The clinician confronted with a patient suspected of having acute pulmonary embolism (PE) faces two immediate challenges. First, coming to the correct diagnosis may be difficult and second, it is essential to start appropriate treatment without delay. As thrombolysis, anti-coagulation and mechanical devices to remove the embolus are potentially dangerous types of treatment, all cases require individual risk stratification. In this issue of NJCC, Salet et al. describe the treatment of a patient with recurrent massive pulmonary embolism (PE) that draws attention to catheter thrombectomy.

PE is a relatively common cardiovascular emergency. The yearly incidence of PE in the Netherlands is unknown, but estimated at 2.6 per 1,000 inhabitants [1]. The clinical presentation of PE ranges from asymptomatic to haemodynamic shock and imminent death. PE must be suspected in patients presenting with new onset of (worsening) dyspnoea and chest pain or sustained hypotension without any other identifiable cause. Clinical prediction scores, such as the revised Geneva score, may help the clinician to assess the likelihood of PE whether acute or not. The revised Geneva score consists of 8 entirely clinically based variables. In the validation set, the prevalence of PE was 8% in the low-probability category (0 to 3 points), 28% in the intermediate-probability category (4 to 10 points), and 74% in the high-probability category (≥11 points) [2].

The diagnostic work-up for PE in haemodynamically stable patients consists of clinical probability assessment, D-dimer testing and multidetector computed tomography (CT) [3]. PE suspected in haemodynamically unstable patients, requires a far more aggressive strategy that is tailored to the need of the individual patient. CT should be performed immediately and is the hallmark for diagnosing acute PE in haemodynamically unstable patients. In acute PE, anatomical obstruction is undoubtedly the most important cause of compromised physiology. CT has a 97% sensitivity for detecting emboli in the main pulmonary arteries [3]. If, however, the patient, as presented by Salet et al., is too haemodynamically unstable for a CT scan to be performed, evidence of right ventricular dysfunction by transthoracic or transoesophageal echocardiography (which can be done at the bedside) may be used to strengthen the diagnosis of PE.

Aggressive management entails simultaneously initiating supportive care (e.g. mechanical ventilation, inotropic support), confirming the clinical diagnosis of PE and guiding immediate treatment by the estimated risk of adverse outcome. Risk stratification is based on clinical features and markers of myocardial dysfunction or injury [3]. Here the monitoring of haemodynamics is of key importance. Acute right ventricle failure with resulting low systemic output is the leading cause of death in patients with PE. In the International Cooperative Pulmonary Embolism Registry, the death rate was nearly 58% among haemodynamically unstable patients and about 15% among haemodynamically stable patients [4]. The markers for right ventricular dysfunction and injury help identify the haemodynamically stable patients at risk. Importantly, one should realize that these markers predominantly have a high negative predictive value.

The first-line treatment in patients with high-risk PE presenting with cardiogenic shock and/or persistent arterial hypotension is thrombolytic therapy [2,3,5]. Although thrombolytic therapy carries a significant risk of bleeding, in a patient with severe life-threatening high-risk PE few absolute contraindications remain relevant for postponing therapy. Nevertheless, as the authors acknowledge, initiation of thrombolytic therapy should be preceded by thorough diagnostic procedures. Thrombolytic therapy rapidly resolves thromboembolic obstruction and exerts beneficial effects on haemodynamic parameters. More than 90% of the patients respond favourably within the first 36 hours. In view of the mechanical device applied in the case report of Salet et al., it is important to define non-responders. Non-responders demonstrate persistent clinical instability, defined as the presence of at least two of the following criteria: refractory cardiogenic shock; systemic arterial hypotension (defined as systolic BP of ≤ 90 mm Hg or a pressure drop of ≥ 40 mm Hg for > 15 min if this has not been caused by new-onset arrhythmia, hypovolaemia, or sepsis); severe hypoxaemia (i.e., room-air pulse oximetry of ≤ 90% or PaO2 without oxygen therapy of ≤ 55 mm Hg); or tachycardia (heart rate ≥ 110 beats/min) and residual echocardiographic right ventricular (RV) dysfunction defined as the persistence of at least two criteria (i.e., RV/left ventricular end-diastolic diameter ratio ≥ 1 in the four-chamber view; paradoxical septal systolic motion and/or pulmonary hypertension, defined as a RV/atrial gradient > 30 mm Hg) [5].

The treatment of PE patients is often ground for bedside discussions and also in the case described by Salet et al. several comments can be made. The authors acknowledge that the decision to initiate thrombolytic therapy should be taken carefully and that the diagnostic work-up was not complete at presentation. Second, although failing of thrombolysis is one of the indications for percutaneous mechanical thrombectomy [2,3,5],
the described patient initially improved haemodynamically after administration of thrombolytic agents. So, one could argue that a second attempt with thrombolysis should have been performed when the patient deteriorated several days later. Finally, a combination of the mechanical approach with intra-arterial pharmacological thrombolysis described by the authors is open for discussion, although this is associated with an increased risk of bleeding at the insertion point.

Despite these relevant issues, the case report usefully draws attention to percutaneous mechanical thrombectomy as an alternative to surgical embolectomy, especially in cases in which immediate access to cardiopulmonary bypass is unavailable. Numerous devices are available today, none with apparent benefit over the other. In a recent meta-analysis of case series, catheter-directed therapy had a clinical success rate of 86% and a rate of major procedural complications of 2.4% (95% CI, 1.9 to 4.3) [6]. It is crucial to terminate the procedure as soon as haemodynamics improve. The haemodynamic improvement can be dramatic in spite of an angiographic result that is minimal.

In conclusion, acute PE in haemodynamically unstable patients will almost always remain a clinical challenge in which aggressive management is of key importance. Thrombolytic therapy is the first line treatment of severe PE with persistent hypotension. Catheter thrombectomy should only be considered in carefully selected patients with absolute contra-indications or failure of thrombolytic therapy. It represents a possible alternative to surgical removal of the embolus.

References