In this issue of the Netherlands Journal of Critical Care, Boendermaker et al. describe two cases of hepatic and renal dysfunction caused by neoplastic pericardial effusion and resulting in cardiac tamponade. The incidence of hepatic and renal dysfunction in critically ill patients is high and can have various causes. Because pericardial effusion, of oncologic origin, is not the most likely cause of these organ dysfunctions, critical care clinicians often omit considering cardiac tamponade as the underlying cause.

In most cases of pericardial effusion, the condition is found more or less by surprise. Once found, the question arises whether the pericardial effusion is symptomatic (i.e. cardiac tamponade) or asymptomatic (i.e. an innocent bystander without therapeutic consequences). The cases described by Boendermaker et al. nicely illustrate the clinical and therapeutic considerations and place of pericardial effusion and/or tamponade in the differential diagnosis of hepatic and renal dysfunction.

The etiology of pericardial effusion is quite diverse: it may be caused by infection (bacterial, viral, fungal, mycobacteria, protozoal), inflammation (auto-immune diseases), myocardial infarction (Dressler syndrome), cardiac surgery, trauma or chemical e.g. uraemia. Pericardial effusion resulting from a malignant process, such as neoplastic pericardial effusion, may occur directly (from the malignant process itself) or as a consequence of therapy e.g. radiotherapy, chemotherapy, necessary to treat the tumour. The signs and symptoms of cardiac effusion may manifest gradually, ranging from overt tamponade to no clinical signs at all. Besides this, most symptoms of cardiac tamponade are nonspecific, like anorexia, cough, hypotension, tachycardia, dyspnoea, tachypnoea and sometimes circulatory collapse. Also, like the cases presented by Boendermaker et al., patients may present with the complications of cardiac tamponade due to reduced organ perfusion resulting in hepatic and renal failure. In cases of pericardial effusion, physical examination may also reveal specific and nonspecific findings. Besides tachycardia, heart sounds may be attenuated, due to the isolating effects of the pericardial fluid. Clinically significant tamponade usually results in hypotension or shock. (i.e. Beck’s triad: hypotension, tachycardia and muffled heart tones). Jugular venous distention is usually present but may be absent in cases of hypovolemia. A key finding in cases of cardiac tamponade is pulsus paradoxus (PP). PP, however, is not pathognomonic for tamponade. It may also be present in cases of massive pulmonary embolism, profound haemorrhagic shock, and obstructive pulmonary diseases. PP is defined as a decline of 10 mmHg or more in systolic arterial blood pressure after inspiration during normal breathing. It may be palpable, but sometimes arterial catheterisation is needed to identify PP.

It has to be emphasized that cardiac tamponade is a clinical diagnosis. Additional investigations, however, may support the diagnosis of tamponade. Electrocardiography (ECG) may reveal signs of pericardial effusion: micro voltages, electrical alternans (as a sign of a swinging heart), PR-segment depression, ST-T segment alterations such as elevation and/or depression. These ECG findings are also not specific but may fit in with the diagnosis of pericardial effusion. Chest radiography may depict an enlarged cardiac silhouette with a tent-like shape. The cardiac silhouette, however, may also remain normal despite a large amount of pericardial effusion. Echocardiography is the principal tool for making the diagnosis pericardial effusion. Echocardiographic signs of cardiac tamponade include collapse of the right atrium and/or right ventricle. Left atrial collapse occurs in approximately 25% of patients and is highly specific for tamponade. Doppler echocardiography may show respiratory variations in transvalvular blood flow. This would be an indication that signs of PP can be demonstrated by Doppler echocardiography. By subcostal echocardiographic examination the inferior vena cava (IVC) can be visualized. In cases of tamponade, the IVC is usually dilated, with little or no collapse after inspiration during normal breathing. CT-scanning, MRI and right and left heart catheterisation may be considered but are generally not needed in the evaluation of pericardial effusion, unless it is necessary to search for underlying diseases such as malignancies.

So, what do we learn from the cases presented by Boendermaker et al.? First, pericardial effusion and tamponade should be considered in patients with hepatic and renal dysfunction that has been caused by diminished hemodynamics, thereby compromising the function.
of multiple organs including the kidneys and liver. Pericardial effusion in itself does not always result in cardiac tamponade: when it develops slowly, large amounts of pericardial fluid (> 2000 mL) may not result in tamponade, whereas small amounts of pericardial fluid (< 100 mL), accumulating in a very short time, may cause severe obstructive cardiogenic shock. Neoplastic pericardial effusion usually develops slowly with gradual clinical deterioration over time due to inflow obstruction; this may eventually result in cardiac tamponade, as described in the presented cases. Second, cardiac tamponade remains a clinical diagnosis. Echocardiography is essential to assess the existence of pericardial effusion and to find clues for the severity of inflow obstruction. It is to be emphasized, however, that even when there are negative signs of cardiac tamponade by echocardiography, a cardiac tamponade may be present. Clinical signs of cardiac tamponade, in the presence of pericardial effusion, are to be diagnosed as cardiac tamponade unless proven otherwise. Removal of the pericardial fluid either by pericardiocentesis or surgery, should not be postponed in cases of clinically manifest tamponade with negative echocardiographic signs of inflow obstruction. In all patients who are hemodynamically unstable, pericardial drainage has to be considered when there is a pericardial effusion.

References