Hemodynamic instability is the major concern in surgical patients with pericardial diseases, since general anesthesia and positive pressure ventilation may precipitate cardiac tamponade. In advanced constriction diastolic impairment and myocardial fibrosis/atrophy may cause low cardiac output during and after surgery.

Elective surgery should be postponed in unstable patients with pericardial comorbidities. Pericardial effusion should be drained percutaneously (in local anesthesia) and pericardiectomy performed for constrictive pericarditis before any major surgical procedure. In emergencies, volume expansion, catecholamines, and anesthetics keeping cardiac output and systemic resistance should be applied.

Etiology of pericardial diseases is an important issue in the preoperative management. Patients with neoplastic pericardial involvement have generally poor prognosis and any elective surgical procedure should be avoided. For patients with acute viral or bacterial infection or exacerbated metabolic, uremic, or autoimmune diseases causing significant pericardial effusion, surgery should be postponed until the causative disorder is stabilized and signs of pericarditis have resolved.

Key words: pericardium, pericardial effusion, cardiac tamponade, constrictive pericarditis, preoperative management

INTRODUCTION

A broad spectrum of pericardial diseases may influence the preoperative and perioperative management, including congenital defects of the pericardium, pericarditis (dry, effusive, effusive-constrictive, constrictive), neoplasms, chylopericardium, and cysts. The underlying background of these disorders may include: infectious, systemic autoimmune, postmyocardial infarction-/post-pericardiotomy syndrome, uraemic, toxic, metabolic and autoreactive aetiologies.

PERICARDIAL SYNDROMES AFFECTING PREOPERATIVE AND PERIOPERATIVE MANAGEMENT

Congenital defects of the pericardium

Congenital defects of the pericardium are detected in 1/10 000 autopsies. Pericardial absence can be partial (left ~70%, right ~17%) or total bilateral (rare). An association with other congenital cardiac, pulmonary or skeletal abnormalities is found in one-third of patients. Most patients with total pericardial absence are asymptomatic. Cardiac displacement and augmented mobility impose an increased risk for traumatic aortic dissection. Partial left-side defects can be complicated by herniation of the heart, which can cause haemodynamic alterations during the general anesthesia or even prolonged ischemic episodes, while partial right defects may cause compression of the vena cava. Before the elective surgical procedure, pericardioplasty is indicated to prevent imminent strangulation.

Acute pericarditis

In the preoperative management, occurrence of acute pericarditis is a clear indication to postpone an elective surgical procedure. Acute pericarditis may be a self-resolving benign disease but it can also be the first manifestation of neoplastic, purulent or tuberculous diseases that require prompt specific therapy. Importantly, careful differential diagnosis between acute pericarditis and acute coronary syndrome or aortic dissection is essential. Generally, pericarditis can be dry, fibrinous or effusive. Major symptoms are retrosternal or left precordial chest pain (which radiates to the trapezius ridge, and varies with posture, being more prominent when sitting than when lying) and sometimes shortness of breath. A prodrome of
fever, malaise, and myalgia is common. The pericardial friction rub can be transient, mono-, bi- or triphasic. Pleural effusion may be present. Heart rate is usually rapid and regular. ECG changes can be non-specific (including normal ECG) or very suggestive changes including PR depression and concave ST-segment elevation (Figure 1). Echocardiography is essential to detect effusion and concomitant heart or paracardial disease.

If associated disease is present in acute pericarditis the likelihood that it is the cause of the pericardial syndrome is very high. In these patients, and in patients with self-resolving forms, only non-invasive diagnostic tests are indicated. Many of them can be managed as out-patients. However, in tamponade without findings of inflammation a likelihood ratio of 3.0 for neoplasia has been demonstrated. A sustained clinical course (> 3 weeks) also increases the likelihood of specific disease. Importantly, purulent pericarditis should be considered in predisposing diseases (pleural empyema, mediastinal infection).

Symptomatic treatment of acute pericarditis is based on chest pain management and anti-inflammatory therapy. The treatment of choice is aspirin (1-4 g/day). Ibuprofen (200-600 mg three times a day) and indomethacin (25-50 mg three times a day) are the preferred alternatives to aspirin because of its rare side-effects and the large dose range. Three months course of Colchicine (0.5 mg twice a day) added to non-steroidal anti-inflammatory drugs (NSAIDs) is effective for the treatment of acute pericarditis and prevention of recurrences. Systemic corticosteroids are indicated for connective tissue diseases, and autoreactive or uraemic pericarditis. Intrapericardial application may be effective and mainly avoids systemic side-effects in patients with large autoreactive and uraemic pericardial effusion that is resistant to conventional treatment. Doses of corticosteroids should be low, as applied in contemporary rheumatology.

**Chronic and recurrent (relapsing) pericarditis**

Preoperative and perioperative management of patients with chronic or recurrent pericarditis is determined by the hemodynamic impact and the volume of pericardial effusion. Moderately large and large effusions (separation of pericardial layers of more than 10 mm in diastole subxyphoidally or in the apical area) should be drained before the elective surgical procedure in general anesthesia. Hypotensive patients with small effusions could be safely operated after volume expansion.

A distinction should be made between chronic pericarditis (which implies inflammatory activity with pericardial pain, fever, etc.) and chronic pericardial effusion. Except for constrictive pericarditis, chronic (> 3 months) inflammatory pericarditis is rare. Tuberculous pericarditis may show a subacute clinical course of several weeks, but not a persistent, sustained evolution. In contrast, pericardial effusion can exhibit a stable chronic course lasting for months or years.

The recurrent or relapsing pericarditis could present as intermittent (symptom-free intervals without therapy) or the incessant disease (discontinuation of anti-inflammato
In large effusions, the heart may move freely within the pericardium ("swinging heart") inducing pseudo-prolapse and pseudosystolic anterior motion of the mitral valve, paradoxical motion of the interventricular septum, and mid-systolic aortic valve closure.

**Cardiac tamponade**

Imminent cardiac tamponade is an absolute contraindication for the surgical procedure in general anesthesia\(^{15,16}\). Additional hypotension responsible for the hemodynamic worsening can be caused by anesthesia-induced peripheral vasodilatation, direct myocardial depression by some anesthetics, or by decreased venous return caused by increased intrathoracic pressure associated with positive pressure ventilation.

Cardiac tamponade is a compressive disorder of the heart (continuum ranging from a mild to a life-threatening condition), caused by effusion accumulation and the increased intrapericardial pressure. In spite of the great value of contemporary echocardiography, cardiac tamponade remains a clinical diagnosis. The classical clinical findings include features of venous hypertension and pulsus paradoxus (inspiratory reduction ≥20 mmHg in systolic blood pressure). Orthopnoea, cough, and dysphagia, occasionally with episodes of unconsciousness, can be observed\(^3,15\).

Up to one-third of patients with asymptomatic large pericardial chronic effusions develop unexpected cardiac tamponade\(^{15}\). Triggers for tamponade include hypovolemia, paroxysmal tachyarrhythmia, and intercurrent acute pericarditis. Indications for pericardial drainage are demonstrated in the Figure 5\(^4\).

Cardiac tamponade is an absolute indication for urgent pericardial drainage in the local anesthesia\(^4,17-20\). Medical treatment could be only a temporary measure until pericardiocentesis or surgical relief (e.g. in dissection of the aorta) can be performed. The benefit of inotropic support for hypotensive patients, with or without vasodilators (e.g. dobutamine), is controversial\(^{11,22}\). Volume infusion, however, is useful for patients with hypovolemia\(^4\). Although we have never noticed such an event in our clinical practice, it was reported that intravenous administration of fluid can even precipitate tamponade in normovolemic or hypervolemic patients\(^{23}\).

Although severely hypoxic patients or those inclining to respiratory arrest must be intubated and ventilated during the preparation for pericardiocentesis, prolonged, positive pressure ventilation should be avoided since it decreases the cardiac output further\(^{24}\). The initial procedural mortality in the large surgical pericardioscopy series from Lille (France) was caused by the precipitation of critical tamponade in general anesthesia and mechanical ventilation\(^25\). In patients with cardiac arrest and a large amount of pericardial fluid, external cardiac compression has no value, before at least a part of the pericardial effusion is evacuated. If the resuscitation is still performed without pericardiocentesis, systolic pressure may even slightly rise, but diastolic pressure will fall and further reduce coronary perfusion pressure\(^{15}\). Intravenous administration of

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FIGURE 2
Preoperative and perioperative management of patients with pericardial diseases 47

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**FIGURE 3.**
A) CHEST RADIOGRAPHY (FRONTAL VIEW) IN A PATIENT WITH LARGE PERICARDIAL EFFUSION WITH GLOBULAR CARDIOMEGALY WITH SHARP MARGINS ("WATER BOTTLE" APPEARANCE) AND B) MASSIVE PERICARDIAL CALCIFICATIONS IN THE LATERAL VIEW IN A PATIENT WITH CHRONIC CONSTRUCTIVE PERICARDITIS - "PANZER HERZ" SIGN.
Constrictive pericarditis

Constrictive pericarditis is a clinical syndrome defined by impaired expansion of the heart by a rigid, chronically inflamed/thickened pericardium. The syndrome may, however, exist in approx. 20% of cases in the absence of pericardial thickening, with ultrastructural changes only.

The predominant form is chronic constriction without pericardial effusion. Effusive-constrictive forms are equally important. Acute/subacute forms, transient constrictive pericarditis, epicardial constriction, and occult/subclinical forms are rather rare.

Patients complain of fatigue, peripheral oedema, breathlessness and abdominal swelling. In decompensated patients venous congestion, hepatomegaly, pleural effusions and ascites may occur, aggravated by a protein-losing enteropathy. In few instances, haemodynamics can be additionally impaired by a systolic dysfunction (myocardial fibrosis or atrophy). Restrictive cardiomyopathy is the condition that may create the most serious differential diagnostic problems. Much less frequently, pulmonary embolism, right ventricular infarction, pleural effusion, or chronic obstructive lung diseases can also be confused with constrictive pericarditis. Physical findings, chest radiography (Figure 3b), echocardiography, computerized tomography, magnetic resonance imaging, haemodynamics, and endomyocardial biopsy contribute to establishing the diagnosis.

Pericardiectomy is the only treatment for permanent constriction. Symptomatic management (diuretics, digoxis, beta-blockers) diminishes congestion and tachyarrhythmias before surgery. Antituberculosis treatment is mandatory in tuberculous constrictions for at least 2 months before the surgery.

Management of anesthesia should include drugs and techniques that minimize changes in heart rate, systemic vascular resistance, venous return, and myocardial contractility. Combinations of opiates, benzodiazepines, and nitric-oxide with or without low doses of volatile anesthetics are acceptable for maintenance of anesthesia. Muscle relaxants with minimal circulatory effects are the best choices, although a modest increase in heart rate as seen with administration of pancuronium is also acceptable.

Preoperative optimization of intravascular volume is essential. When hemodynamic compromise (hypotension) due...
to increased intrapericardial pressure is present prior to surgery, management of anesthesia is as described for cardiac tamponade. Invasive monitoring of arterial and central venous pressure is helpful because removal of adherent pericardium may be a tedious and long operation often associated with significant fluid/blood losses. Cardiac arrhythmias are common, presumably reflecting direct mechanical stimulation of the heart. Intravenous fluids and blood products may be necessary to treat the significant fluid/blood losses associated with pericardiectomy.  

Perioperative mortality is 6-12% in the current series, but can be up to 40% if patients with extensive myocardial atrophy/fibrosis are not excluded. Major complications include acute cardiac insufficiency and ventricular wall rupture. If surgery is carried out early, long-term survival after pericardiectomy corresponds to that of the general population. Postoperative respiratory insufficiency may necessitate prolonged mechanical ventilation. Supraventricular tachyarrhythmias and low cardiac output may complicate the postoperative period. 

Predictors of poor survival are prior radiation, worse renal function, higher pulmonary artery systolic pressure, abnormal left ventricular systolic function, lower serum sodium level, and older age. Surprisingly, pericardial calcification had no impact on survival. In a controlled study of 143 patients with constrictive tuberculous pericarditis, prednisolone therapy as an adjunct to streptomycin, isoniazid, rifampicin, and pyrazinamide reduced the 2-year mortality (4% vs. 11%), decreased the need for repeated pericardial drainage or surgery (21% vs. 30%), and the incidence of late constriction (8% vs. 12%). The 10-year follow-up revealed adverse outcomes in 27% of patients treated with prednisolone in contrast to 38% on placebo, deaths from pericarditis being 3% vs. 11%, respectively. In a multivariate analysis prednisolone reduced the overall death rate, and substantially reduced the risk of death from pericarditis.

**SPECIFIC FORMS OF PERICARDITIS AFFECTING PREOPERATIVE AND PERIOPERATIVE MANAGEMENT**

Several etiological forms of pericardial disease may require specific management before the elective surgery can be safely performed. In acute viral or bacterial infection involving the pericardium or exacerbated metabolic, uremic, or autoimmune disease causing significant pericardial effusion, surgical procedure should be postponed until the underlying disease is stabilized and signs of pericarditis resolved. Treatment of acute idiopathic and viral pericarditis is directed to resolving the symptoms and preventing recurrences including 10 days course of aspirin 4-6x600 mg or ibuprofen 800+800+400 mg concomitantly with colchicine 2x0.5 mg for 3 months.

Purulent pericarditis is a rare, acute, fulminant illness that is always fatal if untreated. The mortality rate in treated patients is 40%, mostly because of cardiac tamponade, toxicity and constriction. Percutaneous pericardiocentesis must be promptly performed. The pericardial fluid obtained should undergo Gram, acid-fast, and fungal staining, followed by cultures of the pericardial and body fluids. Rinsing of the pericardial cavity, combined with effective systemic antibiotic therapy, is mandatory (antistaphylococcal antibiotic plus aminoglycoside, tailored according to pericardial fluid and blood cultures). Intra-pericardial instillation of antibiotics (e.g. gentamycin) is useful but not sufficient. Open surgical drainage and pericardiectomy are required in patients with dense adhesions, loculated and thick purulent effusion, recurrence of tamponade, persistent infection and progression to constriction. Surgical mortality is up to 8%. Instead of surgery, pericardiocentesis and frequent irrigation of the pericardial cavity with urokinase or streptokinase has been applied in a few patients. However, the safety and efficacy of this approach in comparison to surgery remains to be investigated.

Pericarditis in a patient with proven extracardiac tuberculosis is strongly suggestive of a tuberculous aetiology (several sputum cultures should be taken). Tuberculous pericarditis can present as acute pericarditis with or without effusion; cardiac tamponade, acute constrictive pericarditis, subacute constriction, effusive-constrictive, or chronic constrictive pericarditis, and pericardial calcifications. The mortality rate in untreated effusive tuberculous pericarditis approaches 85%. Pericardial constriction in these cases is 30-50%. The diagnosis is made by the identification of M. tuberculosis in the pericardial fluid/tissue, and/or the presence of caseous granulomas in the pericardium. Polymerase chain reaction methods can identify M. tuberculosis rapidly from 1 µl of pericardial fluid. Increased adenosine deaminase activity and interferon-gama concentration in pericardial effusion are also suggestive. The tuberculin skin test may produce a false-negative in 25-33% and a false-positive in 30-40%.
of patients. The more accurate enzyme-linked immunospot (ELISPOT) test detects T cells that are specific for the M. tuberculosis antigen. Pericardioscopy and pericardial biopsy may improve the diagnostic accuracy. Various antituberculous drug combination regimens of different durations (6, 9, 12 months) have been applied. However, only patients with proven or very likely tuberculous pericarditis should be treated. Prevention of constriction in chronic pericardial effusion of undetermined aetiology by e.g. ivanitibus antitubercular treatment was not successful. A meta-analysis of treatment results in effusive and constrictive tuberculous pericarditis suggested that tuberculous treatment combined with steroids might be associated with fewer deaths and less frequent need for pericardiocentesis or pericardiectomy. If given, prednisone should be administered in high doses (1-2 mg/kg/day) because rifampicin induces its metabolism by the liver. This is maintained for 5-7 days and progressively reduced in 6-8 weeks.

Fungal pericarditis may be endemic (Histoplasma, Coccioidioides) or opportunistic (Candida, Aspergillus, Blastomyces, Nocardia, Actinomyces). Diagnosis is obtained by staining and culturing pericardial fluid or tissue or by determination of antifungal antibodies in serum. NSAIDs can support the treatment with antifungal drugs (fluconazole, ketoconazole, itraconazole, amphotericin B).

Most patients with uraemic pericarditis respond to frequent haemodialysis (heparin-free to avoid haemopericardium) with resolution of chest pain and pericardial effusion within 1-2 weeks. Peritoneal dialysis may be therapeutic in pericarditis that is resistant to haemodialysis, or if heparin-free haemodialysis cannot be performed. NSAIDs and systemic corticosteroids have limited success when intensive dialysis is ineffective. Cardiac tamponade and large effusions resistant to dialysis must be treated with pericardiocentesis. Large, non-resolving symptomatic effusions can be treated with intrapericardial instillation of corticosteroids (triamcinolone hexacetonide 50 mg every 6 hours for 2 to 3 days). Pericardiectomy is indicated only in refractory, severely symptomatic patients. Colchicine may worsen the impaired renal function, but benefit was also noted in a resistant case of uraemic pericarditis.

Pericarditis may also occur in several systemic autoimmune diseases. Intensified preoperative treatment of the underlying disease and symptomatic management are indicated.

Post-cardiac injury (postpericardiotomy) syndrome develops within days to months after cardiac/pericardial injury. Cardiac tamponade is more common following valve surgery than coronary artery bypass grafting and may be related to the postoperative use of anticoagulants. Symptomatic treatment is the same as for acute pericarditis and repeat surgery is rarely needed. Benefit of primary prevention using perioperative treatment with colchicine was recently proven in a randomized COPPS trial.

Postinfarction pericarditis occurs as an "early" (pericarditis epistenocardica) or a "delayed" form (Dressler’s syndrome). Epistenocardiac pericarditis, caused by direct exudation, occurs in 5-20% of transmural myocardial infarctions within the first 7 days but is rarely discovered clinically. Dressler’s syndrome arises from one week to several months after myocardial infarction, with manifestations similar to the post-cardiac injury syndrome. It does not require transmural infarction and can also appear as an extension of epistenocardiac pericarditis. Its incidence was 0.5-5% in the past, particularly in the prethrombolytic era, but the syndrome has become a rarity most probably because of advances in treatment of acute myocardial infarction by cardiac interventions. High doses of ibuprofen or aspirin should be given for 2-5 days. Steroids can be used for refractory symptoms but may delay the healing of infarction. Surgical revascularization of the myocardium should be postponed until the signs of Dressler’s syndrome have resolved, except in unstable patients with left-main disease or left-main equivalent.

Of note, a postinfarction pericardial effusion larger than 10 mm is a predictor of ventricular wall rupture (Figure 6). Urgent surgical treatment is life saving. If this is not feasible pericardiocentesis and intrapericardial fibrin-glue instillation could be an alternative. Patients with neoplastic pericardial disease have generally very poor prognosis if the pericardial involvement is caused by metastatic malignant disease. Any elective surgical procedure should be avoided, unless its purpose would be to improve the symptoms or prognosis of the underlying neoplastic illness. The diagnosis is based on the confirmation of the malignant infiltration within the pericardium by cytology or biopsy. Notably, in almost two-thirds of the patients with documented malignancy pericardial effusion is caused by non-malignant diseases, e.g. radiation pericarditis, or opportunistic infections. In neoplastic pericardial effusion without tamponade systemic antineoplastic treatment as baseline therapy can prevent up to 67% of recurrences. Pericardial drainage is recommended in all patients with large effusions/tamponade, but also in patients with moderate effusions 10-20 mm in order to confirm the neoplastic etiology of pericardial involvement. Prevention of recurrences may be achieved by intrapericardial instillation of sclerosing, cytotoxic agents, or immunomodulators. Radiation therapy is effective in patients with radiosensitive tumours (lymphoma and leukaemia). Intrapericardial instillation of cisplatin is most effective in secondary lung cancer and thiotepa is valuable in breast cancer pericardial metastases.

Chylopericardium is caused by a communication between the pericardium and the thoracic duct, predominantly as a complication of trauma, congenital anomalies, or surgery. The pericardial fluid is sterile, odourless and opalescent with a milky white appearance and the microscopic finding of fat droplets. The chylous nature of the fluid is confirmed by Sudan III stain for fat and by high concentrations of triglycerides (5-50g/l), and proteins (22-60g/l). Enhanced computerized tomography, alone or combined with lymphography, can identify not only the location of the thoracic duct but also its lymphatic connection to the pericardium. Chylopericardium after thoracic
or cardiac operation is preferably treated by pericardio-
centesis and diet (medium-chain triglycerides). If produc-
tion of chylous effusion continues, surgical treatment is
mandatory. When the course of the thoracic duct can be
precisely identified, its ligation and resection just above
the diaphragm is effective.4

Pericardial effusion in hypothyroidism occurs in 5–30%
of patients with pericardial disease.2 Fluid accumulates
slowly and tamponade occurs rarely. In some cases cho-
lesterol pericarditis may be observed. The diagnosis is ba-
sed on serum levels of thyroxin and thyroid-stimulating
hormone. Therapy with thyroid hormone leads to the re-
solution of the pericardial effusion.60

CONCLUSION

Pericardial diseases are infrequent, but potentially life-
threatening co-morbidity in patients undergoing surgical
procedures in general anesthesia. Preoperative and peri-
operative care for these patients may be challenging espe-
cially regarding the management of hemodynamic insta-
bility, as well as the underlying infectious, autoimmune or
metabolic diseases. Proper and timely diagnostic asse-
ssment and etiological diagnosis is essential to avoid se-
rious perioperative and postoperative complications.

SUMMARY

PREOPERATIVNA PRIPREMA I PERIOPERATIVNI TRETMAN BOLESNIKA SA OBOLJENJIMA PERI-
KARDA

Hemodinamska nestabilnost je najvažniji problem u
preoperativnoj pripremi bolesnika sa bolestima perikarda pošto opšta anestezija i ventilacija pod pozitivnim pritis-
kom može da izazove tamponadu srca. U odmaškoj kon-
strikciji diastolna disfunkcija, kao i fibroza i atrofija mio-
karda mogu uzrokovati nizak minutni volumen tokom i
posle hirurške intervencije.

Elektivne hirurške procedure treba odložiti kod nestabil-
nih bolesnika sa perikardnim komorbiditetima. Perikardi-
zn izliv treba perikutan drenirati (u lokalnoj anesteziji), a
d kod bolesnika sa kroničnom konstrikcijom uraditi peri-
kardietomiju pre svake velike hirurške intervencije u op-
štoj anesteziji. U urgentnim stanjima treba primiti ek-
spandere intravaskularnog volumena, kateholamine i an-
estetike koji ne obaraju minutni volumen niti sistemska
vascularnu rezistenciju.

Etiologija bolesti perikarda je takodje jedan od važnih
elemenata u preoperativnoj pripremi bolesnika. Bolesnici
sa neoplašćenim bolestima perikarda imaju generalno
loši prognozu i sve velike, elektivne hirurške procedure
treba izbegavati. Kod bolesnika sa akutnom virusnom ili
bakterijskom infekcijom kao i kod metaboličkih, bubre-
žnih ili autoimunih bolesti koje uzrokuju perikarditis, hi-
ruršku proceduru treba odložiti dok se osnovna bolest ne
stabilizuje i znaci perikarditisa ne povuku.

Ključne reči: perikard, perikardni izliv, tamponada
srca, konstruktivni perikarditis,
preoperativna priprema

REFERENCES

1. Maisch B, Ristić AD. Pericardial diseases. In: Fink
M, Abraham E, Vincent JL, Kochanek P., eds. Textbook
of critical care, 5th edition. Elsevier, Philadelphia, PA,
2005, 851-60.
2. Maisch B, Soler-Soler J, Hatle L, Ristic AD. Pericard-
dial disease. In: Serruys PW, Camm AJ, Lüscher TF,
eds. The ESC Textbook of Cardiovascular Medicine.
defect: risk factor for traumatic aortic type A dissection.
on the diagnosis and management of pericardial diseases.
5. Sagrista-Sauleda J, Merce J, Permyaner-Miralda G et
al. Clinical clues to the causes of large pericardial effu-
treatment of acute pericarditis: a management program for
al. Clinical clues to the causes of large pericardial effu-
8. Sagrista-Sauleda J, Barrabes JA, Permyaner-Miralda
G, et al. Purulent pericarditis: review of a 20-year experi-
ence in a general hospital. J Am Coll Cardiol 1993;
22:1661-5.
9. Spodick DH. The pericardium: a comprehensive text-
10. Imazio M, Bobbio M, Cecchi E, et al. Colchicine as
first-choice therapy for recurrent pericarditis: results of the
CORE (COlchicine for REcurrent pericarditis) trial.
11. Imazio M, Bobbio M, Cecchi E, et al. Colchicine in
addition to conventional therapy for acute pericarditis: re-
sults of the COlchicine for acute PEricarditis (COPE)
12. Maisch B, Ristić AD, Pankuweit S. Intrapericardial
treatment of autoreactive pericardial effusion with triam-
cinolone; the way to avoid side effects of systemic corti-
coesteroids for recurrent pericarditis. Eur Heart J 2002;
13. Imazio M, Brucato A, Cumetti D, et al. Corticos-
steroids for recurrent pericarditis: high versus low doses: a
nonrandomized observation. Circulation 2008;
QRS voltage in cardiac tamponade and peri-
cardial effusion: reversibility after pericardiotensis and
after anti-inflammatory drug treatment. J Am Coll Cardiol
16. Modak RK. Pericardial diseases and cardiac trauma.
In: Hines RL, Marschall KE, eds. Stoelting’s anesthesia
and co-existing disease. Saunders/Elsevier, Philadelphia,
PA, 2008:125-133.