CARDIAC DISEASE IN PREGNANCY

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In developing countries cardiac diseases is more common, women with congenital heart disease who have survived into adulthood are more common than rheumatic heart disease and ischemic heart disease has become common cardiac cause of death in pregnancy.

PHYSIOLOGICAL ADAPTATIONS TO PREGNANCY, LABOUR AND DELIVERY:

Normal pregnancy is associated with significant haemodynamic changes. These may not be tolerated in women with heart disease. Cardiac output increases by 40$, reaching a maximum by and second. There is peripheral vasodilation, an increase in heart rate, one fall in systemic and pulmonary vascular resistance. Labour and delivery are associated with further increase in cardiac output. Palpitations, extra systoles, sinus tachycardia and ejection systolic murmur are common in pregnancy but rarely represent underlying pathology. The ECG changes associated with normal pregnancy include a trial and ventricular Q wave and inverted T wave in lead III, ST segment depression and T wave inversion in inferior and lateral heads.

Cardiac out is increased by 40% during pregnancy because of increases heart rate due to decreases vascular resistance and increases in stroke volume.

- cardiac output 15% in 1st stage of labour.
- 50% in2nd stage of labour.

By end of 2nd trimester blood volume and stroke volume have risen by 30 and 50%. Although there is no increase in pulmonary capillary wedge pressure, serum colloid osmotic pressure is reduced, making pregnant women susceptible to pulmonary edema, will be precipitated if there is increase in cardiac preload like infusion and in preeclampsia due to increases in vascular permeability. In late pregnancy in supine position, pressure of gravid uterus on inferior vena cava causes reduction in venous return to heart and fall in stroke volume and cardiac output. Turning from lateral to supine position can result in 25% reduction in cardiac output. Pregnant women should be nursed in left or right lateral position wherever possible. Reduced cardiac output is associated with reduction intrauterine blood flow and placental perfusion, this can compromus-fetuso labour is associated with increases cardiac output 15% in 1st stage 50% in 2nd stage – uterine contraction 300-500 ml of blood back into circulation and sympathetic response to pain and anxiety elevated B.P. Epidural

anesthesia or analgesia cause arterial vasodilation and fall in B.P. General anaesthesia is associated with rise in B.P. and heart rate during induction but cardiovascular stability thereafter. Prostaglandin given to induce labour have little effect on hemodynamic circulation but ergometrine causes vasoconstriction and syntocinon causes vasodilation and fluid retention.

In 3rd stage of labour upto one litre of blood may be returned to circulation due to relief of inferior vema cava obstruction and contraction of uterus. The intra thoracic and cardiac blood volume rises, and cardiac output increases by 60-80% followed by rapid decline to pre labour volume within one hour after delivery. Transfer of fluid from extra vascular space increases venous return and stroke volume. Those women with cardiovascular compromise are therefore most at risk of pulmonary edema during 3rd stage of labour and immediate post partum period.

INCIDENCE:

Although cardiac disease is rate < 1% in U.K. cardiac disease is most common cause of maternal death. Ischemic heart disease also becoming common in pregnancy and death rates are increasing.

ETIOLOGY:

Can be divided into congenital and acquired causes. The most common congenital causes are atrial septal defect (VSA, ASD) and ventricular septal defects, patent ductus arteriosis (PDA) and aortic carticorarctation. These are diagnosed before pregnancy. Acquired causes of cardiac disease include ischemic heart disease, rheumatic heart disease cardiomyopathies and aneurysms and dissection of aorta and its branches.

GENERAL PRINCIPLES:

In pregnant women with heart disease, it is important to remember outcome and safety of pregnancy are related to presence and severity of pulmonary HTN presence of cyanosis, hemodynamic significance of lesion and functional class. The functional class is determined by level of activity that leads to dyspnoea. New York Heart Association (NHA) functional classification:-

NYHA	SYMPTOMS
I	No symptoms and no limitation of physical activity
II	Mild symptom and slight limitation during ordinary activity.

III	Relieved by rest mark limitation in activity due to symptoms and even during less than ordinary activity.
IV	Severe limitation, experience symptom at rest.

In addition, women with previous cardiac events like transient ischemic attack, arrhythmic, pulmonary edema or heart failure and left groin lesion (aortic and mitral stenosis) or myocardial dysfunction are at risk in pregnancy women with congenital heart disease are at increase risk of having baby with congenital heart disease and should be offered detail fetal scanning for cardiac anomalies. During pregnancy women with heart disease require multi disciplinary tarmicase with regular antenatal visits and judicious monitoring to avoid or treat any arenue infection or hypertension. There should be early involvement of obstetric anesthetist and carefully documented plan for delivery.

EISENMENGER'S SYNDROME & PULMONARY HYPERTENSION:-

The most common forms of pulmonary hypertension is encountered in women of child bearing age of idiopathic pulmonary arterial hypertension, Eisenmenger's syndrome (when pulmonary HTN develops secondary to large left to right shunt such as VSD, and the shunt is reversed to become right to left, with consequent cyanosis because deoxygenated blood on right side mix with blood on left side, pulmonary HTN can also develop secondary to chronic pulmonary thromboembolism disease, or secondary to connective tissue disorder like scleroderma, and sickle cell disease also cardiac lesion like mitral stenosis causes left atrial dilatation and hypertrophy and increase pulmonary capillary wedge pressure. Pulmonary HTN from any cause is dangerous and maternal mortality is 25 to 40%. The danger relates to fixed pulmonary vascular resistance and inability to increase pulmonary blood flow with refractory hypoxemia. Most pregnancy related death are cause by thromboembolism hypovolemia or preeclampsia. The maternal mortality has fallen to 17% for pulmonary HTN due to aggressive drug regimens.

Pulmonary HTN is defined as non-pregnant elevation of mean pulmonary artery pressure of 25 m Hg high or more at rest or 30 m Hg on exercise in absence of left to right shunt. Pulmonary artery systolic pressure qis estimated using Doppler ultrasound to measure reguroitant get velocity across tricuspid valve. This should be used as screening test. Women with pulmonary HTN, should be asked to avoid pregnancy and use contraception till resolved.

MANAGEMENT:-

Modern management of pulmonary hypertension includes drugs such as sildenafil vasodilator / tadalafil endothelin antagonist and bosentan and macitentan. With such therapies / pulmonary pressure can be reduced to within normal range and pregnancy may be safely negotiated. In termination should be offered. Since elective termination carries 7% risk of maternal mortality it is important to avoid pregnancy if possible. In patient refuse for termination, then multidisciplinary care, with cardiologist involved elective admission for bed rest, O_2 and thromboprophylaxis with low molecular weight heparin are recommended. Fetal growth should be carefully monitored. There is no evidence that monitoring pulmonary artery pressure before or during delivery improve outcome, insertion of pulmonary artery catheter increases risk of infection, vasodilator with exception of nitric oxide and prostacyclin, will result in lowering of B.P. and exacerbation hypoxemia. There is no evidence that abdominal or vaginal delivery of regional versus general anesthesia improve outcome in pregnant women with pulmonary HTN, greater care must be taken to avoid systemic vasodilation. Patient should be nursed in intensive care unit after delivery. Nebulized prostacyclin can be used to prevent pulmonary vasoconstriction. ergometrine and prostaglandin F2α as it is associated with pulmonary hypertensive crisis. Delivery should be in HDU and with strict control of B.P. fluid balance and O_2 saturation.

AORTIC DISSECTON:

Aortic dissection common cause of cardiac death. In pregnancy, and pregnancy with risk of aortic dissection. Most cases occur in late pregnancy or near term or early puerperium. Certain condition predispose to aortic dissection which include, bicuspid aortic valve, aortic coarctation. turner's syndrome, ehlers danlos syndrome, vascular eds type iv, marfan syndrome. marfan syndrome mortar syndrome is an autosomal dominant condition with skeletal and other features including tall stature, a high arched palate, scoliosis and lens dislocation, progressive aortic root dilatation and aortic root dimension > 4 cm are associate with increase in risk. Conversely in women with minimal cardiac involvement and aortic root < 4cm pregnancy outcome is good. Overall pregnancy is associated with 5 sold increases in risk of aortic complication in women with marfan syndrome.

Management: Aortic, acute dissection should be suspected if women in late pregnancy present with sever chest or inter scapular pain / espig associated with systolic HTN, different B.P. in each arm or an early diastolic murmer, especially if she has predisposing risk factor. Urgent CT, MRI or echocardiography is essential as well as rapid and effective control of blood pressure. Women with aortic root > 4.5 cm should be advised to delay pregnancy. Management of marfan's syndrome or

dilation aortic roots from other cause should include serial ECG and β-blocker vaginal delivery is possible with those with stable aortic root measurements < 4.5cm, but elective C-section with epidural if there is enlarge > 4.5 cm or dilation aortic root. There is higher risk of aortic rupture and maternal death if aortic root > 4 cm.

LEVEL OF RISK OF MATERNAL MORTALITY

- **HIGH RISK RED ALERT – MORTALITY 50%.**

1) Pulmonary HTN, aortic dissection, complicated aortic coarctation, marfan syndrome with significant aortic root involvement and M.I. myocardial infarction.

- **MODERATE RISK:**

Mitral Stenosis, NYHA class 3 and 4, severe aortic sterosis and mechanical heart valves.

- **MINIMAL RISK:**

ASD, VSA, PDA, corrected tetralogy of fallot, tissue prosthetic valve, pulmonary and tricuspid valve disease, arrhythmia mild mitral stenosis.

MITRAL STENOSIS: Rheumatic Heart Disease

- This is most common lesion of rheumatic heart disease (90%).
- Increase risk of pulmonary edema in pregnancy, greatest in labour.
- Increase in heart rate in pregnancy decreases ventricular.

Filling time and increases in pulmonary blood volume leading to pulmonary edema.

- First line Rx for pulmonary edema in pregnancy should be diuretics. β-blockers also used to slow heart rate and improve left arterial emptying add digoxin if in atrial fibrillation) AF.
- Mitral stenosis is most likely lesion to require treatment for pulmonary edema, heart failure or surgery in pregnancy.
- With severe mitral, consider surgery before pregnancy, before pregnancy.
- The risk of thromboembolism is 1.5% in pregnancy, higher with left artrial enlargement and atrial fibrillation. Treat with low molecular weight heparin. History of previous mitral valvotomy does not preclude restenosis. Mitral stenosis is most common rheumatic heart and is important in pregnancy because women may deteriorate secondary to tachycardia, arrhythmia or increased cardiac output, the commonest complication is pulmonary edema, secondary to increased left arterial pressure and precipitated by increase heart rate or increased volume such as occur during 3rd stage of labour. The risk is increased with severe mitral stenosis, moderate or severe symptoms prior to pregnancy and in those diagnosed late in pregnancy. Mitral stenosis, world wide remains most common lethal pre-existing heart condition in pregnancy. There are many pitfalls because (i) an asymptomatic patient with stenosis may deteriorate in pregnancy, (ii) mitral stenosis may have increase in severity since previous uncomplicated pregnancy (iii) stenosis can recur or worsen after valvuloplasty or valvotomy (iv) mitral stenosis that may previously not have been recognized may be , missed during routine antenatal examination because murmur is low-pitched, usually quiet, diastolic and

submammary. Women may deteriorate secondary to tachycardia related to pain, anxiety, exercise or undercurrent infection, arrhythmias or increase cardiac output of pregnancy. Sinus tachycardia at rest should prompt concern. Pulmonary edema may be precipitated by increase volume that occur in 3rd stage of labour or following injudicious intravenous, fluid therapy. The risk are increased with severe mitral mitral stenosis valve area < 1 cm^2 moderate or severe symptoms prior to pregnancy, and in those diagnose late in pregnancy.

The ECG in mitral stenosis show left arterial P wave and R axis deviation. Chest x-ray show small heart with prominence of left arterial QP pemolarge and left atrium and pulmonary congestion or edema. The diagnosis confirm by transthoracic echocardiography.

MANAGEMENT OPTIONS:- Women with severe mitral stenosis, should be advised to delay pregnancy until after balloon, open or closed mitral volvotomy or if valve is not amenable to valvotomy until after mitral valve replacement. β-blockers decrease heart rate, increase diastolic filling time and decrease risk of pulmonary edema. It should be given in pregnancy to maintain heart rate of under 90 beat per minute. Diuretic should be commenced or continued if indicated. It is also important that a woman does not over exert herself. In the event of pulmonary edema, patient should be setup, O_2 should be given and heart rate slowed by relief of anxiety with diamorphine, and intra venous furosemide 20 mg administer digoxin should be used only in case of arterial fibrillation. If medical therapy fails or for those with sever mitral stenosis, balloon mitral volvotomy, may be safely and successfully used in pregnancy if the valve is suitable. Although this will require transfer to hospital with major cardiac facilities. Percutaneous balloon valvotomy carries risk of major complication of about 1% whereas for surgical volvotomy risk as follows:-

- Closed volvotomy: fetal mortality 5-15%, maternal mortality 3%.
- Open volvotomy: fetal mortality 15-33%, maternal mortality 5%.

If an open volvotomy is likely to be required it should be deferred until after delivery.
Women with mitral stenosis should avoid supine and lithotomy position for labour and delivery, fluid overload must be avoided. Even in presence of oliguria, without significant blood loss, the intravenous colloid avoided continuous epidural analgesia or anesthesia is suitable for patients with mitral sterosis as in vaginal delivery suitable but 2nd stage of labour should be augmented with instrumental delivery to limit use of maternal efforts.

AORTIC STENOSIS:

Left ventricular outflow obstruction at any level can cause problem during pregnancy. Pre pregnancy assessment is ideal. Significant obstruction results if aortic valve area less than one cm^2 < 1 cm^2 or if the gradient across the valve > 50 m Hg in non pregnant state is considered severe. Indication that pregnancy will be high risk include impaired left ventricular function and failure to

achieve normal rise in B.P. without development of FT or T wave change during exercise, and chest pain, syncope or presyncope. ECG will normally show left ventricular hypertrophy and Doppler transaortic velocity

will rise during pregnancy. If stroke volume increase in normal fashion. Any patient who develop angina dyspnoea or resting tachycardia should be admitted to hospital for rest. Administration of β-blocker will increase diastolic coronary flow time and left ventricular filling with resultant improvement in angina and left ventricular function. If despite these measure, arginine pulmonary congestion and left ventricular failure persist or progress, than balloon aortic valvotomy needs to be considered.

MECHANICAL HEART VALVES:

Most women with mechanical heart valves have sufficient cardiovascular reserve to accomplish pregnancy safely. These women require life log anticoagulation, that is why they have high risk pregnancy. Pregnancy itself increases risk of thromboembolism, its hypercoagulation state. Warfarin is associated with warfarin embryopathy and increase risk of miscarriage still birth and fetal intracerebral hemorrhage. Heparin even in full doses of anticoagulant doses, is associated with increase risk of valve thrombosis and embolic events. The literature supports, lower risk of thromboembolic events if warfarin is continued through out pregnancy and lower risk of fetal loss, fetal hemorrhage and fetal anomalies is LMW Heparin used. However Warfarin is associated with warfarin embryopathy (chondrodysplasia punctata) if given during period of organogenesis (6-12 weeks gestation) and with fetal intracerebral hemorrhage in 2nd and 3rd trimester. The fetal risk of from warfarin is dose dependent. Women requiring more than 5 mg daily are increase risk of teratogenesis / miscarriage and still birth. Heparin and low molecular weight heparin do not cross placenta and are attractive option but even in full anticoagulant dose, they are associated with increased risk of valve thrombosis and embolic events. Heparin also causes retroplacental hemorrhage so risk of fetal loss is not eliminated.

Low molecular weight heparin (subcutaneous) is better than unfractionated heparin, which is given parenteral and lies short duration of action and narrow therapeutic index, hence L.M.W.H. is preferred has better safety profile in pregnancy and provided there is close monitoring of anti-Xq levels with appropriate dose adjustment and good compliance with twice daily injections and lower risk of thromboembolic events, with use of L.M.W.H. Also low dose aspirin also given and also doses of L.M.W.H. to maintain peak anti Xq level between 0.8 – 1.2 IU / ml. There are three options for anticoagulation:- Aspirin has antithrombotic properties.

1) Continue warfarin throughout pregnancy, stopping only for delivery. There is safest for mother. But maintain INR of 3.0

2) Replace warfarin with high dose L.M.W.H. from 6-12 weeks gestation, to avoid warfarin embryopathy but continue until prior to delivery from 12 weeks till 37 weeks when L.M.W.H. is used again.
3) Use high dose L.M.W.H. throughout pregnancy. The risk of thrombosis is less with newer bi leaflet valves and valves in aortic position. If warfarin is used in pregnancy then, serial fetal scan are indicated to detect embryopathy, and intracerebral hemorrhage. Warfarin should be discontinued and substituted for L.M.W.H. for 10 days. Prior to delivery to allow clearance of warfarin from fetal circulation. For delivey itself L.M.W.H is interrupted, 24 hour prior to delivery but restarted post partum conversion from L.M.W.H back to warfarin back to warfarin should be delayed for 5 days after delivery to minimize risk of obstetric hemorrhage. Warfarin can be restarted from 5^{th} – 7^{th} day postpartum. In event of bleeding or need for urgent delivery in fully anticoagulated patient warfarin reversed with recombinant human factor VI IQ fresh frozen plasma and n + k and heparin with protamine sulphate.

ISCHAEMIC HEART DISESE: MYOCARDIAL INFARCTION: M.I.

Ischemic heart disease in pregnancy is becoming more common as maternal age and obesity increase. Risk of M.I. is increasing due to increase in age at pregnancy and lifestyle factors. Mortality is 20% immediate 32% overall, highest in puerperium.

Risk factor include increase maternal age of pregnancy. Age > 35 years when pregnant, multigravida, those who smoke, and women with diabetes, obesity, hypertension and hypercholesterolaemia. Acute coronary syndrome may be diagnose + F.H. of 1 HD as in non-pregnant women with increase troponin level which are not altered by pregnancy and ECG changes. Infarction occurs in postpartum period. Maternal death rate is 10%. In pregnancy other underlying pathologies form I should be considered like, coronary artery thrombosis or dissection, are most common in pregnancy. There may be a typical symptom like epigastric or abdominal pain vomiting and dizziness.

* Single normal ECG especially in pain free patient does not rule out ischemia, serial ECG should be considered myocardial infarction in pregnancy usually develop without preceding history of typical angina.

MANAGEMENT: Management of acute MI and acute coronary syndrome is same as for non-pregantn woman, coronary angiography should be undertaken without hesitation in order to define pathology and determine management. Intravenous anol intra coronary thrombolysis and percutaneous coronary intervention PCI and stenting have been successfully performed in pregnancy. Percutaneous coronary intervention is better alternative to thrombolysis therapy (streptokinase) for M.I. as it cause less bleeding risk and also allow management of spontaneous dissection with coronary artery stents. For secondary prevention in women with known ischemic

heart disease, both aspirin and β-blocker are safe in pregnancy, clopidogrel also safe in pregnancy statins should be discontinued prior to and for duration of pregnancy.

ENDOCARDITIS PROPHYLAXIS

Infective endocarditic is rare in pregnancy but life threatening for both mother and baby. The current NICE guideline, state that antibiotic prophylaxis against infective endocarditis is not required for child birth in peripartum period. American heart Association have recommended, cover only for patients deemed to be at high risk of developing infective endocarditis such as women with previous IE, and for those who have poorest outcome if they develop IE, such as those with cyanotic heart disease that is tetralogy, traspositioning of rest vessels, also patient with prosthetic and heart valves (mechanical tissues). If antibiotic prophylaxis is used, it should be used with, amoxillin 2 gram IV plus gentomycin 120 mg IV at onset of labour or ruptured membrane, or prior to C-section, followed by amoxicillin 500 mg oral (or 10 m/i.v. depending on patient's condition 6 hour later. For women who are allergic to penicillin vancomycin 1 gram w or teicoplanin 400 mg IV may be used instead of amoxicillin.

HYPER TROPHIC CARDIO MYOPATHY

Most cases of hypertrophic cardiomyopathy (HCM) are familial, inherited, as autosomal dominant. It is characterize by hypertrophy of undiluted left ventricle in absence of abnormal hemodynamic load high risk patient referred because of disabling symptoms or malignant family history. Women may8 be asymptomatic especially diagnosed because of family screening or may experience syncope or angina like chest pain. The danger relates to left ventricular out flow obstruction, which may be precipitated by hypotension or hypovolemia. Provided these are avoided pregnancy is well tolerated. HCM is not infrequently first diagnosed in pregnancy when systolic murmur leads to ECG and echocardiography studies symptoms of shortness of breath, chest pain, dizziness or syncope indicate need for Beta-blocker. Ventricular arrhythmic are common in older patient but uncommon in younger. Sudden deaths, risk factor include non-sustained ventricular tachycardia VT, failure of systolic B.P. to rise during exercise and family history of sudden cardiac death. Beta-blockers should be continued in pregnancy or initiated for symptomatic women.

PERIPARTUM CARDIO MYOPATHY:

The pregnancy specific condition is defined as heart failure secondary to left ventricular systolic dysfunction, towards end of pregnancy or in the months following delivery.

The left ventricle may not be dilated but left ventricular ejection fraction is nearly always reduced (< 45%). Echocardiography may show dilatation of four chambers but dominated by left ventricular hypokinesia. Recognized risk factor for peripartum cardiomyopathy include; multiple pregnancy

hypertension, pre-existing or related to preeclampsia, multi parity, increase maternal age and afro caribbean race. Diagnosis should be suspected in peripartum patient with breathlessness, tachycardia, signs of heart failure pulmonary edema is a major feature precipitated by use of syntocinon or by fluid is given to maintain cardiac output during spinal anesthesia. For delivery chest x-ray should enlarge heart, with pulmonary congestion and bilateral pleural effusion. Sy8stemic embolism from unifal thrembus may can ventricular arrhythmias. The differential diagnosis include preexisting but undiagnosed dilated cardiomyopathy, pulmonary thromboembolism, amniotic fluid embolism, myocardial infraction and pulmonary edema related to preeclampsia or β-2 agonist for preterm labour. Echocardiography confirms diagnosis by implicating left ventricle and exclude pulmonary embolism as cause.

MANAGEMENT:

Treatment is as for other causes, of heart failure with oxygen, diuretics, vasodilators, and angiotensin converting enzyme ACE inhibitors if postpartum. Thromboprophylaxis is imperative. The cautious addition of cardioselective β-blockers may be helpful if tachycardia persist especially if cardiac output is well preserved. The most gravely ill patient will need intubation, ventilation and monitoring with use of isotopes and sometime temporary support from an intra thoracic balloon pump, heart transplant may be only chance of survival in severe cases prognosis and recurrence depend on normalization of left ventricular size and function within 6 months of delivery.

ARRYTHMIAS:

A sinus tachycardia require investigation for possible underlying pathology such as blood loss, injection, heart failure, thyrotoxicosis or pulmonary embolism, but no treatment is required if such cause are excluded. The most common arrhythmic in pregnancy is supraventricular tachycardia SVT. As SVT that does not respond to vagal maneuver may be terminated in pregnancy with aderogine or intravenous verapamil or beta blockers like propranolol. But propafenone and amiodarone is contraindicated because latter interfere with fetal thyroid function.

GENERAL MANAGEMENT DURING LABOUR & DELIVERY

- Spontaneous labour at term is the rule rather than exception.
- Oxytocin & PGE2 can be used for induction of labour
- Lateral supine position
- Pain relief(reduces tachycardia,myocardial work, CO)
- Restriction of IV fluid 75ml/hr
- O_2 inhalation & pulse oxymetry
- Antibiotic prophylaxis where needed.
- Fetal heart monitoring
- Prevention of postpartum pulmonary oedema.
- Avoid IV methergin or bolus oxytocin but control PPH
- Avoid difficult instrumental delivery

MANAGEMENT OF CARDIAC FAILURE IN PREGNANCY

- Propped up position
- O2 administration
- Monitoring with ECG and pulse oxymetry
- Diuretic: Frusemide (Loop) (40–80 mg) IV
- Mechanical ventilation • Injection morphine 15 mg IM
- Digoxin 0.5 mg IM followed by tab digoxin 0.25 mg P.O. (Digoxin crosses the placenta and is excreted in breast milk)
- Dysrhythmias—quinidine or electrical cardioversion
- Tachyarrhythmias—Adenosine (3–12 mg) IV or DC conversion

MANAGEMENT DURING LABOUR

FIRST STAGE

Should be confined to bed with lateral recumbent position to minimize aorto - caval compression.

O2 inhalation 6 ltrs/min.

Analgesia ,majority by epidural

Fluid infusion should not be more than 75ml/hr. to prevent pulmonary edema.

Careful watch of pulse & resp. rate, if pulse
> 100/min b/w uterine contractions, then rapid digitalization may be done

Cardiac monitoring & pulse oximetry can detect arrythymias & hypoxaemia

SECOND STAGE

Delay in 2nd stage of labour to be curtailed by forceps / ventouse application under pudendal and / or perineal block.

Ventouse preferred as it can be applied in lateral recumbent position.

i.v. ergometrine to be withheld after delivery of shoulders.

Prolonged valsalva increase PA pressures, increases R to L shunting

THIRD STAGE

Conventional management to be followed.

It is preferable to administer oxytocin in an i.v. drip to all cases who are not in failure and simultaneously furosemide 40mg. i.v. to relieve volumetric load.

Slight blood loss is beneficial and if in excess oxytocin infusion may be continued.

Episiotomy wound repaired early. Pt kept in propped up position, Inj. morphine & O2 supply through out.

If resp. distress develops secondary to pulm. congestion , should be treated aggressively as pulm edema in I.C.U.

PPH CONTROL

Oxytocin bolus avoided

Methergin & prostodin avoided

Misoprostol can be safely given rectally

Mechanical compression sutures , such as B-Lynch brace sutures , can be used prophylactically in a woman with severe heart disease on CS as an adjunct to medical treatment

MANAGEMENT DURING PUERPERIUM

- Close observation for 1st 24hrs.
- Signs of pulm congestion & Edema to be looked for.
- Sedatives to be given in 1st few days to relieve Anxiety Related tachycardia with Adequate Bed Rest.
- Any Infection however during puerperium should be seriously viewed.
- Not too many visitors.
- Breast feeding is not contraindicated unless there is failure.
- Pts. on warfarin should be allowed to Breast feed as secretion through Breast milk is extremely small.

Infective Endocarditis Prophylaxis in Cardiac patients for Obstetric Procedures

IE Prophylaxis only recommended for High risk Patients-

- Patients with Prosthetic Cardiac valves
- Patients with previous Infective Endocarditis
- Cardiac transplant patients with valvulopathy
- CHD with Unrepaired cyanotic CHD with palliative shunts or conduits, CHD with repaired prosthetic valves less than 6 months and repaired CHD with residual defects at site or prosthetic device

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When **antibiotic prophylaxis** is recommended in high-risk patients, the preferred regimen is ampicillin 2 g i.v. or i.m. plus gentamicin 1.5 mg/kg (maximum 120 mg) i.v. administered within 30 minutes before delivery, followed

by ampicillin 1 g i.v. or i.m. (or **amoxicillin** 1 g [as the trihydrate] orally) six hours later.