SASA 2013 – Port Elizabeth

Refresher Day 1 (Saturtday)

Session 1 – Cardiovascular

Aging and the cardiovascular system – Prof Patrick Wouters, University of Ghent, Belgium

- Maximum age achieved by a human = 122 years. Human idealised total life span is 120 years. Life expectancy is an empirical estimate and evolves rapidly depending on the living conditions and environment.
- Little consensus on what is considered 'elderly' – previously 65 years, now 85 suggested. Elderly portion of the population is increasing rapidly. This is going to massively impact on health economics.
- The older patient presents a significant increase in risk of perioperative morbidity/mortality.
- "Normal" aging is the progressive degeneration of structures and functional reserves. Mechanism is debated; worsening in cell repair systems and/or accumulation of free radicals.
- Vascular aging involves degradation of the elastic connective tissue with collagen, which has 100-1000x the stiffness of elastin, and dysfunction of the endothelium. Accumulation of Advanced Glycation End products (AGE's).
- Stiffening of the arterial conduit arteries – analogy of the 'windkessel' system to change interrupted pulsatile flow into a continuous (sinusoidal) flow. The higher the compliance, the lower the pulse pressure and more consistent the flow. Pulse pressure is a measure of compliance!
- Pulse pressure is also determined by wave refraction. The reflected pressure wave plays a role in diastolic coronary perfusion; arterial stiffening leads to rapid refraction, adding to increased systolic pressure and decreased diastolic perfusion. Double hit to the old heart…
- Increased pulse pressure causes
    - Increased systolic load to ejection
    - High cardiac oxygen consumption
    - LVH with decreased capillary refill
    - Reduced diastolic perfusion pressure
    - Inefficient organ perfusion
- Pulse pressure is an age-independent predictor of stroke (controversial)
- Chronic heart failure is a disease of the elderly. There are two phenotypes – reduced ejection fraction (reduced LV function) and preserved EF. Both types result in similar mortality
- Diastolic HF has preserved EF and accounts for approximately half of cases.
- Very easy to diagnose LV inflow patterns with echo. The early inflow is reduced; maximum filling speed decreases with age, and the atrial kick becomes essential.

- What is the basis of diastolic dysfunction? Replacement of elastic tissue with collagen tightening/stiffening of the sarcomeres.
- Decreasing compliance of the ventricles results in an increase in the gradient of the pressure/volume curve; the elderly heart is more empty at the same pressures as the young heart; conversely, adding the same volumes to an old heart as one would to a young heart will overload it easily.
- There are also the problems of impaired baroreceptor response, impaired parasympathetic control, decreased responsiveness to beta agonists, reduction in effectiveness of ischaemic preconditioning (Nguten et al 2008), etc
- Risk assessment! Comorbidity, pulse wave velocity, pulse pressure, telomere length, exercise. Can we reduce risk?
- Atrial fibrillation is also a degenerative disease, provoked by atrial enlargement (secondary to diastolic dysfunction)
- Summary:
    - Increased vascular stiffness
    - Increased cardiac stiffness
    - Narrow therapeutic goals – maintain diastolic pressure, restrictive fluid status but enough to do the job
- "Youth is a wonderful thing, but what a crime to waste it on children." GBS

Understanding Cardiomyopathies – Prof Justiaan Swanevelder, University of Cape Town

- Cardiomyopathies – 'heart muscle disease', "intrinsic disease of the myocardium associated with cardiac dysfunction". WHO classification:
    - Category I – specific and unclassified cardiomyopathies
    - Category II – established cardiomyopathies with intrinsic disorders of the myocardium
        - DCM
        - HCM
        - RCM
        - ARVD
    - Other classifications – AHA 2006 (Maron BJ Circulation 2006), European Society of Cardiology 2008 (Elliot et al Eur Heart J 2008).
- Specific causes:
    - Ischaemic/hypertensive/valvular
    - Inflammatory
    - Toxic
    - Metabolic
    - Neuromuscular (Duchenne's, Friedrich's ataxia)
- Unspecified – vertricular noncompaction, Tako-Tsubo
- LV non-compaction:
    - LV 'stops growing' in the fetal state (about 8 weeks developmental stage)
    - Deep recesses, spongiform 2-layer appearance (Rehfeldt KH et al 2008)

- DCM – exclusion criteria: abnormal loading conditions, congenital heart disease, excessive alcohol, etc.
- Doppler pattern changes dramatically – very short E (early) wave, prominent A (atrial kick) wave). Atrial fibrillation occurs frequently. 3 functions of the atrium – reservoir, conduit and atrial kick. In cardiomyopathy, the heart becomes more dependent on the atrium.
- RCM is the least common. Primary and secondary causes; amyloidosis is the most common infiltrative type. Atria often larger than ventricles. Interesting paper out of Maputo – see Mocumbi AO et al. Heart 2008. NB – difference between restrictive cardiomyopathy and restrictive pericarditis.
- HCM – good review – Elliot & McKenna, Hypertrophic Cardiomyopathy. Lancet 2004;363:1881-91. Cause of young adult exercise-related death. Familial, autosomal dominant. Can occur at different levels within the heart! Typical 'dagger' shape on pulse wave dopper.

Oxygen: Good or Bad? – Dr Bill Wilson, Adelaide, Australia

- Hypoxia not only stops the machine, it wrecks the machinery (Haldane)
- High saturation past a point only increases the dissolved fraction, not the saturation.
- Oxygen toxicity
    - All mammals are sensitive to exposure to hyperoxaemia
    - Pulmonary toxicity at 1 ATM at 100% - bronchoscopic evidence at 6 hours; cough, pain and lung injury at 24 hours
    - CNS – Hyperbaric air at 8 ATM causes convulsions
- Reactive oxygen species (ROS) – free radicals (unpaired electron) and intermediates (H_2O_2)
- Free radicals are easily converted to hydrogen peroxide in the mitochondria under normal conditions. We have good defence mechanisms (enzymatic and non-enzymatic). Poorly developed in neonates and begin to fail in the elderly
- Massive production of free radicals if there is high dissolved oxygen tension. Reperfusion and mitochondrial injuries follow.
- Physiological role of ROS –
    - Activate guanylate cyclase
    - Control of apoptosis
    - 'Oxygen sensor' in the carotid bodies and for erythropoietin
    - Role in ischaemic preconditioning
- Nunn editorial in BJA Dec 2007 refers
- Preoxygenation – increases O_2 in FRC and reduces the time for desaturation. However, oxygen in a drug and should be treated as such. Concern for ROS, absorbtion atelectasis, etc. Degree of alveolar collapse is significantly increased if 100% O_2 is used. Atelectasis with 100% O_2 – 15% atelectasis; 80% - 3%; 60% - 0.6%
- Grocott HP Anesthesiology Clinics – avoid excessive [O_2] in one lung ventilation
- Austin BMJ 2010 – mortality of 9% in high-flow vs. 4% in titrated O_2 (target SpO_2 88-92%) in patients with COPD

- AVOID trial (underway) – Prehopsital O2 may WORSEN outcome in ischaemic heart disease/ACS/MI
- McNulty et al – coronary blood flow measured by intraluminal Doppler showed significant DECREASE in CBF with 100% O2 as compared to 40%; also showed significant decreased vessel diameter on 100% O2 with angio
- Kilgammon JAMA 2010 – Improved survival with normoxia as opposed to hyperoxia in retrospective analysis of post-cardiac arrest patients.
- Hyperoxaemia increases surgical site infections (although this was a small effect that is minimal compared to other factors).
- NO benefit of supplemental O2 during Caesarean section for elective/routine procedures; no evidence of harm in emergencies (but no real evidence of benefit).
- Neonates resuscitated on oxygen potentially do worse than those rescuscitated on air
- Retinopathy of prematurity, bronchopulmonary dysplasia, functional MRI changes all occur in neonates who become hyperoxic.
- Summary: Oxygen is a drug, hypoxia is BAD; hyperoxaemia may be bad too.

Making PCA Safe – Jeff Coetzee

- Relieving pain decreases the surgical stress response, which in turn decreases the incidence of complications
- Postoperative pain management is still largely dependent on opioids, which have a very variable dose requirement based on individual factors. The MEAC (minimum effective analgesic concentration) is very consistent in INDIVIDUAL patients, but very variable between individuals.
- Avoid peaks and troughs… hence the principle of PCA
- BUT…there are many, many examples of respiratory depression DESPITE the use of PCA
- Causes: lack of training/knowledge, improper prescribing, inadequate monitoring.
- Respiratory depression caused by opioids has three components: decreased respiratory drive, sedation and airway obstruction. Together these cause OIVI: "Opioid Induced Ventilatory Insufficiency"
- Opioids affect the genioglossus muscle, which plays a crucial role in maintaining a patent airway
- Pain 'protects' against the respiratory effects of opioids; opiated patients who then have a nerve block to address their pain rapidly develop respiratory depression.
- The water-soluble nature of morphine creates a powerful concentration gradient; blood concentrations and effect site concentrations can thus differ dramatically.
- Basic components of PCA:
    - Load the patient properly!
    - Achieve the MEAC before commencing the PCA
    - Choose an appropriate PCA demand dose – optimal is generally considered 1mg
    - Out of practice due to no good evidence and/or evidence of harm – background infusions and 1 or 4 hour limits.
- Managing PCA:

    - Morphine loading dose 2-4mg every 5-10min until pain <= 4/10
    - Start PCA with demand dose 1mg, lockout 6-8min
    - If analgesia is successful, continue
    - If the analgesia is not successful, evaluate. If the patient is sedated, use an alternative strategy (add NSAID or regional etc). If the patient is not sedated, improve your loading dose with titrated boluses
    - If this is still ineffective, an infusion with continuous monitoring is advised.
- Standardise dilution (1mg/ml)
- Premixed syringes (from pharmacy) advisable
- Dedicated IV line preferable; non-return valves essential; don't allow 'add-a-line' to be connected to the PCA IV!
- Monitoring PCA –
    - Monitoring respiration rate alone is not sufficient; low rates signal a problem, but insufficient volumes at an adequate rate is a far more common occurrence. Nurses should be trained to assess volume and adequacy of breathing as well as rate
    - Sedation level/score should also be assessed and monitored
    - Sedation – the sixth vital sign?
    - Future monitoring – capnography; breath sounds by automated system?
- Nurses are the cornerstone for safe PCA. Perhaps we should have training and certification in PCA. Must be able to recognise OIVI; inadequate ventilation, etc.
- The time and place to educate the patient about the PCA is not in the recovery room! Warn patients that the device cannot take the pain away completely.
- "We must live with the fact that morphine analgesia is accompanied by respiratory depression; our patients must live in spite of it."

EBM: Do we need gelatins and starches? – Andy Parrish

- Revised Starling equation and the glucocalyx model of transvascular fluid exchange – see Woodcock & Woodcock
- Is there a sub-glycocalyx space?
- Efficacy: choose an appropriate effect measure! Is it surrogate vs. patient-relevant (live longer, get well, get well quicker, less resource-intensive)
- The real hard outcome is only one thing… mortality
- Beware the hazards of interpreting Forest plots
- Gelatins – no demonstrated mortality advantage in any of the trials
- Cochrane groups (prior to the 4 big new trials) showed no advantage in mortality
- Big trials: VISEP, CHRYSTMAS, 6S, CHEST, SAFE, BASeS
- 6S – caveat infusor – subgroup analysis!
- Safety data:
    - VISEP was stopped early for safety
    - CHEST mortality rate was lower than expected
    - Short follow-up misses evidence of harm
- Cognitive psychology and practice patterns…

    - 'Group think' and the 'we all do it' phenomenon
    - Substitution and solving of an evidence problem with a simpler plausibility/belief problem
    - Availability bias: many trials; few good trials
    - Framing: a search for the "right" colloid when we should be searching for whether to use them at all
- Conclusions:
    - Biological models in flux
    - No convincing evidence of mortality benefit
    - Convincing evidence of mortality disadvantage in severe sepsis
    - Concern for harm in terms of increased renal replacement therapy
    - Change in practice is appropriate

Make it Zero – Edwin Turton

- Introduction to the LifeBox product – www.lifebox.org
- Pulse oximeter is the only monitoring device that has been featured on the WHO Surgical Safety Checklist

The Child with a Murmur – Kotie Bester, Red Cross Children's Hospital

- 50-80% of children have a murmur at some time
- Only a fraction of these are pathological
- Murmurs are caused by turbulent flow. Normal flow is laminar. Turbulence occurs if there is abnormal density, diameter, velocity or viscosity (recall Raynaud's Number)
- Children have relatively brisk flow, small vessels, acutely angled branches, and little intervening tissue, so murmurs are more commonly heard.
- Hx – look for family history of congenital heart disease (3-5x increase in risk if there is a family member with CHD), sudden cardiac death or sudden infant death.
- Pre- and perinatal Hx – think of drugs (lithium, SSRIs), alcohol, toxins, intra-uterine infections (NB rubella), maternal diabetes and prematurity
- Associations – chromosomal disorders, syndromes, rheumatic fever, etc
- Symptoms – poor feeding, fatigue, diaphoresis, poor exercise tolerance, failure to thrive, poor growth, history of swelling (eyes in the morning), cyanosis, dizziness, palpitations, chest pain, syncope or near-syncope. Chest pain and syncope are very uncommon, but when they occur they are associated with AS and HCM. Respiratory symptoms – recurrent chest infections.
- Most common symptoms in the emergencu room – dyspnoea, fatigue, nausea & vomiting, dizziness
- Examination tips:
    - You cannot auscultate a crying child

    - Aim for the target – aim for the heart first. Ignore the murmur initially – listen for everything else that will help you make the call (clicks, extra sounds, diastolic sounds, etc).
    - Examine the abdomen to learn about the heart – situs inversus can be indicated by a liver on the wrong side; hepatomegaly is a common feature of failure.
    - Is the child dysmorphic? 25% with heart problems have other dysmorphic features.
    - Check pulses in all limbs; try to check gradient between arms and legs (>20mmHg is abnormal) and check the capillary refill
    - ECG and CXR are not particularly useful….unless the child has LVH, which is associated with AS and HCM.
- Physiological murmur:
    - Intensity changes with changes in flow (valsalva, exercise)
    - Most systolic murmurs are benighn
    - Most pansystolic and diastolic murmurs are pathological
    - 7 S's: Sensitive (changes in position, respiration), systolic (ejection), short, single, small (not radiating), soft (grade 1 or 2), sweet (not harsh).
- Red flags: HCM:
    - Increased intensity on standing or valsalva
    - Decrease or no change on passive leg raise
    - Decrease or no change when going from standing to squatting
- More red flags:
    - Abnormal S2
    - Systolic clicks
    - Increased precordial activity (may be ASD/VSD/PDA)
- Seven innocent murmurs:
    - Still's Murmur – most common, probably LV outflow. LLSB, early systolic, decreases on standing, typically at 2-6 years. Twanging/tuning fork character.
    - Aortic systolic murmur – over aortic area (URSB), ejection systolic.
    - Pulmonary flow murmur – LSB 2-3rd interspace, peaks in mid-systole, dissonant quantity, loudest supine (decreases when upright). Exclude ASD, PS.
    - Peripheral pulmonary stenosis – axilla or back, grade 1-2, low pitch, early to mid-systolic, infants. Exclude PA stenosis
    - Mammary artery soufflé – Caused by arteries and veins running to and from the breasts, high pitch, along chest wall, stretches from systole into diastole.
    - Venous hum – over low anterior neck, lateral to SCM, resolves or changes when supine, continuous whirring quality, accentuated in diastole.
    - Supraclavicular systolic murmur – early brief systolic murmer
- VSD: Holosystolic, 20-25%
- ASD: 8-14%, ejection systolic, ULSB, wide fixed split S2, may have very soft diastolic rumble
- PDA: 6-11%, continuous murmur, ULSB, can radiate to back
- PS: 7.5-9%, ESM, ULSB, radiates to infraclavicular regions, axilla or back
- Coarct: 5-8%, heard at back
- AS: 5-6%, ESM, URSB
- Approach:

- Consider risk, complications to avoid, haemodynamic aims, endocarditis prophylaxis, paradoxical embolism
- Probably innocent (nothing on history, asymptomatic, good effort tolerance) -> Proceed
- Ominous:
    - Less than 1 year (esp < 6 weeks). Under 1 year may be asymptomatic even if the defect is significant.
    - High anaesthetic risk
    - Extracardiac abnormalities
    - Cyanosis, PHT, arrhythmias, PS
- Uncertain about lesion, but asymptomatic, uncomplicated lesion in the differential, and minimal risk procedure -> Probably safe to proceed, but cardiology referral postoperatively.

Rebecca Gray – Muscle disorders

- Weakness can be as a result of problems at any level from the brain to the muscle tissue itself, but this talk will focus on the muscle diseases specifically
- Myotonias:
    - Channelopathies
    - Triggered by pain, cold, stress and diathermy
    - Can be dystrophic or non-dystrophic
    - Steinert's Muscular Dystrophy – respiratory insufficiency, cardiac involvement (DCM) and mental retardation.
    - Assess preoperatively – establish disease severity, avoid triggers, check electrolytes.
    - Muscle relaxation is a challenge. Sux is out! Neostigmine might trigger contraction. Volatiles useful but can cause a lot of respiratory depression. Good analgesia is the key.
    - NOT associated with MH
- Muscular dystrophies
    - X-linked, but up to 1/3 are spontaneous
    - Most common is Duchenne's. 1/3 mentally retarded.
    - Death is usually due to cardiomyopathy.
    - Female carriers are prone to scoliosis and cardiomyopathy
    - Restrictive lung disease picture with poor cough and recurrent infections. Think about pre-op optimisation and post-op support. BiPAP is a useful modality.
    - Cardiac problems are easy to underestimate (patients usually sedentary). Almost impossible to do stress testing, and scoliosis makes echo difficult.