CIRSE Guidelines on Diagnosis and Treatment of Pulmonary Arteriovenous Malformations

Introduction

Pulmonary arteriovenous malformations (PAVMs) are rare congenital vascular anomalies of the lung in which abnormally dilated vessels provide a direct capillary-free communication between the pulmonary and systemic circulations with three main clinical consequences:

1) pulmonary arterial blood passing through these right-to-left shunts cannot be oxygenated, which may lead to arterial hypoxemia;
2) the absence of normally filtering capillary bed allows thromboembolic material to reach the systemic circulation (paradoxical embolism), which can result in transient ischaemic attack and stroke, while bacterial embolisation has been reported to result in brain abscess;
3) rupture of the thin-walled PAVMs can lead to haemoptysis or haemothorax, particularly during pregnancy when hormonal changes may induce a rapid enlargement of PAVMs.

Churton first described PAVMs in 1897 following the autopsy of a cyanotic boy who suffered from recurrent episodes of haemoptysis [1]. In 1938, Rodes described the association between PAVMs and hereditary haemorrhagic telangiectasia (HHT) [2], which is an autosomal dominant inherited vascular disease. PAVMs are about twice as common in women, with a male-to-female ratio of approximately 1:1.5 to 1.8 [3]. The estimated incidence is thought to be around 2-3 per 100,000 (4). A recent analysis of data gathered using thoracic CT scanning determined that PAVMs affect 38 individuals out of 100,000 (approximately 1 in 2,600) [5]. Around 70% of PAVM cases are associated with HHT and 15-35% of patients with HHT will have a PAVM.

Up to 55% of PAVMs are asymptomatic and most of the clinical manifestations can be attributed to hypoxia and to right-to-left shunting. Symptoms related to PAVM often develop between the fourth and sixth decades. The incidence of symptoms is higher in patients with multiple and large PAVMs rather than a single PAVM. Patients with diffuse PAVMs are almost always symptomatic [6]. Incidence of stroke has been reported in about 30% of the patients and brain abscess in 10-20% [7] with a mortality of up to 40% [8]. It is estimated that 18% suffer from a transient ischaemic attack and 10% experience cerebral abscess on presentation of PAVMs [9]. Haemoptysis or haemothorax have been reported in roughly 3-10% of patients [10]. These data illustrate the need for aggressive screening of HHT patients and treatment of PAVMs (level of evidence 2a). These figures might, however, be somewhat overestimated due to selective reporting and publication bias.

The first successful surgical treatment of PAVMs involved a pneumonectomy performed in 1942 [11]. Werner Porstmann described pulmonary fistula in children [12] and performed the first PAVM embolisation in 1977. Embolisation therapy has been considered the gold standard in the treatment of PAVMs since 1983 [13]. Due to ethical concerns, there have been no randomised controlled trials...
specifically examining the efficacy of embolisation therapy for PAVMs compared with surgery and conservative treatment.

Definitions

PAVMs are arteriovenous communications between the pulmonary and systemic circulations causing a right to left shunt through the PAVM bypassing the normal filter function of the lungs. Therefore, PAVMs may cause serious neurological symptoms like cerebral abscess or stroke due to paradoxical embolism. About 80% of PAVMs are classified as simple, fed by a single artery contained within one pulmonary segment. The remaining 20% are deemed complex, with feeding arteries from more than one pulmonary segment [14]. A diffuse involvement of one or more segments or lobes accounts for 5%. HHT is an autosomal dominant disorder characterized by a wide variety of clinical manifestations due to the presence of multiple arteriovenous malformations. The most common clinical manifestation in HHT is spontaneous and recurrent epistaxis in more than 90%. In patients with PAVM, about 40-70% will have functional dyspnoe, 40-50% headache or migraine, 20-35% cerebrovascular incidences, 5-10% hemoptysis but more than 10% will be asymptomatic.

Pre-treatment Imaging/Evaluation

a. Contrast-enhanced echocardiography

In patients with symptoms or signs of a PAVM by history and/or physical examination, contrast-enhanced echocardiography (CE) remains the best initial screening tool due to its high sensitivity and a negative predictive value close to 100% [30] (level of evidence 2a). If the result is negative, no further evaluation is necessary, since the likelihood of a significant PAVM with feeding artery ≥3mm (i.e. requiring embolisation) is very low. However, some advocate using another negative study, such as pulse oximetry or low-dose CT, to increase the negative predictive value. Conversely, all patients with positive CE should be evaluated using CT to identify PAVMs amenable to embolisation [31]. In addition, initial CT will be used as a baseline study that can be compared with post-embolisation examinations [17]. CE is the first-line test in screening patients with HHT for intrapulmonary right-to-left shunting with the appearance of microbubbles after 3–10 heart beats into the left atrium. A significant number of patients with a positive CE will not have PAVMs visible on pulmonary angiography with the indication to treat (with feeding arteries of ≥3mm). In a study from Nanthakumar et al. of 41 patients with positive CE, 8 were negative for PAVMs in following pulmonary angiograms [15]. Detection of PAVMs with a feeding artery of less than 3 mm limits the use of CE as an exclusive screening test for PAVM in cases where there might not be an indication to treat or as control for growth of the PAVM.

b. Chest radiography
Diagnosis of PAVMs may be suspected on chest radiographies (CRX), as abnormal findings have been described in most patients with PAVMs. The most common findings are peripheral circumscribed, non-calcified oval or round lesions connected by blood vessels to the hilus or the presence of nodules often described as coin lesions. However, a normal chest radiograph does not exclude PAVM [16].

c. **Computed tomography**

When CE is positive, computed tomography (CT) should be performed to confirm the diagnosis and evaluate if treatment is indicated. The characteristic appearance of a PAV on CT is a homogeneous, well-circumscribed, non-calcified nodule or the presence of a serpiginous mass connected with blood vessels [17]. The use of contrast media is usually not necessary because CT and multiplanar reconstructions usually allow identification of the feeding artery, aneurysm sac and efferent vein without contrast injection. Iodine-contrast administration may help identify systemic supply to large PAVs via the systemic arteries [18]. In patients with HHT, use of contrast-enhanced CT allows simultaneous evaluation of thoracic and abdominal involvement, which may help confirming the diagnosis of HHT based on the Curacao criteria [19,20]. Low-dose CT plays an important role in children, fertile and pregnant women, and in repeated checks for the growth of small PAVMs until they reach a size that indicates treatment. When CT is negative, repeating the scans at least at 5-year intervals and before pregnancy is recommended to look for PAVM growth [17,20].

d. **Magnetic resonance imaging**

Magnetic resonance imaging (MRI) of PAVMs has been evaluated less than CT. Conventional spin-echo MRI of pulmonary nodules or vascular lesions shows lesions with high signal intensity on T2-weighted images. Several techniques have been recently developed to improve sensitivity to flow. Use of gradient-refocused echo MRI technique or MR angiography with venous or arterial signal elimination or contrast injection has been reported to have a high sensitivity [21].

e. **Pulmonary angiography**

With the intention to treat, selective angiograms of both pulmonary arteries should be performed with imaging in at least two projections of each lung. Although very sensitive, pulmonary angiography (PA) can miss PAVMs smaller than 2 mm as compared with CT. With the introduction of CT allowing high isotropic multiplane reconstructions and the improvement of MRI sequences, the use of PA is no longer recommended as a diagnostic tool.

f. **Indications for treatment**

If one of the following criteria is met, embolisation is indicated:
• Any (solitary or multiple) PAVM with a feeding artery with diameter of 2 mm or larger
• Measurable increase in size of PAVM
• Paradoxical emboli or symptomatic hypoxemia

Pregnancy is a special risk factor in patients with PAVM, especially in the second and third trimesters due to decrease in peripheral vascular resistance and increase in cardiac output by nearly 50% [22]. A recent study in 244 pregnant women with HHT showed major complications in 13%, all in patients who had not been screened or treated for PAVMs prior to pregnancy [23]. Thus, all women with HHT considering pregnancy should be screened for PAVM by CT and eventually treated prior to conception. If women are pregnant and found to have asymptomatic PAVMs, they should not be treated during pregnancy, mainly due to the potential risk of radiation exposure to the foetus. However, if any pregnant woman with HHT develops haemoptysis or sudden dyspnoea, urgent hospitalisation and further diagnosis is recommended; embolisation is then to be considered. For all pregnant women with HHT, antibiotic prophylaxis during delivery is recommended [24].

a. Size of feeding arteries

There is an arbitrary lower limit of 3 mm [25] or 2–3 mm [26] to treat PAVMs. This is empirically defined and based on the fact that patients very seldom experience cerebral events below this size [27]. Technical advances in microcatheter design and embolisation devices may decrease this cut-off size. Antibiotic prophylaxis is recommended if CE is positive, regardless of the size of the feeding artery on CT scan in patients undergoing surgical and dental interventional procedures [28]. However, recently published long-term data from the Irish National HHT Centre on the natural progression of untreated small (< 3 mm) or microscopic (positive CE, negative CT) PAVM enlargement was found to be more infrequent than suspected (7%) [29].

b. Recommendation

The current practice thus recommends embolisation of all patients with treatable PAVM regardless of size (level of evidence 2a).

Contraindications to treatment

There are no absolute contraindications to treat PAVMs. Relative contraindications to treatment include:

- Anaphylactoid reaction to contrast media
- Renal failure
- Pulmonary hypertension (test occlusion before permanent embolisation)
- Hyperthyroidism
- Coagulopathy
- Pregnancy (see chapter IV)
Equipment specifications

Digital subtraction angiography (DSA)

Ceiling- or floor-mounted C-arm (single- or bi-plane) with radiation-reducing software, ideally including pulsed fluoroscopy. High picture framerates (i.e. 4–6 frames/second) are mandatory for the diagnostic runs.

Procedure

When obtaining consent, the patient should be informed about the procedure itself, the potential complications and side effects related to the procedure and the expected outcome as well as available treatment alternatives.

After correct skin preparation, including disinfection of the puncture area and draping the patient, the procedure is performed under local anaesthesia in a sterile fashion. Venous access is performed typically through a common femoral vein or alternatively with an internal jugular vein or cubital vein approach. A 6-7 Fr sheath is introduced and a 4-5 Fr pigtail catheter sequentially to the right or left pulmonary artery. Diagnostic pulmonary angiography is taken with high frame rates (4 - 10 frames/sec) and different angulations as needed to identify all treatable PAVMs and their feeding arteries. Pulmonary artery pressure (PAP) is measured to assess whether pulmonary hypertension (mean pressure > 20 mmHg) is present primarily or secondarily due to liver AVMs, especially in HHT patients.

Carefully avoid any air bubbles in the intravenous line or the catheters, as these can result in paradoxical air embolisation to the heart or brain, possibly causing infarction. Treat one lung at a time to avoid the possibility of bilateral pleurisy if many bilateral PAVMs are present. This recommendation is based on common sense, not on evidence (level of evidence 5).

Catheter equipment and embolic agents:

A 260-cm J-tip stiff guidewire is used for exchange to a dedicated coaxial system (6-7 Fr 65-80 cm long curved sheath or multi-purpose guiding catheter and a 4-5 Fr end-hole angled-tip inner catheter like Cobra/MPA selective catheter, micro-catheters and -guidewires may be required). The guidewire should always be removed from the end-hole catheter under saline to prevent air embolism. The guiding catheter is connected with a continuous saline flush line to minimise the risk of thrombus formation at the catheter tip. The inner catheter is inserted into each feeding artery, and the catheter tip position should be adjusted to avoid catheter wedge and ensure blood aspiration. Selective angiography of the feeding artery is obtained in bi-plane or multi-direction to find a suitable working angle for embolisation device placement. Injection
of contrast material should be careful to avoid air bubble or small thrombi. The target vessel diameter is measured.

**Embolic agents:**

The choice of embolic agent depends on vascular anatomy, vessel size and operator’s preference. Coils 5 – 14 mm depending of vessel size (fibred and non-fibred, hydrocoils, pushable and detachable) or vascular plugs (AVP) 8–20 mm for larger PAVMs (diameter > 10 mm) are available. Microvascular plugs (MVP) are also available in smaller sizes for use through micro-catheters. Careful dense packing and cross-sectional obliteration of the feeder is mandatory. Coils and plugs should be oversized 20–25% compared to vessel diameter.

**Procedure:**

For coil embolisation of simple-type PAVMs, the inner catheter is advanced as close to the venous sac as possible, beyond any branch supplying normal lung, preferably 1 cm within the venous sac to minimise recanalisation or reperfusion of PAVM [32]. The guiding catheter is also placed close to the inner catheter to obtain adequate support during device deployment. In a large feeding artery with a risk of coil migration, the anchor technique, scaffold technique or balloon-occlusion technique can be applied to secure coil stability [33]. If there is difficulty in selective insertion of the inner catheter because of small size, tortuosity or complex anatomy of the target vessel, a microcatheter can be used to deliver microcoils. Detachable microcoils are available which give a possibility to retract and reposition the coils and thus a more precise and secure delivery, but they are more expensive than pushable standard coils [34].

The diameter of the first spiral is recommended to be 20% or 2 mm bigger than the selected target vessel. Further, smaller spirals follow until the afferent artery is tight packed and the PAVM is occluded. A controlled release and exact positioning a longer spiral can also be done by anchoring the first 1-3 cm within a small, distal side branch (so-called anchor technique). Then, by retracting the catheter, the remaining spiral within the target vessel is detached.

For larger diameter PAVMs (> 10 mm), or high flow rate and short supplying artery, (detachable) AVPs have been increasingly used for a faster or safer procedure, although long-term data are still limited [35] (level of evidence 2b).

Similarly to coils, AVPs should be placed as distal as possible in the feeding artery. As an AVP takes certain time until complete blood flow cessation, addition of platinum coils is proposed to prompt occlusion [36] (level of evidence 3b). AVPs may also act as an anchor to prevent coil migration in a large high-flow vessel.

In PAVMs with short feeding arteries and large outflow veins that preclude safe coil placement in feeding arteries, venous sac embolisation can be an alternative to feeding artery embolisation with use of detachable microcoils larger than the diameter of drainage vein. However, it remains controversial whether venous sac embolisation should be routinely performed. Although unproven, venous sac embolisation may reduce the incidence of recanalisation of PAVMs compared to feeding artery embolisation [32] (level of
evidence 3b). On the other hand, venous sac embolisation has disadvantages to increase procedure time, radiation exposure, cost and potential risk of rupture. In addition, the metallic coil artefacts in the sac hinder assessment of sac shrinkage on follow-up CT.

Liquid embolic agents do not play a role in the embolisation of PAVMs. Especially for diffuse PAVM with numerous AV shunts, pulmonary flow redistribution technique might be an option to improve hypoxia, although available data are limited [37] (level of evidence 3b). In this technique, the most severely affected segmental arteries are occluded from peripheral to central by dense coil packing to alter the pulmonary blood flow to the less-affected portion of the lung.

After embolisation, PA with waiting time of 5–10 minutes is performed to confirm the occlusion of PAVM or detect any accessory feeder missed on the baseline angiography. Retrograde filling of the venous sac via the draining vein from normal parenchyma may mimic a residual shunt of PAVM. Multiple treatment sessions are considered in patients with multiple PAVMs, and the duration of each procedure depends on the number and complexity of PAVMs, the amount of contrast material used and patient tolerance.

**Medication and peri-procedural care**

There may be variations in practice regarding the use of intravenous conscious sedation or attendance of the anaesthesiologist during the embolisation procedure. Treatment of paediatric patients should always be performed under general anaesthesia.

During the coil embolisation procedure, heparin (100 units/kg or 3,000-5,000 units) is administered intravenously after the sheath is introduced in the vein. Additional heparin is given every hour to keep an activated clotting time around 200 seconds. It is believed that heparinisation will help to reduce the risk of thrombus formation around the catheter and coils placed within a shunt vessel. However, some practitioners using AVPs do not use heparin to prompt vessel occlusion with a single device [35].

Some recommend antibiotic prophylaxis before the procedure, but it is not based on any evidence.

**Post-treatment evaluation**

Clinical evaluation with combined clinics for these patients by oto-rhino-laryngologists, cardiologists, and interventional radiologists is mandatory.

Imaging follow-up of treated patients in conjunction with clinical and physiological evaluation should be performed to follow involution or reperfusion of embolised PAVMs and to detect growth or enlargement of small PAVMs [38]. Remove intravenous lines at the earliest opportunity to prevent iatrogenic paradoxical embolisation of air or thrombus through residual PAVMs.
Low-dose CT remains the reference imaging modality to assess embolised PAVMs, particularly when comparing with pre-treatment images [17]. Follow-up with CT evaluation one or more years after embolisation showed that 96% of treated PAVMs were either undetectable or reduced in size. Shrinkage of the aneurysmal sac associated with reduction of the diameter of the draining veins are considered key imaging findings for treatment success [17,18].

Outcome

The most relevant data are summarised in Table 1. Post-embolisation morbidity and mortality may occur due to reperfusion of embolised PAVMs or growth of previously non-embolised small untreated PAVMs [29]. Recanalization is the most frequent cause of recurrence and re-treatments of embolized PAVMs. Standard coils should probably not be the first choice for embolization of PAVM, especially not in big-sized feeding arteries and complex PAVMs. If anatomically accessible AVP alone or in combination with coils seems to be the best primary option for embolization. Special coils like hydrocoils and other kinds of detachable coils, microcoils and micro vascular plugs may also be good options but this awaits documentation (51) (level of evidence 3a).

Reperfusion of carefully embolised PAVMs predominantly affects large and/or complex PAVMs [6,37]. Reperfusion may be due to several mechanisms such as insufficient cross-sectional occlusion (coil packing) [6], missed small accessory branches to the PAVM, or recruitment of initially normal branches adjacent to the PAVM [39]. Small branches supplying the embolised PAVM may also be missed during follow-up CT evaluation, particularly in the absence of contrast enhancement or due to coil-related artefacts [39,40].

Bronchial artery hypertrophy has been identified as a cause of reperfusion. Bronchial-to-pulmonary artery anastomoses may enter the pulmonary circulation distally to the embolised pulmonary feeder and may lead to future recanalisation [41]. It is not known if the formation of systemic collaterals may place patients at risk for future haemoptysis [18,39].

Complications and side effects

A pragmatic approach to define and to grade the relevance or seriousness of a complication related to PAVM embolisation is the CIRSE classification of complications [42].

Pleurisy and fever 1-2 days after embolisation is the most common side effect of the therapy, occurring in 15–30% of patients, usually lasting 4-6 days. It can be relieved by non-steroidal anti-inflammatory medication. White reported a group of patients that presented with late-onset (4–6 weeks post-procedure) severe pleurisy and fever [40].
The most feared complication is a paradoxical embolisation of air, thrombus, or an occluding device into the systemic arterial circulation, but in most cases they can be retrieved without consequences to the patient [25,40]. Small air emboli have a propensity to enter the left coronary artery, causing acute chest pain, bradycardia and temporary ECG changes. This usually resolves with sublingual nitro-glycerine; atropine should be immediately available to treat bradycardia. Rupture of the PAVM with haemorrhage is rare and managed by completion of the embolisation.

PAP is usually normal or low in patients with PAVM due to the shunting from the fistula. Rarely, pulmonary hypertension develops in patients who have undergone embolisation of PAVMs; however, the overall state of a pre-existing, pronounced pulmonary hypertension may worsen after embolisation [40], and cardiac failure can develop.

**Conclusion**

PAVM represents a multifaceted disease, with many different causes and manifestations. The majority of the cases are associated with HHT. Patients are typically diagnosed using CXR and CT. The degree of right-to-left shunting of blood caused by PAVMs is best evaluated using CE. Transcatheter embolisation therapy is the preferred therapy in the management of PAVMs. Various embolic agents are available.

However, endovascular treatment of PAVMs is challenging. Proper planning as well as patient and material selection is essential.

Patients with HHT should be screened for PAVM and patients with PAVM for HHT. A systematic follow-up to control of reperfusion of embolised PAVM or growth of small PAVMs should be organised, preferably on intervals up to 5 years.

Table 2 summarises a few key recommendations for the therapy of PAVMs.

**Supplementary Material**

Tables with levels of evidences and the grades of recommendation can be found in the online supplementary material of the article.

**Further reading material:**


References


Figures and Tables

**Figure 1:** Proposed algorithm for the evaluation of patients with suspected PAVM

<table>
<thead>
<tr>
<th>Author (Reference)</th>
<th>Year</th>
<th>Patients</th>
<th>Type</th>
<th>Attempts†</th>
<th>Technical success ‡</th>
<th>Clinical success</th>
<th>Recurrence</th>
<th>Complications</th>
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<tbody>
<tr>
<td>Terry (13)</td>
<td>1983</td>
<td>10</td>
<td>B</td>
<td>NR</td>
<td>58</td>
<td>10</td>
<td>NR</td>
<td>NR</td>
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<tr>
<td>White (40)</td>
<td>1988</td>
<td>76</td>
<td>B &gt; C</td>
<td>276</td>
<td>276</td>
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<td>44</td>
<td>82%</td>
<td>NR</td>
<td>1 DVT</td>
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<td>NR</td>
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<td>100%</td>
<td>NR</td>
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<td>Pollak (45)</td>
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<td>35</td>
<td>B,C</td>
<td>99</td>
<td>96</td>
<td>NR</td>
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<td>53</td>
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<td>NR</td>
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<td>100%</td>
<td>NR</td>
<td>18§</td>
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<td>32</td>
<td>C</td>
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<td>90</td>
<td>NR</td>
<td>2</td>
<td>8, 3 major§</td>
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<td>Andersen (48)</td>
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<td>35</td>
<td>C</td>
<td>106</td>
<td>106</td>
<td>&gt;77%</td>
<td>8</td>
<td>NR</td>
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<td>NR</td>
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<tr>
<td>Andersen (51)</td>
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<td>136</td>
<td>B,C,AVP</td>
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<td>339</td>
<td>91%</td>
<td>30</td>
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*Definition of abbreviations:* B = balloon embolisation; NR = not reported; C = coil embolisation.
†The number of PAVM for which embolisation was attempted.
‡The number of PAVM that were successfully occluded.
§Of 18 complications, 12 were reported as minor, two as moderate (transient confusion) and four as potentially serious (two dislodged coils, one transient cerebrovascular accident and one myocardial puncture). Authors report no lasting sequelae.
$Three dislodged coils
& AVP alone
Table 2: Summary of recommendations

<table>
<thead>
<tr>
<th>Key recommendations</th>
<th>Level of evidence</th>
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<tbody>
<tr>
<td>Aggressive screening of HHT patients for PAVMs</td>
<td>2a</td>
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<tr>
<td>Embolisation of all patients with treatable PAVMs, regardless of size</td>
<td>2a</td>
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<tr>
<td>CE as initial screening tool</td>
<td>2a</td>
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<tr>
<td>For larger diameter PAVMs (&gt; 10 mm), or high flow rate and short supplying artery, AVPs is recommended to achieve a faster procedure</td>
<td>2b</td>
</tr>
<tr>
<td>In a large high-flow vessel AVPs should be used as an anchor to prevent coil migration</td>
<td>3b</td>
</tr>
<tr>
<td>Venous sac embolisation may be considered to reduce the incidence of recanalisation of PAVMs compared to feeding-artery embolisation</td>
<td>3b</td>
</tr>
<tr>
<td>For diffuse PAVMs with numerous AV shunts, pulmonary flow redistribution technique may be considered</td>
<td>3b</td>
</tr>
<tr>
<td>To prevent pericatheter thrombosis 100 units/kg or 5,000 units heparin is recommended to be given at the beginning of the procedure</td>
<td>5</td>
</tr>
<tr>
<td>Antibiotic prophylaxis is not recommended</td>
<td>5</td>
</tr>
<tr>
<td>Treating one lung at a time to avoid possible bilateral pleurisy may be considered</td>
<td>5</td>
</tr>
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</table>

Appendix

List of abbreviations

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
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<tr>
<td>AV</td>
<td>Arteriovenous</td>
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<tr>
<td>AVM</td>
<td>Arteriovenous malformations</td>
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<td>AVP</td>
<td>Amplatzer vascular plugs</td>
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<tr>
<td>CE</td>
<td>Contrast-enhanced echocardiography</td>
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<tr>
<td>CT</td>
<td>Computed tomography</td>
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<tr>
<td>CXR</td>
<td>Chest radiography</td>
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<tr>
<td>DSA</td>
<td>Digital subtraction angiography</td>
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<td>ECMO</td>
<td>Extracorporeal membrane oxygenation</td>
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<td>HHT</td>
<td>Hereditary haemorrhagic telangiectasia</td>
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<tr>
<td>MRI</td>
<td>Magnetic resonance imaging</td>
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<tr>
<td>PA</td>
<td>Pulmonary angiography</td>
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<tr>
<td>PAP</td>
<td>Pulmonary artery pressure</td>
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<td>PAVM</td>
<td>Pulmonary arteriovenous malformations</td>
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<tr>
<td>TTCE</td>
<td>Transthoracic contrast-enhanced ultrasound</td>
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<tr>
<td>V-Q</td>
<td>Ventilation-perfusion</td>
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