According to the American College of Chest Physicians (ACCP) guidelines [1], low-molecular weight heparin and fondaparinux are preferred treatment modalities for acute pulmonary embolism (PE). In addition, in selected patients these guidelines recommend thrombolytic therapy: in patients presenting with an acute PE associated with hypotension and also patients who reveal a high risk of developing hypotension. Of note, patients managed with fibrinolytic agents should not have a high bleeding risk.

Surgical embolectomy or endovascular removal of emboli can be an option in selected patients with submassive acute PE, especially those with emerging hemodynamic or respiratory instability. Also, patients who do not improve after thrombolysis should be considered for surgical or endovascular embolectomy. Recent advances in endovascular techniques, such as aspiration thrombectomy, endovascular thrombus fragmentation devices and rheolytic or rotational thrombectomy, suggest that the endovascular approach can represent an alternative for pharmacological thrombolysis and surgical embolectomy.

**Key words:** pulmonary embolism, endovascular thrombectomy, AngioJet.
Technique of endovascular rheolytic thrombectomy using AngioJet for treatment of acute pulmonary embolism

confirmed and a decision to address it using the rheolytic embolectomy is made, the introducer sheath should be replaced with a bigger one, preferentially an 8F sheath. The embolus should be reached using a multipurpose 8F guiding catheter (the AngioJet system is compatible with 6F and 8F catheters). We suggest using the SiteSeer Judkins Right catheter (Medtronic, Minneapolis, MN, USA) or a long sheath with a special tip, such as the Destination Guiding Sheath (Terumo, Tokyo, Japan). Through this guiding catheter, over a guidewire that should be navigated across the embolus (preferentially, a hydrophilic

Fig. 1. Initial angiography of the pulmonary arteries in a patient with pulmonary embolism (RPA – right pulmonary artery)

Fig. 2. Introduction of AngioJet system into the middle branch of the right pulmonary artery

Fig. 3. Recanalisation of the middle branch of the right pulmonary artery after use of the AngioJet system

Fig. 4. Residual thrombi in the upper branch of the right pulmonary artery (RPA)

Fig. 5. Introduction of AngioJet system into the upper branch of the right pulmonary artery
one, still advanced carefully), the AngioJet system (Boston Scientific, Natick, MA, USA) is introduced (Fig. 2). Using this endovascular device the emboli should be subsequently aspirated from all occluded branches (Figs. 3-6).

In general, embolectomy should be discontinued as soon as the patient’s haemodynamic status stabilizes, even if the angiographic result is not perfect. As emphasized in the introduction, embolectomy of small branches of the pulmonary artery should be avoided, because navigation through these tiny vessels dramatically increases the risk of haemorrhage. If the above-described attempts to restore the patency of pulmonary arteries are not effective, direct local administration of a fibrinolytic agent (on condition that there are no contraindications for such treatment, especially intraprocedural injury of the artery) may be helpful. During aspiration of embolic materials dyspnoea and cardiac decompensation can develop, especially if an aspiration lasts longer than 7-10 s. Thus, longer use of the AngioJet system should be avoided. Also, during the procedure some patients may develop bradycardia. Therefore, some authors recommend insertion of a temporary transvenous pacemaker. Careful preprocedural assessment of the patient should reveal whether such a pacemaker is indeed necessary. It should also be remembered that hemodynamic instability seen in PE patients, especially right ventricle overload, is not solely related to mechanical occlusion of pulmonary arteries, but also is associated with vasoconstriction of the pulmonary vasculature. Although the exact mechanism of such vasoconstriction in the setting of PE has not yet been elucidated, it is thought that haemolysis resulting from PE itself and also from pharmacological treatment with fibrinolytic agents reduces the concentration of nitric oxide (NO) in the pulmonary circulation through scavenging of NO by cell-free haemoglobin [11]. Perhaps other vasoactive substances, such as adenosine and bradykinin, may also play a role in the development of pulmonary vasoconstriction. Furthermore, severe haemolysis can lead to significant hyperkalaemia and also induce renal failure, which should be considered when managing PE patients.

The authors declare no conflict of interest.

References


