high-pressure peak for the increased transvalvular gradient, with a reduction in wall stress thanks to the myocardial concentric hypertrophy. Naturally it leads to a diastolic dysfunction with a consequent increasing in ventricular end-diastolic pressure, which is the preamble for a subendocardial ischemia. Reduction in ejection fraction is due to reduction in myocardial contractility. Myocardial dysfunction and loss of myocardial contractility determine a reduction of ejection fraction, which implies a complex estimation of the severity of aortic stenosis (reduction in valve flow gradient named "low-gradient aortic stenosis").

3.2.1.1 Anesthetic Management

Complications after noncardiac surgery depend on patient-related risk factors (degree of stenosis, ischemic heart failure, etc.), on the type of surgery, and on the circumstances under which it takes place. Moreover, cardiopulmonary resuscitation may not provide an adequate cardiac output in patients with severe aortic stenosis. Aortic stenosis implies for anesthetists a complex management and an accurate patient monitoring to avoid hemodynamic instability or variation in volume status, heart rate, and vascular tone, despite the anesthetic technique [12].

For clinical assessment it is crucial to avoid arrhythmias and to guarantee heart sinus rhythm. In patients with diastolic dysfunction, atrial stroke is essential in maintaining cardiac output. In fact atrial fibrillation leads to the loss of atrial systole with consequent severe hypotension (low cardiac output). In this situation with low cardiac output and hemodynamic instability, clinicians have to quickly perform an electrical cardioversion, also in the preoperative time, to get a hemodynamic stability.

Heart rate variations have to be avoided, also in sinus rhythm. Bradycardia can cause collapse in cardiac output (loss of stroke volume). On the other hand, tachycardia can lead to an increased cardiac stress and work, especially in a hypertrophic heart (as a consequence of stenosis) where discrepancies between oxygen delivery and demand may worsen coronary blood flow. Optimization of fluid load is mandatory. Preload must be increased because of the diastolic dysfunction [13].

Peripheral vascular tone and afterload have to be high, avoiding hypotension and vasodilatation. In case of low arterial pressure and low coronary perfusion pressure,

vasopressors as norepinephrine may help in maintaining vascular tone and cardiac contractility.

Anesthetic management is aimed at ensuring the best compromise between depth of sedation or type of locoregional blockade (avoiding bradycardia or tachycardia) and the management of vascular tone and fluid load to avoid vasoplegia and reduction in preload.

Neuroaxial anesthesia may be contraindicated unless constant maintenance of an adequate afterload is performed [13].

In this setting hemodynamic monitoring is mandatory: heart rate, ECG, oxygen saturation, and invasive blood pressure. In high-risk surgery, continuous central venous pressure (CVP) monitoring or semi-invasive/invasive cardiac output monitoring system may help in the volemic status and cardiac assessment. Intraoperative echocardiography [11], when available, is the most comprehensive monitoring system, which allows to evaluate cardiac contractility and output, heart filling volume, diastolic and systolic function, and volemic status.

In symptomatic patients, aortic valve replacement should be considered before elective surgery. In patients at high risk or with contraindication for aortic valve replacement, balloon aortic valvuloplasty, or, preferably, transcatheter aortic valve implantation (TAVI) may be a reasonable therapeutic option before surgery. Neuroaxial anesthesia (spinal or epidural anesthesia) can be tolerated in the moderate aortic stenosis (especially in asymptomatic patients), but it's not recommended in severe AS, because of the reduction in preload and afterload due to sympathectomy which could lead to a severe hypotension and reduction in coronary perfusion.

3.2.2 Aortic Valve Regurgitation

Aortic valve regurgitation (AVR) depends on an incomplete closure of valve cusps with a regurgitation of an amount of blood in the left ventricle during diastole [12].

Aortic regurgitation can be divided into severe and acute forms due to endocarditis or aortic dissection and chronic regurgitation ones, with a better prognosis. In this last form, regurgitation is due to incompetence of the aortic valve or any defect of the valvular apparatus (leaflets, annulus) for congenital causes, connective degeneration, inflammatory or rheumatic diseases, and annular dilatation (due to aging and hypertension). Coaptation defect allows blood regurgitation to increase the left ventricular end-diastolic pressure [14]. Chronic volume overload leads to a ventricular remodeling with eccentric hypertrophy and dilatation. For this reason, patients may be asymptomatic for many years, till severe regurgitation causes heart failure with fatigue, dyspnea, orthopnea, and nocturnal paroxysms. Reduced diastolic pressure causes angina also without coronary lesions. Stroke volume is increased from low peripheral vascular resistances, so EF% is normal. With the worsening of the ventricular dilatation and the ventricular remodeling, cardiac output fails, with hypotension, increased sympathetic tone, and severity of regurgitation [14].

Echocardiography is the key examination in the diagnosis and quantification of AVR severity, using color Doppler and pulsed-wave Doppler. Echocardiography is also important to evaluate regurgitation mechanisms, describe valve anatomy, and determine the feasibility of valve repair [15].

AR may be mild (regurgitation volume <20%), moderate (volume 20–39%), and severe (volume 40–60%). Entity of regurgitation is due to diastole duration too.

3.2.2.1 Anesthetic Management

In the anesthetic management, the aim is to gain a heart rate of about 90 bpm to shorten the diastolic time and the consequent regurgitation fraction [6]. Mild tachycardia in fact allows to increase diastolic arterial pressure and to reduce end-diastolic left ventricle pressure, with optimization of myocardial perfusion. Drugs reducing heart rate have to be avoided. Sinus rhythm is desirable, but unlike the aortic stenosis, tachyarrhythmia is well tolerated. Preload has to be increased and adequate to the dilated ventricular chamber; reduction in afterload is crucial to allow the antegrade flow through the valve and to avoid regurgitation [6]. Myocardial contractility must be preserved, optimizing the vasodilatation and avoiding the reduction in preload.

Vasoconstrictor drugs increase the afterload with consequent worsening in valve regurgitation and ventricular dilatation. In case of the necessity of inotropic drugs, it's better to choose an inodilator as levosimendan or milrinone, instead of dobutamine. Locoregional anesthesia (subarachnoid or epidural) can certainly represent technique of choice, provided that adequate intravascular volume is maintained.

3.2.3 Mitral Valve Stenosis

Rheumatic fever is the predominant etiology of mitral valve stenosis (MVS), and it has greatly decreased in Europe in the last decades; nevertheless, migration has led to new rheumatic disease cases.

Mitral valve area is about 4–5 cm^2. Moderate reduction in area (<2.5 cm^2) causes symptoms as dyspnea getting worse with exercise, anemia, pregnancy, and fever (need to increase the cardiac output) [16].

The severity of the mitral stenosis is defined by the valve area: mild (valve area 2.5–1.5 cm^2), moderate (1.5–1 cm^2), and severe (<1.0 cm^2).

The diagnosis is usually established by echocardiography and cardiac catheterization. Valve area should be measured using planimetry and the pressure half-time method, which are complementary. Echocardiography also evaluates pulmonary artery pressures, concomitant valve disease, and left atrium (LA) size. In fact the obstruction of the mitral flow determines an increase in left atrial pressure with enlargement of the atrium, risk of atrial fibrillation, and auricular thrombus.

In case of atrial fibrillation, patients need anticoagulation therapy with vitamin K antagonists (VKAs) or non-VKA direct oral anticoagulants (NOACs) or heparin

and rhythm control medications or cardioversion (in rapid-onset fibrillation and hemodynamic instability). Transesophageal echocardiography (TEE) should be performed to exclude LA auricular thrombus before cardioversion or after an embolic episode.

High atrial pressure causes increase in pulmonary pressure with vessels remodeling and postcapillary pulmonary hypertension, consequent enlargement, and hypertrophy of the right ventricle [17].

Slowing in diastolic ventricular filling determines low pressure and low end-diastolic ventricular volume (with reduction in stroke volume). Stroke volume is reduced, especially in case of increased heart rate. Contractility can be maintained, but it is often depressed due to structural alterations of the ventricle and the subvalvular apparatus with a reduction in the compliance of the left ventricle and subsequent diastolic dysfunction. The septal shift for enlargement of the right ventricle also contributes to diastolic dysfunction.

3.2.3.1 Anesthetic Management

The pathophysiology of the SVM involves the control of cardiac rhythm, preload, left ventricular contractile function, and pulmonary hypertension. It is essential to avoid tachycardia (tachyarrhythmia in many cases) because, reducing the diastolic time irreversibly, it compromises ventricular filling. The acute increase in the transvalvular gradient should also be avoided, avoiding the increase of sympathetic tone [5, 6]. Where possible sinus rhythm should be preserved and maintained, favoring the atrial contribution to the determinism of cardiac output. In the case of atrial fibrillation, rhythm control (digital, beta-blockers, even short-acting amiodarone) must be absolutely guaranteed.

An adequate preload must be guaranteed to maintain the trans-stenotic flow; therefore, it must pay attention to the vasodilatation due to anesthetics. However, optimizing the preload involves the risk of further increase in left atrial pressure and the development of pulmonary edema. The excessive reduction of the afterload is not particularly advantageous anyway. Despite normal peripheral vascular resistances and contractility in most patients, it is possible that the empty/unloaded left ventricle develops systolic and diastolic dysfunction. In these patients, it is crucial to monitor pulmonary hypertension, hypoxemia, acidosis, hypercapnia, and the use of nitrous oxide must be avoided [17]. It is also necessary to predict the increase in bleeding linked to anticoagulation. Avoid excessive premedication that compromise ventilation: even mild hypercapnia precipitates hemodynamic stability. Patient with SVM requires monitoring of invasive blood pressure, CVP, and intraoperative echocardiographic monitoring. In high-risk surgeries, monitoring of cardiac output and pulmonary arterial pressure with the Swan-Ganz catheter is recommended, given the simultaneous presence of right ventricular dysfunction. It is essential to avoid hypoxemia and hypercapnia, to maintain optimal acid-base balance, and to reduce pulmonary hypertension with vasodilators, inodilators, and nitric oxide. Locoregional techniques can be poorly tolerated for their effects on systemic vascular resistance; epidural anesthesia is preferable to the subarachnoid one for the most gradual appearances of the sympathetic block.

3.2.4 Mitral Valve Insufficiency

Mitral valve insufficiency (MVI) is a very common valvular disease. It is determined both by changes in the valve and in the subvalvular apparatus including the anomalous remodeling of the left ventricle which leads to pathological valvular coaptation. Mitral valve abnormalities include prolapse, myxomatosis degeneration, rheumatism insufficiency, mitral cleft, and infiltrative/degenerative processes. Functional MVI accounts for about 10–20% of patients with ishemic heart disease [18]. In these patients, morphology of the mitral valve is normal. In conclusion, there are two categories of patients with MVI: those with myxomatous valve degeneration (cordial rupture, cord lengthening, prolapse, flail) and those with ischemic MI (from myocardial ischemia). Valvular regurgitation depends on the regurgitation orifice, the ventricle-atrial pressure gradient, and the duration of the systolic time [18]. Symptoms of chronic MVI range from fatigue and palpitations to heart failure. Echocardiography is essential for the diagnosis and stratification of the degree of mitral insufficiency, but diagnosis, as well as clinical presentation, can also be performed by cardiac catheterization. MVI is defined by the percentage of regurgitation compared to stroke volume as mild (<30%), moderate (30–39%), and severe (40–60%) [8]. The Doppler mode on pulmonary veins helps in quantifying the severity of the MVI. Systolic regurgitation increases pressure and volume of the left atrium, but increase of the pressure is gradual and limited in relation to chronic dilation of the left atrium. Left atrium enlargement determines atrial fibrillation that further alters atrium compliance and leads to pulmonary hypertension [19]. As with MVS, a slower pressure balance is achieved in the small circle with pulmonary hypertension and consequent right ventricular dysfunction. The pathophysiology of MVI is linked to the pressure and volume increase of the atrium and left ventricle. This causes a chronic dilatation of the left ventricle with mild eccentric hypertrophy without increase in ventricular thickness. Stroke volume is guaranteed for a long time, thanks to the reduction of afterload determined by the MVI. Stroke volume begins to decrease as a consequence of the degree of regurgitation which increases the dilatation of the left ventricle itself. The percentage of ejection fraction (EF%) may also be normal considering the amount of regurgitation, but the reduction of EF% significantly compromises hemodynamic status and it requires cardiac surgery.

In patient undergoing high-risk surgery, in selected cases, percutaneous palliation procedure (MitraClip) can be used to reduce the degree of MVI.

Acute MVI from myocardial infarction (rupture of chordae or papillary muscle) or from endocarditis determines a condition of hemodynamics emergency due to the impossibility of atrial adaptation and acute development of pulmonary edema. Usually these patients can be in cardiogenic shock requiring treatment with inotropes, NIV, IABP, and often urgent cardiac surgery.

3.2.4.1 Anesthetic Management

Management of patients with MVI requires maintenance of anterograde stroke volume, with a heart rate between 80 and 100 bpm, which reduces the time of systole

and the amount of regurgitation. Bradycardia can be deleterious because it increases the duration of systole and therefore the regurgitation but also the diastolic time and therefore the overload of the left ventricle. Sinus rhythm should be maintained, but atrial fibrillation is tolerated [3]. Less tolerated are the alterations in preload and afterload due to the effect of anesthesia that can lead to an aggravation of the MVI. As an indication, the slight reduction of the afterload associated with an increase of the preload determines an optimization of the hemodynamics. So an adequate anesthesia depth, a moderate vasodilation, even with inodilators, determines an increase in cardiac output. It is fundamental to avoid hypertension and hypertensive crises which may get MVI worsen [6]. Usually cardiac contractility is maintained, but contractility does not correlate with FE% for the regurgitation quota. In case of hypotension, heart rate (increased) and preload should be improved, absolutely avoiding vasoconstrictor drugs. In case of low EF% and reduced cardiac output, inodilators and inotropes such as dobutamine should be used. Increases in pulmonary arterial pressure should also be avoided. Locoregional techniques are well tolerated, if heart rate control is always monitored.

3.2.5 Antibiotic Prophylaxis of Bacterial Endocarditis

Recommendation for antibiotic prophylaxis in patients with valvular heart disease is complex. Recent guidelines classify different conditions of heart disease by referring to the probability of contracting endocarditis as a secondary effect in high-, moderate-, and low-risk patient groups. Subjects with congenital heart disease, acquired valvulopathies, hypertrophic cardiomyopathy, and mitral prolapse with a regurgitation murmur are included in the moderate-risk group, and they require perioperative antibiotic therapy based on the type, location, and severity of the surgical intervention. In general, antibiotic prophylaxis against *Streptococcus viridans* is necessary for dental, oral, and respiratory tract surgery, while antibiotic prophylaxis toward *Enterococcus faecalis* is indicated for genitourinary and gastrointestinal surgery. Adequate antibiotic therapy must be started before the beginning of surgery.

3.3 Heart Failure

Heart failure is the inability of the heart to pump enough blood to satisfy tissue requests. It occurs in a high percentage of the population, with an increase of 10% in the group over 75 years old, and it is associated with an increase in mortality after anesthesia. Ischemic heart disease is the most common cause. Other causes include hypertension, valvular heart disease, and cardiomyopathies. One third of surgical patients with an ejection fraction of less than 30% die within a year [7].

Cardiac output is lower in heart failure because the systolic output decreases for the same end-diastolic volume of the left ventricle compared to a normal heart.

Because of the decompensated heart has a limited ability to increase the systolic volume, the only response to increased preload is an increase in heart rate, which can cause ischemia. Furthermore, high end-diastolic ventricular pressure hinders blood flow to the endocardium.

In decompensated heart, cardiac output is reduced, and it collapses if the ventricular end-diastolic volume rises to high levels, as in the overload status of heart failure [6].

The goal is to evaluate the severity of the disease and the myocardial contractility. Limited exercise tolerance, orthopnea, and paroxysmal nocturnal dyspnea are indicators of severity. Pharmacological treatments may include ACE inhibitors, diuretics, and nitrates. In some patients with mild or moderate heart failure, cardioselective beta-blockers can be used in an attempt to control heart rate, but the risk is that they can depress sympathetic nerve activity that guarantees myocardial contractility in the decompensated heart. Useful instrumental investigations are ECG (to search ischemia sign), chest radiography, and especially echocardiogram to evaluate the ejection fraction. EF is the percentage of the end-diastolic blood volume ejected from the left ventricle during systole, and values below 30% are index of severe heart failure.

3.3.1 Anesthetic Management

"Safe" anesthesia for patient with heart failure does not exist, but in any case, an optimal conduct is to optimize ventricular filling, keeping in mind that preload can be reduced by the use of diuretics and nitrates. Arterial and central venous blood pressure should be monitored, and sometimes pulmonary arterial pressure should also be monitored. It is advisable to monitor the cardiac output to highlight silent periods of low flow and consequent occult tissue hypoxia. If available, transesophageal echocardiography is a useful tool for visualizing and monitoring overall cardiac performance.

Heart rhythm should be maintained as arrhythmic changes may compromise cardiac output (systolic and diastolic dysfunctions). Especially in advanced diastolic dysfunction (restrictive form), loss of sinus rhythm can lead to fatal episodes, and immediate electrical cardioversion should be performed. Many patients already have an implantable defibrillator and biventricular pacing that needs to be optimized before surgery. However, the heart rate should be maintained in the 80–90 bpm range, as the cardiac output may solely depend on the heart rate.

Proper cardiac contractility must be maintained; in particular, the use of inotropes may be necessary to counteract the cardio-depressive action of anesthetic agents. Reduction of the afterload due to vasodilatation, for example, as a secondary effect of spinal or epidural anesthesia, can have positive effects, as it does not only reduce myocardial work but also help to maintain the cardiac output [6]. However, the benefit of such actions can be limited by falls in blood pressure which can compromise the blood flow to vital organs such as the brain and kidney.

Particular attention should be paid to preoperative therapy with ACE inhibitors, which may result in a massive vasodilation induced by severe vasoplegic action of the anesthetic drugs, difficult to treat [20].

Patients with heart failure should follow a protocol of hemodynamic optimization (optimization of DO_2) intraoperatively and especially in the postoperative time in intensive care where, in addition to arrhythmias monitoring, hemodynamic monitoring and optimization must be continued to ensure better outcomes.

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