SUMMARY

The appropriate management of patients with acute pulmonary embolism (PE) who present with right ventricular (RV) dysfunction but normal arterial blood pressure (i.e., those with submassive PE) continues to be highly controversial. Recently, a number of studies have improved our understanding of subclinical RV dysfunction in PE. Despite the limitations of echocardiographic criteria for diagnosing the enlargement and/or dysfunction of the right ventricle, ample evidence supports the notion that RV dysfunction diagnosed by echocardiography (echo) is an independent predictor of early mortality and complications in normotensive patients with PE. Retrospective studies showed that similar prognostic data can be obtained using multidetector-row chest computed tomography (CT). Moreover, very recent reports indicate that biomarker, particularly troponin, testing followed by echocardiographic or CT imaging of the right ventricle may be an efficient and reliable strategy both for excluding (ruling out) and for predicting (ruling in) a poor outcome in patients with PE. Thus, novel risk stratification algorithms help identify possible candidates for early thrombolytic treatment in PE and thus provide the background for a large controlled trial that will hopefully resolve the 30-year-old debate on the benefits of thrombolysis in normotensive patients with PE and RV dysfunction.
Key Words: Right ventricle; prognosis; biomarkers; echocardiography; computed tomography.

INTRODUCTION

As early as 35 yr ago, McIntyre and Sasahara demonstrated that right ventricular (RV) pressure overload and dysfunction are critical events in the pathophysiology of pulmonary embolism (PE), and that they may be important determinants of the patients’ outcome in the acute phase (1,2). At present, it is widely acknowledged that RV dilation and hypokinesis resulting from acute pressure overload is capable of initiating a vicious cycle of increased myocardial oxygen demand, myocardial ischemia (or infarction), left ventricular preload reduction, and, ultimately, inability to maintain the cardiac index and arterial pressure (3). If left untreated, this sequence of events will lead to cardiogenic shock, and, in fact, most PE-related deaths are due to refractory cardiac (rather than respiratory) failure.

Overt RV failure with hemodynamic instability and shock due to massive, fulminant PE does not pose diagnostic problems, and its detection certainly does not require sophisticated imaging procedures or biochemical tests (see Chapter 8). This condition is associated with a very poor prognosis and mortality rates of up to 65% in the acute phase (4). As a result, there is consensus (5–7), even in the absence of large randomized trials (8), that hemodynamically unstable patients with PE should undergo emergency pulmonary artery recanalization using thrombolytic agents (see Chapters 10 and 11), or by means of surgical (see Chapter 12) or interventional (see Chapter 13) procedures. On the other hand, it remains highly controversial whether the diagnosis of RV dysfunction in normotensive patients with acute PE (so-called submassive PE) should also alert clinicians and prompt them to administer thrombolytic treatment in order to prevent (rather than treat) cardiogenic shock (9–12). In fact, the results of meta-analyses of the randomized trials that tested thrombolysis in PE appear to contradict its (presumed) clinical benefits in this setting (13,14).

This and the following chapter will focus on recent studies that improved our understanding of subclinical RV dysfunction in PE. The data obtained in these trials provide the background for emerging risk stratification algorithms, which will hopefully succeed in identifying high-risk normotensive patients and thus help resolve the ongoing debate on the possible indications for thrombolysis in submassive PE.

CAN THE ELECTROCARDIOGRAM SUGGEST RV DYSFUNCTION?

Although the electrocardiogram (ECG) on admission can, by itself, neither confirm nor exclude the diagnosis of acute PE in a given patient, a number of earlier reports suggested that specific ECG changes may correlate with the severity of PE (15–17). More recently, Geibel et al. provided data to support the notion that simple ECG parameters could serve as a noncostly, ubiquitously available tool for initial triage of patients with PE, i.e., for raising the suspicion of RV dysfunction and for prompting further risk stratification of PE (18). The authors performed multivariate analysis in 508 patients with acute massive or submassive PE derived from the 1001-patient Management Strategies and Prognosis in Pulmonary Embolism (MAPPET) registry (19). The presence of at least one of prespecified ECG abnormalities (atrial arrhythmias, complete right bundle branch block, peripheral low voltage, pseudoinfarction pattern in leads III and aVF, or ST segment changes over the left precordial leads) on admission was associated with an elevated in-hospital