THORACIC VASCULAR INTERVENTIONS

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Evaluation and Treatment of Brachio Cephalic Arterial and Central Venous Diseases in the Chest

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Top 10 Pearls

1. SVC syndrome responds well to metal stent placement with long term primary patency of over 80% and long term secondary patency near 100%. Immediate clinical relief of SVC syndrome is the rule.
2. For central vein stenoses (non-SVC), especially in hemodialysis patients, venoplasty should be performed first with metal stents reserved for angioplasty failures.
3. In venous thoracic outlet syndrome, surgical correction of the costoclavicular constriction is preferred. Percutaneous therapy is directed towards pre-operative thrombolysis and post-operative recurrent venous stenoses.
4. Previous radiation or surgical manipulation of the central venous structures is a risk factor for angioplasty-induced venous rupture and pericardial tamponade or mediastinal/pleural hemorrhage.
5. The most common cause of brachiocephalic arterial (BCA) stenosis is atherosclerosis with the most common location being the proximal left subclavian artery followed by the proximal right subclavian artery and innominate artery origin.
6. Long segment, bilateral BCA stenoses should raise the suspicion of arteritides, such as Takayasu's arteritis or Giant cell arteritis. An elevated erythrocyte sedimentation rate is usually seen in active disease.
7. BCA disease presents with arm claudication, neurologic symptoms and/or angina (when subclavian artery feeds an internal mammary bypass graft to a coronary artery).
8. Technical success of treating BCA stenoses is near 100% with near 80% long term clinical success. The technical success of treating BCA occlusions is lower (70%) and, therefore, surgical treatment should be considered for long or very chronic BCA occlusions.
9. BCA intervention requires longer sheaths and catheters and occasionally brachial artery access, particularly to recannalize subclavian artery occlusions. Use a drug "cocktail" (see below) to minimize spasm and other complications with brachial artery access.
10. For difficult to open venous and BCA occlusions, a "through and through" wire access from the groin to the brachial artery can be used to maximize pushability of over the wire devices.

Part I. Thoracic central venous disease

Clinical Features

- SVC syndrome (SVCS) is typically caused by obstruction or occlusion of the SVC or both the Internal Jugular Vein's (IJV) and/or Innominate veins (INV). Symptoms typically only result from total obstruction to the venous outflow from the head and one open IJV down to the right atrium (RA) will avert symptoms.
- SVCS can be expressed by some or all of the following symptoms:
    - neck and facial swelling (typically around the eyes),
    - dyspnea, cough and hoarseness or change in character of voice
    - Headaches
    - tongue swelling, nasal congestion
    - epistaxis, hemoptysis
    - dysphagia
    - dizziness, syncope
    - lethargy
    - Bending forward or lying down may aggravate the symptoms
- Characteristic physical findings of SVCS:
    - prominent neck veins and increased number of collateral veins on the anterior chest wall
    - edema of the face, arms, and chest
    - cyanosis
- Obstruction of the subclavian veins or axillary veins results in swelling of the affected arm which usually improves without intervention. Similarly, unilateral and isolated innominate vein obstruction or occlusion commonly goes unnoticed as it is well-compensated by the jugular vein flow.
- Thoracic outlet syndrome (TOS), which is muscular or ligamentous compression of the vein as it leaves the chest, can cause persistent arm heaviness and swelling with chronic venous occlusion in typical locations on venography.
    - 1^{st} rib junction with clavicle (costoclavicular ligament)

Diagnostic Evaluation

- Detailed history needs to be obtained:
    - Previous malignant disease
    - Previous surgical history including history of venous catheterization or trauma
    - Previous history of radiation
    - Family history of venous disease such as thrombosis history or hypercoagulable states
    - Iodinated contrast allergy, history of renal dysfunction, history of cardiac valves or pacemaker, and history of claustrophobia in preparation for MRI, CT or venography
- Careful physical examination is mandatory
    - Pulses in affected extremity
    - Diameter discrepancy between affected/unaffected extremity
    - Ulcerations or skin changes
    - Auscultation/palpation for bruits (rule out AV malformations, fistulae)
    - Palpation for adenopathy (rule out lymphedema)
    - Lung/cardiac exam to rule out congestive heart failure or valvular disease as cause of right heart dysfunction and resulting edema

Laboratory

- Patients with unprovoked chronic thrombotic obstruction require careful hematological evaluation for hyper-coagulable states
    - Factor V Leiden deficiency
    - Antithrombin III deficiency
    - Protein S or Protein C deficiency

    - Anti-cardiolipin antibodies
    - Heparin-induced thrombotic thrombocytopenia (HITT)
    - Lupus anticoagulant
- Before any procedure is scheduled, check:
    - Creatinine
    - Electrolytes
    - Prothrombin time (PT) and activated partial thromboplastin time (APTT) if the patient is on anticoagulation
    - Complete blood count with platelet assessment

Imaging:

- Ultrasound plays an important role in the evaluation and diagnosis of the venous system.
    - Deep venous structures in the chest (central veins) may not be accessible with ultrasound but occlusions in inaccessible segments can be inferred by dampened venous signals without cardiac pulsations or response to Valsalva maneuver in the subclavian or jugular veins which are easily imaged by ultrasound.
    - Pulse-wave Doppler and color Doppler analysis of veins provides substantial physiological information (flow volume, valvular incompetence) which other imaging studies do not offer.
    - Acute venous occlusion
        - Hypoechoic clot in distended, non-compressible vein with few collaterals and significant subcutaneous and fascial edema
    - Chronic venous occlusion
        - Linear, hyperechoic, occasionally calcified clot with partial flow and compressibility in venous segment surrounded by multiple collateral veins. Minimal subcutaneous edema evident.
- Venography (conventional)—gold standard imaging study for venous anatomic evaluation.
    - Injection of contrast through a peripheral vein in the affected arm will provide information regarding anatomic structures and the location and degree of obstruction. Tourniquets or local compression allows visualization of deep venous structures or specific superficial venous pathways.
        - Compression of brachial/basilica veins improves opacification of the cephalic veins
    - Venography will not give accurate hemodynamic information. The presence of collaterals bypassing an obstructed vein indicates a hemodynamically significant occlusion.
    - Image quality on venography diminishes in the more central veins near the heart due to contrast dilution.
- Catheter based venography
    - Negotiation of a catheter past an obstructed or stenotic venous segment with injection of contrast through and beyond it gives better images of the obstruction and integrity of the remaining venous outflow to the heart. Pressure measurements can also be obtained through the catheter on both sides of the obstruction (Figure 1A) with a 3mmHg pressure drop considered a significant gradient and indicative of venous flow impairment.
- Intravascular ultrasound (IVUS)
    - Due to the inherent difficulties in visualizing the venous lumen with contrast due to contrast dilution, unopacified blood inflow, and the very thin and compliant nature of the venous wall, IVUS can obtain a more exact estimate of the degree of a venous stenosis as well as its nature (concentric or asymmetric stenoses, presence of clot, etc)
- Computer Tomographic Venography (CTV)
    - Essentially a delayed-contrast enhanced CT when venous opacification is optimal. Provides 3 dimensional and multiplanar venous imaging with excellent evaluation of endoluminal and perivenous pathology.

- Magnetic Resonance Venography (MRV) can be helpful under certain circumstances.
    - Consider this in patients with a serious iodinated contrast allergy and inability to have CTV
    - MRV with phase contrast imaging can provide flow speed, volume and direction information not available by CT
    - MRV can perform real time cine venograpy in any specified plane
        - Requires respiratory and ECG gating

Indications:

- Any symptomatic venous narrowing can be considered an indication for venoplasty and venous stenting.
- After thrombolysis of acute upper extremity venous thrombosis, the underlying venous stenosis/occlusion can undergo venoplasty/stent placement
    - Exception: non-operated venous thoracic outlet syndrome (see below).

Contraindications:

- Stents should not be placed in the subclavian vein across the first rib/clavicle junction in patients with Paget-Schroetter syndrome (venous thoracic outlet syndrome). When the first rib has been resected for this condition and restenosis occurs, a stent can be used.
- Infection is a relative contraindication and stents should not be placed in a patient with bacteremia. This is a relative contraindication as there are exceptionally few reports of vascular stent infections, especially in the venous system.
- Impaired renal functional is a relative contraindication as for all other procedures based on use of contrast agents.

Patient Preparation:

- During the procedure of venoplasty and stent placement the patient is given full anticoagulation using unfractionated heparin.
    - Alternative agents, such as direct thrombin inhibitors (bivalirudin) can also be used
        - May need dosage adjustment for renal/hepatic disease
- The patient should be fasting according to guidelines established for each practice setting.
- An intravenous access for delivery of medications such as sedatives and fluids should be available
- Antibiotics are not routinely given.

Relevant Anatomy:

- **Normal Anatomy**
    - The venous anatomy is more variable than that of the arterial system. Arteries and veins tend to travel side by side but venous aberrancy occurs in several important areas.

- **Aberrant Anatomy:**
    - A duplicated SVC exist in 0.3% of cases where the left SVC drains into the right atrium; usually via an enlarged coronary sinus
    - A left only SVC is less common
    - The left brachiocephalic vein can be compressed by the aortic arch and sternum (aortosternal compression) in vulnerable individuals
        - Aortic aneurysms
        - Aberrant right subclavian vein, especially if associated with a Kummerel aneurysm
        - Other aortic arch anomalies
        - Acquired or congenital sternal deformities
            - Fracture
            - Pectus excavatum
            - Surgical sternotomy
    - Variation in anatomy is more common in the peripheral veins

Equipment:

- Stents: The stents used in the venous system should be self-expanding in most cases.
- Common stent diameters for central venous disease vary from 10 mm to 16 mm.
- Covered stents have limited application in the central chest due to multiple major branching veins which should not be obstructed not only for clinical reasons but also to maintain future avenues for central venous access.
- There are many self-expandable stent types available including nitinol based stents and stainless steel based stents.
- The Gianturco Z-stent is unique for its large cell size and strong radial force
    - Customizable in length—suture stents together
    - Customizable in diameter—create a "flared" end by cutting and re-tying encircling suture at either end of stent
    - Difficult to obtain currently
- Balloon expandable stents are only used when extra radial force is needed and should not be used in superficial areas.
    - Fibrotic SVC stenoses
        - Cancer
        - Radiation
        - Fibrosing mediastinitis
        - Surgically ligated veins
- Catheters: 5 Fr angled angiographic catheters, preferably with hydrophilic coating are optimal for navigating occluded or obstructed veins.
- Guidewire: To recannalize chronically occluded veins or torturous veins try hydrophilic guide wire such as the Glidewire (Terumo). Stiff wires can be very helpful. Once the occlusion is crossed, use stiff braided guide wires as these give more stability.
- Angioplasty balloons: We usually use high pressure balloons to dilated chronically thrombosed veins. Frequently, balloons require > 20 mmHg pressure to "crack" tough venous strictures.

Pre procedure medications:

- Antibiotics are not routinely given
- Anti-anxiety medications (lorazepam, alprazolam) can be given pre-procedurally for very anxious patients

Procedure

Venous Access:

- Use ultrasound to guide venous access in the upper arm, chest or neck
- Selection of venous access depends on the anatomic location of the obstruction to be treated.
    - For the SVC, the common femoral vein (CFV) provides ideal access (right if possible)
    - Right IJV access works well also and may be preferred for anatomic reasons—i.e femoral veins are occluded
    - The subclavian and innominate veins may be easiest accessed from the ipsilateral upper arm veins (basilic or brachial veins) but CFV access can also be used for these veins.
    - Use of both jugular and femoral venous access with "through and through" wire access (achieved by snaring a wire from one vein and out the other) can be a useful technique for advancing catheters and angioplasty balloons through very long or fibrotic venous occlusions/stenoses. This is because control of both ends of a guidewire allows maximum catheter pushability over the taut wire.

Venography

Intra –procedural medications:

- Heparin typically 4000 units to 5000 units with a target activated clotting time (ACT) of 280 seconds to 300 seconds. If the procedure is prolonged, there may be need for additional doses based on periodic ACT determinations.
- Sedation and pain medication is provided by titrating Midazolam Hydrochloride and Fentanyl Citrate. Very rarely is more significant sedation or pain management needed
- Narcotic doses (Fentanyl) should be administered as needed but especially just before balloon angioplasty of venous strictures which can be quite painful.

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Assessing the Narrowing or Occlusion

Pre-existing studies such as ultrasounds, CT scans and MRI will give important information with regards to location and severity. These imaging tools will also help to identify contributing factors such as tumors or aneurysms compressing the vein. The final evaluation is then performed during the procedure by catheter-based venography and hemodynamics (venous pressure gradients).

- **Morphology:** The length and severity can be determined at the time of the catheter based venogram. It is helpful to use catheters with markers so more precise length and diameter measurements can be obtained. The diameter of the stent to be used is based on the diameter of the normal vein adjacent to the lesion.
- The SVC measures about 20-25mm in adults but can be over 30 mm in normal individuals. In practice, symptomatic SVC stenoses rarely require stents over 18 mm in diameter.

- **Location**

  Pressure Measurements: Use of pressure measurements in the venous system is debated. A consensus has built around a pressure difference (gradient) of 2 to 3 mmHg as significant in the venous system. The pressure is measured with a angiographic catheter on each side of the obstruction. In practice, instrument inaccuracy, respiratory variation, and transducer positioning produce errors which exceed this 2-3 mmHg threshold and, therefore, the patient's symptoms and degree of stenosis should guide treatment rather than hemodynamics.

Choosing a balloon or stent:

- As a general rule self-expanding stents are used in the venous system. Balloon-expandable stents should not be used outside of the chest cavity and are rarely used unless extra radial force is needed. Even then, balloon expandable stents are usually deployed inside self-expanding stents when they demonstrate early elastic recoil.
- Compression and deformation of balloon expandable stents does not spontaneously reverse as occurs with self expanding stents. Placing balloon expandable stents where they can undergo such external force can lead to severe consequences such as restenosis or re-occlusion which may be difficult to treat percutaneously.

Performing the procedure

- **Crossing the Lesion:** Chronic thrombotic occlusion of the upper extremity veins is frequently difficult to traverse. The combination of a glidewire and a 4 Fr or 5 Fr glide-coated angiographic catheter works best for this. A stiff angled glidewire works better than a soft or regular glidewire. When the lesion has been crossed a stiff guide wire (braided) can be placed, over which stents and balloons more easily travel.
- **Deploying the balloon/Stent:** Endpoint: Free flow without pressure gradient. Collateral filling is regarded as a reliable sign of persistent hemodynamic narrowing. Cover the entire segment of diseased vessel if possible.
- **Immediate post procedure care:**

- Observe the patient in post anesthesia unit until sedation has worn off (at least one hour).
- Observe for signs of bleeding or pericardial tamponade if superior vena cava stent was placed.
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Follow-up and post procedure medications:

- Upper extremity stents are usually not treated with anticoagulation following the procedure but this depends on the circumstances such as inflow.
- Clopidogrel is often given as 75 mg/day for 6 weeks following venous stent placement.
- For post-stent chest discomfort, pain medication may be needed. None-steroid anti-inflammatory drugs suffice in most cases. The pain wears off in 2 to 3 days in most cases.

Results:

- Central vein angioplasty—frequent causes
  - Dialysis-associated stenoses
    - Only treat those compromising dialysis access flows or with significant symptoms
      - Dilation of insignificant stenoses may damage vein and induce worse/symptomatic lesions in the near future
  - Central venous catheter placement—a growing problem
    - About 40% of upper extremity venous thrombosis is due to subclavian venous injury due to catheterization or cardiac pacer wire insertion
    - About 50% of patients who undergo subclavian venous puncture will develop a venous stenosis (most asymptomatic)
    - Almost half of all subclavian venous occlusions are clinically ignored or silent
  - Studies show that PTA is superior to stent placement in venous strictures that respond well to balloon dilation
    - Mean patency 13 months versus only 5 months in the stented group
  - For venous lesions with elastic recoil, stenting is preferred
    - Mean patency 8.6 months vs 2.9 months for PTA only group
- SVC stenting
  - Clinically rewarding as near 100% of patients experience dramatic and fairly immediate relief
  - Insertion of an SVC stent relieved malignant SVC syndrome in 95%; 11% of those treated experienced reocclusion but recanalization was possible in the majority resulting in a long-term secondary patency rate of 92% and primary patency of 83%
    - Remember, mean survival in malignant SVC syndrome is 6 months
  - For benign disease, SVC stents published primary patency rate is 86% at one year with secondary and assisted-primary patency in the mid 90% range.

Alternative Therapies

- Surgical bypasses are not commonly offered and there are only few centers where expertise exists.
  - For SVCS—
    - Right brachiocephalic vein bypass to the right atrium.
  - For subclavian or brachiocephalic vein occlusion
    - Axillary to brachiocephalic or right atrium vein bypass
    - “necklace” side-to-side brachiocephalic vein bypass
  - A small arteriovenous fistula is often intentionally created in conjunction with large vein surgical bypass in order to improve patency in these low flow vessels.

Complications

- Complications of venous angioplasty and stent placement are few.
    - Bleeding complications are rare.
        - Significant bleeding can occur if the treated area has been radiated or if surgery such as lymph node dissection has been performed recently

    - There are reported deaths from exsanguination or pericardial tamponade due to iatrogenic rupture of the SVC. Thus, the operator should be prepared to act quickly if patients deteriorate immediately or soon following SVC stent placement.

- **How to treat:**

    - Keep in mind that you can diagnose pericardial tamponade quickly with ultrasound and immediate placement of a pericardial drain is usually the treatment. A covered stent or surgery may be required if pericardial hemorrhage is persistent.
    - Re-inflate the balloon in the SVC if extravasation occurs to halt active bleeding and prepare to place a covered stent or initiate surgical repair if extravasation persists after 15-30 minutes of balloon inflation.

- **How to avoid:**
    - Be careful in areas of previous radiation treatment or surgical intervention especially if recent dissection or radiation was directed towards the target vein. Consider a covered stent if treatment is absolutely necessary.
    - Pericardial tamponade is hard to avoid. The length of the pericardial reflection varies much and can extend to the brachiocephalic vein confluence.
        - Be careful not to overdilate. We dilate rarely beyond 14 mm.
        - The primary use of covered stents may be an option.

Part II: Brachiocephalic arteries

Clinical Features:

- Brachiocephalic arterial lesions can involve the proximal portions of any of the major branches off the aortic arch and therefore present with symptoms of cerebral or upper extremity ischemia or both
- In patients with a coronary artery bypass constructed from the internal mammary artery (IMA), brachiocephalic or subclavian artery lesions can present with chest pain (angina) or acute myocardial infarction (MI).
- Most common cause is atherosclerosis due to the existence of multiple risk factors
    - Most common locations (in order)
        - Proximal left subclavian artery
        - Proximal right subclavian artery
        - Origin of innominate artery
- Less common causes--- arteritides with elevated erythrocyte sedimentation rate (ESR)
    - Giant cell arteritis
        - Elderly females
        - Diffuse, long segment, often bilateral brachiocephalic arterial disease
            - Classic is bilateral tapering long segment subclavian/axillary arterial occlusions
    - Takayasu arteritis
        - Younger, often asian females
        - Multiple stenoses occlusions in the brachiocephalic arteries
    - Radiation induced
        - Typically many years after radiation therapy for lymphoma
            - Radiation arterial fibrosis or acceleration of atherosclerosis within the treatment field
    - Fibromuscular dysplasia
        - Rare in the chest but seen occasionally in carotid arteries (2^{nd} to renal arteries in frequency)
    - Arterial thoracic outlet syndrome (ATOS)
        - Fixed or reversible compression of the proximal subclavian artery, generally between the middle and anterior scalene muscle (interscalene triangle)
        - Incidence is debated—between 3-80 cases per 1000 people

Diagnostic Evaluation

Clinical:

- Subclavian arterial disease presents with arm claudication and/or blue fingers or ulcerations on the fingertips
    - Claudication in the arm is different than in the leg, with “tiredness” and weakness more the presenting complaint rather than pain. Patients present earlier if dominant arm affected.
    - Vertebrobasilar insufficiency in lesions proximal to vertebral artery with “subclavian steal”
        - dizziness, unsteadiness, vertigo, and visual changes related to arm activity or neck position
    - Blood pressure differential exists between upper extremities and pulses may be diminished
    - Skin changes from embolic events, Raynaud’s phenomenon, or ulcerations
    - Characteristic severe drop in affected arm blood pressure with Adson maneuver in patients with arterial thoracic outlet syndrome (affected arm abducted and head turned towards normal arm)
- Brachiocephalic arterial disease can present with all of the above, plus:

- Symptoms of anterior (carotid) cerebral circulation ischemia or embolization
  - Transient ischemic attacks (TIA's)
  - Amaurosis Fugax
  - Stroke

Laboratory:

- ESR
  - to rule out/in arteritis
- Creatinine, Electrolytes
  - In preparation for angiography
- Complete blood count
  - Patients with arteritis frequently anemic
  - Check platelets
- PT, APTT
  - If patient anticoagulated
- Refer to vascular surgery for temporal artery biopsy
  - If giant cell arteritis suspected

Imaging:

- CTA/MRA excellent for anatomy and associated conditions
  - Consider provocative maneuver (affected arm abducted) if ATOS suspected
  - Consider delayed scan to see retrograde vertebral artery and distal subclavian artery in patients with suspected subclavian steal phenomenon
  - Not adequate to evaluate forearm arteries or digital arteries affected by embolic events
- Catheter angiography
  - Pigtail flush aortic arch
  - Selective subclavian and brachial angiography with long 5 fr catheter (100-120 cm berenstein, headhunter, davy)
  - Magnified view of hand angiography injecting contrast in brachial artery just above the elbow
    - 4-5 cc's per second for 4-5 seconds with early and delayed filming
    - Consider second hand angiogram after vasodilation
      - 10 mg Diltiazem (best) or 50-100 micrograms Nitroglycerin or 25-50 mg papaverine intraarterial
      - Improves images
      - Confirm whether digital artery occlusions are fixed or reversible
        - Reversible lesions may respond to axillary sympathetic nerve block or chronic oral calcium channel blocker therapy
  - Supplement angiography with pressure gradients across any lesions.
    - Use vasodilation, reactive hyperemia or hand/arm exercise to simulate activity and improve sensitivity for significant lesions
  - Can perform provocative maneuvers to check for ATOS
  - Be sure to visualize the internal carotid arteries
    - Atherosclerosis is common in this location
    - About 23% of patients receiving carotid-subclavian bypass also require carotid endarterectomy for significant occlusive disease

Indications:

- Percutaneous transluminal angioplasty (PTA) and/or stent placement is reserved for those patients with:
  - A hemodynamically significant arterial lesion
  - Significant symptoms attributable to the lesion

Contraindications:

- In patients with ATOS, surgical rib resection and/or scalenectomy is indicated
    - Stent/angioplasty ineffective
    - There is a role for thrombolysis in the case of arterial thrombosis
        - Patient goes to surgery after clot cleared and lesion is located and characterized
    - There is a role for angioplasty/stent in cases of subclavian restenosis after surgical repair of ATOS
    - In cases of a bovine arch, innominate angioplasty/stent carries the risk of bilateral carotid distribution stroke and should be undertaken carefully (relative contraindication
    - Endovascular repair can be attempted in long chronic subclavian occlusions but the success rate is poor and carotid-subclavian bypass is preferred in good surgical candidates.

Patient Preparation:

- Antibiotics prior to stent placement can be given but necessity is doubtful
- Sterile preparation of the groins and also the brachial artery on the affected side
    - May need access from two points
- If puncturing brachial artery, consider administering the “brachial cocktail” intra-arterial after catheterization to reduce spasm and access site thrombosis
    - 80 mg lidocaine (4 ml of 2%)
    - 2.5 mg verapamil (1 ml)
    - 100 micrograms Nitroglycerin (0.5 ml)
    - 0.5 ml sodium bicarbonate solution

Relevant Anatomy:

- 3 great arteries in sequence:
    - Innominate or brachiocephalic artery
        - Bifurcates to right common carotid and right subclavian
        - Bifurcation best seen on right anterior oblique views
    - Left common carotid
    - Left subclavian
        - Best seen on left anterior oblique views
- Aberrant Anatomy:
    - Most common is “bovine arch” where left common carotid comes off at origin of innominate
    - Left vertebral artery originates directly off the aortic arch
        - Protects some patients from subclavian steal if proximal left subclavian artery stenoses or occludes
    - Abberant right subclavian artery as the last branch off the arch
        - goes to the right in the posterior mediastinum, usually behind the esophagus

Equipment:

- Long sheaths in the range of 80-90 cm generally needed
- Similarly, angioplasty and stent delivery devices need 120 cm length
- Larger sheaths and device deployment usually from the groin to minimize sheath size and complications in the brachial artery
- Ultrasound equipment desirable for brachial artery puncture
    - I like to measure the diameter and puncture brachial artery with ultrasound
    - Small brachial arteries (below 5 mm), I might avoid altogether

Procedure:

Assessing the lesion

- Similar procedure to anywhere else but extra care is taken to avoid distal embolization of debris or air into the cerebral circulation
- After diagnostic angiography:
    - 5000 u heparin or 0.75 mg/kg bivalirudin IV
    - Intra-arterial injections of 40-50 micrograms nitroglycerin if spasm may be an issue
        - Not needed in larger arteries, especially if calcified.
- Cross lesion with hydrophilic or other torqueable wire.
- If unable to cross from femoral access, it is reasonable to cross from a brachial access
    - Snare wire afterwards to have through and through access to maximize success at crossing lesion with catheters, balloons, stents.
    - Avoid too much tension on the wire, as it can act like a “cheese cutter” and damage vessels
- Angiography with marker catheter to assess morphology and diameter.
- Check pressure gradient
    - Generally, a 10% systolic pressure drop is significant
- Ideally, advance sheath or guiding catheter beyond lesion, especially if primary stent placement is planned.

Performing the procedure

- If angioplasty affects the cerebral circulation
    - Instruct the patient to count or speak to you during balloon inflation and deflate immediately if neurologic impairment occurs
    - Mild limited chest pain or shoulder cramp normal
        - Severe pain means balloon oversized. Stop
    - Remove balloon over wire and check results with contrast injected through sheath
        - Stent if flow-limiting dissection or poor PTA result with adequate balloon size
        - Repeat PTA if residual stenosis and balloon undersized

Immediate post procedure care:

- Typical post arteriography care with emphasis on:
    - Neurologic checks—r/o stroke
    - Neurovascular checks in the arm, if brachial puncture done.

Follow-up and post procedure medications:

- Most operators give Clopidogrel 75 mg per day for 6-12 weeks
- Some subclavian artery angioplasty sites or stents can be followed by Doppler ultrasound
- Blood pressures in the arms should equalize

Results:

- Data from multiple pooled series—Brachiocephalic stenoses
    - Technical success—96%
    - Long term clinical success—78%
    - Neurologic complications—1%
    - Other complications—4%
- Data from multiple pooled series—Brachiocephalic occlusions
    - Technical success—73%
    - Long term clinical success—78%
    - 20% of patients received stents in these series

Alternative Therapies

- Surgical alternatives
    - Carotid-subclavian bypass
    - Subclavian-carotid transposition
    - Extra-anatomic bypass grafting
    - All the above have <5% morbidity and <2% mortality and long term patency rates of 70-100%

Complications:

- Access site thrombosis/hematoma/pseudoaneurysm
    - Access site occlusion a particular problem in brachial punctures, especially in young women
    - Embolic stroke during angioplasty/stent
        - Fortunately quite rare
        - Vertebral artery does not revert to antegrade flow for about 20 seconds after angioplasty/stent for subclavian steal.
            - Protects posterior circulation from emboli
    - Emboli to arm/hand/fingers
        - Also rare
        - May not respond to thrombolytics as it may be old clot or atheromatous debris
            - Try suction embolectomy if possible
            - Surgical embolectomy may be necessary

How to treat complications:

- Thrombin injection or compression for pseudoaneurysms
    - Can be done carefully in the brachial artery
- Usually surgical exposure and repair for brachial occlusion
    - Thrombolysis has high risk of bleeding from fresh puncture site
- Brachial puncture does not carry risk of neurologic compromise like axillary puncture
    - Hematomas usually self-limited

How to avoid complications:

- Liberal use of anti-spasm medications (“brachial cocktail”) when accessing the brachial artery
- Don’t use brachial artery if small or in young females unless absolutely necessary
- Minimize sheath size in brachial artery
    - Advance larger devices through femoral access
- Anticoagulate to ACT of near 300. Use meticulous angiographic technique
    - Minimize risk of thrombotic or atherosclerotic embolization.
- Monitor patient closely during PTA/stent placement in brachiocephalic arteries
    - Also monitor neurologic status post procedure.

SUGGESTED READING

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Pulmonary Artery Intervention for Pulmonary Embolism

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Top 10 Pearls

1. Pulmonary embolism is a leading cause of death in the United States.
2. In asymptomatic patients, anticoagulation therapy is indicated.
3. For submassive PE (right ventricular dysfunction on echocardiography without attendant shock or hemodynamic instability), anticoagulation only may be indicated.
4. For massive PE, additional treatment such as systemic thrombolysis, surgical thrombectomy, or percutaneous catheter directed therapy are indicated.
5. Criteria for massive PE are arterial hypotension and cardiogenic shock.
6. Pulmonary artery intervention is reserved for massive PE.
7. Systemic IV thrombolysis for massive PE has been shown to reduce the risk of death or recurrent PE by 55%.
8. Catheter directed thrombolysis for massive PE has been shown not to offer any advantages over systemic IV thrombolysis.
9. Surgical embolectomy and thromboendarterectomy for massive PE are performed if there is no response or contraindication to thrombolytic therapy.
10. Percutaneous thrombectomy devices resulted in immediate hemodynamic improvement in greater than 80% of patients with massive PE, however, the majority of cases combined thrombolysis.

Pulmonary embolism (PE) is a leading cause of death in the United States. The true incidence of PE is not known, but it is estimated that there are 530,000 cases of symptomatic PE[1], and 150,000 cases of acute massive PE annually.[2] The 30 day mortality rate for massive PE is almost 30%,[3]and the presence of shock in these patients results in a 3-7 fold increase in mortality, with a majority of deaths occurring within one hour of presentation.[4]

Therapeutic options are anticoagulation, systemic thrombolysis, percutaneous catheter directed therapy and surgical thrombectomy. In asymptomatic patients, anticoagulation therapy is indicated. For submassive PE (right ventricular dysfunction on echocardiography without attendant shock or hemodynamic instability), anticoagulation only may be indicated, however, this issue remains controversial. A systematic review and meta-analysis performed by Ramakrishnan of 494 patients undergoing thrombolytic therapy for submassive PE revealed a lack of clear mortality benefit, combined with the potential for harm and increased health care cost with the use of thrombolytics.[5] Stein, Beemath, et al. concluded in the evaluation of 76 hemodynamically stable patients with PE and right ventricular enlargement that right ventricular enlargement alone does not adversely affect prognosis and is not an indication for thrombolytic therapy.[6]

For massive PE, additional treatment beyond anticoagulation such as systemic thrombolysis, surgical thrombectomy, or percutaneous catheter directed therapy are indicated. Criteria for massive PE are arterial hypotension and cardiogenic shock. Arterial hypotension is defined as a systolic arterial pressure <90 mm Hg or a drop in systolic arterial pressure of at least 40 mm Hg for at least 15 minutes. Shock is manifested by tissue hypoperfusion and hypoxia, including an altered level of consciousness, oliguria, or cool, clammy extremities. Early mortality in patients with massive PE is at least 15% and the degree of hemodynamic compromise is the most important predictor of in-hospital death.[7]

Pulmonary artery intervention is reserved for massive PE. Once massive PE is suspected, high dose heparin, larger than usual, should be administered because standard doses frequently do not reach therapeutic levels. Crystalloids should be administered to increase cardiac output,[8]however, caution should be applied to prevent fluid overload. PE related shock is addressed with pressors.

Systemic IV thrombolysis for massive PE has been shown to reduce the risk of death or recurrent PE by 55%.[9] Thrombolysis has been shown to have a high risk of bleeding complications, however. 22% of the 304 patients enrolled in the International Cooperative Pulmonary Embolism Registry developed major bleeding and 3% developed intracranial bleeding.[10]Alteplase (rt-PA) is the preferred agent, administered as a 100 mg continuous 2 hour infusion or accelerated dosing of 0.6 mg/kg over 15 minutes. Alternatively, Urokinase 4400 IU/kg as loading dose over 10 minutes, followed by 4400 IU/kg/hr over 12-24 hours or 3,000,000 IU over 2 hours. Streptokinase 250,000 IU as loading dose over 30 minutes, followed by 100,000 IU/hr over 12-24 hours.[11]

Catheter directed thrombolysis for massive PE has been shown not to offer any advantages over systemic IV thrombolysis. Although it would seem that there are fewer hemorrhagic complications with catheter-directed thrombolysis, there is no supportive evidence in the literature. Thrombolytic regimens: rt-PA-bolus of 10 mg followed by 20 mg/hr over 2 hours (total of 50 mg), or 100 mg over 7 hours; Urokinase-infusion of 250,000 IU/hr over 2 hours, followed by an infusion of 100,000 IU/hr for 12-24 hours.[12]

Surgical embolectomy and thromboendarterectomy for massive PE are performed if there is no response or contraindication to thrombolytic therapy. Sternotomy is performed with opening of the main pulmonary artery for clot retrieval with a long forceps.[13]Although modification in anesthesia and circulatory bypass technology have greatly improved, significant morbidity and mortality persist.[14]Mortality rate after surgical embolectomy is about 30%.[15]Patients with pulmonary hypertension and right heart failure secondary to chronic thromboembolic disease may benefit from pulmonary thromboendarterectomy.[14]

33% of patients with massive PE are not candidates for thrombolysis due to major contraindications (i.e. prior surgery, trauma, stroke, or advanced cancer).[7] Few centers perform urgent surgical embolectomy for patients with massive PE and contraindications to thrombolysis. Only 1% of patients with massive PE and cardiogenic shock underwent surgical embolectomy upon review of the two largest PE registries.[7,10,15]For patients with contraindication to thrombolysis or where surgical embolectomy is not available or feasible, percutaneous catheter thrombectomy is the only other option. Small cohort studies[16] have suggested that the clinical outcome of percutaneous catheter intervention is similar to that of surgical embolectomy, however, no controlled clinical trials have been performed that compare the two.

Three criteria should be present prior to consideration of catheter thrombectomy for acute PE.

1. Hemodynamic instability, defined as a systolic arterial pressure of <90 mm Hg, a drop in systolic arterial pressure of at least 40 mm Hg for at least 15 minutes, or ongoing administration of pressors for the treatment of systemic arterial hypotention.[17]
2. Occlusion of the right, left or main pulmonary artery demonstrated by chest CT or pulmonary angiography.
3. One or more of the following contraindications to thrombolysis[18]: active bleeding; history of intracranial bleeding, head injury, ischemic stroke, brain tumor, or neuro-surgery; surgery, delivery, organ biopsy, or puncture of a noncompressible vessel within 10 days; GI bleeding within 15 days; major trauma within 15 days; active cancer with known hemorrhagic risk; platelet level of >50,000 cells/uL or international normalized ratio of > 2.0; and pregnancy.

Some percutaneous thrombectomy devices are designed to remove clots, whereas others fragment, macerate or aspirate. The purpose of these devices is to provide rapid relief of central obstruction. Most of the devices break clot down into smaller fragments, which migrate peripherally into the pulmonary artery with resultant clearing the main pulmonary artery and improving perfusion. The cross-sectional area of the distal arterioles is greater than four times than that of the main pulmonary arteries and the volume is two times. By redistributing the larger central clots into the periphery, cardiopulmonary hemodynamics may acutely improve with increase in pulmonary blood flow and right ventricular function. Follow-up catheterization and

pulmonary perfusion studies after percutaneous redistribution have provided evidence for reduced pulmonary pressure and improved perfusion. Immediate hemodynamic improvement was demonstrated in greater than 80% upon review of the available literature, however, interpretation of the results is difficult because the majority of reported cases combined thrombolysis.[19,20,21]

Percutaneous thrombectomy devices are frequently combined with thrombolytic therapy. Thrombolytic therapy prior to percutaneous thrombectomy may soften clot, speeding up debulking and fragmentation.[21]Thrombolytic therapy following percutaneous thrombectomy exposes fresh surfaces of clot to thrombolytics, facilitating emboli reduction.[22]Certain thrombectomy devices are used with thrombolytics simultaneously. The end-point of the procedure should be hemodynamic improvement, regardless of the angiographic result.[23]

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Endovascular therapy of the thoracic aorta (TEVAR)

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Top 10 pearls

1. TEVAR should be considered for the treatment of aortic pathology involving the descending thoracic aorta.
2. Aortic lesions involving the aortic arch and thoracoabdominal aorta can be treated by hybrid endovascular procedures or by branched devices.
3. The diameter threshold for intervention on the descending aorta is 5.5cm.
4. In most cases, insertion of these devices is straightforward and does not involve a long procedure time.
5. A femoral arteriotomy is usually required, although percutaneous insertion using closure devices is feasible.
6. The technical success rates are very high and approach 100% in most series.
7. The conversion rate to open surgery is negligible.
8. The paraplegia rate is around 2% and the stroke rate is approximately 4% in most series.
9. The most common complication is a type 1 endoleak. This requires reintervention and usually involves the insertion of additional endografts.
10. The technique is relatively new and follow-up by imaging should be lifelong.

The development of stent-grafts and their use in the thoracic aorta has significantly changed the management of diseases involving the descending aorta and the aortic arch in the last decade or so. Thoracic aortic aneurysms, Type B dissections, and traumatic transections can now be treated without the need for thoracotomy and cardiopulmonary bypass with the obvious potential advantages in terms of reduced morbidity and mortality.

<u>Indications for thoracic aortic stent grafts</u>

- Descending thoracic aneurysms >5.5cm
- Complicated type B dissection – rupture, aneurysm formation, visceral ischaemia.
- Traumatic aortic transaction
- Aortobronchial fistula
- Penetrating aortic ulcers
- False aneurysms.

<u>Inclusion criteria for endografting:</u>

- The maximum diameter for a landing zone is 42mm and the minimum is 18mm. The landing zones should be at least 20mm in length.
- It is possible to extend the length of the proximal landing zone by intentionally covering the left subclavian artery with the stent-graft, usually after carotid subclavian bypass surgery. The landing zone can be further elongated by elective bypass of the left common carotid artery to the right common carotid artery.
- The distal landing zone length can be increased by bypass of one or more of the celiac, superior mesenteric and renal arteries.

<u>Commercially Available Devices (Europe)</u>

a. Valiant (Medtronic, Santa Rosa, CA, USA).

- External skeleton of Nitinol stents sewn to thin-walled polyester graft material by non-absorbable sutures.
- Available in a good range of lengths and wide diameters. Sizes range from 24-46mm in 2mm intervals and three lengths between 110mm and 215mm (covered length). The

delivery system varies from 22-25 French depending on the diameter of stent graft used. The recommended oversizing is 10-20% for aneurysms and up to 10% for dissections.

b. TAG thoracic Endoprosthesis (W.L. Gore, Flagstaff, AZ, USA.) (ONLY device approved for use in the USA)

- Nitinol exoskeleton heat sealed to non porous expanded polytetrafluroethylene (PTFE).
- The delivery system is inserted through a 30cm introducer sheath (20-24 French depending on stent diameter).
- The stent is released by traction on a cord which unzips the restraining sleeve and deploys from its center outwards in less than 1 second. This reduces the possibility of the cardiac output causing stent malposition during deployment.
- The TAG device is available in diameters of 26-40mm. Lengths between 10 and 20cm are available.
- Its ease of use and rapid delivery is advantageous in acute aortic rupture, where speed is important.
- The "all or nothing" nature of the delivery system is also its major disadvantage and it cannot be repositioned once deployed.

c. Zenith TX2 endograft (William Cook Europe, Bjaeverskov, Denmark)

- The stent graft is constructed of stainless steel Z stents sutured to polyester fabric graft. Fixation is enhanced by both proximal and distal barbs.
- The stent graft is mounted on a central core within a braided sheath. Trigger wires hold the device in position even when the sheath has been retracted.
- The TX2 endoprosthesis is available in 28-42mm diameters and in both straight and 4mm tapered proximal components. Both the straight and tapered components are available in at least two lengths for each diameter ranging from 108 to 216mm. The device should be oversized by 10-25% and at least a 3cm landing zone. The device has the lowest profile sheath, only 20 French for diameters of up to 34mm, and 22 French for the larger sizes.

Insertion Procedure:

The procedure is performed under general or regional anaesthesia although stents can also be inserted under local anaesthesia alone. A pigtail catheter is placed in the aorta via the contralateral femoral artery or a brachial artery to perform angiography prior to device deployment.

After a femoral arteriotomy has been performed, a 260cm long extra stiff guidewire (e.g. Lunderquist wire, William Cook) is advanced so that the tip is placed in the low ascending aorta. The stent-graft on its delivery system is advanced over the guidewire to the desired site of deployment. Accurate positioning is achieved by serial aortography. When the correct position is achieved, the stent-graft is released. Balloon dilation may be performed after stent deployment but is by no means routine and is contraindicated in patients with dissections because of the risk of rupturing the dissection flap.

Follow-up

Patients are followed up by serial CT scans and plain radiography predischarge, at 3months, at 12 months and at annual intervals thereafter. Most problems at followed up can be solved by additional interventions without the need for open surgery.

Outcomes of TEVAR

Endovascular repair of the thoracic aorta (TEVR) has become an established treatment modality despite a relative paucity of evidence, with a lack of the randomized trials that have accompanied other new vascular procedures. The majority of available data are obtained from case series and registries.

Descending thoracic aneurysms: The survival of patients with untreated TAA is bleak and is estimated to be 13-39% at 5 years (1). The results of open repair in centres of excellence are good with 3 day mortality rates for all types of thoracic aneurysm below 12% and paraplegia rates

below 4% (2). Community results which incorporate several centres are more realistic and demonstrate 30-day mortality rates approaching 20% (3).
Outcome data for endovascular repair of thoracic aneurysms are available from several sources. Leurs *et al* on behalf of the EUROSTAR collaborators reported data in 249 patients with 30-day mortality for elective TEVR of 5.3% and paraplegia of 4% (4). A cohort of patients with TAA who underwent endografting with the Gore TAG device was compared retrospectively with the results of a cohort of 94 patients who underwent open surgical repair. The perioperative mortality (2.1% vs 11.7%), paraplegia (3% vs. 14%) and freedom from major adverse event (48% vs. 20%) rat.es were all better in the endovascular group (5).
Similarly, the European Talent Registry reported technical success of 98%, in-hospital mortality in 5% (4.1% and 7.9% for elective and emergency procedures, respectively), paraplegia in 1.7% and stroke in 3.7% (6) . Similar outcomes have been reported for the newest generation of endografts, despite the fact the patients in these later data series had more challenging anatomy compared with earlier series (7).

Thoracic Dissection and Acute Aortic Syndrome.
The management of acute Type B dissections is principally medical, with surgery reserved for complications. Overall, medical management of patients with acute aortic dissection has a mortality rate of just over 10%. Surgical intervention in patients with complicated dissection has a mortality rate of approximately 30% (8).
The early results of endovascular repair of acute complicated Type B dissections were vastly better than the open surgical alternative. Most series reported mortality rates below 10% with paraplegia rates of less than 3% (4,9-12). These findings stimulated a rapid change in management and most vascular centres would now regard endovascular therapy to be the first line treatment for acute complicated Type B dissections.
Indications for repair of chronic dissections have usually been limited to the onset of complications, and an aortic diameter exceeding 5.5-6.0 cm. The availability of data regarding the outcomes of EVR for chronic dissections is very poor. In the series to date the mortality rates have been acceptable but the long-term success in preventing aortic expansion is unclear.

Traumatic Aortic Injury.
In the last decade, due to its low complication rate, TEVR has in many centres superseded surgery for TAI. The procedural time is short and the operation confers very little in terms of additional morbidity to these severely ill patients. Due to the focal nature of the injury, only a short length of aorta requires covering with an endograft.
Although TEVR seems to have become the gold standard for TAI, there are relatively limited data on outcomes. However, the procedural mortality is less than 10% throughout and the reported risk of paraplegia is negligible (13-15), outcomes much improved compared with surgery. The main drawback concerns the unsuitability of the devices available for the patients who require TEVR. TAI occurs in a relatively young population with narrower aortas and more angulated aortic arches than older patient. None of the devices currently available conform very well to angulated arches

In summary, the advent of endovascular repair for thoracic aneurysms has changed practice. In my view, there is enough evidence to suggest that TEVR should be used as first line therapy for most thoracic aneurysms involving the descending aorta and probably the aortic arch. Regarding dissection, endovascular repair should be considered the gold standard for complicated acute Type B thoracic dissections. There appears to be no justification for the repair of uncomplicated acute dissections. The indications and methodology for the treatment of chronic dissections remain undefined. TEVR for TAI has reduced complication rates compared with surgery, however improvement in the devices is required before it can be recommended as first line therapy for this indication.

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