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Physiological pericardial effusion

Skip Nav Destination Pericardial effusion is a common finding in clinical practice either as incidental finding or manifestation of a systemic or cardiac disease. The spectrum of pericardial effusions ranges from mild asymptomatic effusions to cardiac tamponade. The aetiology is varied (infectious, neoplastic, autoimmune, metabolic, and drug-related), being tuberculosis the leading cause of pericardial effusions in developing countries and all over the world, while concurrent HIV infection may have an important promoting role in this setting. Management is guided by the haemodynamic impact, size, presence of inflammation (i.e. pericarditis), associated medical conditions, and the aetiology whenever possible. Pericardiocentesis is mandatory for cardiac tamponade and when a bacterial or neoplastic aetiology is suspected. Pericardial biopsy is generally reserved for cases with recurrent cardiac tamponade or persistence without a defined aetiology, especially when a bacterial or neoplastic aetiology is suspected and cannot be assessed by other conventional and less invasive means. A true isolated effusion may not require a specific treatment if the patient is asymptomatic, but large ones are at risk of progression to cardiac tamponade (up to one third). Pericardiocentesis alone may be curative for large effusions, but recurrences are also common and pericardiectomy or less invasive options (i.e. pericardial window) should be considered with recurrent cardiac tamponade or symptomatic pericardial effusion (either circumferential or loculated). The aim of this paper was to summarize and critically evaluate current knowledge on the management of pericardial effusion. Pericardial effusion, Aetiology, Diagnosis, Management, Pericarditis Probably no serious disease is so frequently overlooked by the practitioner. Postmortem experience shows how often pericarditis is not recognized or goes to resolution and adhesion without attracting notice (Osler, The Principles and Practice of Modern Medicine, 1892).Pericardial effusion is a common finding in clinical practice either as incidental finding or manifestation of a systemic or cardiac disease. The spectrum of pericardial effusions ranges from mild asymptomatic effusions to cardiac tamponade. Moreover, pericardial effusion may accumulate slowly or suddenly.1–3 Unfortunately, there are few epidemiological data on the incidence and prevalence of such effusions in the clinical setting. In Maria Vittoria hospital, an urban general hospital in Torino and an Italian referral centre for pericardial disease, the mean annual incidence and prevalence of pericardial effusion have been, respectively, 3 and 9% in a 6 years experience of the echo laboratory (2000–05).4 Such data mainly depend on the epidemiological background (especially developed vs. developing country, where tuberculosis is a leading cause of pericardial disease and concurrent HIV infection may have an important promoting role),5 the institutional setting (tertiary referral centre vs. secondary and general hospitals), and the availability of specific subspecialties (especially nephrology, rheumatology, and oncology).No specific guidelines and management recommendations have been issued by medical societies beyond the 2000 national Spanish guidelines6 and the 2004 European Society of Cardiology guidelines on the management of pericardial diseases,7 and some narrative reviews on the topic.4,8,9 whereas no specific guidelines have been issued from the American Heart Association (AHA) and American College of Cardiology (ACC).It is the aim of this review to critically evaluate current knowledge on the management of pericardial effusion. A thorough literature review has been performed without language restriction with the MeSH term 'pericardial effusion' or 'pericardial'[All Fields] and 'effusion'[All Fields]) or 'pericardial effusion'[All Fields]. After the initial identification of 1520 papers, 139 papers were selected for detailed review based on novelty or important data leading to the final inclusion of 50 papers in the final reference list. Initial evaluation and pathophysiological issues When a pericardial effusion is detected, the first step is to evaluate its size and haemodynamic importance, as well as the possible association with concomitant diseases. Echocardiography is the first diagnostic tool for this assessment as also acknowledged by the AHA/ACC guidelines on the use of echocardiography, that gave a class I indication for the use of echocardiography in any case of suspected pericardial disease.10Pericardial effusion may be classified based on its onset (acute, subacute vs. chronic when dating >3 months), distribution (circumferential or loculated), haemodynamic impact (none, cardiac tamponade, effusive-constrictive), composition (exudates, transudate, blood, rarely air, or gas from bacterial infections), and especially by its size as mild, moderate, and large (Table 1) based on a simple semiquantitative echocardiographic assessment (Figure 1) that has been demonstrated useful also to estimate the risk of specific aetiology and complications during follow-up.4,7,9,11,12 Table 1Classification of pericardial effusion Onset Acute (1 week but 3 months) Size Mild (20 mm) Distribution Circumferential Loculated Haemodynamic effect Without cardiac tamponade With cardiac tamponade Effusive-constrictive Composition/type Transudate Exudate Hydropericardium (transudate, plasma ultrafiltrate) Haemopericardium (blood in pericardial space) Chylopericardium (chylous pericardial fluid) Pyopericardium (purulent pericardial effusion) Pneumopericardium (air in the pericardium) Open in new tabDownload slide(A) The semiquantitative assessment of the pericardial effusion size in a case of idiopathic chronic pericardial effusion. A large effusion of >30 mm in diastole is shown in parasternal long-axis view. (B) Acute pleuropericarditis with ST segment elevation and sinus tachycardia on ECG strip. A large pericardial effusion >20 mm with concomitant pleural effusion is also evident posteriorly to the aorta. Pe, pericardial effusion; pl, pleural effusion; Ao, aorta.The normal pericardial sac contains 10–50 mL of pericardial fluid acting as a lubricant between the pericardial layers. Surprisingly, little is known about the formation and removal of pericardial fluid, because of the paucity of comprehensive studies, especially in human subjects, and methodological difficulties to distinguish between the dynamics of normal pericardial fluid and those of a pathological effusion (Supplementary material online, Reference A). Nevertheless, normal pericardial fluid is generally considered an ultrafiltrate of plasma (Supplementary material online, Reference B). The arrangement of lymphatic vessels is complex and has been described in human cadavers (Supplementary material online, References C and D). The lymphatic vessels follow different pathways according to ventral, lateral, and posterior surfaces, but, in any case, terminate to mediastinal, tracheobronchial, or luxtaesophageal lymph nodes. On the ventral surface, the lymphatics of the parietal pericardium connect to lymphatics in the pericardial fat and areolar tissue. On the lateral and posterior surfaces, the lymphatics of the parietal pericardium anastomose with lymphatics of the reflected mediastinal pleura (Supplementary material online, Reference C). Lymphatic drainage of the pericardium to the mediastinal and tracheobronchial lymph nodes and interactions with pleural lymphatics (Supplementary material online, Reference D) provide the anatomical basis for pathological involvement of the pericardium in specific diseases (i.e. pleuro-pulmonary diseases such as pulmonary tuberculosis and lung cancer).Any pathological process usually causes an inflammatory process with the possible increased production of pericardial fluid (exudate). An alternative mechanism of the formation of pericardial fluid may be the decreased reabsorption due to increased systemic venous pressure generally as a result of congestive heart failure or pulmonary hypertension (transudate). If pericardial fluid is free to move within the pericardial sac following the gravity forces, it usually starts accumulating posteriorly to the left ventricle when the patient is laying on his/her left side for echocardiographic evaluation (mild effusion detected initially as posterior), then circumferentially in the case of moderate to large pericardial effusions. A mild pericardial effusion may also be detected close to the right atrium because this is the cardiac chamber with the lowest pressures within the cardiac cycle and thus fluid accumulation is easier in this position (Figure 2). An isolated mild anterior pericardial fluid is unusual on echocardiography without previous pericardial scarring as following surgery or chronic pericarditis, and should be regarded as fat rather than pericardial fluid.13 Computed tomography (CT) or cardiac magnetic resonance (CMR) may confirm the finding in specific cases14 (Figure 3). On the contrary after pericardial scarring (i.e. after cardiac surgery or chronic pericarditis, or bacterial infections), pericardial fluid may not have a uniform distribution within the pericardial space and may give rise to loculated effusions that should be evaluated with multiple cardiac views.14,15 Open in new tabDownload slidePresentation of a mild (A) vs. moderate to large pericardial effusions (B) on echocardiography. Mild pericardial effusion is evident adjacent to the right atrium in four-chambers view and only posterior in parasternal long-axis view (A). As fluid accumulates, the effusion becomes circumferential (B). Pe, pericardial effusion; RA, right atrium; Ao, aorta. Open in new tabDownload slideDifferential diagnosis of pericardial effusion vs. epicardial fat. (A) Echocardiographic view. Anterior pericardial echo-free space. No effusion was evident adjacent to the right atrium in four-chambers view. (B) Epicardial fat and thickened pericardium with contrast enhancement on CT scan.The pressure–volume curve of the normal pericardium is a J-shaped curve: after an initial short shallow portion that allows the pericardium to stretch slightly in response to physiological events, such as changes in posture or volume status, with minimal pressure increase, then the pericardium does not allow further sudden increases of the volume without a marked increase in the intrapericardial pressure. Thus a sudden increase of pericardial volume of 100–200 mL, as in haemopericardium, may elevate pericardial pressure till 20–30 mmHg with acute cardiac tamponade (acute or surgical cardiac tamponade). On the contrary a slowly accumulating pericardial fluid may allow pericardial distention till the accumulation of 1–2 L of pericardial fluid without the development of cardiac tamponade (Figure 4) till advanced stages often because of intercurrent events (chronic cardiac tamponade reported symptoms and signs are often related to the speed of pericardial fluid reaching cardiac tamponade only after larger volumes (B). Aetiology A wide variety of aetiologic agents may be responsible of pericardial effusions, since all known causes of pericardial disease may be causative agents (Table 2). The more common causes of pericardial effusions include infections (viral, bacterial, especially tuberculosis), cancer, connective tissue diseases, pericardial injury syndromes (post-myocardial infarction effusions, post-pericardiotomy syndromes, post-traumatic pericarditis either iatrogenic or not), metabolic causes (especially hypothyroidism, renal failure), myopericardial diseases (especially pericarditis, but also myocarditis, heart failure), aortic diseases, especially aortic dissection extending into the pericardium, and selected drugs (i.e. minoxidil). Hydropericardium, a non-inflammatory transudative pericardial effusion, may occur not only in heart failure, but also advanced hypoalbuminaemia, such as in cirrhosis and nephrotic syndrome, when Starling forces promote the accumulation of a plasma ultrafiltrate across the pericardium as well as other membranes (e.g. pleura and peritoneum). Table 2Causes of pericardial effusion . Infectious Viral (most common: Echovirus and Coxsackievirus (usual), Influenza, EBV, CMV, Adenovirus, Varicella, Rubella, Mumps, HBV, HCV, HIV, Parvovirus B19 and Human Herpes Virus 6 (increasing reports) Bacterial [most common: tuberculous (4–5%), Coxiella burnetii, other bacterial rare may include Pneumo-, Meningo-, Gonococcosis, Haemophilus, Staphylococci, Chlamydia, Mycoplasma, Legionella, Leptospira, and Listeria] Fungal (rare: Histoplasma more likely in immunocompetent patients, Aspergillosis, Blastomycosis, Candida more likely in immunosuppressed host) Parasitic (very rare: echinococcus, toxoplasma) Non-infectious Autoimmune and autoinflammatory Systemic inflammatory diseases (more common in systemic lupus erythematosus, Sjögren syndrome, rheumatoid arthritis, systemic sclerosis, systemic vasculitides, Behçet syndrome, Sarcoidosis, Familial Mediterranean Fever) Pericardial injury syndromes (post-myocardial infarction, post-pericardiotomy syndrome, post-traumatic) Autoreactive Cancer Primary tumours (rare, especially pericardial mesothelioma) Secondary metastatic tumours (lung, breast cancer, lymphomas, and melanoma) Metabolic (Uraemia, Myxedema) Trauma Direct injury (penetrating thoracic injury, oesophageal perforation, and iatrogenic) Indirect injury (non-penetrating thoracic injury, and radiation injury) Mediastinal radiation, recent, or remote Drugs and toxins (rare): Procainamide, hydralazine, isoniazid, and phenytoin (lupus-like syndrome), Penicillins (hypersensitivity pericarditis with eosinophilia), Doxorubicin and daunorubicin (often associated with a cardiomyopathy, may cause a pericardiopathy). Minoxidil. Immunosuppressive therapies (e.g. methotrexate, cyclosporine) Haemodynamic Heart failure, pulmonary hypertension, and hypoalbuminaemia In the last 20 years, five major surveys have been published on the characteristics of moderate to large pericardial effusions (Table 3).17–21 Obviously, the relative frequency of different causes depends on the local epidemiology (especially the prevalence of tuberculosis), the hospital setting, and the diagnostic protocol that has been adopted. Many cases still remain idiopathic in developed countries (up to 50%), while other common causes include especially cancer (10–25%), pericarditis and infectious causes (15–30%), iatrogenic causes (15–20%), and connective tissue disease (5–15%), whereas tuberculosis is the dominant cause in developing countries (>60%), where tuberculosis is endemic.22 In the setting of pericarditis with pericardial effusion, the prevalence of malignant or infectious aetiologies ranges from 15 to 50% depending on published series.12,17–21 Table 3Aetiologic diagnosis of moderate to large pericardial effusions according to major published series Feature . Corey et al.17 . Sagrista-Sauleda et al.18 . Levy et al.19 . Reuter et al.20 . Ma et al.21 . Patients 57 322 204 233 140 Study years 1993 1990–96 1998–2002 1995–2001 2007–09 Country USA Spain France South Africa China Effusion size >5 mm >10 mm NR NR Moderate to large Cardiac tamponade NR 37 NR NR NR Evaluation Subxiphoid pericardiotomy TB, ANA, TSH, pericardiocen-tesis, and biopsy BCx, TSH, ANA, Q fever, viral rectal, and throat swabs HIV, sputum, TB, BCx, blood chemistry, and serology Pericardiocen-tesis Idiopathic (%) 7 29 48 14 9 Cancer (%) 23 13 15 9 39 Infections (%) 27 2 16 72 29 Connective tissue diseases (%) 12 5 10 5 6 Metabolic (%) 24 6 11 0 0 Iatrogenic (%) 0 16 0 0 9 At present, the largest cohort of patients with moderate (echo-free space of 10–20 mm during diastole) and large pericardial effusions (echo-free space >20 mm) comes from Spain and includes 322 cases (mean age 56 years, 52% males). Cases were collected retrospectively from 1990 to 93 and then prospectively from 1994 to 96 in a 637-bed university general hospital in Barcelona. The authors adopted a three-stage protocol including a basic evaluation for all (stage I including clinical history, physical examination, electrocardiography, chest X-ray, echocardiography, evaluation for tuberculosis, measurement of serum antinuclear antibodies and thyroid hormones, and other targeted studies according to specific presentation), followed by stage II with pericardiocentesis and pericardial fluid analysis for those with cardiac tamponade, suspicion of purulent pericarditis, or chronic large pericardial effusions. Stage III (surgical biopsy of the pericardium) was limited to those with persistent or recurring tamponade after pericardiocentesis, and when the effusion lasted for >3 weeks after admission and without an aetiologic diagnosis. The most common diagnoses included: acute idiopathic pericarditis (20%), iatrogenic effusions (16%), cancer (13%), and chronic idiopathic pericardial effusion (9%). In 60% of cases, the cause of pericardial effusion was a known medical condition.18 Clinical presentation The clinical presentation of pericardial effusion is varied according to the speed of pericardial fluid accumulation as mentioned in the introduction, and the aetiology of the effusion with possible symptoms that may be related to the causative disease. The rate of pericardial fluid accumulation is critical for the clinical presentation. If pericardial fluid is quickly accumulating such as for wounds or iatrogenic perforations, the evolution is dramatic and only small amounts of blood are responsible of a quick rise of intrapericardial pressure and overt cardiac tamponade in minutes. On the contrary a slowly accumulating pericardial fluid allows the collection of a large effusion in days to weeks before a significant increase in pericardial pressure becomes responsible of symptoms and signs.3 Classical symptoms include dyspnoea on exertion progressing to orthopnoea, chest pain, and/or fullness. Additional occasional symptoms due to local compression may include nausea (diaphragm), dysphagia (oesophagus), hoarseness (recurrent laryngeal nerve), and hiccups (phrenic nerve). Non-specific symptoms include also cough, weakness, fatigue, anorexia and palpitations and reflect the compressive effect of the pericardial fluid on contiguous anatomic structures or reduced blood pressure and secondary sinus tachycardia.23The classical findings of cardiac tamponade have been described by the thoracic surgeon Beck in 1935.24 Beck identified a triad including hypotension, increased jugular venous pressure, and a small and quiet heart. This triad was classically identified in 'surgical tamponade' with acute cardiac tamponade due to intrapericardial haemorrhage because of trauma, myocardial or aortic rupture. The Beck triad may be lacking in patients with 'medical tamponade' with slowly accumulating pericardial fluid. Hypotension is absolute or relative. Acute cardiac tamponade is usually associated with low blood pressure (10 mmHg during inspiration.In the absence of cardiac tamponade reported symptoms and signs are non-specific and may include: shortness of breath, dyspnoea on exertion, fever, chills, chest pain, paroxysmal nocturnal dyspnoea, orthopnoea, cough, and oedema.23 All these symptoms, and signs may be secondary to the underlying cause of the effusion (i.e. systemic disease and pericarditis) or the initial mechanical interference of pericardial fluid with cardiac function or surrounding anatomical structures. Diagnosis The diagnosis of pericardial effusion is generally performed by echocardiography, that also allows the semiquantitative assessment of the pericardial effusion size and its haemodynamic effects as outlined in the paragraph on the initial approach to pericardial effusion. The diagnosis of cardiac tamponade is essentially a clinical diagnosis requiring echocardiographic confirmation of the initial diagnostic suspicion. In most patients, cardiac tamponade should be diagnosed by a clinical examination that shows elevated systemic venous pressure, tachycardia, dyspnoea, and paradoxical arterial pulse. Systemic blood pressure may be normal, decreased, or even elevated. The diagnosis is confirmed by an echocardiographic demonstration of moderately large or large circumferential pericardial effusion and in most instances, of right atrial compression, abnormal respiratory variation in right and left ventricular dimensions, and in tricuspid and mitral valve flow velocities usually associated with inferior vena cava plethora (Table 4 and Figure 6) (Supplementary material online, References H).27 Sign . Sensitivity . Specificity . Large pericardial effusion with swinging heart n.a. n.a. Diastolic collapse of right atrium (RA) 50–100% 33–100% Duration of RA inversion time index (duration of inversion/cardiac cycle length); for values >0.34 >90% 100% Diastolic collapse of right ventricle (RV) 48–100% 72–100% Variations in E velocities during respiration across the mitral valve, tricuspid valve, and pulmonary outflow that are greater than 25, 50, and 30% n.a. n.a. Inferior vena cava (IVC) plethora (dilatation >20 mm and 25% (A) and inferior vena cava plethora (B; dilatation >20 mm and 60, and >80% with HIV infection)28,29 that should be ruled out in such context, as well as in immigrants and HIV-infected patients. Non-idiopathic and non-viral aetiologies (especially bacterial and neoplastic) are associated with an increased risk of cardiac tamponade and large effusion, and pericardiocentesis is mandatory when cardiac tamponade or such aetiologies are suspected.4,6,7Analyses of pericardial effusion can establish the diagnosis of infectious and neoplastic pericardial effusions. Cytology and tumour markers [carcinoembryonic antigen (CEA), alfafeto protein, carbohydrate antigens CA 125, CA 72-4, CA 15-3, CA 19-9, etc.) should be performed in suspected malignant disease. In suspected tuberculosis acid-fast bacilli staining, mycobacterium culture or radiometric growth detection (e.g. BACTEC-460), adenosine deaminase (ADA), interferon (IFN)-gamma, pericardial lysozyme, and as well as PCR analyses for tuberculosis should be performed (indication class I, level of evidence B). Differentiation of tuberculous and neoplastic effusion is virtually absolute with low levels of ADA and high ADA levels of CEA. In addition, high ADA levels may predict the evolution towards pericardial constriction. In suspected bacterial infections, at least three cultures of pericardial fluid for aerobes and anaerobes as well as the blood cultures are mandatory (indication class I, level of evidence B). PCR analyses for cardiotropic viruses have been suggested (indication class IIa, level of evidence B), but are rarely used in clinical practice. Analyses of the pericardial fluid specific gravity (>1015), protein level (>3.0 g/dL), fluid/serum ratio >0.5, LDH >200 mg/dL, serum/fluid >0.6, and glucose can separate exudates from transudates, but are not directly diagnostic (class IIb). Nevertheless, purulent effusions with positive cultures have significantly lower fluid glucose levels than non-infectious effusions. White cell count is highest in inflammatory and infectious diseases, and lowest in myxedema. Technical advances in instrumentation, introduction of pericardioscopy and contemporary pathology, virology, and molecular biology techniques have improved the diagnostic value of epicardial/pericardial biopsy. Pericardioscopy performed through air instead of fluid, made it possible to inspect large areas of pericardial surface, select the biopsy site, and take numerous samples. Targeted pericardial/epicardial biopsy during pericardioscopy was particularly useful in the diagnosis of neoplastic pericarditis. No major complications were reported with the use of flexible pericardioscopes (Supplementary material online, References I and J). Such techniques and approaches are warranted in skilled tertiary referral centres for selected cases, when a specific diagnosis, that may require a targeted therapy (i.e. neoplastic pericardial effusion), is suspected and cannot be diagnosed by traditional diagnostic means.In the 2004 ESC guidelines, a specific definition is provided for autoreactive pericarditis/pericardial effusion. The diagnosis of autoreactive pericarditis is established using the following criteria: (i) increased number of lymphocytes and mononuclear cells >5000/mm3 (autoreactive lymphocytic), or the presence of antibodies against heart muscle tissue (antisarcolemmal) in the pericardial fluid (autoreactive antibody mediated); (ii) signs of myocarditis on epicardial/endomyocardial biopsies by >14 cells/mm2; (iii) exclusion of active viral infection both in pericardial effusion and endomyocardial/epimyocardial biopsies (no virus isolation, no IgM titer against cardiotropic viruses in pericardial effusion, and negative PCR for major cardiotropic viruses); (iv) tuberculosis, Borrelia burgdorferi, Chlamydia pneumoniae, and other bacterial infection excluded by PCR and/or cultures; (v) neoplastic infiltration absent in pericardial effusion and biopsy samples; (vi) exclusion of systemic, metabolic disorders, and uraemia. This is essentially a diagnosis of exclusion for pericardial effusions with an autoimmune pathogenesis not related to a known systemic inflammatory disease, and that may be efficiently treated by corticosteroids.A standard recommended approach for the analysis of pericardial fluid is reported in Table 5. Table 5Routine analyses to be performed on pericardial fluid Analysis . Test . Aetiology or feature . General chemistry Specific gravity> 1015, protein level >3.0 g/dL, fluid/serum ratio >0.5, Exudate LDH >200 mg/dL, fluid/serum ratio >0.6a Glucose, blood cell count Cytology Cytology (volumes of fluid, centrifugation, and rapid analysis improve diagnostic yield) Cancer Biomarkers Tumour markers (i.e. CEA >5 ng/mL or CYFRA 21-1 >100 ng/mL) Cancer Adenosine deaminase >40 U/L, IFN-gamma TBC Polymerase chain reaction (PCR) PCR for specific infectious agents (i.e. TBC) TBC Microbiology Acid-fast bacilli staining, mycobacterium cultures, aerobic, and anaerobic cultures TBC Other bacteria Among specific causes of pericardial effusion on the inflamed pericardium (B, see red arrows). Therapy The therapy of pericardial effusion should be targeted at the aetiology as much as possible.37–41 In ~60% of cases, the effusion is associated with a known disease,18 and the essential treatment is that of the underlying disease. When pericardial effusion is associated with pericarditis, management should follow that of pericarditis.30,37Nevertheless, when diagnosis is still unclear or idiopathic, and inflammatory markers are elevated, a trial of aspirin or a non-steroidal anti-inflammatory drug (NSAID) can be prescribed also to evaluate the response (Table 6). A viral or idiopathic form is often responsive to such empiric therapy. For the management of recurrent inflammatory cases, the first step is considering the combination of aspirin or a NSAID plus colchicine,42 while corticosteroids at low to moderate doses may be considered for specific indications (i.e. systemic inflammatory diseases and pregnancy),43 and in case of intolerance, contraindications, or failure of aspirin/NSAID; other therapies are based on less solid evidence: less toxic and less expensive drugs (e.g. azathioprine or methotrexate) should be preferred, tailoring the therapy for the individual patient and the physician experience.44 Table 6Empiric anti-inflammatory therapy for inflammatory pericardial effusions Drug . Attack Dose . Treatment length . Level of evidence . Acetylsalicylic acid 750–1000 mg t.i.d. 1–2 weeks B Ibuprofen 600 mg t.i.d. 1–2 weeks B Indomethacin 50 mg t.i.d. 1–2 weeks C Prednisone or steroid at equivalent dose 0.2–0.5 mg/kg/day First episode: 2 weeks B Recurrence: 4 weeks Colchicine 0.5 mg b.i.d (≥70 kg) First episode: 3 months B 0.5 mg once (10 mm may be associated with haemopericardium, and up to two-thirds of these patients may develop tamponade/free wall rupture. In this setting, urgent surgical treatment is life saving. However, if the immediate surgery is not available or contraindicated, pericardiocentesis, and intrapericardial fibrin-glue instillation could provide an alternative immediate treatment, as proposed in the 2004 ESC guidelines.7In the setting of autoreactive pericardial effusion, growing evidence supports the possible use of intrapericardial therapies to reduce side effects related to the oral use of corticosteroids (Supplementary material online, Reference K). Systemic corticosteroids offer an effective treatment option for autoreactive pericarditis; however, their use is limited by adverse effects and they are an independent risk factor for pericarditis recurrence. In a recently published systematic review (Supplementary material online, Reference L), one case series and three open-label trials evaluating intrapericardial triamcinolone for the management of autoreactive pericarditis were reviewed. Included studies were limited by small sample sizes (n = 2–84), lack of control groups, short durations of follow-up (24 h–12 months), use of adjuvant agents, lack of patient demographic data, subjective report of symptom relief, and lack of consistent dose of intrapericardial triamcinolone. Despite these limitations, available data suggest symptom resolution and reduced pericarditis recurrence with the administration of intrapericardial triamcinolone to patients with autoreactive pericarditis (Supplementary material online, Reference L).When a pericardial effusion becomes symptomatic without evidence of inflammation or when empiric anti-inflammatory drugs are not successful, drainage of the effusion should be considered. Pericardiocentesis with prolonged pericardial drainage till 80% of cases, stretching of the pericardium is often painful so appropriate analgesia is recommended.37,38The 2004 ESC guidelines gave a class IIa recommendation to pericardiectomy for frequent and highly symptomatic recurrences resistant to medical treatment. Other reported indications include repeated recurrences with cardiac tamponade, and evidence of serious steroid toxicity.7 Although surgical experiences are not always concordant, pericardiectomy is generally considered as a therapeutic option of doubtful efficacy in recurrent idiopathic pericarditis or pericardial effusion and should be considered only in exceptional cases. Chronic permanent constriction remains the major indication for such intervention.37 However, incessant pericarditis, as distinguished from recurrent intermittent pericarditis, may respond favourably to surgical removal, especially in the presence of recurrent pericardial effusion.45 An idiopathic chronic pericardial effusion is defined as a collection of pericardial fluid that persists for >3 months and has no apparent cause; large effusions have a risk of progression to cardiac tamponade (up to one-third, according to a Spanish study). On this basis some authors have advocated the need for pericardiectomy for such cases, whenever a large effusion recurs after pericardiocentesis.46 Since drainage is relatively safe and easy in some cases with guided pericardiocentesis, drainage has been recommended for large subacute effusions, that do not respond to empiric therapy, and are stable after several weeks (e.g. 6–8 weeks), especially when there are signs of right-sided collapse, in order to prevent the possible progression of the effusion towards tamponade following additional events (e.g. pericarditis, bleeding following chest trauma).47Unfortunately, there are no proven effective medical therapies to reduce an isolated effusion; in the absence of inflammation, NSAID as well as colchicine and corticosteroids are generally not efficacious.42 Pericardiocentesis alone may be necessary for the resolution of large effusions but recurrences are also common and pericardiectomy or less invasive options (i.e. pericardial window) should be considered whenever fluid reaccumulates, becomes loculated, or biopsy material is required.37 The feasibility of pericardiocentesis is high (>90%) in patients with anterior effusion, while the rate of success is 95% of cases. The most serious complications of pericardiocentesis are laceration and perforation of the myocardium and the coronary vessels. In addition, patients can experience air embolism, pneumothorax, arrhythmias (usually vasovagal bradycardia), and puncture of the peritoneal cavity or abdominal viscera. Internal mammary artery fistulas, acute pulmonary oedema, and purulent pericarditis were rarely reported. The safety was improved with echocardiographic or fluoroscopic guidance. Recent large echocardiographic series reported an incidence of major complications of 1–1.6%. In a large series of fluoroscopy-guided percutaneous pericardiocenteses, cardiac perforations occurred in