The role of IABP in Cardiogenic Shock;

Get pumped about the noble balloon

Nepean WTET summary 15/9/20

Background and Rationale

- The intra-aortic balloon pump (IABP) provides mechanical assistance in cardiogenic shock by reducing left ventricular (LV) afterload (↓s myocardial work) in systole and increasing coronary perfusion pressure (CPP; ↑s myocardial oxygen delivery) in diastole, improving stroke volume (SV) and thereby cardiac output (CO)
- Improved CPP may be of particular use for incomplete revascularisation in an acute coronary event
- A balloon inserted via the femoral artery is placed in the descending aorta to lie below the left subclavian artery and above the renal arteries. It is inflated using helium (low viscosity gas) in diastole and deflated during systole. Differing volume balloons based on patient height (affects SV)

Advantages and Disadvantages

- Advantages
    - Many pharmacological inotropic agents increase myocardial work +/- oxygen consumption thereby potentiating further a mismatch between supply-demand and worsening any pre-existing myocardial ischaemia, the IABP avoids this for reasons explained above
    - Proarrhythmogenic consequences of high dose catecholamines can be avoided
    - IABP is of particular benefit in those with ventricular septal defects (VSD) and severe mitral regurgitation (MR e.g. secondary to papillary muscle rupture in acute MI) above and beyond pharmacological agents due to improvement in forward flow (rather than just 'contractility')
    - Veno-arterial extracorporeal membrane oxygenation (VA ECMO) another mechanical support device increases LV afterload and thereby work (it also increases LV distension; IABP can be used here also as an adjunct to reduce the harmful effects of this)
    - Unlike ECMO, the impella and ventricular assist devices (VADs) IABP maintains pulsatile flow
- Disadvantages
    - Contraindicated in moderate-severe aortic regurgitation (increases regurgitant jet), aortic dissection; relatively contraindicated in severe peripheral arterial disease, severe coagulopathy, AAA/grafted, infection at potential insertion sites
    - Complications; from insertion (e.g. vascular injury), malposition (e.g. impaired renal blood flow → AKI), physical presence (acute limb ischaemia and thromboembolic phenomena e.g. CVA); equipment failure (failure to unwrap and balloon rupture → gas embolism)
    - Less effective in (tachy)arrhythmias, mistimed inflation/deflation → low efficacy / harm

Key studies

IABP vs standard care

- TACTICS trial 2005 J Thrombosis & Thrombolysis
    - MC (non-PCI only) RCT; Acute MI complicated by hypotension (of presumed cardiogenic aetiology), severe heart failure (frank pulmonary oedema and hypoperfusion - cool/clammy/oliguric/↓GCS) or CI<2.2 (<2.5 if on inotropic drugs) randomised to thrombolysis +/- IABP within 3h thrombolysis for 48h 1:1 then wean. N=57

    - Primary outcome; all-cause mortality at 6m 43% in fibrinolysis only group, 34% in IABP-fibrinolysis group, adjusted P=0.23. Subgroup benefit (P=0.05) if severe (Killip class 3/4) where 80% mortality if fibrinolysis alone
    - Of 27 in no IABP group, 9 crossed over to IABP, 3 in IABP group did not receive (2 died, 1 unable to insert). Equal numbers referred for bypass surgery. 2x CVA in IABP group
- IABP-SHOCK trial 2010 Crit Care Med
    - SS RCT; Primary PCI for cardiogenic shock secondary to acute MI randomised to standard care (inotropes / vasopressors /ventilation) +/- IABP. N=45 (40 analysed)
    - Primary outcome; change in APACHE2 by day 4; no difference (but no difference in cardiac index, CI, between groups either….)
- IABP SHOCK 2 trial 2012 NEJM
    - MC RCT; acute MI complicated by cardiogenic shock (BPs<90 for >30mins or needing catecholamines to maintatin BPs>90mmHg; with pulmonary congestion and impaired end organ perfusion) with planned revascularisation (PCI/CABG); excl. those who needed CPR. Randomised to +/- IABP either before or after intervention at discretion of clinician. N=600
    - Primary outcome; 30d all-cause mortality no difference (39.7% in IABP group vs 41.3% in control, 95% CI 0.79-1.17, P=0.69). Trend to higher VAD insertion in control group. Safe

IABP vs other mechanical support

- Thiele 2005 Eu Heart J
    - SS RCT; Cardiogenic shock (BPs <90mmHg unsupported, end organ failures and elevated LV filling pressures with CI<2.1L/min) complicating acute MI and intention to revascularise (PCI / CABG) randomised to percutaneous VAD (Tandem heart; inflow cannula via transseptal puncture into LA and centrifugal pump returns via femoral arterial cannula) vs IABP. N=41
    - Primary outcome; haemodynamic improvement – significant increase in cardiac power index from baseline with VAD as compared to IABP, however significant increase in complications (severe bleeding and limb ischaemia). Similar 30d mortality 43% vs 45%
- Burkhoff 2006 Am Heart J
    - MC RCT; Persistent cardiogenic shock <24h (included patients failing in situ IABP), randomised to Tandem heart pVAD vs IABP. Excl R heart failure. N=42
    - Primary outcome; ?frequency at which haemodynamic profile reversed (did not die during or <24h device removal, CI>2.2L/kg/min, PCWP =/<24mmHg, MAP=/>70mmHg) 14% IABP vs 37% pVAD (↑CI by 0.6L/m2, ↓PCWP 10mmHg, ↑MAP 8mmHg). 30d mortality no difference
- Seyfarth 2008 JACC
    - MC(2) RCT; Acute MI <48h and cardiogenic shock (as per SHOCK trial criteria) randomised to post PCI insertion of IABP or impella LP2.5 (via femoral artery transaortic microaxial pump apspirates blood from LV and expels into ascending aorta; max flow 2.5L/min). N=25
    - Primary outcome; haemodynamic improvement at 30m after device implantation defined as change in CI from baseline. Increased ∆CI with impella 0.49L/min/m2 vs IABP 0.11L/min/m2; p=0.02
    - Significantly more haemolysis with impella. No difference in 30d mortality. LV ejection fraction at discharge 35% (impella) vs 45% (IABP) p=0.34

Summary (my practice)

- The IABP has a unique set of qualities no other pharmacological or mechanical assist device offers – notably reducing myocardial work whilst also increasing coronary perfusion pressure
- It is not a device that can be used as a one-size fits all for patients with cardiogenic shock which is the main problem with the trials to date – nuances of cardiogenic shock and patient profile need to be considered alongside the pros and cons of alternative approaches (pharmacological or other mechanical devices) in individual patients
- The IABP continues therefore to have a role in critically ill patients with cardiogenic shock
- Use in peri-procedure support e.g. complex PCI is not discussed (e.g. PROTECT 2; IABP vs impella)