The right ventricle is a thin-walled, low-pressure cavity with a lower metabolic demand than its left counterpart. The right ventricle receives its arterial blood supply mainly from the right coronary artery, however, because of its thin walls, it also receives oxygenation directly from the RV cavity and from the cardiac veins. It is, therefore, less commonly damaged in the setting of coronary artery occlusion. RV involvement during an ischemic event, however, is responsible for a typical clinical picture, and mandates particular attention in order to be properly diagnosed and treated.

Right ventricular (RV) infarction requires particular attention in order to recognize and treat it accordingly. The mechanical complications of myocardial infarction generally have a rapid clinical course with high mortality and require early diagnosis in order to properly manage the patient and refer to a center with cardiac surgery.

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RV infarction generally occurs in the setting of right coronary occlusion. The incidence is quite variable (between 10% to 50%), depending on the technique used for diagnosis, but is probably clinically relevant in approximately 15% of inferior myocardial infarctions. The clinical picture is also very variable, and depends mostly on the extent of myocardial damage to the right, as well as the left, ventricle.

**Clinical Features**

It is necessary to maintain a high clinical level of suspicion in order to make a prompt diagnosis of RV infarction. In the context of an acute inferior infarction, the presence of hypotension should lead one to suspect a possible RV infarction. The most common clinical findings are listed in Table 1. The classical clinical presentation consists of the triad of hypotension, distended jugular veins and clear lungs on auscultation. Because RV compliance is decreased, the increase in venous return on inspiration causes an increase in jugular venous distension, also called Kussmaul’s sign. Other findings are an RV heave, a systolic murmur of tricuspid regurgitation and the presence of a right S3, the latter two of which become more severe with inspiration.

The sensitivity and specificity of clinical findings are variable. Although the classical clinical

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**Kussmaul sign is an increase in internal jugular distension during inspiration, due to a decrease in right ventricular compliance seen in right heart infarction.**
The triad has a high specificity (96%) for diagnosis of RV infarction, its sensitivity is low (25%). The presence of elevated jugular veins alone has a sensitivity of 88% and specificity of 69%, however, the addition of Kussmaul’s sign increases specificity to almost 100% without decreasing sensitivity. Physical and hemodynamic findings, however, can be masked by volume depletion. Internal jugular veins are considered elevated or distended when their upper limit is greater than 5 cm above the sternal angle when examining the patient at a 45º angle. This would translate to a central venous pressure of at least 10 cm H₂O.

**Table 1**

**Clinical Findings in Right Ventricular Infarction**

<table>
<thead>
<tr>
<th>Findings</th>
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<tr>
<td>Jugular venous elevation</td>
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<td>Kussmaul sign</td>
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<td>Hypotension</td>
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<tr>
<td>Clear lungs</td>
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<tr>
<td>Right ventricular lift</td>
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<tr>
<td>Tricuspid regurgitation murmur</td>
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<td>Bradycardia</td>
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<td>Atrio-ventricular (AV) Block</td>
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Electrocardiography (ECG)

RV infarction most commonly occurs in the setting of an inferior left ventricular infarction, hence an ECG will show ≥1 mm (0.1 mV) ST-segment elevations in the inferior leads (II, III and aVF), combined with ST-segment abnormalities in leads V₁ and V₂. Typically, right chest leads V₃R and V₄R will have ≥1 mm ST-segment elevation (Figure 1). V₃R and V₄R are obtained by switching the precordial electrodes V₃ and V₄ from their respective positions on the left to the right side of the sternum. ST-segment elevation ≥1mm in V₃R has a sensitivity and specificity of approximately 80% for the diagnosis of RV infarction.³,⁴ Diagnostic accuracy is high when an ECG is performed early during acute infarction. Other common ECG findings are bradycardia and variable degrees of atrial-ventricular conduction disturbances.

**Physiology and Hemodynamics**

The hemodynamic findings in RV infarction are due to a combination of systolic and diastolic dys-
function of the right ventricle. The myocardial damage incurred by the right ventricle will lead to a proportional decrease in systolic function, which is partially responsible for the low output state that can be found in this condition. Right myocardial ischemia and necrosis also will lead to diastolic dysfunction with elevated RV end-diastolic pressure and increased central venous pressure. At catheterization, right atrial pressure generally will be above 10 mmHg. The ratio of right atrial pressure/pulmonary capillary wedge pressure generally will be equal to or above 0.8.\textsuperscript{1,2,5} Due to inter-ventricular dependence, RV dilatation also will interfere with left ventricular function, therefore, exacerbating the effect on lowering cardiac output.

Echocardiography

In general, it is not necessary to perform right-heart catheterization in order to diagnose RV infarction. Diagnosis usually is made on clinical presentation and ECG. Echocardiography permits non-invasive confirmation of the diagnosis and a complete evaluation of bi-ventricular and valvular function. It is also very useful in the differential diagnosis of RV infarction (see section on Differential Diagnosis).

Classic echocardiographic findings of RV infarction are a dilated and hypokinetic RV with a distended and non-compliant inferior vena cava. Tricuspid regurgitation is also a common finding. Generally, there will be segmental wall motion abnormalities in the inferior wall of the left ventricle.

Differential Diagnosis

The main diagnoses to be considered for a hypotensive patient with myocardial infarction are RV infarction, cardiogenic shock, cardiac tamponade and mechanical complications. In cardiogenic shock secondary to significant damage to the left ventricle, patients are generally tachycardic and have evidence of pulmonary congestion. They usually will present with anterior myocardial infarction. In cardiac tamponade, as with RV infarction, patients will be hypotensive with elevated central venous pressure, but they will usually be tachycardic and have pulsus paradoxus (inspiratory decrease of greater than 10 mmHg of arterial systolic blood pressure). The latter finding can, however, also be found in RV infarction. Electrocardiography could show low voltage and electrical alternans. Clinical evolution in tamponade is often dramatic. Echocardiography is the gold standard diagnostic tool. Mechanical complications are discussed below.
Management

The basic approach to therapy in RV infarction is the same as that for infarction of the left ventricle (i.e., rapid use of anti-platelet drugs and institution of reperfusion therapy, either with thrombolytics or primary angioplasty). The hemodynamics of RV infarction, however, requires certain therapies that are different from those used in the context of left ventricular infarction without RV involvement. Agents that lower blood pressure and veno-dilators, such as nitrates, morphine and diuretics, should be avoided as much as possible. Saline infusion (up to 2 L in the first 12 to 24 hours) often is required, and positive inotropic agents, such as dobutamine or milrinone are sometimes necessary if volume loading fails. Bradycardia and atrio-ventricular conduction disturbances could require atropine or sequential atrio-ventricular pacing with a transvenous pacemaker.

Prognosis

Prognosis in RV infarction is quite variable, depending on studies. Morbidity and mortality have been reported to be between 10% and 50%, and controversy surrounds whether RV involvement truly increases mortality. Overall prognosis is dependent on the degree of damage sustained by both ventricles during the infarction. Prognosis is improved by early reperfusion of the obstructed coronary artery. In the majority of survivors, RV function returns to normal.

Post-infarction Mechanical Complications

Mechanical complications can arise after a left ventricular myocardial infarction. These consist of myocardial rupture/tamponade (RT), ventricular septal defect (VSD) and acute mitral regurgitation (AMR). These complications generally present between one and six days after the infarct, have a high morbidity and mortality, and require rapid recognition and intervention. Although the first step in treatment is medical, their definite treatment requires surgical intervention. Patients with suspected or confirmed mechanical complications, therefore, should be treated in centers performing cardiac surgery.

Mechanical complications are uncommon, but are responsible for approximately 15% of the cases of mortality due to myocardial infarction. In a recently reported large registry of patients with cardiogenic shock, the incidence of RT was 1.4%, VSD 3.9% and AMR 6.9%. These complications had respective mortalities of 55%, 87% and 55%.11
Patients generally require medical stabilization with inotropic agents and vasodilators, such as nitroprusside, and left ventricular support with an intra-aortic counter-pulsation device. Myocardial rupture and tamponade require immediate intervention. Acute mitral regurgitation generally requires surgery within the ensuing hours. Optimal surgical timing is controversial in ventricular septal defects, however, it is generally agreed that earlier surgical intervention leads to a better prognosis.

Myocardial Rupture

Myocardial rupture occurs in approximately 1% of myocardial infarctions. Its incidence probably has decreased with the advent of reperfusion therapies. It generally occurs within the first five days post-myocardial infarction, and can affect any myocardial wall, but most commonly involves the lateral segment. Rupture generally occurs within a segment of necrotic tissue that is adjacent to normal myocardium. Clinical evolution is usually very rapid, and patients present with hemodynamic compromise and clinical features of tamponade, possibly resulting in sudden death with electromechanical dissociation. When diagnosed, the only possible treatment is rapid surgical intervention, which, nevertheless, carries a poor prognosis. The role of pericardiocentesis is controversial.

Rupture may be sub-acute with warning signs of pericardial pain and hypotension with jugular
venous elevation preceding acute hemodynamic compromise.\textsuperscript{12} Patients with suspected myocardial tamponade or rupture of the left ventricular free wall should undergo immediate two-dimensional and Doppler echocardiography in order to establish a diagnosis.

A left ventricular pseudo-aneurysm or false aneurysm represents a contained myocardial rupture by inflammatory pericardium. It is diagnosed with echocardiography or left ventricular angiography. This is a different entity from a true left ventricular aneurysm, which represents a necrotic and thinned bulging segment of myocardium that contains endo-, myo- and epi-cardium. The wall of the false aneurysm is made only from pericardium and hematoma. Due to the relatively high risk of eventual rupture, a false left ventricular aneurysm should be surgically corrected promptly.

**Ventricular Septal Defect**

Ventricular septal defect (VSD), also called ventricular septal rupture, resulting from myocardial infarction, occurs in approximately 2% of cases, usually presenting during the first days post-infarction. Its incidence probably has decreased in the post-thrombolytic era.\textsuperscript{13} The VSD varies in dimension, from 1 mm to a few centimeters. VSD results in a left-to-right shunt that leads to left ventricular volume overload, and the clinical picture of pulmonary edema and hemodynamic compromise. Patients with VSD usually will have a new, loud, pansystolic murmur, often associated with a palpable thrill. VSD is located in the apical septum when resulting from an anterior myocardial infarction, or in the basal posterior septum when resulting from an inferior myocardial infarction. Right heart blood sampling will show oxygen saturation step-up at the level of the right ventricle (O\textsubscript{2} saturation increases by 5% or more between the right atrium and ventricle). The diagnosis, however, is generally made with two-dimensional and color Doppler echocardiography (Figure 2). Medical management alone of VSD generally carries a dismal prognosis with mortality rates greater than 90%.\textsuperscript{13} Patients with cardiogenic shock and VSD can survive only if operated on, however, even with surgical intervention, mortality is approxi-
mately 50%. The optimal timing of surgery in patients without cardiogenic shock is controversial. The clinical course for patients who are stable with VSD is often marked by rapid deterioration. Therefore, it is generally agreed that early surgical intervention is more appropriate.

**Acute Mitral Regurgitation**

Post-infarction acute mitral regurgitation (AMR) results from partial or complete rupture of posterior or medial papillary muscle. Contrary to the anterolateral papillary muscle that receives dual blood supply from the left anterior descending and circumflex coronary arteries, the postero-medial papillary muscle receives its blood supply solely from the posterior descending coronary artery. An AMR, therefore, generally will be observed in patients with inferior myocardial infarction, and it presents within the first week post infarction. Patients with papillary muscle rupture will show rapid clinical deterioration with acute pulmonary edema and, often, hypotension. Complete rupture of the papillary muscle is usually fatal. Partial rupture, however, can be managed with initial medical therapy consisting of diuretics, inotropic agents, pulmonary arterial vasodilators and an intra-aortic counter pulsation device. Of note, because of pressure equalization between the left atrium and ventricle, a systolic mitral regurgitant murmur is present in only about 50% of cases. Rapid clinical deterioration with pulmonary edema, therefore, should lead one to suspect the presence of significant mitral regurgitation, which is readily diagnosed with transthoracic or transesophageal echocardiography (Figure 3). As with other mechanical complications, medical therapy alone carries a very poor prognosis, with a mortality rate of approximately 90%. Early surgical intervention generally is required.

Ischemic mitral regurgitation without papillary muscle rupture can occur in up to 50% of cases. Although, the prognosis is not nearly as poor as with papillary muscle rupture, the presence of peri-infarct mitral regurgitation is associated with a poorer prognosis. In such cases, the treatment is aimed at the underlying coronary disease.

**Summary**

Patients with RV infarction and post-infarction mechanical complications generally present with typical clinical features. Awareness of the clinical features and diagnostic tests will lead the clinician to a prompt diagnosis and proper management in order to improve the outcome in these patients, who generally have a poor prognosis.

**References:**

76:44-51.

Put Your Knowledge to the Test

Answer the questions in our quiz found on page 183 and send the response card to the University of Calgary for CME credits.