

MANAGEMENT OF PRE-ECLAMPSIA AND ECLAMPSIA

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Fits occurring in pregnant women were recognized as early as the 4th century B.C. by Hippocrates.¹ The condition was termed eclampsia a Greek Word which translates literally as "shine forth" implying a sudden development. Little was known about eclampsia until 1843, when Lever of Guy's Hospital found that many of the women who had fits also had albumin in their urine.² It was by the wide spread use of sphygmomanometer for measurement of blood pressure, that the condition was known to be associated with hypertension.

Because it was recognized that albuminuria and hypertension could precede the onset of fits the term pre-eclampsia was used although only a small proportion of patients with pre-eclampsia subsequently developed eclampsia.³

DEFINITION AND CLASSIFICATION

The American college of obstetrician and Gynaecologists separates the hypertensive disorders of pregnancy into three general categories:

- i. Chronic hypertension that precedes pregnancy.
- ii. Chronic hypertension with super imposed pre-eclampsia/eclampsia.

- iii. Pregnancy induced hypertension (PIH).

By definition, women who become hypertensive during pregnancy have PIH and those who also develop proteinuria or pathologic oedema after the 20th week of gestation in addition to hypertension have pre-eclampsia.

Table I illustrates the criteria for applying the diagnosis of pre-eclampsia. Eclampsia is simply seizure or coma that occurs during pre-eclampsia and that is not caused by concurrent neurological disease such as epilepsy.

EPIDEMIOLOGY

Mild pre-eclampsia occurs in approximately 15% of pregnancies, moderate to severe pre-eclampsia in around 8% and severe pre-eclampsia in about 1-2%.⁵

Eclampsia is a rare but serious complication of pre-eclampsia. It complicates about 1:2000 deliveries in Europe and the developed countries⁶ and from 1:100 to 1:1700 deliveries in developing countries.⁷ The higher incidence in countries with poor economies is thought to result from inadequate antenatal care with late presentation of patients to hospital. In the UK, 38th of

DIAGNOSIS OF PRE-ECLAMPSIA AND SEVERE PRE-ECLAMPSIA

Pre-eclampsia	Severe Pre eclampsia
<p>Hypertension Diastolic blood pressure > 90 mmHG (or) systolic blood pressure > 140 mmHg (or) Rise of > 15 mmHg diastolic or 30 mmHg Systolic blood pressure</p> <p style="text-align: center;">And</p> <p>Proteinuria >300 mg protein/24 hr (or) >1 gm/L in tow random specimens at least 6 hr apart.</p> <p style="text-align: center;">And or</p> <p>Oedema Generalized oedema (>1+) after 12 hr of bed rest (or) Weight gain > 5 pounds in 1 week.</p>	<p>Pre eclampsia + Any of These Complications.</p> <p>Blood Pressure Immediate diagnosis if diastolic blood pressure is > 110 mmHg on two occasion at least 6 hours apart.</p> <p>Proteinura 3-4 + on semi-quantitative urine analysis (or) > 5 gm in a 24 hr urine collection.</p> <p>Pulmonary Oedema Diagnosis of pulmonary oedema or cyanosis.</p> <p>Abdominal pain Epigastric or right upper quadrant pain (liver oedema – lesion capsule stretch/pain)</p> <p>Hepatic Rupture</p> <p>Impaired liver Functions.</p> <p>Cerebral or Visual disturbances Headache, blurred vision, loss of consciousness (including seizures-by definition, eclampsia)</p> <p>OLIGURIA</p> <p>Thrombocytopenia <150,000 platelets/dl</p> <p>HELLP Syndrome Hemolysis, elevated liver enzyme, low platelets</p>

TABLE -1

convulsions occur before the diagnosis of pre-eclampsia is made and 44% occur in the post partum period⁸ and contributes to 10% of maternal deaths.⁹

MORTALITY RISK

Table 2 lists the cause of maternal deaths in the recent maternal mortality study from the UK¹⁰ that were judged to be related to hypertensive disease. Most maternal deaths were due to either cerebral haemorrhage or adult respiratory distress syndrome, and most importantly, care was considered substandard in 24 to 27 cases (89%).

Table 2: causes of maternal deaths related to hypertensive disease in UK (1988-1990).

The most common factor leading to maternal death was delay in treating the patient's disease aggressively. Other lapses in clinical care include poorly controlled blood pressure, delay in delivery, ignoring worsening symptoms and inadequate post partum follow up and management.⁹

PATHOPHYSIOLOGY

Pre-eclampsia is a syndrome, which affects virtually all-maternal organ systems. The underlying pathophysiology is still not fully elucidated but the common pathological feature in the placenta, kidneys and brain is vascular endothelial damage and dysfunctions,¹¹ the major pathological changes are in the placental bed.

CAUSES OF MATERNAL DEATHS RELATED TO HYPERTENSIVE DISEASE IN UK (1998-1990)

Cerebral Haemorrhage	
Intracerebra	10
Subarachnoid	02
Infarction	01
Oedema	01
Total Cerebral Deaths	14
Pulmonary	
Adult respiratory distress syndrome	09
Oedema	01
Total Pulmonary Deaths	10
Hepatic	
Necrosis	01
Other	02
Total Death	27

TABLE - 2

In normal pregnancy, the endothelium, internal elastic lamina and muscular layer of the media of the spiral arteries supplying the placenta is replaced by trophoblast and amorphous matrix containing fibrin.¹¹ These changes transform the vascular supply to a low pressure and high flow system, which meets the needs of the fetus and placenta. Pre-eclampsia is associated with failure of or incomplete trophoblastic invasion of these vessels, resulting in narrowed spiral arteries and subsequent placental ischaemia. The abnormal placenta may release one or more factors which damage vascular endothelial cells throughout the mother (figure 1), leading to multi system dysfunction^{12,13} (fig 2).

CARDIOVASCULAR SYSTEM

Hypertension is an early sign of pre-eclampsia and this may result partly from severe vaso-spasm. Responses to circulating,

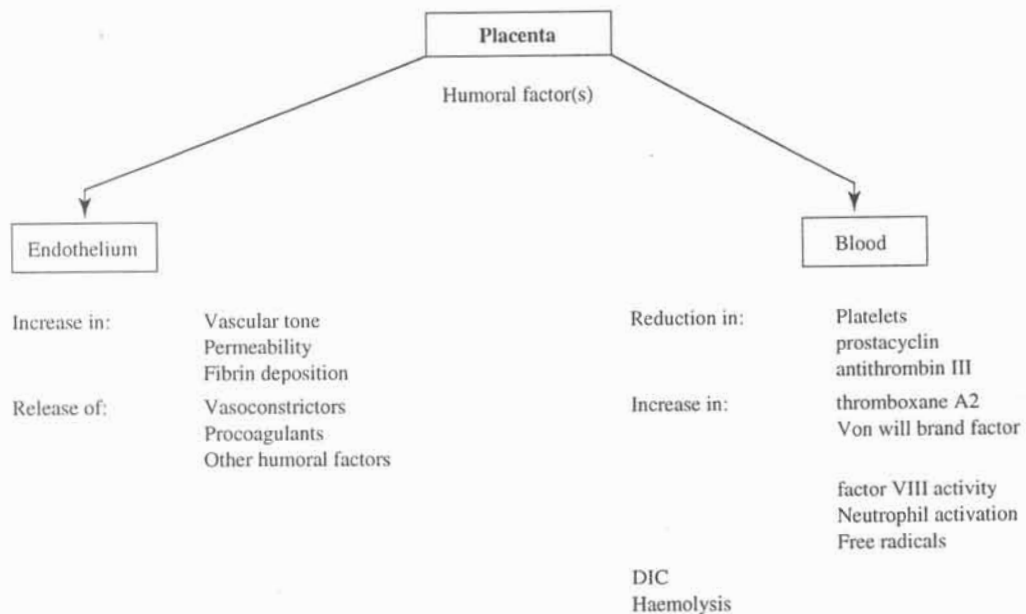


Figure 1: Major initiating changes in the actiology of pre-eclampsia. The primary trigger may be placental ischaemia (DIC= disseminated intravascular coagulation)

MINIMAL TESTS TO BE PERFORMED IN PRE ECLAMPTIC PATIENTS.⁴²

Full blood count including platelet count	
Full coagulation screen	Thrombin time
	Fibrinogen degradation products (F.D.Ps)
	Fibrinogen
Urea, electrolytes, creatinine	
Cross match in delivery by abdominal route is planned or coagulopathy is present.	
Obtain coagulation replacement factors, including platelets, if necessary.	
Liver function tests including serum albumin.	
24 hours urine protein collection.	

TABLE - 3

adrenaline, nor-adrenaline and angiotensim II are exaggerated.¹⁴

The haemodynamic state in pre-eclampsia using both invasive and non-invasive^{15,16,17} techniques is as shown in fig: 2.

The plasma volume is reduced in pre eclampsia and is more obvious clinically in patients with severe disease.¹⁸ However Sibai and colleagues found that the plasma volume was normal in patients with mild disease.¹⁹

Plasma albumin is reduced as a result of loss in urine and loss across the leaky capillaries. Consequently, colloid osmotic pressure (COP) is reduced²⁰ found as 20 mmHg and 15 mmHg in moderate and severe pre eclampsia respectively compared with COP of 22 mmHg in normotensive patients at term by Ovain and colleagues.²¹

RESPIRATORY

Pulmonary oedema is not uncommon in pre eclampsia.²² Sibai found an incidence of 2.9% in patients with pre eclampsia and eclampsia, 70% of the cases developed pulmonary oedema 71 hours after delivery. It occurred more commonly in association

with multiple organ dysfunction than as an isolated complication.

Pulmonary oedema occurs as a result of (a) a low COP in association with increased intravascular hydrostatic pressure and (b) increased capillary permeability.²³ Rackow and colleagues²⁴ studied 17 non-pregnant, critically ill patients with non-cardiogenic pulmonary oedema and found that a COP-PCWP difference of 4 mmHg or less was always associated with pulmonary oedema.

CENTRAL NERVOUS SYSTEM

The pathogenesis of eclampsia continues to be the subject of extensive investigation and speculation. The mechanisms suggested include vasospasm, ischaemia,

ANTI HYPERTENSIVE DRUGS COMMONLY USED IN PRE-ECLAMPSIA.

Hydralazie:	Most effective. Direct vascular smooth muscle dilator, tachycardia, Increased cardiac output, and renal and uterine blood flow enhanced.
Methyldopa:	Central action. Drowsiness, depression, postural hypotension.
Beta Blockers:	Renin suppressors, adverse effects on fetal circulation and fetal glucose.
Labetalol:	Combined alpha and beta blocker. Not extensively studied.
Calcium antagonist's:	Increasing patients are presenting on these drugs. Can cause marked hypotension and may adversely affect the fetus.
Diuretics:	Deplete intravascular volume further and may cause marked. Hypotension should only be used when circulating blood volume is adequate.

TABLE - 4

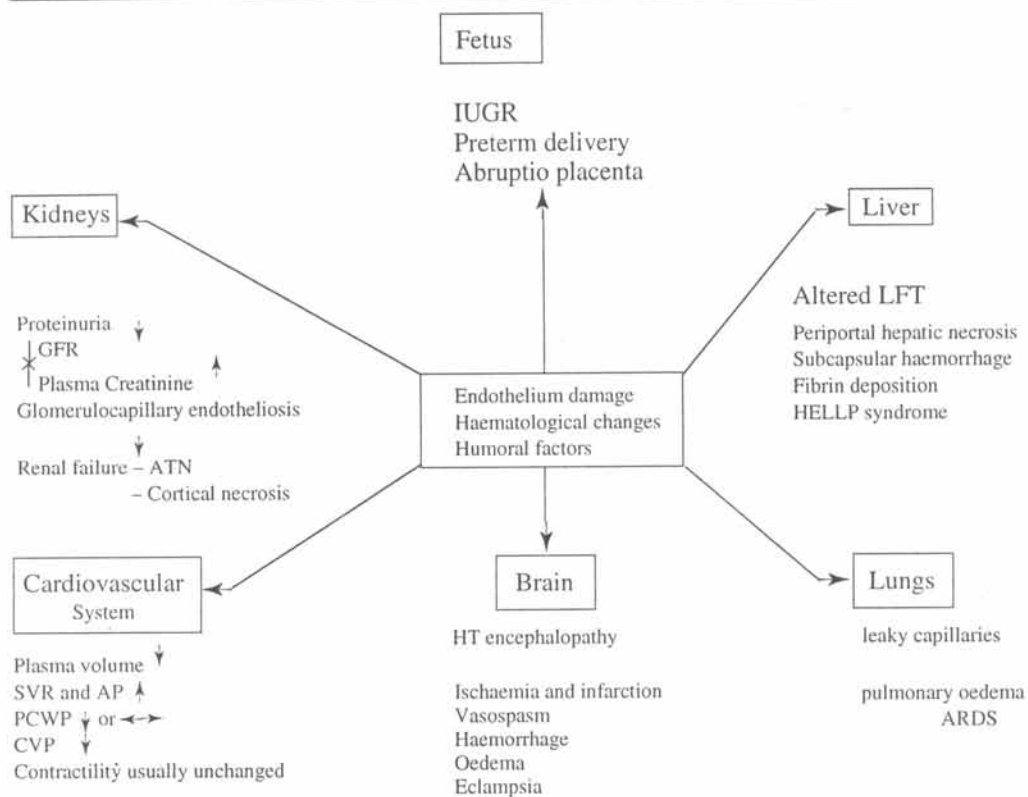


Figure 2: Multisystem changes occurring in pre-eclampsia. (GFR – glomerular filtration rate. ATN = acute tubular necrosis, SVR = systemic vascular resistance, AP = arterial pressure, PCWP = pulmonary capillary wedge pressure, CVP = central venous pressure, IUGR = intrauterine growth retardation. HT = hypertensive, LFT = liver function test, HELLP = haemolysis, elevated liver enzymes and low platelets, ARDS = adult respiratory distress syndromes)

haemorrhage, hypertensive encephalopathy, disseminated intravascular coagulation and cerebral oedema.²⁵ Magnesium sulphate a known cerebral vasodilator controls eclamptic convulsions very effectively and this lends support to the aetiology of cerebral vasospasms.

Diffuse cerebral oedema²⁶ haemorrhages²⁷ and infarcts²⁸ have been demonstrated in patients with eclampsia using computed tomography (CT) scanning. While most recently, magnetic resonance image (MRI) studies of brains of eclamptic women have revealed focal changes characteristic of Ischaemia.²⁹

RENAL

The type of proteinuria in pre eclampsia is non-selective; the increased permeability allows proportionately larger molecular weight proteins such as transferrin and glubulin to appear in the urine than occurs in renal disease with selective proteinuria.³⁰ The renal perfusion and glomerular filtration are reduced with correspondingly elevated serum creatinine concentration due to glomerular capillary endotheliosis. Acute tubular necrosis is often the cause of reversible renal failure with a good prognosis.³¹ Abruptio-placentae, DIC and

hypovolaemia usually precede the renal failure. Cortical necrosis, fortunately uncommon, cause permanent renal failure.

COAGULATION

Thrombocytopenia is a common finding in pre eclampsia, occurring in one third of all patients.³² Fortunately significant thrombocytopenia ($< 100 \times 10^9/\text{litre}$) occurs in only 15% of women with severe disease,³³ caused by increased platelet consumption associated with a low grade DIC.³⁴ Increased levels of fibrin peptide, factor VIIc activity, Von Willebrand factor and reduced antithrombin III are present. Maternal vascular prostacycline is reduced in pre eclampsia and platelet thromboxane α_2 production is increased leading to enhanced platelet activity and vascular damage. Haemolysis may occur especially in association with the abnormal liver function. These changes occur in the form of HELLP syndrome.³⁵

HEPATIC

The cause of hepatic dysfunction is not clear, but it may result from periportal hepatic necrosis, subcapsular haemorrhages or fibrin deposition in hepatic sinusoids. Spontaneous liver rupture is a rare but potentially lethal complication.³⁶ Abnormal liver function tests may be part of the HELLP syndrome.

FETUS

Impairment of placental perfusion caused by placental disease and vasospasm is almost certainly the major reason for the high incidence of fetal loss, intrauterine growth retardation (IUGR), small for dates infants and perinatal mortality. The incidence of respiratory distress syndrome is higher in neonates born to mothers with pre eclampsia. Placental abruption, more com-

mon in pre eclamptics is associated with a high perinatal mortality.

PREDICTION OF PRE ECLAMPSIA

In spite of many clinical, biophysical and biochemical tests that have been reported in the literature to predict the development of pre eclampsia, there is no ideal predictive test that fulfills all desired criteria. The two most important predictive factors remain nulliparity and family history. Pre eclampsia is present in 5.8% of primigravidas but only 0.45 of secundagravidas.³⁸ There is considerable evidence to support a significant genetic contribution to pre eclampsia.³⁹

Doppler ultrasound studies of the deep lying arcuate arteries at 16-18 weeks of gestation may diagnose impaired trophoblastic invasion of the spiral arteries and may have potential in predicting pregnancies destined to become complicated by re eclampsia.⁴⁰

PREVENTION

The aetiology and pathophysiology of pre eclampsia are still not understood fully and this has hindered development of effective preventive measures. So far, preventive strategies have concentrated on areas thought to be involved in the pathogenesis of pre eclampsia, i.e. endothelium and prostaglandins. Anti platelet therapy in the form of low dose aspirin have been tried but the results were conflicting.

Magnesium, zinc, fish oils and calcium supplements have been attempted to reduce the incidence of pre eclampsia but needs further large controlled multicentre trials to assess any beneficial or negative effects of these regimens. Recently it is suggested that pre eclampsia may be prevented by increas-

ing the duration of sexual cohabitation before the first pregnancy with that partner.⁴¹

MANAGEMENT

Management is directed at the maintenance of maternal and fetal well-being, the control of hypertension and the prevention of eclampsia.

HOSPITALIZATION

Pre eclamptic patients are admitted to hospital for careful observation of blood pressure and proteinuria, so that prompt and appropriate therapy can be started as necessary. Prognostic factors indicating disease severity include hyperuricaemia, which presents earlier than proteinuria. Other cause of hyperuricaemia e.g. renal impairment and diuretic drugs should be excluded. Thrombocytopenia also indicates serious disease (platelet count $<100 \times 10^9$ / Litre). Other investigations are listed in table-4.

BED REST

There is no evidence that bed rest is beneficial and in fact pre eclamptic patients are commonly noted to be most hypertensive between midnight and 6 a.m.

Table-III. Minimal tests to be performed in pre eclamptic patients.⁴²

- Full blood count including platelet count
- Full coagulation screen
 - Thrombin time
 - Fibrinogen degradation products (F.D.Ps)
 - Fibrinogen
- Urea, electrolytes, creatinine
- Cross match in delivery by abdominal route is planned or coagulopathy is present.
- Obtain coagulation replacement factors, including platelets, if necessary.

Liver function tests including serum albumin.

24 hours urine protein collection.

CONTROL OF BLOOD PRESSURE

Cerebral haemorrhage is a major cause of maternal deaths (60%) from pre eclampsia or eclampsia.⁹ When mean arterial pressure (MAP) exceeds 140 mmHg (equivalent to 180/120 mmHg), there is a significant risk of maternal cerebral vascular accident (CVA).⁴³

Therefore, it is recommended that arterial pressures of greater than 170/110 mmHg should be treated with urgency with the aim of maintaining pressure below 170/110 but above 130/90 mmHg. The aim should be to prevent intracerebral haemorrhage while not affecting uteroplacental blood flow and maternal renal function. Precipitous reductions in arterial pressure should be avoided by adequate volume expansion before the use of vasodilators.

There are numerous drug regimens available and the most common anti-hypertensives are listed in table-4.

FLUID THERAPY

Pre eclampsia is associated with a reduction in intravascular volume, haemoconcentration and hypoproteinemia but pulmonary oedema occurs in some cases of pre eclampsia and therefore there is some controversy on optimum fluid therapy. It is generally accepted that plasma volume should be corrected by volume expanders before vasodilation with either drugs or extra dural analgesia/anaesthesia. The crystalloid colloid controversy is still debated in the management of pre eclamptic patients, because the condition is complicated by both low COP and leaky capillaries and they are therefore at risk of

developing non cardiogenic pulmonary oedema.²²

Crystalloids may be given at a rate of 1-2 ml/kg/hr and altering subsequent fluid therapy according to the patients clinical condition and urine output in addition to CVP and PCWP measurements where necessary. Infusion of crystalloids alone decreases oncotic pressure further, while the use of colloids may result in high CVP and PCWP values and the onset of pulmonary oedema particularly in the postpartum period. The level of monitoring required should be tailored to each patient. However, adequate hydration and intravascular volume expansion with balanced salt solution is found beneficial.

TREATMENT OF OLIGURIA

The pathogenesis, definition and management of oliguria in severe pre eclampsia is still not well defined. The definitions of oliguria used in clinical practice include a urine output of <30 ml/h for 3 hours, <30 ml/h for 2 hours, <500 ml/24 hours. Many patients with a transient decrease in urine output to <30 ml/hr often resume normal urine output spontaneously.⁴⁵ Poor renal functions in these patients have a good prognosis.³¹ The oliguric patients may be treated with a fluid challenge of 500 ml to 1000 ml crystalloid. If there is no response to a fluid challenge, the CVP should be monitored in the knowledge that this does not always correlate with PCWP when CVP is >6 mmHg.²³ Low dose dopamine infusion 1-5 g/kg/min and nifedipine 10 mg orally every 4 hours have been found to increase urine output without any adverse maternal or fetal side effects.⁴⁶ Repetitive unmonitored fluid administration should be avoided as this may lead to pulmonary oedema specially in the partum period.⁴⁴

MANAGEMENT OF ECLAMPSIA

Because the pathogenesis of eclamptic convulsions is still poorly understood it is not surprisingly that there is disagreement on how to treat the condition.

Diazepam was considered appropriate in the past which is now challenged⁴⁷ chlormethiazole has become less popular due to the need of large volume of fluid administration and over sedation with loss of airway reflexes.

Phenytoin is considered the agent of choice in UK. Magnesium sulphate has been the main choice in the USA for over 60 years and also in south Africa. In the recent collaborative eclampsia trial, mothers allocated to receive MgSO₄ had a significant lower risk of recurrent convulsion than those allocated to either diazepam or phenytoin.⁴⁷ 99% of the units in USA use prophylactic MgSO₄ in all women with pre eclampsia in labour.

In the UK, 16% of units do not prescribe prophylactic anticonvulsants. Prophylactic anticonvulsants are only worth while if the benefits outweighs the harm from side effects.

HELLP SYNDROME

Pritchard and colleagues first described an association of coagulation and liver enzyme abnormalities with pre eclampsia in 1954 and Weinstein in 1982 coined the term HELLP (haemolysis, elevated liver enzymes and low platelets).³⁵

It complicates 0.3% of all pregnancies and between 4 and 20% of those with severe pre eclampsia. Maternal and fetal outcomes are poor with maternal mortality of upto 24% and perinatal mortality of 33%.⁴⁸ Thirty percent of the cases occur in the post partum period. The clinical signs and symptoms include epigastric pain, upper abdominal tenderness, proteinuria, hypertension, jaun-

dice, nausea and vomiting. The disease may progress to haematuria, oliguria, acute tubular necrosis, cortical necrosis and panhypopituitarism. Other complications of HELLP syndrome are DIC, abruptio-placentae, pulmonary oedema, subcapsular liver haematoma, retinal detachment, intra-abdominal bleeding, pleural effusion, ARDS, cerebral oedema and transfusion of blood products. Laboratory investigations reveal thrombocytopenia, abnormal liver function tests, elevated plasma concentrations of haptoglobin and haemolytic anaemia. Sibai suggested that the following standard laboratory values be used to diagnosis HELLP syndrome.¹ haemolysis, defined by abnormal peripheral blood smear,² Increased bilirubin (> 1.2 mg/dl) or increased lactate dehydrogenase (> 600 u/litre),³ Increased serum aspartate aminotransferase (> 70 U/litre),⁴ Low platelet count defined as platelet count $> 100 \times 10^9$ /litre. Aggressive treatment is necessary and delivery should be carried out by caesarean section if necessary.

Vaginal delivery is not contraindicated but fetal thrombocytopenia in 20% of cases may make this hazardous.

Platelet transfusion is recommended if platelet is $< 20 \times 10^9$ /litre for vaginal delivery and $< 50 \times 10^9$ /litre for caesarean section.

A transfusion of fresh whole blood is recommended if the Hb is less than 10 gm/dl. Blood glucose should be monitored to avoid severe hypoglycaemia.

ANAESTHETIC CONSIDERATIONS

Anaesthetic involvement usually start when a decision is made to terminate the pregnancy, either for fetal or for maternal welfare. This includes considerations of the mode of analgesia and anaesthesia, the institution of adequate monitoring and peripartum care.

CHOICE OF ANALGESIA

Provided there is no contra indication epidural analgesia is the method of choice as it also provides cardiovascular stability particularly in the second stage.

Analgesia should be maintained throughout labour, so that the conversion of the block to one that is sufficient to permit surgery can be rapid and the risks of general anaesthesia avoided.

Hypotension should be avoided by adequately correcting the hypovolaemia of the patient and pre-loading with crystalloid solution. Central venous or pulmonary capillary wedge pressures should guide fluid replacement.

When a patient refuses epidural analgesia, despite advice to the contrary, the conventional methods (intra muscular pethidine or pethidine via patient controlled Analgesia or entonox or a combination of these may be used.

Eclampsia itself does not preclude epidural analgesia, local anaesthetic agents pre se do not precipitate eclampsia in a hyper-reflexic patient. Convulsion occur only if an inadvertent intra vascular injection is given.

POST DELIVERY CARE

All severe pre eclamptic patients should be nursed in a high dependency unit. Analgesia can be provided by the epidural route, preferably as a continuous infusion or by bolus.

Epidural narcotics are also eminently suitable for post operative analgesia.

THE ROLE OF SPINAL (SUBARACHNOID) ANAESTHESIA

Subarachnoid blockade is becoming more common for routine caesarean sections. However, in the pre eclamptic patients

the major problem is extensive sudden sympathetic nervous system block with marked maternal hypotension and resultant fetal distress. Therefore, it is not recommended for caesarean section except in mild pre eclampsia. Saddle blockade with its minimal cardiovascular changes is a feasible choice when rapid analgesia for vaginal delivery is required.

GENERAL ANAESTHESIA

Regional anaesthesia is undoubtedly safer for patients with pre eclampsia but general anaesthesia may be necessary when fetal distress requires immediate delivery or On occasions when regional analgesia has either failed or is contra indicated. General anaesthesia in patients with pre eclampsia presents risks to the mother and baby.

The hazards of general anaesthesia include.¹ Potentially difficult intubation, laryngeal Oedema may not become apparent until laryngoscopy² Potential aspiration of gastric contents,³ difficulties related to neuromuscular blockers,⁴ pressure response to laryngoscopy and intubation and⁵ impaired intervillous blood supply.

PROBLEMS WITH INTUBATION

Rapid sequence induction is mandatory. Intubation is more difficult in parturients (later tilt, expanded chest, large tongue, full dentition) and especially, so in those with pre eclampsia as laryngeal oedema may exist. Appropriate skilled anaesthetic help will be required, and a selection of endotracheal tubes and introducers down to small sizes should be available.

Antacid therapy (and H_2 receptor blocker, where possible) should have been administered. As laryngeal oedema may develop during the intra operative period, anaesthetist need to take care when extubating the trachea of these patients.

PRESSOR RESPONSE TO INTUBATION AND EXTUBATION

The pressor responses to laryngoscopy, intubation and extubation in pre eclamptic. Patients may lead to life threatening rises in blood pressure and dysrhythmias. This may increase the risks of cerebrovascular accidents, increase myocardial oxygen requirements, induce pulmonary oedema and reduce uterine blood flow. Prophylactic drugs including lignocaine 1 mg/kg, labetalol 1 mg/kg (given in 10-20 mg boluses), alfentanil 0.05 mg/kg, fentanyl 2.5 g/kg, or a combination of entanyl 200 g and droperidol 5 mg. Esmolol, MgSO₄ 40 g/kg are found effective in attenuating the pressor responses to laryngoscopy, intubation and extubation. Problems with neuromuscular blockers where the mother has received MgSO₄, the non depolarizing neuromuscular blocker are potentiated and they should be used with caution, MgSO₄ inhibits calcium facilitated presynaptic transmitter release. Fasciculations may not occur after administration of suxamethonium to a mother treated with MgSO₄.

Induction and Maintenance of General Anaesthesia

Thiopentone with its specific anti-convulsion and cerebral metabolic depressant effects is the agent of choice for induction. Halothane and Isoflurane are safe when used in appropriate obstetric concentrations for maintenance of anaesthesia. The respiratory depressant effect of opioids may be potentiated by concurrent administration of anti-convulsants and sedatives.

Monitoring: The level of maternal and fetal monitoring should be tailored to the severity of the disease. Central venous pressure and intra arterial pressure should be monitored in severe pre eclampsia.

A swan ganz catheter may be needed if cerebral oedema or cardiac failure coexist.

Urine output and conscious level must be recorded pre-operatively.

POST DELIVERY MANAGEMENT

Post delivery problems are common in patients with severe pre eclampsia, regardless of the mode of delivery and anaesthesia. Continued attention must be paid to airway management and prevention of aspiration, anti hypertensive therapy, anticonvulsant therapy, careful fluid balance, assessment and maintenance of renal function.

Following general anaesthesia, elective ventilation ensures adequate oxygenation while allowing time for other organ systems to stabilize before extubation. Other important factors to consider in post delivery management are as follow.

Hypoproteinaemia, may be corrected by infusion of 20% albumin solution to maintain adequate CVP (5-10 cm of H₂O), clotting problem need adequate haematological advice, infusino of FFP, platelet or individual clotting factors be based on the coagulation studies.

Eclampsia: When convulsions occur a department protocol should be put into practice. A "crash team" of anaesthetists, obstetricians and mid wives should immediately be called, who will carry out the simple resuscitative measures of airway maintenance and oxygen therapy, while administering the necessary anti-convulsant, anti hypertensive, diuretic and steroid therapy. Once convulsions have been controlled, the mode of fetal, delivery must be decided while maternal treatment continues. If convulsions persist, and the fetus is alive, then emergency caesarean section with elective postoperative ventilation in the intensive care unit is required. Eclampsia can occur upto 48 hours post natally in 30% of patients.

CONCLUSION

Hypertensive disease in pregnancy is the most common cause of death in parturition. The aetiology is not yet fully understood. It is however, a multisystem disorder which may progress to the failure of numerous maternal systems, and ultimately to death. As the pathophysiological changes become better understood and clinicians become mor aware of these changes, it is to be hoped that the outcome will improve.

Ideally, epidural analgesia is the mode of choice for both operative and vaginal delivery. However, certain patients will present for general anaesthesia. The need for adequate assessment, investigation, monitoring, and intensive care for these patients, ante, peri and post partum cannot be over emphasized and the necessity for pooled knowledge and team work has to be stressed.

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