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SEVERE VALVULAR HEART DISEASE IN PREGNANCY

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CONTENTS

"Oh my heart…I'm having a baby"

SEVERE VALVULAR HEART DISEASE IN PREGNANCY

INTRODUCTION

Valvular heart disease in pregnancy is a vast topic with many case reports and limited reviews available. In my institution, I have witnessed various anaesthetic techniques being demonstrated for the same category of patient. Varying degrees of success and complications were noted across the range. It piqued my interest to establish what the global perspective on the topic was and if any evidence-based practice guidelines were in place that individuals/departments were utilising. For this presentation, severe valvular heart disease is defined as a mitral valve area (MVA) of less than $1cm^2$ and an aortic valve area of less than $1cm^2$. This is based on the American Society of Echocardiography recommendations which are documented in the 2014 ACC/AHA Management of patients with Valvular heart disease Guidelines.

My aims are to:

- Provide a historical perspective
- Contextualise the burden of disease in South Africa
- Review the implications of cardiovascular changes in pregnancy
- Provide an approach to the perioperative management of these patients including risk stratification
- Review the current anaesthetic options for caesarean delivery

HISTORICAL PERSPECTIVE

Previously, when parturients with valvular heart disease required anaesthesia, it was general anaesthesia that was the chosen technique. The anaesthetist sought to avoid hypotension due to the sympathectomy from regional anaesthesia which resulted in a decrease in preload, systemic vascular resistance and coronary perfusion. Consequently, there were very few publications on the topic. One primarily bad outcome was published as a case report(1), randomised control trials were not performed in part due to the small number of cases being managed and also because of potential ethical issues that may have been raised.

The 1980s saw a small number of case reports detailing the challenges faced with non-pregnant patients with idiopathic hypertrophic subvalvular aortic stenosis requiring surgery for a femur fracture(2). Spinal anaesthesia with 8mg tetracaine and 100mcg fentanyl resulted in ST depression, tachycardia and hypotension leading to the operation being cancelled. One week later, under general anaesthesia, the operation was successfully conducted. In a second case with a 56 year old patient with idiopathic hypertrophic aortic stenosis requiring a herniorrhaphy, epidural anaesthesia with 15mls 2% lidocaine resulted in a sensory block to T10,

hypotension(70/40mmhg) and bradycardia(20bpm) with an adequate response to atropine 1.2mg iv.

In 1987, a case report documenting a 36 year old primigravida patient with predominant aortic incompetence and pre-ecclampsia (uncontrolled blood pressures) had an epidural placed for an elective C/S. After a test dose of 2mls 0.5% bupivacaine, a bolus of 18mls 0.5% bupivacaine was administered. The patient became hypotensive which initially corrected with Ringers Lactate. Despite a T4 sensory level, there was no sacral anaesthesia for which a further bolus of 6mls 0.5% bupivacaine resulted in severe hypotension unresponsive to fluid or vasopressor therapy. The mother was intubated, ventilated and underwent cardiac arrest protocol whilst delivery of the baby took place. The patient demised and following this the authors concluded that epidural anaesthesia should be contraindicated for a caesarean section delivery should in patients with severe VHD.

Following this, came the acquisition of new knowledge and further non-obstetric cases with valvular heart disease being performed successfully under meticulous anaesthetic technique of a gradual regional block together with appropriate monitoring.

THE BURDEN OF DISEASE

A recent publication shows that there is a range of aetiology for valvular disease that differs in lower-to-middle income countries(LMICs) versus higher income countries(HICs).(3) Rheumatic heart disease contributes more than 30% of the burden in pregnancy in LMICs. There is an annual incidence of 250 000 deaths in these countries. This is compared to congenital heart disease disease in the HICs.(3). According to the Saving Mothers Report (2011-2013)(4), there were 169 notified deaths due to cardiac disease (34% of all deaths due to 'underlying medical and surgical disease'). It has been shown that this has surpassed the routinely reported short term post-partum period of 42 days.(3) The IMMR is the number of maternal deaths among 100 000 deliveries in health facilities/institutions. There has since been a steady increase in the Institutional Maternal Mortality Ratio (IMMR) for cardiac disease in South Africa. The IMMR for 2005-2007 was 3.73 and this has increased to 5.64 for 2008-2010. It is now at 5.78 per 100 000 in this triennium.

Cardiac disease is the second most common cause of indirect maternal death after non-pregnancy related infection. The causes of these cardiac deaths were complications of rheumatic heart disease, peripartum cardiomyopathy and dilated cardiomyopathy.

However pregnant women are likely to be seen and missed/missed diagnosed at antenatal care visits. They are being seen by midwives at their local clinics only to present later in pregnancy when the progression of disease has reached a decompensated stage. This is the stage that anaesthetists at both district and regional level facilities are encountering such patients for the first time and perhaps in an

emergency situation. It is therefore vital that we as a multidisciplinary team of obstetricians, physicians and anaesthetists are able to diagnose early, manage early and prevent complications.

CARDIOVASCULAR CHANGES IN THE NORMAL VERSUS CARDIAC PARTURIENT

Table 1: Cardiovascular changes in pregnancy

Normal Pregnancy	
CO	Sharpest rise at beginning of first trimester. By 24 weeks, up to 45% increase due to bigger stroke volume (SV) than in 1st trimester and increased heart rate later.
BP	DBP and MAP drop more than SBP more in early pregnancy (6-8weeks). Arterial pressures increase by 3rd trimester. Return to preconception values postpartum.
HR	Increase progressively during pregnancy (20-25% above baseline) reaching maximum in 3rd trimester.
Blood volume	Double pre-pregnancy values at 32 weeks. Proportionately higher than red cell mass (due to erythropoiesis) resulting in "physiological anaemia".
Cardiac function	Diastolic function unchanged. Systolic function increases in second trimester. Temporary eccentric LVH. Mild 4 chamber dilatation(right more than left) with transient trivial MR and physiological TR and PR.
Aortocaval Sx	Supine: compression from 13-16 weeks. Near complete IVC obstruction at term. 15% women experience Supine Hypotension Syndrome: bradycardia and substantial drop in BP when supine.
Postpartum	
CO	75% above pre-delivery levels from vena-caval relief reduced lower extremity venous pressure. Pulmonary oedema in at risk pts. Falls below pre-labour values at 24hrs and pre-pregnancy levels at 12-24weeks.
HR	Falls rapidly after delivery and reaches pre-pregnancy levels by 2 weeks postpartum

“Nature’s stress test”, as pregnancy is sometimes called, occurs because previously asymptomatic cardiac patients have their disease process unmasked when they are unable to adapt to normal physiological changes of pregnancy. The main changes in pregnancy are an increased cardiac output, blood volume and red cell mass and a decrease in peripheral and systemic vascular resistance.

Mitral stenosis is the most common valvular defect in pregnancy(5). Labour, delivery and the immediate pueperium appear to be the period associated with the greatest risk for adverse cardiac events including heart failure and arrhythmias. Important

changes associated with the left-sided stenotic lesion include obstruction to flow from the left atrium to the left ventricle, an increase in left atrial pressure as well as pulmonary capillary venous pressure(1). This increase in pulmonary blood volume leads to an increase in pulmonary capillary pressure that exceeds colloid osmotic pressure. The chances of pulmonary oedema are therefore increased(6). Clark *et al.* theorised that an associated increase in pulmonary capillary wedge pressure postpartum was due to the loss of the low resistance placental circulation. This, together with the "autotransfusion" from the now empty uterus, pelvis and lower extremities, could lead to decompensation.(7)

Studies regarding cardiac (mal)adaptation have mainly been conducted in the healthy parturient or those with hypertension in pregnancy but there is little longitudinal data in those parturients with valvular heart disease(8) There is also a paucity of evidence on the gradation of reversibility of these haemodynamic changes and post-partum cardiac function.(8)

Cornette *et al* in their prospective observational trial of thirty five women with structural heart disease showed an attenuated cardiovascular adaptation with a reduced systolic function and diastolic dysfunction that persisted after 6 months postpartum. Despite having an already increased pre-pregnancy left ventricular (LV) mass there was a statistically significant increase during pregnancy. This was compared to normal healthy pregnancies. The gradual decline in ejection fraction (EF) and fractional shortening (FS) continued until 6 months postpartum.

This was in contrast to a study by Uebing(9) who did not observe a deterioration in LV function. The authors acknowledged the intrinsic limitations that echocardiography volume estimations may have and suggested that MRI analysis of systolic function is relevant in confirming findings.

Diastolic dysfunction was evidenced by a further increase in the E/E′ ratio from baseline with progress in gestation. In the normal parturient, an increase in load leads to compensatory hypertrophy of the myocardium. But due to an elevated baseline, there is a reduced capacity for expansion in LV mass thereby leading to increased filling pressures as seen with the E/E′ ratio.

In patients with underlying cardiac disease, factors such as underlying cardiac pathology, gestational age, intravascular fluid status, positioning of the patient, anaesthetic agents, route, dose and choice of uterotonic agents can all affect haemodynamic status during and immediately after delivery.

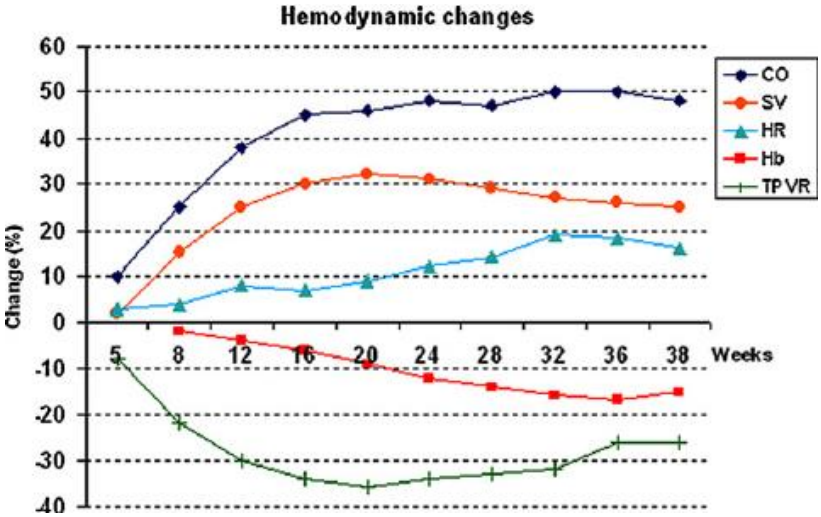

Fig.1 Hemodynamic changes in pregnancy. CO - cardiac output; SV - stroke volume; HR - heart rate; Hb - Haemoglobin; TPVR - Total Peripheral Vascular Resistance. Pregnancy and delivery in cardiac disease (29)

PRECONCEPTUAL COUNSELLING

Apart from the obstetrician and anaesthetist, the multidisciplinary team should include a cardiologist with proficiency in heart disease and the parturient. The process of consultation should occur prior to conception, early, and regularly in pregnancy.

Pre-conceptual counselling is beneficial for all women with cardiovascular disease. According to the recent Saving Mothers Report 2011-2013,there should be preconceptual risk assessment clinics established at tertiary centres whereby women with underlying cardiac disease can be reviewed.

The preconception evaluation should ideally include advice on risk prediction as well as contraception. Some may require optimisation of their cardiac disease and function before falling pregnant. They may have potentially teratogenic cardiac medication changed to safer alternatives.(10) Table 1 details issues that should be addressed at the time of preconceptual counselling.

This will include(11):

1. Anatomy and physiology of the underlying cardiac lesion(s).
2. Maternal functional status.
3. Maternal risk score.
4. Lesion-specific risk.
5. Fetal and neonatal risk

Pregnancy risk stratification • Maternal cardiac risk • Maternal obstetric risk • Fetal and neonatal risks
Long-term effects of pregnancy on the heart
Maternal life expectancy
Genetic consultation
Contraception safety and efficacy
Modification of cardiac medications
Optimization of cardiac status
Planning for pregnancy*

Figure 2: Pre-conceptual counselling considerations(5)

PRE-OPERATIVE ASSESSMENT AND PERIOPERATIVE PLANNING

Many healthy pregnant women may show features suggestive of cardiac failure during their pregnancy and/or at delivery. This may include dyspnoea, reduced exercise capacity and fatigue. Signs such as distended central veins, audible flow murmurs or a third heart sound may indicate volume overload.

History

It can be normal to experience some degree of dyspnoea in pregnancy but profound dyspnoea, deterioration in functional class, paroxysmal nocturnal dyspnoea(PND) and orthopnoea may herald cardiovascular pathology. The NYHA Class is an important predictor of maternal cardiovascular morbidity and fetal mortality(11). It is therefore important to obtain a baseline functional status. A history of previous cardiac events including heart failure, cerebro-vascular events or arrhythmias should be obtained as these escalate the risk of maternal cardiovascular events.

Examination

A Grade 2 ejection systolic murmur or mildly elevated jugular venous pressure may be normal. The presence of cyanosis, clubbing, prominent murmurs and decreased oxygen saturation should alert the physician. Signs suggestive of pathological heart disease include: (12) loud fourth heart sound, any diastolic murmur, fixed splitting of the second heart sound and an opening snap. Other abnormal signs include cyanosis, clubbing. The presence of one or more of these signs would prompt the need for an echocardiogram.

Electrocardiogram (ECG)

Marked sinus tachycardia, ventricular tachycardia or atrial fibrillation may suggest severe underlying disease.

Echocardiography

If there is no recent pre-conception echocardiography, one should obtain a first trimester Echocardiogram to evaluate risk of the pregnancy, the valve lesion and its severity. The need for repeat examinations should be prompted by decreasing functional class, moderate to severe pulmonary hypertension or right ventricular dysfunction. Normal echo changes in pregnancy include 10% increase in cardiac chambers and normal mitral and tricuspid annuli dilatation.(7)

Cardiopulmonary Stress Testing

This is said to be useful pre-conceptually as a more objective evaluation of functional capacity compared to history alone. Studies have been conducted in healthy parturients but there has been no safety data presented for those patients with cardiovascular disease.

A baseline serum B-type natriuretic peptide(BNP) can be incorporated into pregnancy assessment in those women at risk of developing heart failure (HF) during pregnancy.(11) This may be followed by serial BNP levels.

Maternal risk score

Risk was initially assessed by predictors such as cyanosis and functional class But with time and large pregnancy cohorts, pregnancy risk indices were developed(13) as seen in Table 2.

a- CARPREG (Cardiac Disease in pregnancy) was the first risk index and it examined both acquired and congenital heart disease. 4 predictors of adverse maternal cardiac events were identified: prior cardiac events, poor functional status (NYHA Functional class 3 or 4) or cyanosis, left heart obstruction or systemic ventricular ejection fraction < 40%.

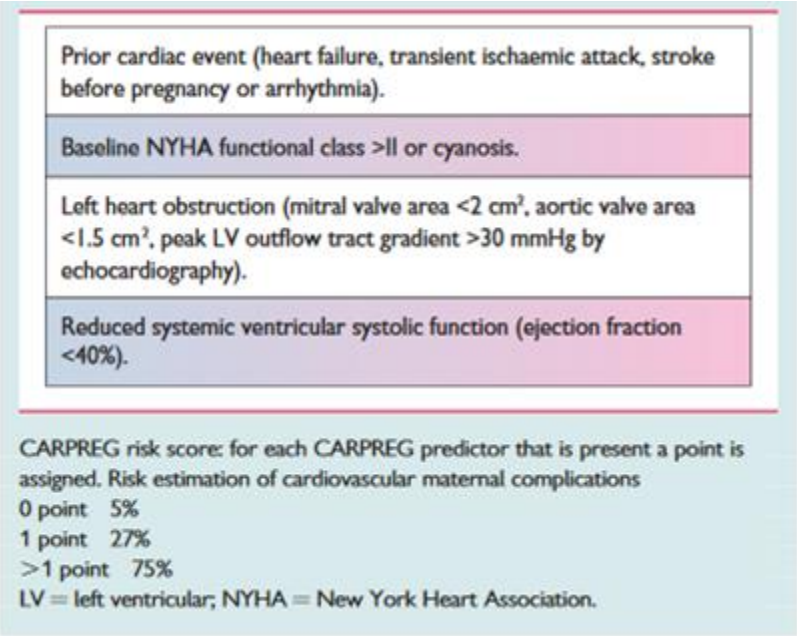

Figure 3: Predictors of maternal cardiovascular events and risk score from the CARPREG study. Adapted from (14)

b- The ZAHARA (Zwangerschap bij vrouwen met een AangeborenHARtAfwijking-II [translated as “Pregnancy in women with CHD II risk index”]).It is a weighted risk score.

ZAHARA I	Points
• Prior arrhythmias	1.5
• NYHA functional class > II	0.75
• Left heart obstruction	2.5
• Cardiac medication before pregnancy	1.5
• Systemic AV valve regurgitation	0.75
• Pulmonary AV valve regurgitation	0.75
• Mechanical valve prosthesis	4.5
• Cyanotic heart disease	1.0

Drenthen 2010

Total points	Risk %
0	2.9
0.5 - 1.5	7.5
1.51 - 2.50	17.5
2.51 - 3.50	43.1
> 3.51	70.0

Figure 4: Predictors of maternal cardiovascular events identified in congenital heart diseases in the ZAHARA study

c- A lesion-specific risk classification was developed by a British working group using a modified World Health Organisation (WHO) Classification. Lesions are characterised as low, medium, high risk and lesions in which pregnancy is contraindicated (WHO I - IV).
An article by Balci et al stated that the modified WHO classification was the best tool for assessing cardiovascular risk in pregnant women with congenital heart disease. This was in comparison to the CARPREG , ZAHARA I, and WHO classifications.
The Saving Mothers Report 2011-2013 make a recommendation that patients with cardiovascular disease need to be stratified based on the Modified WHO Classification.

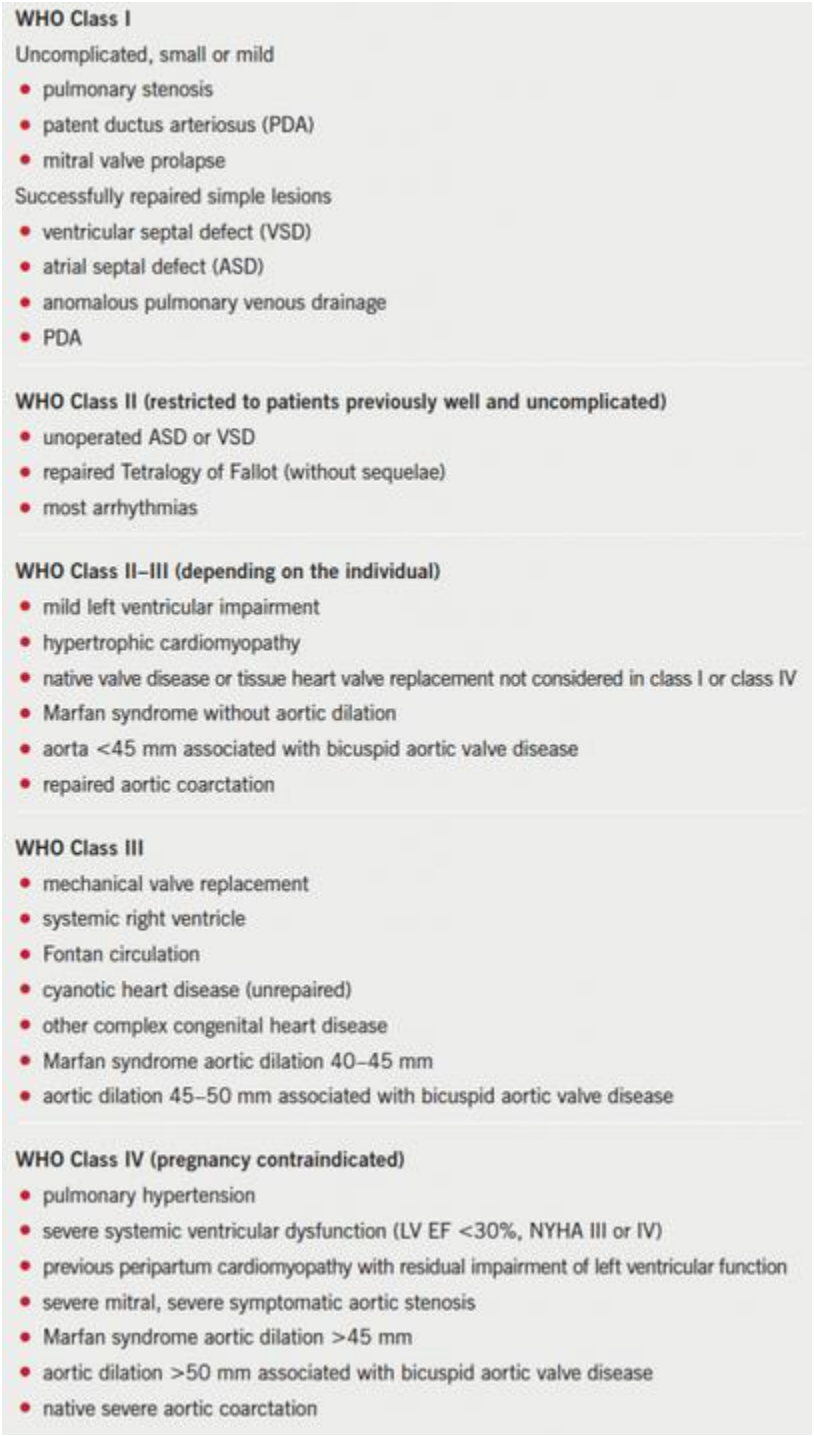

Figure 5: Modified WHO classification of maternal CVS Risk principles (14)

Risk class	Risk of pregnancy by medical condition
I	No detectable increased risk of maternal mortality and no/mild increase in morbidity.
II	Small increased risk of maternal mortality or moderate increase in morbidity.
III	Significantly increased risk of maternal mortality or severe morbidity. Expert counselling required. If pregnancy is decided upon, intensive specialist cardiac and obstetric monitoring needed throughout pregnancy, childbirth, and the puerperium.
IV	Extremely high risk of maternal mortality or severe morbidity; pregnancy contraindicated. If pregnancy occurs termination should be discussed. If pregnancy continues, care as for class III.

Figure 6: : Modified WHO classification of maternal CVS Risk applications (14)

Despite advances in the development of risk assessment tools and our understanding of this risk, clinical judgement still remains an important aspect in decision making.

A management plan should be made and discussed with the patient. This will include:

- Provision of neuraxial or parenteral labour analgesia (including pharmacological considerations)

- Possibility of decompensating during labour and delivery

- Possible anaesthetic techniques depending on the delivery plan (including monitoring and IV access)

- Post operative management and care

PARENTERAL LABOUR ANALGESIA

There is a lack of relevant evidence to structure clear management planning of parenteral labour analgesia in the cardiac parturient. Most studies have been conducted in healthy women.

Rocke and Rout in their Chapter entitled Anaesthesia and cardiac disease (1999, Obstetric Anaesthesia, 2nd Ed) recommend a continuous intravenous infusion of alfentanil starting early in labour with a loading dose of 30mcg/kg over 10 minutes followed by an infusion of 1mcg/kg/min. This is said to provide excellent first stage analgesia and additional boluses may be required for the second stage. The neonate should receive intravenous naloxone if necessary upon delivery and observed in the NICU post-delivery for 12 hours.

Intravenous patient controlled analgesia with remifentanil or fentanyl is not advisable in the high risk cardiac patient as one may have suboptimal results(12). This may lead to an inadequately mitigated catecholamine release. This may also require the administration of an opioid dose that suppresses ventilation and results in the sequelae of respiratory depression- hypercarbia and respiratory acidosis. Both of these features together with catecholamine release result in exacerbation of pulmonary hypertension, ischemia, decompensated heart failure and arrhythmias.

NEURAXIAL LABOUR ANALGESIA

Epidural analgesia is recommended as it provides excellent analgesia which minimises pain and anxiety-induced catecholamine release. This reduces intrapartum haemodynamic instability and lowers pulmonary artery and left atrial pressures.(10) Ideally the epidural catheter should be placed early in labour. The epidural can be easily titrated to meet the increasing analgesic requirements as labour proceeds. If caesarean section is required, it is possible to establish a denser block. The addition of opioids such as fentanyl improves the quality of analgesia but without influencing the sympathetic block.

Opioids may be used alone in the epidural or intrathecally in the critically ill patient(15). If an epidural has not been placed, Kuczowski and colleagues recommend a low spinal anaesthetic to allow for a controlled second stage.(15)

Hypotension is a major complication of an epidural for labour analgesia. This can be avoided or treated by careful infusion of crystalloid and vasoconstrictors as well as left uterine displacement. (4) These patients will need to be monitored in a high care setting by a dedicated nursing staff member.

INTRAOPERATIVE PLAN

Anaesthetic general principles

Additional continuous monitoring (other than CTG and NIBP) is advisable in the high risk cardiac patient. This would include:

- Ideally a 5-lead ECG with computerised ST segment trending and multiple lead monitoring.
- Pulse oximetry with audible and visual waveform display
- Intra-arterial catheter for beat-to-beat analysis of the effects of uterine contractions and expulsive maternal efforts on haemodynamics. It allows early detection of hypotension, facilitates vasoactive drug administration and measurement of arterial blood gases. In the high risk case, the catheter should be inserted prior to regional anaesthesia or induction of general anaesthesia.
- Central venous catheter for vasoactive drug administration in the unstable patient. E.g. postpartum haemorrhage, pulmonary oedema and hypotension.
- Pulmonary artery catheter(12)- the risks of insertion far outweigh the potential benefits. It is rarely indicated in the cardiac parturient. Complications include pulmonary artery rupture, incorrect data interpretation and arrhythmias. Scenarios where insertion may be an option include the severe pulmonary hypertension where titration of pulmonary vasodilatory agents such as nitrous oxide is necessary.
- Echocardiography: during general anaesthesia, transoesophageal echo best assesses volume status, regional and global cardiac function. Perioperative transoesophageal echo or transthoracic echo is indicated to assess pre-operative or intrapartum cardiac function or unexplained instability.

  Other equipment:

  -syringe drivers for IV infusion of vasopressor and inotropic agents. There should be bolus doses of phenylephrine and ephedrine prepared in advance.

  -External defibrillator pads.

ANAESTHETIC TECHNIQUES

Successful anaesthetic management of high risk cardiac pregnancies depend more on attention to haemodynamics than actual anaesthetic technique. Decision to proceed with a regional or general anaesthetic technique should not be determined by the degree of stenosis. Each case should have its own perioperative management plan involving the most senior and experienced anaesthetist.

With the evolution of monitoring and advances in intra- and post-op surveillance, the traditionally held belief of neuraxial techniques being contraindicated in the patient with a fixed output is slowly being challenged. High risk patients requiring caesarean section delivery may be reasonable candidates for a catheter technique either a low-

dose combined spinal epidural technique, a slowly titrated epidural anaesthetic or a continuous spinal. Time is required to assess and establish a limited extent of the block. Prior to institution of the block, it is essential that an intra-arterial catheter be inserted continuous monitoring arterial blood pressure. A single shot spinal technique cannot be supported as the rapidly evolving sympathectomy will result in severe hypotension which may prove fatal to the at risk patient. E.g. aortic and mitral stenosis where maintenance of preload and afterload is crucial.

Epidural Anaesthesia

Epidural anaesthesia has been shown to decrease catecholamine levels in the parturient (Ueland *et al*). It is also the recommended option as it expands the peripheral vasculature, reduces venous return, provides optimal analgesic effect while relieving the burden on the heart, and prevents an aggravation of cardiac failure. (16)

This is particularly relevant in the patient with mitral stenosis who does not tolerate an increase in heart rate and cardiac output (sympathetically mediated) as well as the normal pregnant female.

Evidence

In a case report by Hemmings et Al(17), a parturient with mitral stenosis underwent haemodynamic monitoring after having received a titrated epidural to T10 level for vaginal delivery. What the study showed was that epidural anaesthesia was beneficial during the first stage of labour. Induction of epidural anaesthesia caused the pre-induction rise in PVR, SVR and PAP to decrease while CO increased towards baseline. This was maintained throughout the first stage. Epidural anaesthesia may have also contributed to the stable heart rate control during labour secondary to sympathetic control.

Gomar *et al*(18) recommends careful graded epidural for patients with mitral stenosis. This was to be followed by cautious use of fluids and phenylephrine if a vasopressor was required. In a recent article(19), investigating the epidural anaesthesia management for caesarean section in parturient with rheumatic heart disease and mitral stenosis, various haemodynamic parameters were analysed. The 48 patients all remained haemodynamically stable. Epidural anaesthesia provided sufficient analgesia and muscle relaxation and avoided aggravation of cardiac failure.

Low dose Combined Spinal Epidural

The Low dose Combined Spinal Epidural technique provides excellent analgesia both intra- and post-operatively. There is a slower onset of the block which assists with the goal of maintenance of haemodynamics but one has the reliability of the subarachnoid block.

Evidence

Langesaeter et al (2010), in an observational study of parturients with various categories of cardiac disease noted that the arguments against regional anaesthesia maybe invalid with several case reports demonstrating fair outcomes. The reduction in systemic vascular resistance (SVR) after spinal injection can be counteracted with

a smaller dose of local anaesthetic, and co-hydration with a crystalloid infusion of Ringers Lactate. A spinal technique with 8-10mg bupivacaine and sufentanil 5mcg or fentanyl 20mcg with either a spinal or combined spinal epidural (CSE) showed no adverse maternal or fetal mortality at 6 months.

Langesaeter et al stated that with a general anaesthetic (GA) however, there is potential risk of intubation failure (although rare) and it was being performed by inexperienced staff at inconvenient times with less monitoring. The Confidential enquiries into maternal deaths 2000-2002 showed 6 deaths due to GA in females without cardiac disease. A GA can cause hyper/hypotension, tachycardia which is less controllable, and the risk of positive pressure ventilation affecting pulmonary artery pressures. The authors stated that a GA should be chosen when there is a contraindication to a regional or in the emergency setting otherwise they recommended regional anaesthesia. The conclusion was that haemodynamic stability could be obtained by incrementally-dosed regional anaesthesia, an infusion of phenylephrine and small repeat boluses of oxytocin. These should be directed by invasive monitoring.

Gomar-2005(18) fixed dosing and standard techniques should be avoided. A well-controlled CSE whereby the fast onset of the sympathetic block is avoided with incremental dosing should have invasive monitoring instituted early. A CSE with opioid is recommended to allow for a reduced dose of local anaesthetic. Lateralisation of the uterus, avoidance of intravascular volume loading and judicious use of vasopressors for hypotension is detailed.

In one of the first case reports Van Helder (1998) of a CSE technique for labour and caesarean section in a patient with mitral and aortic stenosis and insufficiency, it was stated that a CSE gives excellent pain relief with minimal sympathetic action. Successful anaesthetic management depends on attention to haemodynamic changes. For labour epidurals, Intrathecal sufentanil with 2.5mg bupivacaine, given at the onset of contraction followed by a continuous infusion of local anaesthetic at low doses maintained analgesia without haemodynamic instability. If the patient requires a caesarean section, the epidural should be functioning well and can be topped up with boluses of 2% lignocaine in 5ml increments over 15 minutes to a total of 15mls.

Continuous Spinal analgesia and anaesthesia

The only currently available catheters for CSA are epidural catheter kits with a 17 or 18-g Tuohy-type needle(1). The disadvantage of this would be the incidence of PDPH. The second potential kit would be the smaller “paediatric” epidural catheters which are 24-g and placed through a 20-g needle. This should reduce the incidence of post dural puncture headache.

There are no randomised control trials comparing agents to be used In CSA, for labour analgesia or caesarean section. Whether a local anaesthetic-opioid or opioid only mixture is used, they should be preservative free. Majority of case reports have used plain local anaesthetic which should remove concerns about laminar flow in the CSF.

There should be dedicated tubing and infusions pumps for this purpose. There should be strict adherence to an aseptic technique to reduce the risk of infection.

It is difficult to recommend practice guidelines regarding the removal of the intrathecal catheter as there is poverty of evidence for this issue.

“Regrettably, continuous spinal anaesthesia will probably continue to be an infrequently used option in the obstetric population for the foreseeable future because of the unavailability of suitable catheters. PDPH rates that accompany use of currently available catheters and needles are unacceptably high to advocate its routine use.”(1).

This technique maybe useful for patients with significant valvular heart disease. It provides excellent anaesthesia and analgesia without causing significant haemodynamic effects. This is ideal for the fragile physiology of the cardiac pregnant patient. Surgical anaesthesia can be administered with incremental dosing allowing the anaesthetist to monitor and respond to undesirable effects timeously.

Neurological problems associated with microcatheters are likely due to neurotoxicity from the local anaesthetic rather than the catheters themselves.

Evidence

Usage of the continuous spinal technique in the obstetrics dates back almost seven decades. It was initially described in 1906 as an intermittent injection, continuous spinal technique for surgical anaesthesia by leaving the needle “in the canal during the operation”(20). Almost 3 decades later, William T Lemmon described the use of a 17/18g needle attached to a stopcock and rubber tubing to administer local anaesthetic as required. A unique mattress with an opening for the in-dwelling needle was required (1). Lemmon and Hager further reported the use of the technique in 140 caesarean sections stating the benefit of haemodynamic stability but the unfortunate “rather severe but transient headache”.

This technique was also limited by the necessity to secure the needle in the subarachnoid space. The advent of catheters and microcatheters with the aim to reduce post dural puncture headache (PDPH) came at infrequent intervals. In 1987, Hurley and Lambert researched and later published in manuscript form, the use of a 32-g “microcatheter” which was passed through a 26-g spinal needle. Rigler et al went on to describe 4 cases of cauda equina syndrome associated with microcatheter usage for CSA.

Arkoosh *et al* published the largest randomized clinical trial of continuous spinal anaesthesia for labour with a 28-g catheter through a 22-g Sprotte-type spinal needle. There was no permanent neurological changes encountered suggesting that there was a less than 1% risk of neurological damage in the CSA group. Compared to the patient with the epidural catheter, the CSA patients had pain scores that were lower in the first 60mins after commencement. They had less of a motor block with higher

maternal satisfaction after delivery. There was no difference in obstetric or neonatal outcomes. There were however, higher pruritus scores and a trend towards more PDPH in the CSA group. A case report in 1997 by Pittard and Vucevic(21) detailed the first successful use of a subarachnoid microcatheter technique for an aortic stenosis patient requiring caesarean section delivery.

General Anaesthesia

In high risk cardiac patients, haemodynamic stability is foremost on the list of priorities during the case. Specific drug usage depends on the encountered cardiac lesion. The patient should be well-preoxygenated, cricoid pressure applied and the induction performed cautiously. Obtundation of the intubation response may be key and if opiates are to be used, the paediatrician should be informed beforehand. Nitrous oxide should be avoided if the patient has pulmonary hypertension.

Evidence

Iyer *et al* (2011) details that the choice of anaesthetic technique should be made on an individual basis and that a general anaesthetic should be considered for the severe lesions. It is felt that a high dense regional blockade for operative delivery decreases ventricular preload. A general anaesthetic allows effective control of preload, pulmonary artery pressures (PAP) and response to surgical stimulation. There is the option of administration of inhaled/nebulised pulmonary vasodilators. An opioid-based technique will allow reduction in pulmonary artery pressures during laryngoscopy and avoidance of the negatively inotropic effect of inhalational agents.

Anaesthetic techniques for Caesarean Section Delivery			
Low Dose Combined Spinal Epidural(LCSE)		**Continuous Spinal Anaesthetic(CSA)**	
Advantages	**Disadvantages**	**Advantages**	**Disadvantages**
> Excellent analgesia intra and postoperatively > Slower onset > Reliability of subarachnoid block	> Large volumes of local anaesthetic required >Time required to establish block > Unpredictable results > May lead to CVS instability	> More haemodynamic stability > Faster insertion > Titratable onset	> Difficult advancement > Potential for Cauda Equina Syndrome > Maldistribution of local anaesthetic > Increased potential dor Post Dural Puncture Headache
Incremental Epidural(EPI)		**General Anaesthesia(GA)**	
Advantages	**Disadvantages**	**Advantages**	**Disadvantages**
> Excellent analgesia > Reduces catecholamine levels > slower onset of block, able to monitor haemodynamics	> Large volumes of local anaesthetic required >Time required to establish block Unpredictable results	> Physiological stability > Availability of cardiac theatre and CABG facilities	> Increase in pulmonary vascular resistance > Reduced venous return during positive pressure ventilation > negative inotropy with inhalational agents

Table 2: Comparison of Anaesthetic techniques for Elective Caesarean Section Delivery

ANAESTHETIC GOALS

Regardless of the anaesthetic technique one has chosen to proceed with, the haemodynamic goals do not change.

Goal number 1:
AVOID TACHYCARDIA TO OPTIMISE LEFT VENTRICULAR DIASTOILIC FILLING TIME.
Low heart rates are required as an increased diastolic filling time is required to ensure adequate preload. This is compounded by the reduction in left ventricular filling time associated with tachycardia that may be a normal occurrence of pregnancy. This contributes to increases in left atrial pressure.

Goal number 2:
MAINTAIN HIGH TO NORMAL PRELOAD, CONTRACTILITY AND AFTERLOAD.
If there are reductions in systemic vascular resistance, this may result in an unwanted compensatory tachycardia.

In the event of haemodynamic instability, the choice of vasopressor should be individualised to the patient's anaesthetic goals. Ephedrine may cause a tachycardia. Phenylephrine is a good option for restoration of haemodynamic stability with minimal to no untoward effects on the circulation. Noradrenaline will provide inotropic support

without a significant increase in the heart rate. Vasopressin will spare the pulmonary vasculature.

UTEROTONICS

There is a poverty of evidence regarding the usage of uterotonics in the cardiac pregnant patients presenting for Caesarean section either as prophylaxis or treatment of postpartum haemorrhage. Some advocate not using any intravenous oxytocin(23) whilst others use infusions.(22, 23) Langesaeter et al(24) looked at the anaesthetic management of the cardiac pregnant patient. Ultra-low doses of oxytocin (0.05-0.5IU) were administered and repeated as required to avoid haemodynamic instability and excessive intravenous fluid to avoid congestive cardiac failure. Haemodynamic changes were not only transient and minor but did not aggravate any symptoms. Seemingly, there is no consensus guideline on the matter. The main issue with oxytocin is hypotension as a result of vasodilatation. This is mediated by calcium-dependant nitric oxide release.(25). Chlorobutanol, a preservative may have some negatively inotropic effects on the atrial myocytes.

More concerning, is the repeated development of reproducible ST depression(coronary vasoconstriction) on electrocardiogram in those patients receiving intravenous oxytocin boluses.(25) This appeared to be a dose dependant phenomenon with statistically more ST depression in the patients given a 10iu bolus versus 5iu.(Jonnson). This together with the increase in cardiac output secondary to both heart rate and stroke volume may be detrimental in patients vulnerable to cardiovascular instability and cannot mount an increase in output. This adverse effect may be accentuated in situations where women are already hypovolaemic, when higher bolus doses are administered and when it is rapidly administered.

There have been 3 relevant oxytocin dosing studies that assessed uterine tone following administration of a bolus of oxytocin during caesarean delivery.(26) It is important to assess whether studies address usage of oxytocin for prophylaxis (initiating adequate uterine tone) or for the treatment of uterine atony. This may be via infusion, bolus or both).

Carvalho et al(27) found that the ED90 for oxytocin at elective caesarean section was 0.35iu. Butwick et al(28) concluded that a dose of 0.5-3iu oxytocin was adequate to achieve sufficient uterine tone at 2 minutes. This suggests that in the event of a high risk cardiac patient requiring caesarean section, minimal dosing is required for adequate uterine tone. One speculates that this would avoid the adverse effect of hypotension and tachycardia in a patient requiring absolute haemodynamic vigilance. Oxytocin varies in its cardiovascular effects according to dosing and mode of administration (26) A slow infusion of 5 IU results in less haemodynamic changes compared to a 5 IU bolus.

Co-administration of phenylephrine after a slow bolus of oxytocin obtunds the peripheral CVS effects.

The haemodynamic effects of oxytocin following a second does are attenuated as compared to after the 1st effect.

POSTPARTUM CARE

Postoperatively, patients with severe cardiac disease should be kept in the high care unit / ICU for aggressive monitoring of

- Haemodynamics
- Oxygen saturation
- Fluid therapy

During the first 24-72 hours significant fluid shift may occur, which may cause congestive heart failure and dysrhythmias especially in patients with diminished left ventricular function(29) It is recommended that the high risk patient be monitored for signs of heart failure for several days. A post-delivery echocardiogram may be warranted in high risk individuals paying close attention to the affected valve in a resource-rich environment.(29)

In those patients at low risk for cardiac failure and normal ventricular function, a shorter period of observation (up to 48hrs) might be adequate.

There is also an increased risk of thromboembolic complications postpartum and anticoagulation should be adjusted accordingly.

Most adverse events occur in the first week following delivery but this can extend to 3-4 weeks postpartum. The level of postpartum monitoring is dependent on the underlying cardiovascular condition as well as any obstetric or cardiac events that occurred during labour and delivery. Re-establishment of anticoagulation should occur timeously.

Postoperative analgesia should be provided with opioids or combination with NSAIDS and opioids intravenously. Patient controlled analgesia(PCA) or continuous epidural analgesia is an adequate alternative in cardiac patients

CONCLUSION

In South Africa, cardiac disease in pregnancy is the commonest medical disorder leading to maternal mortality. Twenty six percent (26%) of those deaths have been attributed to valvular heart disease complications.(30) “Nature’s stress test” or pregnancy can precipitate symptoms of cardiac disease in those parturients that were previously asymptomatic. A collaborative effort between obstetrician, cardiologist and anaesthetic team is best started early to reap the benefits of prevent the complications of the disease. Antenatal risk stratification is important for specific management planning.Stenotic lesions carry a higher risk of maternal and foetal complications than regurgitant lesions ones.(30)

There are currently no evidence based guidelines on the preferred anaesthetic technique for caesarean section. Options include Incremental epidural dosing, Low-dose Combined spinal epidural, continuous spinal technique and general anaesthesia. All of the above have their advantages and issues and key to decision making depends on confidence and experience with a particular technique, individualising care and above all maintenance of perioperative haemodynamic stability regardless of one’s choice.

RECOMMENDATIONS FOR THE REGIONAL LEVEL ANAESTHETIST

- Open communication with senior anaesthetist experienced in cardiac disease pregnancy
- High care/ ICU Bed available for post-op
- The consultant obstetrician should be performing the C/S.
  Theatre preparation is vital! Equipment including difficult airway devices, CSE kits, functioning syringe drivers or infusion pumps, invasive monitoring of blood pressure, various sizes of blood pressure cuff. Pressure wedge, emergency drugs, uteronics-must be precalculated. Many of these may not be available and alternatives should be discussed at a higher level.
- Anaesthetic goals:
  - SLOW HR(60-80bpm),
  - AVOID TACHYCARDIA AND ALL ITS CAUSES(drop in SVR or drugs),
  - NO DECREASE IN AFTERLOAD (consider starting a phenylephrine infusion pre-induction),
  - AVOID LARGE SINGLE BOLUS INJECTIONS
- Keep patient at their baseline The benefit of utilising uterotonics in the setting of postpartum haemorrhage outweighs the drop in the blood pressure which is treatable.
- Ultra-low dose uterotonics and an infusion thereafter.
- Post-delivery complications can be catastrophic-BEWARE pulmonary oedema, arrhythmias, thromboembolic events!
- Patient may need prolonged monitoring in recovery. DISCHARGE WHEN YOU ARE SATISFIED WITH HAEMODYNAMICS AND UTERUS.

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