Pearls & Pitfalls of Acute Aortic Syndrome Resuscitation

I. Disease Overview and approach to initial management
   a. Acute aortic syndrome comprises 3 specific aortic diseases:
      i. Acute aortic dissection
      ii. Intramural hematoma (IMH)
      iii. Penetrating atherosclerotic ulcers (PAU)
   b. In symptomatic patients, all 3 of these should be treated just like an aortic dissection in the ED.

II. Critical elements of initial resuscitation
   a. Minimize aortic dP/dT
      i. Minimize "dP": Decrease the steepness of the rise in aortic pulse pressure, creating a laminar blood flow in the aorta to reduce sheer stress on the injured aortic wall.
         1. Reduce the systemic arterial pressure as low as possible without compromising visceral perfusion – in some ways, analogous to the concept of damage control resuscitation in trauma.
         2. **Target SBP:** < 120
      ii. Minimize "dT"
         1. Reduce the frequency of contractile force on the aorta by reducing the patient's heart rate
         2. **Target HR:** 60 – 70

III. Pharmacologic pearls & pitfalls
   a. Vascular access: Invasive vascular access is not *required*
      i. Venous: all meds can be delivered with 2 peripheral IVs
      ii. Arterial access – If I am placing an arterial line, where should it go?
         1. Pulse deficits – present in 19-30% of Type A dissections[3]
         2. Place your a-line in the arm that does not have a true/false lumen.
            a. Subclavian branch vessel dissections can often cause reduced limb blood flow causing "pseudohypotension"
            b. Can lead to under resuscitation & hemodynamic control
            c. If you feel comfortable, use your CT scan to delineate the path of the false lumen
            d. If unsure, use the arm with the higher blood pressure to titrate vasoactives

b. Pain management first
    i. Early and aggressive narcotic administration can significantly reduce patient's intrinsic sympathetic response, reducing the amount of vasoactives needed to achieve resuscitation end points.
c. Heart rate control second: Esmolol vs. labetalol
    i. No head to head evidence, however must be familiar with dosing.
    ii. **Labetalol**: α- and β-blockade
        1. Loading dose: 20mg IV
        2. Infusion: -.5 – 8 mg/min, titrate by 1mg/min q10 min
        3. Pros: Simpler approach, simpler approach to both BP and HR control
        4. Cons: longer half-life of about 4.5 hours, so can linger if patient decompensates.
    iii. **Esmolol**: pure β_1-blockade – HR control only
        1. Loading dose: 0.5 mg/kg IV over 2-5 minutes
        2. Infusion: 50 mcg/kg/min, titrate by 50 mcg/kg/min q4 min
        3. Pros: Short half-life, only about 4-5 minutes
        4. Cons: More challenging dosing administration if unfamiliar with medication.

d. Systolic arterial pressure reduction: Nicardipine, Labetalol
    i. **Nicardipine**: Pure α-blockade, arterial vasodilator
        1. Dose: 2.5 mg/hr IV, titrated by 2.5 mg/hr, max 15 mg/hr
        2. Limitations
            a. Beware in patients with concurrent heart failure (i.e aortic insufficiency) as can cause pulmonary shunting and hypoxemia
            b. Beware starting first, as can lead to reflex tachycardia (to maintain cardiac output).
    ii. **Nitroprusside**: potent arterial/venous dilator by increasing nitric oxide release
        1. No longer recommended, unless no other drugs available – we have better drugs in 2018.
        2. Side effect is literally poison! Patients, particularly those with liver dysfunction can develop cyanide toxicity
        3. In addition, antihypertensive effect can be unpredictable, can cause coronary steal, excessive reflex tachycardia[1,2]

IV. **Clinical Pearls & Pitfalls**
a. **Failing to consider acute aortic insufficiency early in patients with medically refractory tachycardia**
    i. Cause of aortic insufficiency: Aortic root dilation, malcoaptation of aortic leaflets, detachment of one or more aortic valve leaflets, or intussusception of intimal flap into left ventricular outflow tract

ii. Prioritize blood pressure goal over rate control
iii. Aggressive rate control can often dramatically reduce cardiac output and significantly increase aortic regurgitation, pulmonary edema, and hypoxemic respiratory failure.

b. Avoid early intubation the patient with a Type A dissection and pericardial effusion if possible

i. Often recommended to intubate if "hemodynamically unstable"
ii. Cardiac filling & output are often dependent on negative transpleural pressure that occurs with spontaneous filling in the setting of early tamponade.[4]
iii. Positive pressure ventilation, analgesia, & sedation can reduce venous return and cause hemodynamic collapse in the setting of a worsening pericardial effusion.[5]
iv. Judicious fluid resuscitation to improve cardiac filling may temporize patient to the operating room.

v. Is pericardiocentesis safe?
    1. Controversial, in general, time better spent getting to OR.
    2. If patient hypotensive/peri-arrest & refractory to initial fluid resuscitation, consider performing a "controlled" pericardial drainage (CPD).[6,7]
        a. Case series of 18 patients, mortality 16.8% - excellent compared to reported mortality of Type A dissection with tamponade (often in excess of 50%)
        b. Hayashi approach to CPD: Prone position, local anesthesia, 8 Fr pigtail inserted under ultrasound.
        c. Goals: 5-10 mL of drainage performed with continuous BP monitoring.
        d. **Resus Target**: Systolic BP 80-90 mmHg.
        e. After CPD performed, transferred directly to operating room for aortic repair (< 60 min)

c. Pitfall: The belief that type B dissections are a medical disease

i. Long term outcomes from INSTEAD-XL trial[8]
    1. RCT of elective TEVAR vs. medical management
    2. Improved all-cause mortality at 2 & 5 years (4.1% versus 28.1%)
    3. Reduced progression of dissection and false lumen thrombosis (90.6%) with elective TEVAR + medical management
ii. If vascular surgery not available locally, consider transfer to high volume center in conjunction with critical care management.

Selected References

1. Oparil S, Aronson S, Deeb GM, et al. Fenoldopam: a new parenteral antihypertensive: consensus roundtable on the management of perioperative hypertension and hypertensive crises. *Am J Hypertens*. 1999;12(7):653-664.
2. Varon J, Marik PE. The diagnosis and management of hypertensive crises. *Chest*. 2000;118(1):214-227.
3. Hagan PG, Nienaber CA, Isselbacher EM, et al. The International Registry of Acute Aortic Dissection (IRAD): new insights into an old disease. *JAMA*. 2000;283(7):897-903.
4. Faehnrich JA, Noone RB, White WD, et al. Effects of positive-pressure ventilation, pericardial effusion, and cardiac tamponade on respiratory variation in transmitral flow velocities. *J Cardiothorac Vasc Anesth*. 2003;17(1):45-50. doi:10.1053/jcan.2003.9
5. Ho AM-H, Graham CA, Ng CSH, et al. Timing of tracheal intubation in traumatic cardiac tamponade: a word of caution. *Resuscitation*. 2009;80(2):272-274. doi:10.1016/j.resuscitation.2008.09.021
6. Hayashi T, Tsukube T, Yamashita T, et al. Impact of controlled pericardial drainage on critical cardiac tamponade with acute type A aortic dissection. *Circulation*. 2012;126(11 Suppl 1):S97-S101. doi:10.1161/CIRCULATIONAHA.111.082685
7. Adler Y, Charron P, Imazio M, et al. 2015 ESC Guidelines for the diagnosis and management of pericardial diseases: The Task Force for the Diagnosis and Management of Pericardial Diseases of the European Society of Cardiology (ESC)Endorsed by: The European Association for Cardio-Thoracic Surgery (EACTS). *Eur Heart J*. 2015;36(42):2921-2964. doi:10.1093/eurheartj/ehv318
8. Nienaber CA, Kische S, Rousseau H, et al. Endovascular repair of type B aortic dissection: long-term results of the randomized investigation of stent grafts in aortic dissection trial. *Circ Cardiovasc Interv*. 2013;6(4):407-416. doi:10.1161/CIRCINTERVENTIONS.113.000463