Femoral Artery Pseudoaneurysm
Current diagnosis and treatment

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ACKNOWLEDGMENT

The authors give special tribute to Prof. Essam Baligh, the founder of the non-invasive vascular lab in Kasr Al-Ainy Medical School. Over two decades, this amazing physician taught several generations the techniques of vascular imaging in different vascular beds. This work was strongly influenced by his teaching and guidance.
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Preface

A pseudoaneurysm (PSA) is a consequence of disruption of all three layers of arterial wall i.e. contained rupture. PSA may occur after catheterization, at the site of native artery and synthetic graft anastomosis, after trauma and infection.¹ PSAs are recognized complications of liver transplantation², heart transplantation,³ obstetric procedures such as dilatation and curettage, cesarean section. Blunt and penetrating trauma may also cause pseudoaneurysms of the carotid, extremity, splenic, and hepatic arteries. Inflammatory conditions such as pancreatitis or infection may also lead to pseudoaneurysm formation.⁴

This monograph will focus mainly on PSAs that occur after cardiac and peripheral endovascular procedures. A post catheterization PSA is one of the most common vascular complications of cardiac and peripheral angiographic procedures. There are different diagnostic modalities but the most convenient is usually duplex ultrasound. Until the early 1990s, the only treatment available for PSA was surgery. Since that time, ultrasound-guided compression repair (UGCR), ultrasound-guided thrombin injection (UGTI), and a whole host of other treatment modalities such as compression devices,⁵ adhesives⁶, coil insertion⁷, fibrin or balloon occlusion have been used with variable success.
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<th>Description</th>
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<tr>
<td>AAA</td>
<td>Abdominal Aortic Aneurysm</td>
</tr>
<tr>
<td>ABI</td>
<td>Ankle Brachial Index</td>
</tr>
<tr>
<td>ACC-NCDR</td>
<td>American College of Cardiology's National Cardiovascular Data Registry</td>
</tr>
<tr>
<td>ACD</td>
<td>Arteriotomy Closure Device</td>
</tr>
<tr>
<td>AF</td>
<td>Atrial Fibrillation</td>
</tr>
<tr>
<td>ALI</td>
<td>Acute Limb Ischemia</td>
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<tr>
<td>ASA</td>
<td>Acetyl Salicylic Acid</td>
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<tr>
<td>ATA</td>
<td>Anterior Tibial Artery</td>
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<tr>
<td>A-V fistula</td>
<td>Arterio-Venous Fistula</td>
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<tr>
<td>BP</td>
<td>Blood Pressure</td>
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<tr>
<td>BMI</td>
<td>Body Mass Index</td>
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<tr>
<td>CA</td>
<td>Coronary Angiography</td>
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<tr>
<td>CABG</td>
<td>Coronary Artery Bypass Graft</td>
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<tr>
<td>CCA</td>
<td>Common Carotid Artery</td>
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<tr>
<td>CDI</td>
<td>Color Doppler imaging</td>
</tr>
<tr>
<td>CFA</td>
<td>Common Femoral Artery</td>
</tr>
<tr>
<td>CIA</td>
<td>Common Iliac Artery</td>
</tr>
<tr>
<td>CT</td>
<td>Computed Tomography</td>
</tr>
<tr>
<td>CVD</td>
<td>Cerebrovascular disease</td>
</tr>
<tr>
<td>CVI</td>
<td>Color velocity imaging</td>
</tr>
<tr>
<td>CW</td>
<td>Continuous wave Doppler</td>
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<tr>
<td>DF</td>
<td>Damping factor</td>
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<tr>
<td>DM</td>
<td>Diabetes mellitus</td>
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<tr>
<td>DUAM</td>
<td>Duplex ultrasound arterial mapping</td>
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<tr>
<td>DVT</td>
<td>Deep venous thrombosis</td>
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<tr>
<td>ECG</td>
<td>Electrocardiogram</td>
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<tr>
<td>EIA</td>
<td>External iliac artery</td>
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<tr>
<td>EP</td>
<td>Endoluminal Protheses</td>
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<tr>
<td>F</td>
<td>French</td>
</tr>
<tr>
<td>FAP</td>
<td>Femoral Artery Pseudoaneurysm</td>
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<tr>
<td>HF</td>
<td>Heart failure</td>
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<tr>
<td>HTN</td>
<td>Hypertension</td>
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<tr>
<td>IHD</td>
<td>Ischemic Heart Disease</td>
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<tr>
<td>Abbreviation</td>
<td>Description</td>
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<tr>
<td>IMT</td>
<td>Intima Media Thickness</td>
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<tr>
<td>INR</td>
<td>International Normalized Ratio</td>
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<tr>
<td>IV</td>
<td>Intravenous</td>
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<tr>
<td>LMWH</td>
<td>Low molecular weight heparin</td>
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<tr>
<td>LVH</td>
<td>Left ventricular hypertrophy</td>
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<tr>
<td>MRA</td>
<td>Magnetic resonance angiography</td>
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<tr>
<td>PA</td>
<td>Peroneal artery</td>
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<tr>
<td>PASI</td>
<td>Para-aneurysmal saline injection</td>
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<tr>
<td>PAD</td>
<td>Peripheral arterial disease</td>
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<tr>
<td>PCI</td>
<td>Percutaneous Coronary Intervention</td>
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<tr>
<td>PI</td>
<td>Pulsatility Index</td>
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<tr>
<td>PSA</td>
<td>Pseudoaneurysm</td>
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<td>POP A</td>
<td>Popliteal artery</td>
</tr>
<tr>
<td>PRF</td>
<td>Pulse repetition frequency</td>
</tr>
<tr>
<td>PTA</td>
<td>Posterior tibial artery</td>
</tr>
<tr>
<td>PCI</td>
<td>Percutaneous coronary intervention</td>
</tr>
<tr>
<td>PW</td>
<td>Pulsed wave Doppler</td>
</tr>
<tr>
<td>Re</td>
<td>Reynolds number</td>
</tr>
<tr>
<td>RI</td>
<td>Resistance Index</td>
</tr>
<tr>
<td>SC</td>
<td>Subcutaneous</td>
</tr>
<tr>
<td>SCAI</td>
<td>Society of Cardiac Angiography and Intervention</td>
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<tr>
<td>SFA</td>
<td>Superficial femoral artery</td>
</tr>
<tr>
<td>SPSS</td>
<td>Statistical Package for the Social Science</td>
</tr>
<tr>
<td>SVS</td>
<td>Society of vascular surgery</td>
</tr>
<tr>
<td>US</td>
<td>Ultrasound</td>
</tr>
<tr>
<td>UGCR</td>
<td>Ultrasound guided compression repair</td>
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<tr>
<td>UGTI</td>
<td>Ultrasound guided thrombin injection</td>
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</table>
**Anatomy**

The external iliac artery (EIA) continues around the side of the pelvis to the level of the inguinal ligament, it lays anteromedial to the psoas muscle and is normally superficial to the external iliac vein.

The common femoral artery (CFA) runs from the inguinal ligament to its division into superficial and deep femoral arteries in the upper thigh; this division is usually 3-6 cm distal to the inguinal ligament. The deep femoral artery or profunda femoris artery passes posterolaterally to supply the major thigh muscles. The importance of the profunda femoris lies in its role as a major collateral pathway in patients with significant superficial femoral artery disease. Several other branches arise from the external iliac, common femoral and profunda femoris arteries and occasionally one of these may be mistaken for the profunda femoris artery, especially if it is enlarged as a collateral supply.

![Figure 1. Arteries of lower limb](image)

The superficial femoral artery (SFA) passes downwards along the anteromedial aspect of the thigh lying anterior to the vein; in the lower third of the thigh it passes into the adductor canal, deep to sartorius and the medial
component of quadriceps femoris. Passing posteriorly behind the lower femur it enters the popliteal fossa and becomes the popliteal artery, which lies anterior to the popliteal vein and gives off several branches, the largest of which are the superior and inferior geniculate arteries. Below the knee joint the popliteal artery divides into the anterior tibial artery and the tibioperoneal trunk, although the exact level of the division may vary; after 2-4 cm the latter divides into the posterior tibial artery and the peroneal artery (Figure 1).
Chapter {1}: Access complications of cardiovascular procedures

Arterial puncture and sheath insertion by the modified Seldinger technique have become the standard method for invasive cardiovascular procedures. With improvement in techniques and devices, approximately 7 million invasive cardiovascular procedures are performed annually worldwide, and this number is expected to increase with the aging of the population.

Despite the growth of radial artery utilization, the vast majority of procedures are still performed via femoral access. Because the number of cardiovascular procedures performed via the femoral approach continues to increase, effective arterial hemostasis is essential. Vascular access complications, reported to be as high as 6% in some series, remain a leading cause of morbidity after a cardiac catheterization procedure.

Manual compression has been considered the traditional technique to achieve closure of the arteriotomy site, requiring close observation and hours of immobilization for success. Arteriotomy closure devices (ACDs) were introduced in 1995 to decrease vascular complications and reduce the time to hemostasis and ambulation. Subsequently, several generations of passive and active ACDs have been introduced that incorporate suture, collagen plug, nitinol clip, and other mechanisms to achieve hemostasis. Despite the widespread use of both passive and active ACDs, there are incomplete data on their safety and efficacy. Additionally, there are few published recommendations regarding the indications for the use of these devices and their comparative effectiveness versus manual compression.
Choice of site and arterial access

To perform both diagnostic and interventional percutaneous cardiovascular procedures, arterial access is usually obtained at the common femoral or radial artery. There has been significant recent interest in the use of the transradial approach for cardiac catheterization and endovascular interventions in the United States, a practice that is standard in many other countries.\textsuperscript{11} Of note, a recent review of the American College of Cardiology’s National Cardiovascular Data Registry (ACC-NCDR) found that fewer than 2% of all cardiac catheterization procedures are performed from the radial site.\textsuperscript{12}

![Image of femoral access]

Figure 2. Femoral access

Proper techniques regarding identification, puncture, and location of sheath insertion into the common femoral artery are important predictors of access site complications.\textsuperscript{13} In clinical practice, the arterial puncture site is usually
verified by the radiographic landmarks of the femoral head or, rarely, by the use of a femoral angiogram (Figure 2). Therefore, the importance of operator experience and meticulous technique cannot be overemphasized.

**Incidence of femoral access complications**

Although complications of vascular access are an inevitable part of the practice of interventional cardiology, patients’ morbidity and mortality can be minimized by anticipation of these potential complications, as well as by their prompt diagnosis and management. In one series, diagnostic cardiac catheterization was associated with an overall 3.4% rate of serious vascular complications.\(^{14}\) The rates of non-coronary vascular complications after interventional catheter based procedures vary from 2% to 6%\(^{15}\) (Table 1).

<table>
<thead>
<tr>
<th>Complication Type</th>
<th>Rate (%)</th>
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<tbody>
<tr>
<td>Diagnostic cardiac catheterization (SCAI Registry 1990)*: vascular complications</td>
<td>0.40</td>
</tr>
<tr>
<td>Interventional coronary procedures</td>
<td></td>
</tr>
<tr>
<td>Femoral-access hematoma (&gt; 6 cm)</td>
<td>5–23</td>
</tr>
<tr>
<td>Retroperitoneal hematoma</td>
<td>0.15–0.44</td>
</tr>
<tr>
<td>Pseudoaneurysm</td>
<td>0.5–6.3</td>
</tr>
<tr>
<td>Arterio-venous fistulae</td>
<td>0.2–2.1</td>
</tr>
<tr>
<td>Infection</td>
<td>&lt;0.1</td>
</tr>
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SCAI indicates Society for Cardiac Angiography and Interventions.

*Pepine et al.\(^{16}\)
A meta-analysis of more than 35000 patients identified an overall vascular complication rate of 3.3% for femoral artery access with manual compression for hemostasis.\textsuperscript{17} A multicenter registry of more than 18000 patients undergoing PCI also reported a 3% vascular complication rate. Vascular complications increase with the complexity of the procedure and with the intensity of anticoagulation and antiplatelet therapy. The predictors of increased vascular complications during PCI include:\textsuperscript{17,18}

Increasing age, women and small body frame

History of CABG

Congestive heart failure

Bleeding disorders: (hemophilia, thrombocytopenia, or disseminated intravascular coagulation)

Stroke / peripheral arterial disease

Diabetes

Chronic obstructive pulmonary disease

Renal failure or a creatinine>2 mg/dL

Liver failure

Immunosuppression

Higher patient acuity (procedural priority and indication)

Greater number of diseased coronary vessels

Higher lesion complexity

Multi-vessel interventions

Use of an intra-aortic balloon pump

Periprocedural use of thienopyridines and/or Glycoprotein IIb/IIIa blockers
Risk stratification for vascular access complications

There are three broad groups of risk categories:

A) - Low risk: Diagnostic angiographic procedures (<1% complication rate)

Patients undergoing diagnostic cardiac catheterization and/or peripheral angiography without intervention are at low risk for vascular complications. As noted, patient characteristics associated with fewer vascular complications include male sex, younger age, normal renal function, and increased body size. These procedures are often elective and characterized by the use of a smaller sheath size (5F), shorter overall procedural time, and little or no concomitant anticoagulation. The expected rate of all vascular complications in this group undergoing diagnostic only procedures has been reported to be < 1%, with registry data noting the rate to be near 0.4% to 0.7%.19

B) - Moderate risk: routine percutaneous intervention (1% to 3% complication rate)

Patient populations undergoing routine percutaneous coronary or peripheral intervention with their associated anticoagulation regimens should be considered at moderate risk for developing vascular complications. These patients with moderate risk are likely to be older, are more often female, and may have evidence of renal dysfunction. The procedure is generally notable for increased sheath sizes (6F to 7F), procedure time that may be prolonged compared to diagnostic procedures, and the use of adjunctive anticoagulants and antithrombotic regimens.20 Recent clinical trial and registry data would
suggest the rate of significant vascular complications in patients undergoing percutaneous intervention ranges between 1% and 3%.  

C) - **High risk (>3% complication rate)**

Patient-specific features and/or certain clinical indications can help identify the group considered at high risk for vascular complications. Patients at the highest risk for vascular complications include those with known peripheral arterial disease, advanced age, female sex, liver disease, coagulopathy, immunosuppression, status after valve replacement, and renal dysfunction. High-risk clinical indications include emergent procedures such as primary percutaneous intervention for acute myocardial infarction, prolonged multivessel intervention, or procedures that require larger sheath sizes (e.g. 8F). The major vascular complication rate in these patients was observed in clinical trials and registries to be >3%.22,23

**Post catheterization Arteriotomy Closure Devices (ACDs)**

In an attempt to reduce some of these potential complications, ACDs have been developed as adjuncts or alternatives to manual compression for hemostasis. The other potential benefits of ACDs include improved patient comfort and satisfaction, faster hemostasis, shorter time to ambulation, and shorter duration of observation. However, ACDs also introduced unique vascular complications that require highly specialized clinical care. Severe groin infections have been described that often present with unusual manifestations and time frames. Embolization of devices and collagen material requires rapid recognition and urgent therapy. Although low in frequency, the severity of these events relative to the indications for the use of ACDs must be considered in each individual case.20
ACDs are either passive or active. Passive closure approaches have focused on enhanced manual compression utilizing external patches with prothrombotic coatings (Syvek Patch, Marine Polymer Technologies, Danvers, Massachusetts), wire-stimulated track thrombosis (Boomerang Wire, Cardiva Medical, Mountain view, California), or assisted compression with mechanical clamps; the passive closure devices do not afford immediate (<5min) hemostasis. {Dauerman, 2007 #992} 
doi:10.1016/j.jacc.2007.07.028

Active closure devices cause immediate closure with suture devices, clips, and collagen plug devices. ACDs have a gradient of risk for vascular complications based on their respective mechanisms of action. Although small studies have identified differing rates of vascular complications for specific devices, there are no definitive data across a wide spectrum of patients. The existing evidence in the literature emanates largely from small and moderate sized clinical trials (100 to 650 patients) that compared ACDs with manual compression, which has historically been considered the “gold standard.” Many of these studies were single center trials that enrolled predominantly low and medium risk patients. These studies were likely underpowered to detect differences in the rates of infrequent vascular complications. Other studies lacked defined allocation concealment, blinded outcome assessment, or intention-to-treat analyses. Finally, standardized definitions of what constitutes a vascular complication or an effective outcome of ACDs are lacking. Given the limitations and somewhat conflicting safety data of the randomized studies, physicians have resorted to large registries and databases to confirm the efficacy and safety of ACDs and test them against manual compression and against each other.
Chamberlin and colleagues compared Vasoseal, Perclose, and mechanical compression with FemoStop in a cohort of 185 patients undergoing PCI with periprocedural abciximab. In this small analysis, Vasoseal and Perclose showed comparable safety to FemoStop but achieved significantly lower initial rates of successful hemostasis (79% and 86% versus 100%, respectively). Another retrospective, nonrandomized study of 827 ACDs (245 Angio-Seal and 582 suture-based) after PCI showed similar success and low and comparable vascular complication rates between the devices. A larger retrospective analysis compared outcomes of 5892 uses of manual compression with 516 ACD uses (mostly Angio-Seal, n=371, and Techstar [suture-based], n=101) after diagnostic and PCI procedures. This single center experience demonstrated that the use of ACDs was associated with significantly more hematomas, a greater hematocrit drop, and a greater need for vascular surgical repair at the access site. However, after multivariable analysis; age, body surface area, and sex, but not the use of ACDs, were independent predictors of vascular complications. Duffin et al compared Perclose and Angio-Seal with manual compression in 1500 patients and found Angio-Seal use to be associated with faster hemostasis and ambulation than Perclose. Perclose use, on the other hand, was associated with more access-site complications than Angio-Seal and manual compression.

Femoral closure devices in the setting of primary PCI for STEMI were tested in 558 consecutive patients in a prospective registry from 2003 to 2008. Major vascular complications were defined as a composite of fatal access site bleeding, access site complication requiring interventional or surgical correction, or access site bleeding with ≥3 g/dL drop in hemoglobin or requiring blood transfusion. 83.2% of patients received a closure device
and 16.8% had manual compression. Major vascular complication occurred in 5.2% of patients. The risk was significantly lower with closure devices compared to manual compression (4.3% vs. 9.6%, $P = 0.036$). Multivariate logistic regression showed that closure device use was an independent predictor of lower rates of major vascular complications (odds ratio 0.38, 95% CI 0.17-0.91).

The largest registry of data from the ACC-NCDR\textsuperscript{14} compared the relative risks associated with the use of ACDs versus manual compression. This database included a total of 166680 ACD uses during diagnostic and interventional cardiac procedures in 214 sites, of which 25495 were suture-mediated ACDs, 28160 were collagen-mediated ACDs, and the rest were manual compression. The registry demonstrated that the use of ACDs was associated with a lower risk of vascular complications in men than in women and after diagnostic versus PCI procedures. On multivariable analysis, ACD use was associated with lower risk of any vascular complication (adjusted odds ratio [OR] 0.83, 95% confidence interval [CI] 0.75 to 0.91). This was driven predominantly by lower rates of bleeding and pseudoaneurysm formation and was seen exclusively with diagnostic procedures.\textsuperscript{14}

Unfortunately, this analysis from ACC-NCDR did not capture access-site related infections or neurological injury and was based solely on site-reported complications, which may be subject to underreporting and potential recall bias. Overall, the nonrandomized observational registry data appeared to confirm the efficacy of ACDs but provided no definitive evidence as to their safety vis-à-vis manual compression or of their safety and efficacy relative to each other.
In contemporary practice, ACD failure is rare but is dependent on specific device choice and patient characteristics. In general, ACD deployment failure significantly increases the subsequent risk of vascular complication rates. Suture-based ACD demonstrated a lower risk of vascular complications when compared with other ACD, irrespective of the success of ACD deployment.  

**Recommendations regarding ACD use**

The available literature is limited and characterized by small studies in select populations that are often underpowered to detect meaningful differences in clinical rates of vascular complications. The available evidence allows the following conclusions to be made regarding the consideration and deployment of arteriotomy closure devices.

1. Patients considered for deployment of ACDs at the femoral artery site should undergo a femoral angiogram with identification of sheath insertion site and other features (atherosclerosis, calcification, etc.) to ensure anatomic suitability for their use (Class I; Level of Evidence C).

2. Facilities with standard manual compression regimens should aim to achieve the reported low vascular complication rates (<1%) in patients undergoing uncomplicated 5F diagnostic angiography (Class I; Level of Evidence C).

3. Use of ACDs is reasonable after invasive cardiovascular procedures performed via the femoral artery to achieve faster hemostasis, shorter duration of bed rest, and possibly improved patient comfort. The use of these devices should be weighed against the risk of increased complications in
certain patient subsets and also take into account body habitus, location of arteriotomy, size and condition of the parent vessel, sheath size, and presence or absence of systemic disease in the patient (Class IIa; Level of Evidence B). This recommendation is based on a meta-analysis and several small-scale trials; however, in these studies, there are trends toward higher rates of complications.

4. ACDs should not be used routinely for the specific purpose of reducing vascular complications in patients undergoing invasive cardiovascular procedures via the femoral artery approach (Class III, Level of Evidence B).
CHAPTER {2}: ARTERIAL PSEUDOANEURYSMS (PSAS)

A pseudoaneurysm (PSA) is a contained rupture after a disruption of all 3 layers of the arterial wall. Under the influence of sustained arterial pressure, blood dissects into the tissues around the damaged artery and forms a perfused sac that communicates with the arterial lumen (Figure 3. Post-catheterization femoral artery pseudoaneurysm). The perfused sac is contained by the media or adventitia or simply by soft tissue structures surrounding the injured vessel. The formation of multiple lobules has been attributed to the natural evolution of simple pseudoaneurysms when they are subjected to continuous increased arterial pressure; it is a way of diminishing intraluminal pressure in a pseudoaneurysm according to Laplace's law.

![Figure 3. Post-catheterization femoral artery pseudoaneurysm](image)

Post catheterization PSA is one of the most common vascular complications of cardiac and peripheral angiographic procedures. The incidence of PSA after diagnostic catheterization ranges from 0.05% to...
When coronary or peripheral intervention is performed, the incidence increases to 2% to 6%. Despite a low incidence, PSAs are commonly encountered when more complex coronary and peripheral interventions are performed, especially with the use of potent antithrombotic and antiplatelet therapy. Although modern imaging modalities have made the diagnosis of pseudoaneurysms more common, the increase in the number of surgical and angiographic procedures performed has led to a real increase in the prevalence of pseudoaneurysms.

PSAs may occur under 4 circumstances:

(1) After catheterization

(2) At the site of native artery and synthetic graft anastomosis: Anastomotic pseudoaneurysms occur with an incidence of 2-5%, are encountered most commonly as a late complication of synthetic aorto-femoral bypass grafting, inevitably continue to enlarge if left untreated, and may require arteriography before repair. Surgery can cause pseudoaneurysms through direct injury to the vessel or the introduction of infection.

(3) Trauma: Blunt and penetrating trauma may also cause pseudoaneurysms of the involved artery. Affected arteries include the carotid, extremity, splenic, and hepatic arteries. Inflammatory conditions such as pancreatitis or infection may also lead to pseudoaneurysm formation. PSAs are also recognized complications of liver transplantation, heart transplantation, obstetric procedures such as dilatation and curettage, cesarean section, and endovascular stent graft implantation for aortic aneurysms, in addition to percutaneous biopsy or drainage.
(4) Infection (e.g. mycotic PSA): Infected femoral pseudoaneurysms may occur as the result of arterial puncture during drug abuse and must be treated by extensive operative debridement, often in conjunction with either autogenous in situ reconstruction or extra-anatomic bypass grafts to avoid chronic limb ischemia.\textsuperscript{34}

This review will focus mainly on PSAs that occur after cardiac and peripheral endovascular procedures.

**Factors Associated With Pseudoaneurysm Formation**

Several patient and procedural factors contribute to the formation of PSA.\textsuperscript{35} The complexity of interventions such as coronary stenting, atherectomy, intraprocedural thrombolytic therapy, and repeat coronary angioplasty have been shown to increase the risk of vascular complications.\textsuperscript{36} Other factors include:

- Antiplatelet agent (often aspirin and clopidogrel)
- Anticoagulant
- Large sheath size > 8
- Age > 65
- Obesity
- Poor post procedural compression
- Simultaneous artery and vein catheterization
- Hypertension
- Peripheral arterial disease
- Haemodialysis
- Complex intervention
- Low or high puncture sites

We prospectively enrolled all patients who underwent coronary angiography, left heart catheterization and/or PCI in our center from 2009 to 2011. We encountered 120 pseudoaneurysms out of 3500 procedures (3.43%). Female gender, hypertension and obesity were more frequent in patients with FAP compared to controls. Diagnostic studies, multiple arterial punctures and the use of dual anti-platelet or antithrombotic therapy were also more common in pseudo-
aneurysm patients. By univariate analysis, hypertension, diagnostic procedure, multiple punctures, puncture a vessel other than the CFA, the use of anticoagulation, the use of dual antiplatelet therapy or both were significantly associated with increased risk for developing pseudoaneurysm.\(^{37}\)

Operators should always perform the puncture with fluoroscopic guidance to locate the level of the head of the femur. Femoral puncture guided by palpation and by superficial landmarks as the anterior superior iliac spine and the inguinal crease result in puncturing the SFA in obese individuals with a “redundant” inguinal skin crease. Another factor for pseudo aneurysm development is the common practice of removing the sheath in the cathlab immediately after the procedure. Most patients are given heparin at the start of the procedure but only a minority of operators orders an activated clotting time (ACT) test before removing the groin sheath after the diagnostic angiography. It may be necessary in some patients to reverse the effects of heparin with protamine.

Table 2. Risk-adjusted multivariate logistic regression analysis concerning incidence of FAP\(^{37}\)
Female gender arose in our study to be an independent risk factor for pseudoaneurysm formation. This could be explained by the higher incidence of bleeding in general and of local hematomas with PCI in females. Hypertension also came as an independent risk factor probably due to the difficulty in compressing an artery with an elevated intra-luminal pressure. In fact, the strongest risk factor for FAP formation (OR 2.89) was puncture below the CFA (Table 2). The best manual compression forces are achieved for the compression of arterial segments located at the femoral head, i.e., for CFA. Compression exerted below this structure lacks sufficient support which limits its efficacy.  

Of particular importance is the increased incidence of femoral artery PSAs when the puncture site is not in the common femoral artery, but rather in the superficial or deep femoral artery or the external iliac artery. There are no good prospective data to predict who will develop a PSA. However, it makes intuitive sense that the accuracy of the initial puncture and the expertise and duration of compression of the puncture site after the sheath is removed may be important factors in the development of PSAs. Vascular complications are less common when the interventionist uses ultrasound or fluoroscopy with localization of the femoral head to puncture the femoral artery in the correct location and with the first attempt (e.g., no posterior wall puncture, especially in obese patients and those with weak pulsations.  

The Natural history of PSA:
PSAs may thrombose spontaneously. In one study, spontaneous thrombosis occurred in 72 of 82 patients with PSA < 3 cm at a mean of 23 days, whereas in another study 9 of 16 patients had spontaneous thrombosis at a mean of 22 days. Failure to thrombose was associated with FAP diameter > 2 cm (volume > 6cm³) and concomitant use of anticoagulation or antiplatelet agents. Some authors have
suggested the age and size of the lesion and the length and width of the track are predictive variables in these situations.\textsuperscript{29,41} That’s why observation of small PSAs (2.0 cm) is reasonable in the absence of severe pain or complications.

The most catastrophic complication of PSA is rupture. Although the exact rate is unknown, the risk of spontaneous rupture of PSA is related to a size >3 cm, presence of symptoms, large hematoma, or continued growth of the sac.\textsuperscript{39,42} Although most post-catheterization PSAs are sterile, infection of a PSA significantly increases the risk of rupture as well as the risk of septic embolization.\textsuperscript{43}

**Diagnosis of pseudoaneurysms:**

A) - Clinical Features:

Pseudoaneurysms may be asymptomatic and detected only incidentally during radiologic investigation of other conditions or during surgery. The presence of pain or swelling in the groin after catheterization is the most common presentation of a PSA. Swelling from a large PSA or hematoma may also lead to compression of nerves and vessels with associated neuropathy, venous thrombosis, claudication, or, rarely, critical limb ischemia. Local ischemia of the skin may lead to necrosis and infection (Figure 4).

![Figure 4. Femoral artery pseudoaneurysm with overlying skin infection and necrosis.](image)
On physical examination, there may be a palpable pulsatile mass or an audible systolic bruit. However, sometimes none of these findings may be present. That’s why, any patient who experiences pain that is disproportionate to that expected after a percutaneous procedure should undergo an ultrasound examination to exclude the presence of a PSA regardless of the presence of a bruit. The advent of new radiologic techniques with a greater sensitivity for asymptomatic disease has allowed more frequent diagnosis of pseudoaneurysms. Conventional angiography remains the standard of reference for diagnosis but is an invasive procedure, and noninvasive diagnostic modalities (e.g., ultrasonography, computed tomographic angiography, magnetic resonance angiography) should be included in the initial workup if possible.

A complete workup will help in determining the cause, location, morphologic features, rupture risk, and clinical setting of the pseudoaneurysm; identifying any patient co-morbidities; and evaluating surrounding structures and relevant vascular anatomy, information that is essential for treatment planning.

B) - Imaging Features

_Ultrasonography (US):

Role of duplex in assessment of aneurysms

- **True aneurysms**

True aneurysms are abnormal dilations of arteries. The term ectasia is often used to describe a moderate dilation of arteries. The abdominal aorta is one of the commonest sites for aneurysms to occur.

Rupture of abdominal aortic aneurysm (AAA) is a common cause of death in men over the age of 65 years. Ultrasound is a simple noninvasive method of
detecting aneurysms and can be used for serial investigations to monitor any increase in size.

However, if surgical intervention is being considered, other imaging techniques, such as CT and MRI, may be required to demonstrate the relationship of an aneurysm to major branches and other structures within the body.

There have been significant developments in the treatment of aneurysms over the last several years, with the introduction of endovascular devices to repair aortic aneurysms and covered stents to exclude flow in aneurysms in other areas of the body.

It has been suggested that an aneurysm is a permanent localized dilation of an artery having at least a 50% increase in diameter compared to the normal, expected diameter. Ectasia is characterized by a diameter increase >50% of the normal, expected diameter. It is worth remembering that there is considerable variability in the normal diameter of arteries among individuals, and this will be dependent on factors such as physical size, sex and age.

The mechanism of aneurysm development is still uncertain but may involve a multifactorial process leading to the destruction of aortic wall connective tissue. There is evidence that increased local production of enzymes capable of degrading elastic fibers as well as interstitial collagens is associated with AAA.

The lumen of aneurysm is often lined with large amounts of thrombus, which can be a potential source of emboli. This is also why arteriograms, which only demonstrate the flow lumen, are not accurate for estimating the true diameter of an aneurysm, as the flow lumen can be significantly smaller than the diameter of the entire vessel. Dissection of an aneurysm can occur following a
tear in the intima, and blood can leak into the space between the intima and media, sometimes creating a false flow lumen.

Aneurysms can also be caused by a variety of infections, such as bacterial endocarditis, and are termed mycotic aneurysms. These can occur anywhere in the body.

Popliteal aneurysms may be the source of distal emboli. They can also occlude, leading to symptoms of acute lower limb ischemia. This should always be considered as a potential cause of the acutely ischemic leg, especially in patients with no other obvious risk factors.

- **False aneurysms**

  False aneurysms occur predominantly in the femoral artery following puncture of the arterial wall for catheter access. In this situation, blood continues to flow backward and forward through the puncture site into a false flow cavity outside the artery.

  True femoral artery aneurysms occur less frequently and are usually associated with aneurysmal disease elsewhere. However, aneurysmal dilations can occur where graft anastomoses have been performed.

  False aneurysms, also known as pseudoaneurysms (PSAs), primarily occur following arterial puncture for catheter access, due to poor control of arterial bleeding following the procedure. This is usually due to insufficient pressure being applied over the puncture site or pressure being applied for too short a time.

  They may also occur following trauma. Blood flows backward and forward through a hole in the arterial wall into the surrounding tissue, forming a flow cavity.
in the tissue adjacent to the artery. The false lumen often contains thrombus, which may be layered. False aneurysms can increase in size over time.

Color flow imaging should be used to confirm flow in the false lumen. The color flow image typically demonstrates a high-velocity jet originating from the defect in the artery wall, which is associated with a swirling pattern inside the false lumen, similar to the ‘yin-yang’ sign. Spectral Doppler usually demonstrates an equal forward flow and reverse flow component to the arterial jet as flow enters the false aneurysm during systole and exits during diastole.

The audible Doppler signal is very characteristic, with high-frequency Doppler shifts heard in the forward and reverse phases across the neck.

The common femoral artery is the main vessel in which false aneurysms occur, as it is the commonest site for catheter access. False femoral aneurysms maybe very large, and bleeding into the retroperitoneal cavity can be a serious complication, leading to shock and death.

-Scanning false femoral aneurysms

The patient should lie as flat as possible. The procedure should be started by scanning the common femoral artery in transverse section. A mid frequency 5 MHz or broad-band equivalent, flat linear array transducer will usually provide an adequate image. However, in some cases an abdominal curved array transducer may be required, especially if the patient is obese or if the puncture has been very high. In addition, areas of hematoma lying over the vessel, associated with the puncture site, can make the imaging difficult.

The common femoral artery should be identified and scanned along its length in transverse section using color flow imaging. The proximal few centimeters of the
superficial femoral artery and profunda femoris artery should also be examined, as low punctures can result in false aneurysms of these vessels. A potentially confusing situation can occur if the inferior epigastric artery, a superficial branch of the common femoral artery, runs close to an area of hematoma or swelling, as this might be mistaken for a small leak. Spectral Doppler recordings taken from the superficial epigastric artery will demonstrate a peripheral arterial type wave form with overall flow in the forward direction as opposed to the high forward and reverse flow components seen in the necks of false aneurysms (Figure 5).

Figure 5. US assessment of PSA, (a) color Doppler before treatment. (b) Pulsed Doppler over the neck, (c) Thrombosed PSA after therapy

US is the best modality for imaging PSAs especially in the femoral arteries, easily distinguishing them from haematoma or arterio-venous fistulae, and will give precise anatomical information, as well as the velocities within the sac and the neck. Ultrasound is portable, readily available, inexpensive, and fast, involves no ionizing radiation or renal toxic contrast material, and is noninvasive. Ultrasound has been reported to have a sensitivity of 94% and a specificity of 97% in the detection of post-catheterization pseudoaneurysms. However, ultrasound is operator dependent, and the evaluation of deep vessels may be difficult. The classic picture by gray-scale US is a hypoechoic cystic structure adjacent to a supplying artery (Figure 6).
Figure 6. B-mode ultrasound of PSA with echolucent sac.

The size of the pseudoaneurysmal sac, the number of compartments (lobes) in the sac, the connection of the sac to the artery, and the length/width of the pseudoaneurysmal neck can be assessed with gray-scale US (Figure 7).

Figure 7. (a) Doppler US image depicts PSA with bidirectional flow within the neck (N). (b) Color US image clearly depicts the PSA neck (N).

Pseudoaneurysms may be simple (one lobe) or complex (two or more lobes separated by a patent tract with a diameter smaller than the minimal dimension of the smallest lobe). Septa within the lobe or lobes of a pseudoaneurysm may also be seen (Figure 8).45
Figure 8. Color ultrasound of PSA. There are 2 chambers and a long track

In addition, concentric layers of hematoma are occasionally seen within the pseudoaneurysm. However, gray-scale US is not diagnostic, since these findings are seen in a number of conditions, the most common being simple and complex cysts and hematomas. Doppler US helps establish the diagnosis. Blood flow within a cystic structure is characterized by a typical swirling motion called the “yin-yang sign”. However, this pattern of flow may also be seen in saccular aneurysms, so that a diagnosis made on the basis of this finding alone may prove to be inaccurate. The hallmark of the diagnosis is the demonstration of a communicating channel (neck) between the sac and the feeding artery with a “to-and-fro” waveform at duplex Doppler US. The “to” component represents blood entering the pseudoaneurysm in systole; the “fro” component represents blood exiting the pseudoaneurysm during diastole.  

46 (Figure 9)
In addition, placing the imaging findings in the clinical context (i.e., the history of the cause of the pseudoaneurysm) allows the diagnosis of a pseudoaneurysm versus a saccular true aneurysm. There are an increasing number of procedures performed from the arm, which thus increases the likelihood of PSAs in this location. The same principles apply to diagnosis PSAs in the arm and leg.

One common mistake during ultrasound examination is to image too superficially. It is important to increase the depth on the ultrasound machine so that deep PSAs (> 4 cm from the skin) are not overlooked. A complete examination should include imaging of the mid and distal external iliac artery, common femoral artery, and proximal portions of the superficial femoral and profunda femoral arteries.

**Computed Tomographic angiography (CT):**

CT angiography, especially with multidetector row helical scanners, is another valuable diagnostic tool. Unenhanced CT scans may demonstrate a low-attenuation rounded structure arising from the donor artery. Intermediate or high attenuation (hemorrhage) adjacent to the pseudoaneurysm indicates pseudoaneurysm rupture (Figure 10), which may vary in attenuation depending on whether it is chronic or acute.
Figure 10. (a) Axial contrast material enhanced CT angiogram demonstrates a ruptured splenic PSA with adjacent hemorrhage (arrowheads). (b) Axial contrast-enhanced CT scan shows intrasac thrombosis of the PSA (arrow) and communication with the donor artery (arrowhead).

The wall of the pseudoaneurysm is usually smooth and well delineated except in a mycotic pseudoaneurysm, whose wall is thickened, irregular, or ill defined. Contrast-enhanced CT may demonstrate a sac filled with contrast material. However, the entire pseudoaneurysm may not fill with contrast material; a low-attenuation area will remain within the pseudoaneurysm, a finding that indicates partial thrombosis. The donor artery is adjacent to the pseudoaneurysm and can usually be seen communicating with it.

CT angiography has advantages over other imaging modalities. Although US and magnetic resonance (MR) imaging are also noninvasive, CT angiography is not as operator dependent and has a shorter acquisition time (1 minute). Post-processing of the raw data to generate three-dimensional (3D) images may be time consuming; however, diagnostic information sufficient for surgical planning can be obtained from the axial images. (Figure 11)
Three-dimensional CT angiography allows visualization of the lesion from all angles, which is not possible with angiography. CT virtual endoscopy is an additional feature that multidetector row helical CT scanners provide but requires a dedicated workstation for image creation and interpretation. In addition, CT provides a global perspective on the entire vasculature, including adjacent vascular beds; angiography is limited to selected vascular territories, which can lead to overlooking synchronous pseudoaneurysms or other vascular diseases. CT angiography has a high sensitivity and specificity for detecting arterial injuries. For example, in a study by Soto et al, CT angiography had a sensitivity and specificity of 95.1% and 98.7%, respectively, in detecting pseudoaneurysms in the proximal extremities.

The usefulness of CT angiography is still limited by imaging artifacts caused by bullet fragments or other metallic objects. The spatial resolution of CT angiography is inferior to that of conventional angiography, leading to limited detectability of subtle abnormalities. In addition, it may be difficult to distinguish between pseudoaneurysms and true aneurysms in small visceral arteries at CT.
general, more contrast material is needed for CT angiography than for angiography. In addition, endovascular therapy cannot be performed at the time of diagnosis.

*Magnetic Resonance angiography (MR):*

MR imaging techniques are numerous, and a comprehensive discussion of the use of these techniques for vascular imaging is beyond the scope of this review. We will make reference only to certain sequences and the advantages they offer. Three dimensional gadolinium enhanced MR angiography allows visualization of a lesion in any projection (Figure 12). Furthermore, unlike 3D CT angiography, no iodinated contrast material or ionizing radiation is necessary\(^5\), making 3D contrast-enhanced MR angiography a valuable tool in the imaging of pseudoaneurysms in patients with impaired renal function and allergies to CT contrast material.

![Figure 12. (a) Axial gradient-echo T1-weighted MR angiogram demonstrates flow void in a pseudoaneurysm (arrowhead) due to turbulent flow. (b, c) Coronal spoiled gradient-echo (b) and conventional (c) T1-weighted MR images demonstrate a pseudoaneurysm of the right internal iliac artery (arrowhead).\(^4\)](image)

Figure 12. (a) Axial gradient-echo T1-weighted MR angiogram demonstrates flow void in a pseudoaneurysm (arrowhead) due to turbulent flow. (b, c) Coronal spoiled gradient-echo (b) and conventional (c) T1-weighted MR images demonstrate a pseudoaneurysm of the right internal iliac artery (arrowhead).\(^4\)
Axial spoiled gradient echo or spin-echo T1-weighted MR imaging allows visualization of intraluminal thrombus and the assessment of pseudoaneurysmal sac size (Figure 12b).\textsuperscript{52}

However, MR angiography remains time consuming compared with CT or US. It is not practical in the emergency setting because (a) it has limited availability, (b) proper monitoring of the patient in the magnet may be difficult, and (c) patients are often connected to imaging incompatible medical equipment.\textsuperscript{51} The usefulness of MR angiography may also be limited due to (a) artifacts caused by patient motion, (b) metallic artifacts due to surgical clips or orthopedic hardware, (c) vessel tortuosity, (d) turbulent flow, or (e) pulsatility.\textsuperscript{53}

\textit{Conventional Angiography:}

Angiography remains the standard of reference for the diagnosis of pseudoaneurysms despite the advent of new imaging technologies.\textsuperscript{54}

Figure 13. Axillary artery PSA after a diagnostic arteriographic procedure. Disruption in the vessel wall (white arrow) and the larger PSA sac (black arrow).  

A significant advantage of angiography is its capacity for real-time hemodynamic assessment of a particular vascular bed, which includes identifying
collateral vessels to assess the expendability of the donor artery. Such assessment is important in treatment planning (Figure 13).

Figure 14. Conventional angiography showing PSA.⁵⁵

Figure 15. A multilobed PSA is seen arising from SFA.⁵⁵

Other pseudoaneurysms not seen at US, CT, or MR imaging of the vascular bed in question may also be identified at angiography. The donor artery can be accurately identified and selective angiography performed to identify the characteristics of the pseudoaneurysm, including the size of its neck. Lesions that may have a similar appearance at CT (e.g., pseudoaneurysms, arteriovenous fistula, and vascular malformations) are better differentiated with angiography.⁵⁶ In
addition, angiography provides a diagnostic tool with concomitant therapeutic potential if indicated.

The principal disadvantage of angiography as a diagnostic modality is its invasive nature and the increased risk of procedure-related complications. The overall prevalence of a major vascular complication from angiography is reported to be 0.02%-9%. These complications include the development of pseudoaneurysms, hematomas, arterio-venous fistula, distal embolization, arterial spasm, ischemia, intimal dissection, and vessel thrombosis. Furthermore, angiography makes use of ionizing radiation and iodinated contrast material, each with its own risks and complications. Another limitation of angiography is that it may underestimate the size of a pseudoaneurysm that contains a thrombus (Figure 14). In addition, vascular lesions, including other pseudoaneurysms, can be overlooked if particular vascular beds are not evaluated during catheter-directed angiography. As a result, angiography should be used as a focused diagnostic modality to complement and to compensate for pitfalls of other diagnostic modalities such as CT angiography and as a prelude to endoluminal treatment of pseudoaneurysms.

Figure 16. (a) Selective digital subtraction angiogram of the phrenic artery shows contained contrast material extravasation (arrowhead). (b) Contrast-enhanced CT scan shows a large PSA of the phrenic artery (arrow). PSA was underestimated at angiography.
Treatment of Pseudoaneurysms

Symptomatic pseudoaneurysms should be treated. However, the decision to treat asymptomatic pseudoaneurysms is controversial due to the unclear and variable natural history of pseudoaneurysms, particularly when factoring in the anatomic locations of various pseudoaneurysms, the clinical setting, and patient comorbidities. Pseudoaneurysms may undergo spontaneous thrombosis, and some investigators have advocated observation for small asymptomatic pseudoaneurysms. However, there is currently no way to predict which pseudoaneurysms will undergo spontaneous thrombosis.

The controversy in the literature over spontaneous thrombosis of pseudoaneurysms mostly concerns post-catheterization extremity pseudoaneurysms and post-traumatic visceral (hepatic and splenic) pseudoaneurysms. Close observation of small, asymptomatic pseudoaneurysms is recommended and treating them only if they enlarge, do not resolve, or become symptomatic. However, the risk of spontaneous rupture of extra-organic visceral pseudoaneurysms is very high regardless of their size, and the mortality rate for such ruptures in morbid postsurgical patients has been reported to approach 100%. Therefore, some authors believe that definitive treatment should be administered in all such cases.

The therapeutic options (including observation) for the treatment of pseudoaneurysms should be tailored to the site, rupture risk, and clinical setting of the pseudoaneurysm as well as to patient comorbidities. Traditionally, pseudoaneurysms have been treated with surgical repair; however, traditional surgical treatment is invasive and is often associated with significantly higher morbidity and mortality rates. Over the past few years, minimally invasive
radiologic treatments have been developed as alternatives to surgery, including ultrasound-guided compression, direct percutaneous management (including US-guided thrombin injection), and endoluminal management.\textsuperscript{60,63,64}

We will provide an overview of the various therapeutic approaches of post catheterization arterial pseudoaneurysms and their respective merits, drawbacks, and indications.

I–Surgery
In general, surgical management of pseudoaneurysms varies widely and includes resection with a bypass procedure, arterial ligation, and partial or complete organ removal.\textsuperscript{65} The latter obviously involves mainly the intra-organic visceral pseudoaneurysms, which include renal pseudoaneurysms (partial or complete nephrectomy), hepatic pseudoaneurysms (segmentectomy), and splenic pseudoaneurysms (spleenectomy). Surgical treatment of pseudoaneurysms involving vital arteries includes pseudoaneurysm resection with patch repair of the vital donor artery and arterial ligation with a bypass procedure. As a result of technologic advances in US guided and endoluminal management of pseudoaneurysms, there is an ongoing paradigm shift toward minimally invasive management of pseudoaneurysms.

However surgical management of PSA is still an important and necessary management strategy in a minority of patients with post catheterization PSA with the following features:

Infected PSA
Rapid expansion
Failure of other therapies
Skin necrosis
Compressive syndromes:

Neuropathy
Claudication
Critical limb ischemia

The technique used for surgical repair depends on the presence or absence of concomitant infection.

A) - Non-infected pseudoaneurysm:

The conventional approach is to gain proximal and distal control of the PSA, open the fibrous pseudo-capsule and evacuate the haematoma. The puncture site is identified and one or two sutures of a synthetic, non-absorbable suture are placed transversely to avoid narrowing. If the vessel is severely damaged or diseased, formal arteriotomy with endarterectomy and vein patch angioplasty may be required with systemic heparinisation. With experience, the technique may be accelerated to simply incising the sac and evacuating the pulsatile clot. The subsequent brisk arterial bleeding can be controlled by digital pressure over either the puncture site or the vessel proximally, or by introducing a 3Fr catheter through the arterial hole and inflating the balloon. This 'blind' technique is especially useful where the anatomy of the area and feeding vessels is distorted and there are important neighboring structures, e.g. complex carotid or subclavian pseudoaneurysms.

Radiological balloon occlusion can assist the surgeon by providing haemostasis and allowing the traumatised artery to be accessed through the sac.

B) - Infected pseudoaneurysm
The goals of surgical management here are the eradication of infection and maintenance of distal perfusion. The former is achieved using high doses of intravenous antibiotics based on pre-operative blood cultures or operative tissue culture, together with excision of the infected aneurysm sac (Figure 17 & Figure 15). The most commonly isolated bacteria are Staphylococcus aureus and Salmonella species. Since bacteraemia may persist following surgery, antibiotics should continue for at least 6 weeks.

Simple ligation of infected PSAs is associated with a 30% amputation rate with a further 25% suffering incapacitating leg ischaemia.\(^6\) It is, therefore, desirable to perform primary bypass wherever possible prior to excision of the aneurysm. Synthetic grafts should be avoided and autogenous vein bypasses should run through remote and healthy tissue.

The opinion of a plastic surgeon should be sought if the skin overlying the pseudoaneurysm is at risk of necrosis. A management plan should be devised and discussed with the patient and the other teams involved with care of the patient, so that timing of surgery, planning of incisions, anticoagulation regimens, type of anesthesia and other aspects of care can be worked out.
Any PSA that occurs at the site of a vascular anastomosis (e.g., aorto-bifemoral bypass) should be repaired surgically because it results from a disruption at the suture site and may be caused by infection. Likewise, spontaneously occurring PSAs are often mycotic and should be repaired surgically. Compression on underlying structures by an expanding pulsatile mass, which causes claudication, neuropathy, or critical limb ischemia, requires urgent surgical decompression and resection of the PSA. Rarely, the PSA is so large that it has or will cause skin necrosis. In this situation it is imperative to decompress this area surgically.\textsuperscript{39,47}

However, surgery is rarely employed to treat the usual post-catheterization PSA. The disadvantages of surgery are that it requires anesthesia and an incision usually in the groin, an area known to become infected easily after a surgical procedure. Lumsden and colleagues reported a surgical complication rate of 20% after PSA repair. Complications included bleeding, infection, neuralgia, prolonged hospital stay, perioperative myocardial infarction, and, rarely, death.\textsuperscript{40}

II – Ultrasound-guided Compression repair (UGCR)

Since first being described in 1991 by Fellmeth et al,\textsuperscript{69} ultrasound-guided compression of pseudoaneurysms has rapidly replaced surgery in the treatment of post-catheterization pseudoaneurysms. Compression is performed with the US transducer itself, a procedure that permits direct and continuous visualization of the vessels.\textsuperscript{63} Pressure is applied to pseudoaneurysms in various locations depending on lesion accessibility (Figure 18).
The patient should be lying supine, and some form of analgesia or sedation should be administered as the procedure can be uncomfortable. The distal ankle artery signals should be assessed with continuous wave Doppler to ensure there is good distal perfusion of the leg before beginning the procedure. Firm transducer pressure is applied over the point of communication between the true lumen and false lumen to occlude the arterial jet.

Compression is maintained for 10 min intervals with a brief release of pressure to allow for distal perfusion. It may take over an hour to successfully thrombose the aneurysm.

Typical protocol includes an initial 10–20 minute compression of the pseudoaneurysm neck; if this is not feasible, the pseudoaneurysm itself may be compressed.\textsuperscript{35,59} Cycles are repeated for up to 1 hour. Anticoagulation therapy decreases the success rate; therefore, anticoagulants may be discontinued prior to the procedure if possible. Compression should eliminate flow within the pseudoaneurysm but permit arterial perfusion to the extremity. Although it would be advantageous to use manual compression devices, the vertical angle created by
the device does not allow selective compression of the PSA chamber and tract. Non-selective compression leads to longer compression times, more discomfort to the patient, and a lower success rate, in addition to an increase in the likelihood of complications such as deep venous thrombosis.\textsuperscript{59}

UGCR is limited by patient and operator discomfort due to the long compression time required. Compression is painful to the patient, necessitating the administration of analgesics. The use of two operators and a mechanical C arm compression device has been described,\textsuperscript{70,71} but a recent innovation using a Femostop\textsuperscript{TM} (RADI Medical Systems AB) device filled with saline to act as a sonic window for the ultrasound probe offers great promise (Figure 19, Figure 20).\textsuperscript{72} The significance of this recent improvement is that the operator does not physically compress the PSA for the entire duration as the Femostop accomplishes this, and yet the precise position of compression can be regularly checked to ensure that it is satisfactory.

Figure 19. The belt wraps around the patient for support. The pneumatic bubble is inflated over the arteriotomy to compress the artery. Image courtesy of St.Jude Medical\textsuperscript{73}
Similarly, measuring the oxygen saturation of the hallux may be useful to ensure distal arterial flow is not compromised. Only fairly superficial pseudoaneurysms can be treated with this method, such as those affecting the femoral, axillary, and brachial arteries. In obese patients, this therapeutic application can be hindered even at these sites. Although UGCR may control expanding haematomas, this external pressure may compromise skin viability.

UGCR has been shown to have a success rate of 75% to 98%. In patients on anticoagulation, the success has been reported to be in the range of 30% to 73%. Other factors affecting the success of this method include anticoagulation status, pseudoaneurysm size, whether the pseudoaneurysm is simple or complex, age of the pseudoaneurysm, and the length and width of the pseudoaneurysmal neck. Rare complications such as venous thrombosis, skin necrosis, and pseudoaneurysm rupture have been reported. Local arterial thrombosis and distal embolization due to occlusion of the underlying artery caused by the pressure required to compress the pseudoaneurysm have also been reported. The failure rate of this technique
has been reported to be as high as 15%–38%, with a recurrence rate after initial success as high as 20%–30% in patients who have received anticoagulants.35,75,76

III -US guided Percutaneous Thrombin Injection (UGTI)

In 1986, Cope and Zeit77 described a new technique for “clotting aneurysms” by direct injection of diluted thrombin. They conceived the idea based on the use of coils, cyanoacrylate glue, and balloons in other aneurysms, and the mixed results with compression of femoral false aneurysms. Thrombin converts inactive fibrinogen into fibrin, leading to thrombus formation. In 1987, Walker et al78 reported the successful treatment of a large deep femoral artery pseudoaneurysm in a 33-year old man with the use of a thrombin injection. Despite the excellent results achieved by Cope and Zeit and Walker et al, this technique was not fully embraced until the work of Kang et al was published in 2000.63 At many institutions, US guided percutaneous thrombin injection has replaced US guided compression as the therapeutic method of choice for treatment of post-catheterization pseudoaneurysms.64,76 Thrombin preparation consists of adding sterile normal saline solution to the commercially available sterile thrombin powder. Bovine thrombin is available in a commercial kit, and it is generally reconstituted in normal saline to a concentration of 1000U/mL. Several authors have advocated lower concentrations of thrombin (e.g., 100 U/mL)79,80 or the use of human thrombin because of the theoretical concept that it may produce a lower likelihood of allergic reactions.81

After complete US evaluation of the pseudoaneurysm is performed, and under complete antiseptic precautions, the color Doppler is turned off and a 20 gauge coaxial needle is inserted into the cavity of the pseudoaneurysm using real-time sonographic guidance with a ‘freehand’ technique. The needle tip is directed away from the neck of the pseudoaneurysm. The needle is positioned in the center of the
sac, and thrombin is injected at a constant rate while flow within the sac is monitored with Doppler US. The thrombin is continuously injected until flow within the sac ceases, usually within seconds. The volume of injected thrombin ranges from 0.5 to 1.0 mL (Figure 21).  

Figure 21. Ultrasound guided thrombin injection technique

The goal of UGTI is to produce complete obliteration of flow in the chamber and tract (Figure 22). Under no circumstances should an injection be made directly into the tract, as this will increase the likelihood of thrombosis of the native artery.

Figure 22. A) PSA that arises from the common femoral artery. B) Image after UGTI. There is no flow within the chamber.
There is some controversy about how to approach patients with multi-chambered aneurysms. Injection into the deeper chamber will almost certainly cause all other chambers to thrombose. However, the needle placement is closer to the native artery, and the potential for complications may be higher. It is preferred to inject into the most superficial chamber first. This usually results in thrombosis of all chambers. However, if flow is still present in the deeper chambers, a second injection may need to be performed.\textsuperscript{1} US guided percutaneous thrombin injection is not limited to use in superficial arteries; pseudoaneurysms in deep visceral arteries may also be treated with this method,\textsuperscript{83} especially when the donor artery is endoluminally inaccessible and its occlusion is not a viable option. Arteries above the inguinal ligament can also be treated with this method, which is contraindicated in UGCR due to the potential risk of rupture.\textsuperscript{84}

The most serious complications associated with thrombin injection are the development of deep venous thrombosis (if the thrombin is inadvertently injected into the vein), pulmonary embolism, or thrombosis of the artery (if thrombin is injected into the PSA tract or the artery itself). This is most likely to occur if an injection is made directly into the neck of the PSA.\textsuperscript{86} Although some clinicians take the patient immediately to surgery should this complication occur, other patients have been observed while anticoagulated and reported spontaneous resolution with no significant clinical sequelae.\textsuperscript{87,88} It has been suggested that short wide tracts may increase the risk of thrombin injection. For this reason, it has been proposed that a lower concentration of thrombin (100 U/mL) be used. Some authors advocate the use of balloon occlusion of the pseudoaneurysm neck prior to thrombin injection. However, such occlusion makes the procedure more invasive, and the related complications outweigh the theoretical potential benefit. Although thrombin is the preferred agent for percutaneous embolization of
pseudoaneurysms, other materials such as coils may be used, either independently or in conjunction with thrombin.

The possibility of inadvertent injection of thrombin into native vessels can be reduced by accurate anatomical evaluation prior to thrombin injection, by directing the needle tip away from the pseudoaneurysm neck, by avoiding ‘over-injection’ and by excluding patients with a very large pseudoaneurysm with a short, wide neck and a small native vessel. Post-procedural monitoring of peripheral pulses and the ankle-brachial index allows early detection of peripheral thromboembolism downstream from the injection site. Allergic reactions and anaphylaxis have been reported in patients previously exposed to bovine thrombin. \(^{87,88}\) Pope and Johnston recommend skin testing in patients with prior exposure to bovine thrombin. However, this has not been observed in most reports and skin testing is rarely performed. However the immediate and long term immunological impacts of bovine thrombin injection are unclear. Despite its antigenic potential, allergic-type responses appear to be extremely uncommon. Nevertheless, a recent report of a prolonged generalized urticarial reaction following thrombin injection of a pseudoaneurysm is cause for concern. \(^{87}\) Moreover, there is evidence to suggest the presence of immunoglobulin G and M antibodies to bovine thrombin following topical application. \(^{89}\) Since repeated exposure appears to increase the risk of developing antibodies to coagulation factors, most authors consider prior exposure to thrombin to contraindicate its use for treating pseudoaneurysm. Unfortunately, this may be difficult to confirm as the patient is unlikely to know of prior exposure, and previous detailed surgical records are not always available. As more patients undergo this procedure, future thrombin use may be limited due to concerns about allergic-type reactions. There have been previous reports of antibodies that form against bovine factor V contamination of thrombin preparations. This has potential
for serious hemorrhagic complications in humans; however, it has not been observed in any of the series of patients treated with UGTI. The overall complication rate from UGTI is 1.3% with an embolic rate of 0.5%.

Contraindications to UGTI include a history of allergic reaction to thrombin, unfavorable anatomy of the PSA and its neck, local infection and distal limb ischemia.\textsuperscript{75} Also UGTI should only be performed in patients who develop PSA secondary to a catheterization procedure. If a PSA occurs spontaneously, a mycotic PSA should be suspected, and UGTI should not be undertaken. Additionally, a PSA that occurs at the anastomosis of a synthetic graft and native artery should be treated surgically and not with UGTI. The size of the PSA chamber is not, in of itself, a contraindication to UGTI. However, if the PSA is large enough to cause skin necrosis or compression of nerves or blood vessels, then surgery should be performed instead of thrombin injection.

Technical success rates with UGTI in the setting of post-catheterization pseudoaneurysms ranges from 90% to 100%, even in patients undergoing anticoagulation or antiplatelet therapy, making this method superior to UGCR.\textsuperscript{32,35} More than 45 individual series have been published on the safety and efficacy of this therapy. The cumulative overall success rate in 1329 PSA injections was 97%. The success and complications of ultrasound thrombin injection in the largest series is summarized in Table 3.\textsuperscript{62,86,90–94}
Table 3. Summary of ultrasound thrombin injection series

<table>
<thead>
<tr>
<th>First Author</th>
<th>Year</th>
<th>No.</th>
<th>Bedrest (Hours)</th>
<th>Percent Success</th>
<th>Dose (Units)</th>
<th>Recurrence</th>
<th>Complications</th>
<th>Embolic</th>
<th>Description of Complications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Knezevic</td>
<td>2001</td>
<td>52</td>
<td>...</td>
<td>94</td>
<td>357 vs 638</td>
<td>4</td>
<td>0</td>
<td>0</td>
<td>Thrombotic complication of native artery</td>
</tr>
<tr>
<td>Carbon</td>
<td>2001</td>
<td>54</td>
<td>93</td>
<td>1000</td>
<td>4</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>Vaso-vagal</td>
</tr>
<tr>
<td>Sheinman</td>
<td>2003</td>
<td>61</td>
<td>...</td>
<td>405.0</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>Sided deep vein thrombosis on follow-up with cell pain</td>
</tr>
<tr>
<td>Etemadi</td>
<td>2000</td>
<td>20</td>
<td>94</td>
<td>1150.0</td>
<td>3</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>Brachial artery thrombosis that resolved spontaneously</td>
</tr>
<tr>
<td>Kang</td>
<td>2000</td>
<td>83</td>
<td>99</td>
<td>820.0</td>
<td>7</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>Pulmonary embolus unrelated to procedure</td>
</tr>
<tr>
<td>Mohler</td>
<td>2001</td>
<td>91</td>
<td>NR</td>
<td>98</td>
<td>500 to 1000</td>
<td>2</td>
<td>1</td>
<td>0</td>
<td>Local erythema at injection site (foxin); groin pain &gt;2 (humans)</td>
</tr>
<tr>
<td>Yasquez</td>
<td>2005</td>
<td>96</td>
<td>99</td>
<td>1000 vs 300</td>
<td>0</td>
<td>3</td>
<td>0</td>
<td>0</td>
<td>Infected abscess; blue toe; pain in leg and buttocks; anaphylaxis</td>
</tr>
<tr>
<td>Malheux</td>
<td>2003</td>
<td>101</td>
<td>...</td>
<td>260.0</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>2 intra-arterial thrombin injections with critical limb ischemia; PTA rupture after thrombosis</td>
</tr>
<tr>
<td>Paulson</td>
<td>2001</td>
<td>114</td>
<td>96</td>
<td>366.0</td>
<td>0</td>
<td>4</td>
<td>2</td>
<td>0</td>
<td>Allergic reaction (fever); decreased posterior and anterior tibial artery Doppler velocity</td>
</tr>
<tr>
<td>Khoury</td>
<td>2002</td>
<td>131</td>
<td>96</td>
<td>NR</td>
<td>9</td>
<td>3</td>
<td>2</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Greve</td>
<td>2004</td>
<td>132</td>
<td>99</td>
<td>669.0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Knutson</td>
<td>2005</td>
<td>240</td>
<td>6 to 24</td>
<td>100</td>
<td>6</td>
<td>2</td>
<td>1</td>
<td>0</td>
<td></td>
</tr>
</tbody>
</table>

**IV - Para-Aneurysmal Saline Injection (PASI)**

This is a relatively novel technique, first described first by Gehling et al.\textsuperscript{95} Physiologic saline is injected -ultrasound-guided- extravascular near the neck of the pseudoaneurysm. The technique involves compression of the pseudoaneurysm neck with tissue swelling produced by the injected saline until flow within the neck is obliterated, avoiding a compromise of flow in the underlying artery (Figure 23). Para-aneurysmal saline injection offers an alternative, minimally invasive, very simple, effective, and safe treatment approach for the closure of pseudoaneurysms after cardiac catheterization.
PASI was studied only in three series:

Gehling et al\textsuperscript{95}: PASI was used in treating six patients with post-catheterization pseudoaneurysms, with mean duration of 29±20 min and mean saline volume of 52±33 ml saline injected beneath the communication tract of the PSA yields to rapid occlusion in the six patients. During 4 weeks of follow up, the PSAs remained occluded in all patients. However this study included only six patients with small sized PSAs. This series was intended to be a pilot study and the true value of this technique has to be evaluated in a larger randomized study in comparison with other approaches (as mentioned by the others).

Finkelstein et al\textsuperscript{96}: Sixty-four consecutive patients with pseudoaneurysms after cardiac catheterization were treated using normal saline (0.9% sodium chloride 25 to 60 ml) injected into the tissue surrounding the tract connecting the pseudoaneurysm with the femoral artery, followed by manual pressure of short duration. In none of the patients was concomitant antithrombotic therapy (aspirin [n=63], clopidogrel [n=45], unfractionated or low molecular weight heparin [n=23], and warfarin [n=5]) discontinued during the closure attempt. Fifty-nine of the 64 pseudoaneurysms (92%) were successfully occluded using saline injection.
In 5 patients in whom saline injection failed, the pseudoaneurysms were successfully treated with thrombin injection (n=4) or ultrasound-guided compression (n=1). In all 64 patients, pseudoaneurysm closure was confirmed by ultrasound at 24 hours. The patients very well tolerated the procedure, and no side effects or complications were noted.

ElMahdy et al\textsuperscript{97} compared ultrasound-guided compression with para-aneurysmal saline injection in 80 patients with post-catheterization femoral aneurysms. The patient’s skin at the puncture site was prepared with povidone iodine, and sterile drapes were placed in the inguinal area. The sonography transducer was covered with a sterile laminated bag, and sterile gel was applied to the patient’s skin to prevent infections. After administration of subcutaneous anesthesia with 10 ml of 2\% lidocaine, an 18 gauge needle mounted on a plastic syringe filled with 0.9\% saline (drawn from a 500-ml saline bag) was advanced and positioned within 2 to 5 mm along the neck communicating the femoral artery and the pseudoaneurysm. After confirmation of extravascular and extra-aneurysmal needle position(Figure 52), saline was slowly injected until the resultant tissue swelling completely obliterated the pseudoaneurysm neck. Puncture of the femoral artery and the pseudoaneurysm was avoided in all patients. Repositioning of the needle and reinjection on the opposite side of the neck was performed if ultrasound monitoring indicated insufficient compression of the pseudoaneurysm neck. After saline injection, manual compression was applied for a 5-minute period.\textsuperscript{104}

Primary success was considered if the following two conditions are justified: complete thrombosis of the pseudoaneurysm cavity with no residual blood flow, using the highest possible color gain and lowest possible PRF (Pulse Repetitive Frequency) to detect any minimal residual flow. Obliteration of the neck, with loss of the characteristic Doppler waveform (to and fro) at the previous site of the neck.
If one or both of the previous conditions are not justified by the end of the attempt, so another one (for UGCR or PASI) or two attempts (for UGCR only) are planned within 24 hours, during which the thigh is wrapped with tight dressing and the patient was instructed to be kept in bed and maintain stretched limb. In cases with successful thrombosis of FAP, a dressing was wrapped around the thigh, and the patient was instructed to maintain bed rest following the procedure until the first follow-up examination. Final success: reflects the number of cases that were successfully treated with UGCR or PASI, fulfilling the criteria mentioned in the primary success and regardless the number of attempts needed.

PASI was done by single injection in 30 (75%) patients, while double injections were needed in 10 (25%) patients. The mean amount of injected saline was 50±33ml. All patients in the 2 groups had well felt pedal pulsations with triphasic Doppler waveform in the infrapopliteal arteries prior to the procedure and none of them showed altered waveform after treatment. The primary success rate was 75% (30/40) and 87.5% (35/40) for UGC and PASI respectively, (p=0.43). Figure 4 In the UGC group, 10 patients needed a second attempt and 3 of the 10 needed a third attempt. In the latter 3 patients, we failed to obliterate the FAP. Two of the failed cases were treated successfully by PASI and 1 case had surgical repair. In the PASI group, 5 patients needed a second attempt. In 3 of them, the FAP was obliterated successfully and in 2 of them, PASI failed to obliterate the FAP and the patients were repaired surgically. The final success rate was 92.5% (37/40) and 95% (38/40) for UGC and UPASI respectively, (p=0.75). In successfully treated patients, there was no reperfusion of the FAP in the follow up studies I and II in both groups, also there were no new symptoms, thromboembolic complication, infection at the site of arterial puncture and the Doppler waveform in the pedal arteries was triphasic during the follow up studies (I & II) in both groups.
We found that UGC and PASI have similar primary and final success rates. Both PASI and UGC were safe in terms of the absence of embolization and ischemic complications. However, we found that PASI can be accomplished in a significantly shorter time, almost half that time required for successful UGC. There was also a strong trend towards fewer self-limited vasovagal attacks with PASI. These differences should result in greater convenience for the operator and the patient. We found the technique of PASI to be easy and required short period of training. The duration of the procedure declined as we proceeded in the study till it became <20 minutes in the last cases. The procedure time in our study (30.33±8.56 minutes) is close to that reported by Gehling et al (29±20 minutes). The final success rate of PASI in our study (95%) is comparable to that reported by Finkelstein et al (92%). We were unable to specify factors associated with failure of PASI because of the small number of failures. Also we observed that patients with multilocular FAPs, who represent a problem during thrombin injection, could be successfully treated by PASI. The technique of PASI also fits well the labs with limited financial resources in the developing world, as it does not require any sophisticated equipment or expensive drugs. We conclude that ultrasound-guided para-aneurysmal saline injection is an effective method for treatment of femoral pseudoaneurysms. Compared to ultrasound-guided compression, it is faster, less likely to cause vasovagal reactions and can be more convenient to patients and physicians.

V–Endovascular management

PSAs may be excluded by embolisation, perfusion balloons, and by placement of covered stents/endoluminal prostheses (EP). On rare occasions, uncovered stents may be used in conjunction with embolic agents in aneurysms with wide necks to prevent embolic material leaving the sac.
Embolisation of PSAs has been carried out with a variety of agents including coils, thrombin, collagen and cyanoacrylate. The PSAs can be accessed via the neck or by direct puncture. Various techniques of direct puncture have been described including the placement of an angioplasty balloon across the neck reducing inflow and aiding haemostasis in the event of sac rupture. In a similar fashion PSAs can be temporarily excluded using a perfusion balloon; blood flows through a central channel at a rate sufficient to keep the limb perfused, while the neck of the PSA is excluded from the circulation.

Both coils and thrombin can be injected percutaneously into PSAs; the advantage of the latter is that it only requires a fine needle puncture. In the second technique, the parent artery can be embolised distal and then proximal to the PSA; some authors have recommended packing the sac in addition to proximal and distal occlusion (Figure 24 & Figure 25).

Figure 24. Completion angiogram demonstrating occlusion of the PSA, but normal flow down the SFA and PFA.  

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In the 1980s, the treatment with collagen insertion was first introduced, it is a nonsurgical, minimally invasive method to close the PSA percutaneously with collagen. This approach enabled the use of biodegradable adhesive bovine collagen to facilitate clotting within the false aneurysm and, thus, achieves the closure. When collagen comes in contact with blood, platelets aggregate on the collagen and release coagulation factors that, together with plasma factors, result in the formation of a fibrin matrix. Once implanted into tissues of an organism, collagen is ultimately degraded and progressively resorbed by granulocytes and macrophages (Figure 26).
Figure 26. Angiogram via the contralateral site. (A) Bilobulated PSA with its neck arising from the SFA. (B) PSA is completely visualized. (C) Direct injection of collagen through a needle. (D) Final angiographic control.

The advantages of collagen lie in its physical-chemical properties. The fact that it consists of long paste fibers allows the collagen to remain within the PSA cavity, which putatively reduces the risk of migration through the neck of the FAP or through a fistula. The injection of collagen is done under fluoroscopic guiding because this made better angiographic visualization possible, but as with thrombin injection, ultrasound-guided injection of collagen is certainly feasible. Insertion of collagen plugs in PSAs requires the use of large introducer sheaths (11F). Subsequently, the development of a paste-like application form of collagen permitted the use of smaller introducer sheaths (9F).102

The fibrin adhesive consists of a substrate; containing human fibrinogen, factor XIII and bovine aprotinin, and a “starter” containing human thrombin and calcium chloride in solution. The substrate and starter react within the
pseudoaneurysm to produce fibrin while the blood within the pseudoaneurysm should provide further substrate for the reaction, leading to thrombosis of the pseudoaneurysm.\textsuperscript{103} Fibrin adhesive injection may be done with or without balloon occlusion of the pseudoaneurysm neck. Currently, however, there is no evidence showing superiority in the treatment of pseudoaneurysms of fibrin adhesive over thrombin alone obtained from these proprietary preparations or that obtained from pooled blood products.\textsuperscript{104}

Indications for covered stents include: aneurysms that cannot be accessed via their necks or by direct puncture or where the overlying haematoma is to be surgically evacuated. The contraindications for these prostheses include infection, presence of the aneurysm at the site of a flexion or aneurysms related to arteries <7mm in diameter. The majority of these prostheses reported in the literature have been used for the exclusion of atherosclerotic aneurysms; however pseudoaneurysms have been treated in this manner.\textsuperscript{41,99,105,106}

Endovascular prostheses are available commercially or can be made on an individual basis. They must be placed across the neck of the IPA to exclude inflow to the sac (Figure 27).

\begin{figure}[h]
\centering
\includegraphics[width=0.8\textwidth]{image1.png}
\caption{A) DSA obtained to delineate the exact anatomy, B) Post treatment image demonstrates satisfactory exclusion following insertion of endoluminal prosthesis.\textsuperscript{107}}
\end{figure}
All vessels that might backfill the sac should be embolised prior to placement of the EP. For example, with PSAs of the iliac system, the internal iliacs must be embolised or the sac will fill by crossover flow leading to continued expansion of the sac and possible rupture.

US or spiral CT should be used to ensure sac thrombosis; continued filling should be regarded as treatment failure and the risk of rupture is not reduced.

Not all PSAs can be treated by embolisation and the contraindications include infection, essential parent artery, and uncontrolled outflow (for liquid agents.

**ACC/AHA Guidelines for the Management of Patients with Peripheral Arterial Disease** recommends the following regarding the investigation and therapy of catheter related femoral artery pseudoaneurysms:

**Class I**

1. Patients with suspected femoral pseudoaneurysms should be evaluated by duplex ultrasonography. (Level of Evidence: B)

2. Initial treatment with ultrasound-guided compression or thrombin injection is recommended in patients with large and/or symptomatic femoral artery pseudoaneurysms. (Level of Evidence: B)

**Class IIa**

1. Surgical repair is reasonable in patients with femoral artery pseudoaneurysms 2.0 cm in diameter or larger that persist or recur after ultrasound-guided compression or thrombin injection. (Level of Evidence: B)
2. Re-evaluation by ultrasound 1 month after the original injury can be useful in patients with asymptomatic femoral artery pseudoaneurysms smaller than 2.0 cm in diameter. (Level of Evidence: B)

Although a pulsatile mass is an obvious indication that a pseudoaneurysm may be present, a diagnostic duplex scan should be obtained whenever the diagnosis is even suspected.

In the absence of antithrombotic therapy, several studies have indicated that catheter-related pseudoaneurysms that are less than 2.0 cm in diameter tend to heal spontaneously and usually require no treatment. Figure 26 illustrates the spontaneous closure rate of selected pseudoaneurysms that were not immediately repaired, 90% of which resolved within 2 months.\textsuperscript{3429}

\begin{figure}[h]
\centering
\includegraphics[width=0.5\textwidth]{figure26.png}
\caption{Spontaneous closure rates of selected pseudoaneurysms.}
\end{figure}

Accordingly, small asymptomatic pseudoaneurysms probably can be managed conservatively unless they are still present on a follow-up duplex scan 2 months later.
At the opposite extreme, large pseudoaneurysms can rupture into the retroperitoneal space or the upper thigh or cause venous thrombosis or painful neuropathy by compressing the adjacent femoral vein or the femoral nerve.

Urgent surgical repair clearly is necessary if any of these serious complications occur, and until recently, it was the mainstay of treatment for most catheter-related femoral artery injuries. Many reports now have demonstrated, however, that the majority of uncomplicated pseudoaneurysms can be managed non-operatively with either ultrasound-guided compression therapy or the injection of miniscule amounts of thrombin directly into the pseudoaneurysm cavity.

Problems with ultrasound-guided compression therapy include pain at the site of compression, long compression times, and incomplete closure, each of which is more problematic with large pseudoaneurysms. Pseudoaneurysms ranging in size from 1.5 to more than 7.5 cm may be successfully obliterated by the injection of thrombin, 100 to 3000 international units, under ultrasound guidance.²⁹

The algorithm illustrated in Figure 28 presents an approach to the management of catheter-related femoral artery pseudoaneurysms
Figure 28. Diagnostic and treatment algorithm for femoral pseudoaneurysm.
Cases

In this section, examples for cases included in the prospective study conducted in the cardiology department, Cairo University, Cairo, Egypt from 2010 till 2013, comparing the safety and efficacy of PASI to UGCR are illustrated by figures.\textsuperscript{97} Cases were diagnosed after clinical suspicion of a vascular complications (hematoma, pulsatile mass, atypical pain, or a new femoral bruit) by vascular ultrasound scanning. All the patients with documented FAPs, have either large size PSA and/or having symptoms, so they were subjected to one of the two proposed treating modalities in the study (UGCR/PASI).\textsuperscript{97}
Case I (Successful UGCR)

Figure 29. Typical Doppler waveform at the neck of FAP

Figure 30. Totally thrombosed FAP after UGCR, with no residual blood flow could be detected with color box
Case II (Failed UGCR)

Figure 31. Typical Doppler waveform at the neck of FAP

Figure 32. Persistent FAP with minimal lining thrombi after second UGCR attempt
Case III (Successful PASI)

Figure 33. Post catheterization PSA arises from PFA

Figure 34. Complete obliteration of FAP after PASI
Case IV (Failed PASI)

Figure 35. US gray scale showing FAP

Figure 36. Persistent FAP after PASI
References


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