HEAMODYNAMICS OF PACING

NASPE Training
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Lauren Butler – Lancashire & South Cumbria Cardiac Network
Objectives

- Physiology
- AV synchrony & pacemaker syndrome
- AV Intervals
- Pacing for cardiomyopathies, 1st degree block, neuro-cardiogenic syndromes
- Rate Modulation
  - Basic concepts
  - Haemodynamics
- Biventricular pacing
Physiology
Normal physiology

- Increase cardiovascular demand and metabolic demand due to exercise or emotion
- Increase HR and SV
  - Usually an initial increase occurs in which?
  - athletes \( \uparrow \) which predominantly?

**Definition SV**

- The amount of blood ejected with each ventricular contraction
- End diastolic volume - End systolic Volume
- What Does End diastolic volume depend upon?
- What Does End Systolic volume depend upon?
Relationship EDV & SV

- Hyperdynamic Systolic function
- Normal Systolic function
- Impaired Systolic Function

End Diastolic Volume

SV
Abnormal Physiology

- Disease affecting HR, SV or both
- Chronotropic Incompetence
  - Isolated sinus node dysfunction
  - Autonomic dysfunction
  - Drugs that blunt chronotropic response to physiologic stress
- Those with impaired function have poor SV effects/responses and rely largely on increase HR and thus may be more symptomatic with chronotropic incompetence
- Patients with LV dysfunction (poor contractility) depend more on pre-load & after-load to optimize SV
How complicated is it?

- **LV dysfunction**
  - Metabolic abnormalities, chronic acidosis, hypoxia, hypercarbia (affect contractility)
  - Autonomic dysfunction (affects HR & SV)
  - Concomitant renal failure, diabetes, hypertension (affect pre-load, after-load, autonomic function, contractility)
  - Drugs for treatment dysfunction, AF, CAD (suppress HR)
  - Lets not forget associated conduction abnormalities!
AV synchrony & Pacemaker syndrome
AV synchrony proven to contribute towards cardiac performance

- Increases Stroke volume by as much as 50%
- Decrease LA pressure 25 – 30%
- Normal LV function – greatest absolute degree of improvement
- LV dysfunction – relative improvement
AV synchrony (BP & CO)

- In some patients, loss AV synchrony leads to a drop in BP & occasionally causes syncope.
- Assess supine & standing (symptoms exacerbated standing due to reduction venous return and \( \therefore \) reduction end diastolic volume)
- Loss atrial kick (pacing population 15 – 25% cardiac output)
- VA conduction and all associated with it!
Commonly Ventricular pacing, may be seen in any mode where AV dissociation occurs

Atrial contraction against closed AV valve

- VA conduction & contraction
- Without VA conduction poor hemodynamics occur when atrial contraction is mistimed to occur simultaneously with ventricular systole

Incidence VA conduction

- Occurs 90% patients with SSS
- Occurs 15 – 35% patients AV block
Activation mechanical stretch receptors within atrial walls & Pulmonary Veins, leads to afferent central activation, leads to peripheral vasodilation

Release neuro-hormone, atrial natriuretic peptide (Potent vasodilator, inhibit sodium re-absorption & indirectly promotes salt loss which indirectly reduces BP)
**AV synchrony (atrial pressures)**

- Elevation atrial pressures – due to contraction atria against closed valve
- Large ‘A’ or cannon waves
- Present in RA, LA, Pulmonary veins & PCW
- Phenomena emphasized in patients with already elevated atrial pressures
- Remember patients – poor LV often have high End Diastolic Volumes, LA, PCW and PA pressures
- Some patients - symptoms with no VA cond
  - due to the intermittent nature - do not establish a tolerance
Small number patients may be symptomatic due to production or worsening of MR & TR

Oklahoma study – 30% slight worsening degree MR

PRIMARY MECHANISM - due to atria contracting against closed valve
Other factors – AV vs V pacing

- Ejection fraction – SV & EDV lower with loss AV synchrony but because both numerator & denominator are affected EF does not alter.
- Mortality rates - many studies show little effect on mortality although all do show lower mortality rates in DDD/AAI modes vs VVI.
- Many studies show V pacing can increase incidence of AF & development heart failure (Rosenqvist, Hesselson & CTOPP) and mortality may be related more closely to development of atrial arrhythmias & associated complications.
Pacemaker syndrome

- Any combination of variety of symptoms & signs (most often as a result of VVI pacing) due to AV dyssynchrony (even AAI with long PR intervals)
- BP related – syncope, malaise, fatigue, weakness, lightheaded, dizziness
- Atrial pressures – dyspnea, orthopnea, sensation fullness, pulsations in neck/chest, palpitations, chest pain, nausea, Clinical signs – relative or absolute hypotension, neck vein distension, cannon waves, pulmonary rales, peripheral oedema
Evaluation & treatment

- Prevalence – 7 –10 % (Heldman – crossover VVI to DDD & reported some level of syndrome in 83%, implies awareness due to comparison)
- Evaluate at implant – drop 20mmHg during V pacing likely to lead to p/m syndrome & check for retrograde conduction
- Upgrade DDD, lower base rate, use hysteresis, withdrawl medications that impair SA nodal function
- For Dual chamber patients – ensure atrial capture, avoid atrial pacing (VDD) if possible
AV Intervals
AV interval too long – mitral valve closure occurs early & impairs diastolic filling

AV interval too short – atrial contribution may be impaired
Paced & sensed AV delays

- Mechanical AV delay varies between atrial paced and atrial sensed AV delays
- Due to position on the P wave that sensing occurs
- Also due to intrinsic delay of atrial activation occurring after atrial pacing stimulus (from atrial pacing to atrial depolarisation)
- Difference intra-atrial conduction times
  - between atrial sensing & atrial pacing
  - Between individual patients due to underlying conduction/myocardial disease
- Mean intra-atrial conduction delay – 27ms (Ausubel)
Paced & Sensed AV delay

PAV = 200ms

SAV = 200ms

What would we reduce to ensure equivalent AV delays?
Rate responsive AV delays

- Reduction AV interval with an increase in rate
- Linear relationship exists – 4 msec decrease AV delay with every 10 bpm increase in heart rate
- Mimic physiological response to SR (have better hemodynamic indices) and also allow higher tracking rates
Heamodynamic effects

Cardiomyopathy
1\textsuperscript{st} degree block
Neuro-cardiogenic syncope
Cardiomyopathy

- Varying degrees – septal hypertrophy, outflow tract obstruction, MR due to displacement mitral valve apparatus
- Treatments include drugs, surgical septal myectomy, septal ablation, pacing
- Primarily used to reduce outflow tract obstruction and reduces intraventricular gradients
Altered septal activation due to RV apical pacing, reducing outflow obstruction

Short AV delay to ensure full ventricular activation from pacing stimulus (? AV nodal ablation to ensure ventricular pacing activation)

Studies show – reduction symptoms, improvement NYHA class, reduction outflow gradient, possibility LV remodelling as benefits continued when pacing was terminated

PIC (pacing in cardiomyopathy), M-PATHY (multicentre study of pacing therapy for Hypertrophic myopathy)
1\textsuperscript{st} degree AV block

- Hemodynamic compromise due to 1\textsuperscript{st} degree block documented (Barold, Kuniyoshi)
- Due to loss of optimal AV relationships only and cannot be directly compared to pacemaker syndrome
- Consideration to pace Mobitz I patients in whom hemodynamic compromise is established rather than bradycardia alone
Neuro-cardiogenic syncope & CSS

- **CSS** - exaggerated response to carotid sinus reflex (Brignole 9% recurrence syncope with pacing, 57% recurrence without)
- **Vaso vagal syncope** - head up tilt
- **Cardio-inhibitory** (Sinus bradycardia, Sinus arrest, AV block), vaso-depressor or mixed (most common)
- **Dual chamber pacing** beneficial – cardio-inhibitory response, not always preventing but often ameliorating symptoms
- **VVI pacing** – avoided, loss AV synchrony aggravates peripheral vasodilatation
Rate drop therapies

- **VPS -1 (North American Vasovagal pacemaker study)** - compared pacing with therapy (17% syncopal incidence) to 59% without any pacing therapy

- **Remember** – DDI with hysteresis, does not rate smooth down to allow intrinsic conduction to resume
Rate Modulation

Basic concepts

Heamodynamics
Basic Concepts

- Activity/accelerometer sensors dominate the market – respiratory & QT sensors also commonly used
- Ideal system should provide pacing rates proportional to level of metabolic need
- Speed of rate change should be similar to normal sinus nodal function
- Demonstrate sensitivity - respond to exercise and non-exercise increases (anxiety, emotion etc)
- Demonstrate specificity – failure of the sensor to be unaffected by stimuli not requiring/causing an increase in rate
Technical considerations

- Stability of sensor
- Size
- Biocompatibility
- Ease of programming
- Drain battery life
- Placement additional electrodes
- Sensor response (intrinsic properties and algorithms used)
Accelerometers & activity sensors

- Detect mechanical vibrations based on association between increasing body motion and exercise
- Detect low frequency vibrations – 4Hz (resonant frequency of human body)
- Piezoelectric crystal – strain gauge, bonded to can (activity) or circuit board (accelerometer) and generates electrical current as it flexes and deforms in response to mechanical vibration
- Relates to both frequency and amplitude of the vibrations
Activity sensors

- Activity sensors characterised by a rapid response to exercise
- Over respond to those activities walking, running
- Under respond, cycling, swimming
- Also give higher rate pacing descending than ascending stairs!
- Susceptible to environmental noise, transport, tapping device
Accelerometer

- Bonded to circuit board
- Allows detection body motion
- Isolated from case (preventing response from mechanical pressure against can)
- Respond to movement in antero-posterior axis
- Piezoelectric produce electrical signals
- Piezoresistive measure changes in resistance that occur with mechanical deformation of the crystal
- Accelerometers give better response to cyclists, less response whilst descending stairs and are more proportional to exercise workload
Respiration sensors

- Minute ventilation – emission small, sub-threshold charge of known current (1 mA)
- Emission from can – measure resulting Voltage at lead tip (and resistance calculated)
- Emission from ring electrode – resultant voltage measured at can (and resistance calculated)
- Transthoracic impedance increases with inspiration and decreases with expiration
- Measure frequency (& :: respiration rate) of fluctuations in impedance
- Emission every 15 msec – 50 msec
Combination Sensors

- Dual sensor – advantage of rapid response due to activity and sustained response associated with MV
- Bipolar leads needed – MV
- ELA medical – Chorus RM (atrial bipolar lead to measure impedance), ELA – Talent allows use of unipolar lead
- Guidant pulsar max & Medtronic Kappa 400 have combination sensors with emphasis placed on accelerometer – low level activity & MV – high level activity and also have cross checking facilities and automatic adjustment
**QT interval**

- Autonomic activity and heart rate affect the stimulus - T interval (affected by emotion)
- Shortens at increased pacing rates
- QT interval varies between individuals but is consistent for each individual at rest
- Affected by drugs and electrolytes
- Measured - stimulus to apex T wave in the IEGM
- Early devices – T wave undersensing (improved with T wave filters) and large polarisation impulses also interfered with measurement (lower polarisation with improved electrode design)
Other sensors

- Temperature – thermistor in RA portion lead, slow response to exercise
- Pre-ejection interval – shortens as exercise increases (pacing stimulus to onset ventricular contraction)
- Stroke volume – placement of impedance catheter, alters pacing rate to keep SV constant
- RV Pressure increase – transducer incorporated into distal portion lead
Other sensors

- Wall motion and small changes of contractility - associated with metabolic changes
- Mixed Venous Oxygen saturation - hemorefectance oximetry placed in RV portion lead
- Paced Depolarisation Integral - measures ventricular depolarisation gradient (vector)
- Peak Endocardial acceleration (PEA) - incorporates an accelerometer in tip lead, correlates to changes in sympathetic tone (early signal to rate drop episode)
Heamodynamics

- Chronotropic competence most important contributor to cardiac output – Improve HR and exercise capacity (every 40% increase pacing rate – 10% increase exercise capacity)
- Important at higher levels of activity, AV synchrony at lower levels
- Diagnosis chronotropic incompetence
  - 70–85% predicted maximum (difficult due to limited exercise capacity many patients)
  - Wilkoff chronotropic assessment exercise protocol (CAEP) (2 minute stages gradual increase speed and incline)
Summary

- AV – Synchrony important all patients, atrial transport, stroke volume, affects hemodynamics
- AV synchrony more important at rest than exercise
- Chronotropic incompetence reduces exercise capacity, Rate modulation important at peak exercise (sensor types)
- Remember choice of mode - underlying patho-physiology as well as ECG indication
- Different pacing sites (Septal) may have effect, yet to be confirmed by further studies