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Pregnancy induced hypertension--anaesthetic considerations

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Pregnancy Induced Hypertension - Anaesthetic Considerations

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The American College of Obstetrician and Gynecologists have classified the hypertensive disorders of pregnancy into the following four categories:

1. Pre-eclampsia or pregnancy induced hypertension (PIE) is defined as the triad of hypertension, proteinuria and generalized oedema, developing after the 20th week of gestation.

2. Chronic hypertension is the presence of sustained hypertension prior to pregnancy and continuing there into.

3. Superimposed pre-eclampsia implies the development of increased blood pressure, proteinuria, and/or oedema in a gravida with chronic hypertension.

4. Transient gestational hypertension, refers to development of hypertension without proteinuria or oederna in a previously normotensive gravida followed by return to normotension within 10 days postpartum.

Pre-eclampsia remains the most frequent, most serious and least understood of these disorders. The terminology used to define pre-eclampsia is confusing and atleast eight terntsare used in obstetric literature today[1]. Pre-eclampsia is a disease unique to human pregnancy;predominantly affects the young multiparas. (Frequency is 5 times higher in mothers of less than 20 years, compared to older than 20 years)[2] usually occurs after the 20th week of gestation (may appear earlier in trophoblastic diseases); the prevalence is 2.5-7% of all the pregnancies in USA and accounts for atleast 20-40% maternal mortality recorded in U.S.A. and Englan[3,4].

Pre-eclampsia is manifested by hypertension, proteinuria and oedema. Oedema is nota reliable sign[1]. Pre-eclampsia can be mild, moderate and severe. In severe pre-eclampsia, more than one of the following must exist:

1. Systolic B.P. >160 torr; DBP >110; MBP >120 ton.

2. Proteinuria in excess of 5 g/24 hours (+3->+4 by dipstick test).

3. Oliguna <500 ml/24 hours.

4. Headache, visual disturbances, cerebral disturbances.

5. Epigastnc pain.

6. HELLP syndrome (haemolysis; elevated liver enzymes; low platelets).

When grandmal convulsions occur in pre-eclampsatic patients, the syndrome is called eclampsia.

1. Pathophysiology

The cause of this multisystemic disorder is yet to be elucidated. Whether the initiating factor of this disorder is immunological, genetic or simply a decrease in uterine blood flow, is unknown. One proposed theory is immunological rejection of fetal tissues by the mother which causes placcental vasculitis and ischaemia. It appears that there are abnormalities in production and effects of circulating vasoconstricting (angiotensin and thromboxane) and vasodilating (PGE2; prostacyclin and EDRF) substances, perhaps due to widespread endothelial (immunoreactive) damage[5]. The abnormal changes in these hormonal levels produce generalized artenolar vasoreactivity (spasm), retention of salt and water, and altered coagulation profile (Figure).
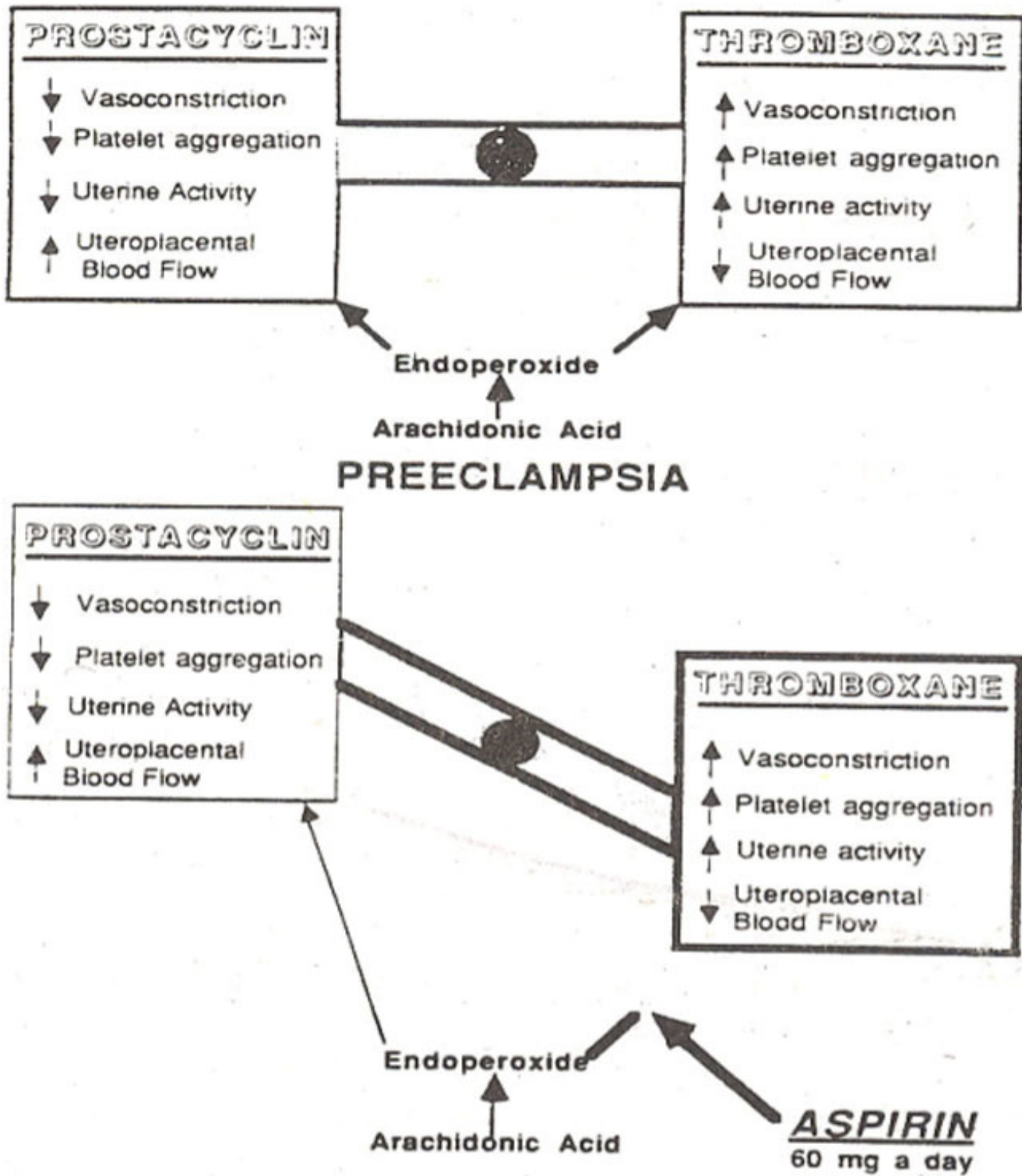
PROSTACYCLIN

- ↓ Vasoconstriction
- ↓ Platelet aggregation
- ↓ Uterine Activity
- ↑ Uteroplacental Blood Flow

THROMBOXANE

- ↑ Vasoconstriction
- ↑ Platelet aggregation
- ↑ Uterine activity
- ↓ Uteroplacental Blood Flow

Endoperoxide

Arachidonic Acid

PREECLAMPSIA

PROSTACYCLIN

- ↓ Vasoconstriction
- ↓ Platelet aggregation
- ↓ Uterine Activity
- ↑ Uteroplacental Blood Flow

THROMBOXANE

- ↑ Vasoconstriction
- ↑ Platelet aggregation
- ↑ Uterine activity
- ↓ Uteroplacental Blood Flow

Endoperoxide

Arachidonic Acid

ASPIRIN
60 mg a day

Figure. Anaesthetic considerations in preeclampsia-eclampsia.

II. General management

The management is symptomatic, but the definite therapy is delivery of fetus and placenta. An early deliveiy has been stressed as crucial in avoiding serious sequelae. The main aim of management should

be directed towards minimizing vasospasm, improving circulation to uterus, placenta and kidneys, improving the intravascular volume, correcting the acid base and electrolytes imbalance and above all decreasing reflex hyperactivity and central nervous system activity.

1. Bed rest: Hospitalization and bed rest in lateral decubitus position is strongly advised.

2. Hydration: Adequate hydration and intravascular volume expansion with balanced salt solution is beneficial[6]. Sodium intake should be adequate as salt restriction may result in increased production of renin, angiotensin and aldosterone. The use of thiazide diuretics should be discouraged[7].

3. Anti convulsant therapy: Convulsions in these patients could produce deleterious effects on the mother and fetus. Commonly used anticonvulsant drugs are Benzodiazepines, Phenytoin and Magnesium Sulphate.

a. Benzodiazepines

Benzodiazepines are the first line of treatment to control convulsions in eclamptic patients. The dose for Diazepam is 5-10 mg increments until desired effects are obtained. Prophylacitically Intravenous (fly) infusion of 10 mg/hour of Diazepam can be used. Excessive sedation and airway management problems for the mother, while flaccidity, hypothermia, severe respiratory depression, low apgar score are the major disadvantages for the neonate.

b. Phenytoin

Phenytoin is gaining popularity in place of Diazepam due to lack of serious sedative side effects[8]. The therapeutic level is 40- 100 micro molll; which could be achieved by a loading dose of 10 mg/kg in 100 ml saline infused intravenously at a rate of 50 mg/mm (7 ml/min in a 70 kg patient), followed by a second bolus at 5 mg/kg, 2 hours later. Maintenance therapy is commenced 12 hours after the second bolus, at a rate of 200 mg/S hourly, I.V or orally.

c. Magnesium Sulphate (Mg SO$_4$)

The agent is used for anti-convulsant and uterine vasodilatory effects. It is effective centrally and peripherally[9]. Magnesium Sulphate affects the neuromuscular junction by inhibiting the presy naptic calcium-facilitated transmitter release. The patients receiving $MgSO_4$ are sensitive to Peuromuscular drugs especially non-depolarizing blockers[10]. The enhanced effects of Succiny lcholine is due to deficiency in plasma pseudocholinesterase rather than $MgSo_{411}$. Magnessium sulphate decreases peripheral resistance, increases cardiacoutput[12] atemporaxy decrease in blood pressure which is offset by an increase in heart rate.

The therapeutic level is 4-6 m Eq/L which can be monitored by deep tendon reflexes and blood levels. Dose is 40-80 mg/kg followed by an infusion of 2 g/hour. In severe pre-eclampsia for the seizure prophylaxis magnesium sulphate appears to be a better choice when compared with a drug such as Phenytoin, as magnesium is an effective vasodilator even of cerebral vessels during cerebral vasospasm, while Phenytoin acts primarily by suppressing electrical activity[13]. In another study the best pennatal outcome was established with diphenylhydantion[14].

4. Antihypertensive drugs

Commonly used drugs are Hydralazine, beta-blockers (Propranolol, Atenolol), Ca-Channel blocker (Vempamil, Nifedipine) Nitrates (Nitroglycerin), Labetalol (alpha and mainly beta blocker with predominantly beta effects), Sodium Nitroprusside.

a. Hydralazine

The primary actions of Hydralazine is to decrease precapillary arteriolar resistance. It increases cardiac output and causes reflex tachycardia. The renal blood flow is also increased. Dose is 5 mg bolus increments till the desired effects are obtained or a maximum dose of 40 mg has been administered. It is followed by a continuous IN infusion of 5-20 mg/hour(50-350 microgram/min). Maximum effect is in 20-30 minutes after I.V injection and duration is 2-3 hours. As the onset of action is slow, so

repeated dosage should not be given at less than 20 minutes intervals. If sufficient time is not allowed between injections severe hypotension may ensure[15].

b. Methylodopa

Methylodopa is one of the most commonly used drug in pregnancy induced hypertension. Improved fetal outcome has been reported in various studiesJt does cross from maternal to umbilical plasnta but does not cause any ill effects on the fetal heart rate patterns orfetal peripheral resistance. It is used in 1-3 G/day dosage. Over dosage will cause drowsiness, depression and postural hypotension.

c. Calcium Antagonists

1. Nifedipine 2. Verapamil

1. Nifedipine

Nifedipine is a calcium channel blocker and is effective as a safe antihypertensive agent in pregnancy particularly in Toxaemia of pregnancy[16]. Nifedipine causes a decrease in blood pressure. It improves uterine blood flow and fetal oxygenation[17], increases renal blood flow[18] and urinary output. Nifedipine is a potent uterine muscle relaxant so is useful as tocolytic agent, but could calese post-partum haernorrhage. Dosage is 10mg sublingual Nifediipin every 2 20 minutes till 30 mg is given. Also it can be used oralty. After oral administration peak effect is in 1-2 hours, witla plasma half life of 5 hours[19]. A combination of calcium chan blockers and $MgSo_4$ should be used very cautiously, as hypotension respiratory difficulty and cardiac toxicity has been reported[20].

2. Verapamil

When Verapamil was administered as 5-10 mg/hr via I/V infusion in patients with pregnancy induced hypertension, after fluid repletion to a pulmonary wedge pressure of 14-16 nun Hg, showed a reduction in blood pressure in a controlled manner. Both systemic and pulmonary resistance fell but cardiac output was maintained without any significant side-effects on mother and fetus[21].

d. Beta-Blockers

The use of beta-blockers during pregnancy is a controversial issue. The non-selective beta-blocker are feared to be associated with increased uterine activity, decreased uterine and placental blood flow, decreased fetal heart rate and decreased fetal tolerance to hypoxia, unacceptable levels of intrauterine growth retardation and fetal mortality.

Chronic use of cardio-selective beta-blockers, such as Atenolol, appears to be effective to treat moderate to severe hypertension during pregnancy. A daily dose of 50 mg to a maximum 150 mg/day when used after 24-3 1 week of pregnancy, resulted in a better control of blood pressure with mild adverse side effects to mothers and new borns[22]. In anotherprospective randomized and double blind study, when Atenolol was used upto a maximum of 200 mg/day, starting from the end of the first trimester, the drug was associated with significant lower birth weight[23].

c. Sodium Nitroprusside and Nitroglycerine

These drugs are to be used only in acute malignant hypertension (hypertensive emergencies). They might be useful at time of induction and intubation for a rapid control of blood pressure. Esmolol (0.5 mg/kg bolus followed by 0.3 mg/kg/min) or Labetalol 5 mg incremental bolus dosages till 1-2 mg/kg also have been administered with success mainly at induction and intubation times[24].

III. Anaesthetic considerations

One should keep in mind the following points while assessing/managing a patient inlaborwith severe pre-eclampsia.

1. Cardiovascular systems

There is reduced circulating blood volume with haemoconcentration which will mask the presence of anaemia. The volume depletion may lead to an exaggeration of the response to aortocaval compression, therefore, left uterine displacement must be employed. Intravenous fluid loading under the continuous monitoring of CVP or pulmonary capillary wedge pressure and urine output is necessary before the

conduction of regional or general anaesthesia. There is still controversy about the type of fluids (crystalloid versus colloid) for preloading and replacement. Volume loading should be done with caution as these patients have leaking capillaries, decreased oncotic pressures[25] and reduced ventricular compliance. Nisell et al[26] examined the effects of acute plasma volume expansion on atrial nathuretic peplide concentration in 16 pre-eclamptic patients and compared with 16 healthy control patients. Atrial natriuretic peptide (ANP) has diuretic smooth muscle relaxing and hypotensive properties and is known to reduce blood volume by increasing capillary permeability. As a result of volume load this peptide is secreted. There is an enhanced response to ANP in preeclamptic patients, also there is an increased level in preeclamptic patients. AMP is slowiy cleared owing to poor renal function and endothelial damage. The increased level of ANP may further exacerbate the capillary leak enhanced fluid loss and protein into extravascular space. The data strongly suggests against raising the right atrial pressure to "normal levels". Woods et al[27] found a six-fold increase in incidence of CCF and four-fold increase of acute respiratory distress syndrome within 24 hours postoperatively in the pre-eclamptic patients as a result of over enthusiastic fluid loading. Rapid infusion of crystalloid fluid may significantly lower the colloid oncotic pressure for 24 hours[28], which may not be tolerated by these patients and end up in pulmonary ocdema[29]. 500-1000 ml of 5% Albumin in severe pre- eclamptics increases cardiac output, decreases systemic vascular resistance with no or minimal effects on the mean arterial blood pressure[30]. The colloidal solution (plasma protein fraction or Albumin 5%) seems to be a preferable choice in severe preeclamptic patients. Both fluid regimes have their proponents and opponents. Crystalloid fluids should be without Dextrose. A balanced crystalloid solution or 0.9% Nacl solution upto a volume load of 500 ml-l000 ml is well tolerated in these patients with epidural analgesia upto T10 level. A combination of colloid/crystalloid could be a better choice in these patients.

2. Laryngeal oedcma
Retention of sodium and waterand exaggerate effects of normal pregnancy hormones lead to the development of laryngeal oedema in pre-eclamptic patient. This could cause intubation and post-operative ainvay management difficulties.

3. Coagulopathy
In the severe pre-eclamptic/eclamptic patients, coagulation disorder might be markedly present. At admission it is necessary to obtain a complete blood picture with platelets count, followed by serial platelet counts when monitoring intra-partum coagulation indices. Evaluation of PT, PTT and flbrinogcn to be added only if platelet count is less than 100.000 per mm3[31]. A rapid change in platelets count is a more significant event rather than an absolute value[32]. Platelet functions, rather than number is more important[33], but tests (Thromboelastography) to check the function are time consuming and expensive. About 10 to 25% of pre-eclamptic patients with normal platelet counts have prolonged bleeding time[33,34].

Schindler et al[34] recommended that bleeding times should be performed on all patients with platelet count between 50,000- 100,000/mm^3 prior to invasive regional techniques but some feel that bleeding time is not a good indicator for coagulation disorders. Fibrin D-Dimer analysis has been proposed as a more sensitive and early indicator on intravascular coagulation and fibrinolysis than standard coagu lation tests[35]. Only if significant coagulation disorderexist with a platelet count falling under 100,000 mm^3. a prolonged bleeding time or an abnormal TEG (Thromboelastography) then regional anaesthesia is relatively contraindicated[36]

Epidural is absolutely contraindicated when platelets are less than 50,000 mm^3. While when platelets are between 50,000 to 1,00,000 per mm^3 risk benefit balance should be assessed as possibility toepidural haematoma incidence is 1:10,000 cases. There are yet no clear guidelines foracceptable safe limits but a level of platelets >100,000 mm^3 or a bleeding time of<10 minutes is commonly used[37]. In

the presence of coagulopathy
significant bleeding can occur during laryngoscopy for oral intubation. Attempts should be very gentle and careful.

4. Drugs interactions

There could be significant effects on the mother's cardiovascular, muscular, CNS and other systems, due to the interactions between therapeutic drugs and anaesthetic drugs/techniques.

a. Magnesium sulphate and muscle relaxants

The interactions between magnesium sulphate and muscle relaxants is well documented. The reduction in the dosage and a continuous monitoring of neuromuscular function with the help of peripheral nerve stimulator is advisable. b. Magnesium sulphate and regional anaesthesia

The neuromuscular weakness and potential hypotensive effects of magnesium sulphate might be slightly exaggerated with sympathetic blockade, but the protective and beneficial effects on the uterine and umbilical blood flow due to epidural anaesthesia eliminates the increased fetal risk[21], so combination is assumed to be safe. Subarachnoid block for higher level is not widely recommended due to rapid onset of sympathetic block and risk of severe hypotension[38,39]

c. Use of low dose aspirin and epidurals

The use of low dose aspirin (60 mg/day after the 12th week of pregnancy is based on the rationale that at this dosage aspirin produced an inhibition of lipid peroxide and thromboxane production but not prostacyclin production, thus protecting against both vasoconstriction and pathological clotting). Concerns have been expressed about placing epidurals in patients receiving aspirin as their antiplatelet actions might increase risk of epidural haematoma. A large multicentral[40] randomized placebo control trial to evaluate the effects of low dose aspirin to prevent and treat pre-eclampsia failed to show any significant adverse effects on coagulation profile of these patients. There is also no data to recommend routine bleeding tests on these patients as bleeding time does into indicate the risk of bleeding. Orlikowsk et al[41] noted that chronic use of low dose of Aspirin does not seem to be associated with TEG (Thromboelastography) changes.

5. Antihypertcnsive drugs and anaesthetic drugs When anaesthetic drugs are administered to the patients who are on antihypertensive treatment cardiovascular depression may be more pronounced and marked. The careful choice of anaesthetic drugs and dosage is mandatory. There is a need for titration of the dose of anaesthetic drugs.

IV. Hyperexaggerated Response

There is increased vascular reactivity which results man intensified response such as tachvcardia, severe hypertension, due to the release of catecholamines. This particular problem is apparent during with laryngoscopy, in light general anaesthesia and at extubation. Such sudden rises in blood pressure may increase the risk of cerebral haemorrhage and oedema, myocardial infarction, pulmonary oedema or cardiac failure[39]. Techniques and drugs should be used to obtain or completely abolish these responses. The general recommendations are to use drugs such as short and rapidly acting anti hypertensives, xylocaine intravenously or high doses of narcotics, before induction and laryngoscopy. The stress responses to laryngoscopy could be obtunded by nitroprusside 0.1 to 3 micrograms/kg/min, just prior to induction[42]. Labetalol 20 mg given intravenously foliwed by 10 mg increments to a total dose of 1 mg/kg resulted, in reduction in mean artery blood pressure and heart rate and the cardiovascular response to laiyngoscopy and intubation were reduced.

Nitroglycerine 0.1 micrograms/kg/min was not effective in preventing the hypertensive responses to laryngoscopy while Esomolol could prevent the hypertensive response but has caused fetal hypoxemia in animal studies[43]. Fentanyl I micrograms/kg or Alfantanil 10 micrograms/kg given at induction attenuated these responses but did not abolish them completely[44].

V. Pre-anaesthetic management/monitoring

Before any procedure is undertaken, it is mandatory to optimize the condition of the patient. We should try to control the blood pressure, restore blood volume, improve renal function, institute anticonvulsant therapy (if indicated) and initiate proper monitoring. The minimal monitoring employed should be blood pressure by a non-invasive method, heart rate and urine output measurements. Direct intra-arterial blood pressure monitoring is indicated in patients with severe pre-eclampsia or eclampsia. Central venous pressure monitoring is indicated in severe pre- eclamptic and eclamptic patients as a minimal invasive monitoring. They have clearly reduced blood volume and are fluid deficient. Volume loading is often necessary, which could cause pulmonary oedema. Some anaesthesiologists recommended the use of pulmonary arter catheter (P.A.C) for effective management of patients. Informations gathered from P.A.C. can be used to institute therapy such as fluid challenges, potent vasodilator infusion and beta adzcnergic blockade to optimize the patients haemodynamic status[45]. P.A.C. is also indicated in pre- eclarnptic/eclampsia patients with unresponsive oliguria, pulmonary oedema and in those cases where epidural anaesthesia is contemplated. However, some workers do not think that pulmonary catheterization is strongly indicated and useful[46]. Electronic monitoring of fetal heart rate and uterine contractions is essential, when labour is artificially induced or epidural analgesia is instituted. Fetal blood analysis for PH may be indicated when significant fetal heart rate abnormalities occur.

VI. Choice of Analgesia

Choice of analgesia will depend on obstetrical situation, the status of the patient and the condition of the fetus (if still in utero). Narcotics provide good analgesia but have no anticonvulsant and antihypertensive properties. Tranquilizers in combination with narcotics are in use and have variable success with significant side effects. Patient controlled analgesia (PCA) with Fentanyl (loading dose of 50-100 micrograms followed by 25-50 micrograms hourly) or Meperidine (25-50 mg followed by 10-15 mg/hour) may be used. A mixture of N20 and ox-ygen(50:50%) provides good analgesia and increases oxygenation. This combination is effective during the late first stage and at delivery time. Addition of 0.5-0.75% Enflurane or 0.3-0.5% Isoflurane or 0.5% Halothane to N20 plus 02 admixture could produce the effective desired results for delivery. Local infiltration of perineum or Pudendal nerve block provides satisfactory analgesia for most forceps deliveries.

Epidural analgesia

The use of continuous lumbar epidural analgesia in severe pre- eclamptics/eclamptics whose convulsions are under control is widely accepted and recommended[45,47-49] unless contraindicated due to gross coagulation abnormalities, or marked untreated hype- volumia, or maternal septicemia. Properly conducted epidural analgesia for labourand delivery, decreases maternal oxygen requirenients, prevents maternal hyperventilation, significantly decreases circulating levels of Epinephrine[50]: increases intervillous blood flow[51]; appears to protect against eclamptic convulsions[52]; chances of pulmonary aspiration are minimized and stabilizes the blood pressure at modestly lower levels. All these effects cause an improvement in mother's clinical status; moral attitude and fetal well being. Apgar score in neonates born to mothers with Epidural was far better than non-epidural group. Concerns expressed by others are that epidural analgesia should not be used in severe pre-eclamptic patients[53]. Sudden hypotension which could be deleterious to already compromised fetus due to further decreases in uteroplacental blood flow[54]. Also anaesthetics used could decrease fetal beat-to-beat variability and impede the diagnosis of fetal hypoxia and distress[55]. Due to cOagulopathy, in pre-cclamptic patients formation of epidural haematoma is a possibility during invasive regional techniques.

Studies have shown that. properly conducted epidural analgesia/anaesthesia does not cause significant hypotension or fetal distress[56]. The decrease in blood pressure is comparable with non pre-eclamptic normal patients[57,58]. A sequential technique, of raising the block in stages. rather than in a bolus is the

generally favoured approach. Ephedrine is the drug of choice for hypotension[59]. Animal and human studies, including studies in pre-eclamptic parturients showed that intervillous blood flow was maintained or improved with the use of epidural analgesia[61,60]. Anecdotal reports of epidural or subarachnoid block inadvently administered to patients with platelets counts <100,000 mm^3 have not described any epidural haernatorna as a consequence[61]. Epidural block administered before delivery in pre-eclamptic patients have not resulted in epidural haematoma even if the platelet numbers have decreased below 50,000 mm^3 after delivery. It is advisable to leave the catheter in place till the coagulation profile including platelets are within normal acceptable limits.

The use of adrenaline in epidural space is controversial. Administration of adrenaline mixed local anaesthetic could cause severe hypertensive response in patients with preeclampsia/eclampsia, due to increased sensitivity to catecho lamine. However, others[62] think adrenaline is safe as only B-adrenergic effects are seen. Lidocaine does cause a slight decrease in fetal heart beat to beat variability, while Bupivacinc use is not associated with decrease in fetal beat to beat variability[63]. Ideally, in patients with severe pre- eclampsia institution of epidural analgesia should be proceeded by placing a CVP or Pulmonary Artery Catheter[64]. Such patients should receive a balance salt solution and/orcolloid until CVP is +3/+4 ems of water or pulmonary capillary wedge pressure is 5- 12 mmHg[65].

In view of the vast favourable clinical experience and recent investigational work, the evidence supports the use of epidural analgesia for labor, vaginal delivery and Cesarean section in most mothers with severe pre-eclampsia or controlled eclarnpsia[59].

General anaesthesia

While planning for general anaesthesia, the hazards and possible pmblems which could be encountered in severe pre-cclamptic patients should be kept in mind. The major hazards are difficulties in airway maintenance, excessive and aggregated response to laiyngoscopy and endotracheal intubation. interaction of anaesthetic drugs with drugs used by obstetrician particularly magnesium sulphate. Despite various hazards, some authorities feel that risks of rneral anaesthesia are less than those of regional anaesthesia[66] while others feel that general anaesthesia has markedly increased risks[67].

Operation of the patient for general anaesthesia

a) P re-induction period

The earlier involvement of the anaesthesiologist is very important. The clinical condition of the patient should be optimized before surgery. Blood pressure and convulsions should be properly controlled. All antihypertensive and anticonvulsant drugs to be continued till patient is in the operating room. It is advisable to bring patient to the operating room in the lateral position and have oxygen via face mask during transportation. The usual doses of antiemetics (Metocloprarnide 10 rug IM) and antacids (Sodium Citrate 0.30 mmol, 30 ml) or a H2 histamine receptor antagonist (Ranitidinc or Cimetidine or Famotidine) be given orally 1-2 hours prior or 40-60 minutes earlier intravenously.

b) Induction period

In case of severe fetal distress or compromise, intravenous induction with agents such as Thiopentone sodium or Propofol is likely to reduce placental perfusion with further detrimental effects to the fetus. Ketamine is contraindicated while Etomidate has been condemned for undesirable effects on adrenocortical axis, but is a relatively cardio stable drug. There may be ajustification for the use ofbenzodiazepines for induction especially, when a specific benzodiazepine receptor antagonist (Flumazenil) is available. The exaggerated pressor responses to laryngoscopy and intubation could be attenuated by various techniques using drugs such a Hydralazine, Labetalol or Sodium Nitroprusside. A rapid sequence technique should be used to secure the airway.

Intra-operative l)CflOd

Institution of LPPV can adversely affect the uterine blood flow by reducing cardiac output especially in dehydrated or volume depleted patients. These patients should be kept normocapnic or somewhat

hypcrcapnic to improved placental blood flow and decrease shunting. Hyperventilation should be avoided as it will lead to deterioration in uterine/placental blood flow due to decrease in cardiac output, vasoconstnction and respiratory alkalosis which will shift the oxygen dissociation curve to the left. It is well known that Nitrous Oxide has low lipid solubility and it has relatively rapid transplacental passage which can cause some degree of neonatal depression during C-section. There is probably a slight but definite real risk that the already hypoxic infant may be adversely affected by this agent. However, if an unacceptable risk of maternal awareness at operation is to be avoided due to low nitrous-oxide concentration or no nitrous oxide imposed by a high FIo2, additional vapor (inhalational) supplementation will be required. In general the available evidence suggest that Halothane in a concentration of 1.0 MAC or less has little or no effect on placental perfusion or fetal well-being, while higher concentration result in deterioration[68].

Various studies[50,69] failed to show any adverse effects due to Halothane or Enflurane on the fetus as determined by neonatal apgar score, acid-base status or earlier neuro-behavioral score when compared with the other group which reccivedoxygen/nitrous oxide maintenance. Also 0.5%orless Halothane is not associated with increased blood loss. Enflurane in low concentration is devoid of any adverse effects on neonates when used as a supplementary agent in subanaesthetic concentrations during labour[50,69] Isoflurane has gained popularity due to properties of low blood solubility, apparent freedom from hepatic and renal toxicity and relative cardiovascular stability. At equipotent concentrations it's effects on uterine contractility is similar to Halothane and Enflurane[70], while maintaining normal maternal and neonatal conditions. In another study, Isoflurane 0.75%was found to be preferable to 0.5% halothane during cesarean section because of more rapid recovery, less bleeding and less uterine relaxation. Intra-operative muscle relaxation could be maintained by Suxamethonium infusion (4 mg/min or small boluses of a depolarizing agent in incremental doses, e.g., Vecuronium (1-2 mg) or Atracrium (10-15 mg) adjusted as necessary using a nerve stimulator.

Extubation

The cardiovascular changes due to extubation could be as severe as pressor responses to intubation. At this moment, neither narcotics due to their respiratory depressant effects nor Magnesium Sulphate due to its neuromuscular blocking properties are likely to be ideal. The idea of extubating these patients in the deeper planes should be addressed very carefully due to high potential for airway management difficulties and aspiration. There is sonic role for vasodilators or beta-blockers especially short acting such as Esmalol at this stage.

Post-operative care

The delivery of the fetus and placenta is the treatment but the full recovery may take 10 days to 2 weeks. The airway management may be more difficult in the postoperative period due to the worsened laryngeal oedema situation. Respiratory embarrassment may be severe and airway maintenance may be impossible without anendotrachealtube. The antihypertensive and anticonvulsant drugs should be continued in the postoperative period as long as indicated. It is very important to keep these patients pain free with good analgesia. Epidural analgesia is an ideal choice, if epidural catheter is in place. These patients should be closely and maximally nionitored (both invasively and non-invasively) in the Intensive Care Unit at least for the first 24 hours.

Key points for anaesthetic considerations

1) Essential monitoring:
a. Non-invasive monitoring. e.g.. ECG. pulse rate, blood pressure, pulse oximetrv. urine output.
b. Invasive monitoring, e.g., CVP. Swan Ganz Catheter, intraarterial blood pressure.
2) Prevention and control of convulsions by use of Magncsium Sulphate, Diazepam and Phenytoin.
3) Optimal stabilization of cardiovascular system by careful fluid balance and use of vasodilators.
4) Proper pre-anaesthetic evaluation of various systems with specific attention towards cardiovascular, renal coagulation and hepatic system.

5) Anaesthetic choices:

a. Regional anaesthesia

A carefully administered epidural blockade is strongly advised, unless gross contraindications exists. Epidural is absolutely contraindicated when platelets are less than 50,000/mm3. while between 50.000-100,000/mm3, the risk balance should be assessed as possibility of haematoma incidence is 1:10,000 cases.

Subarachnoid block is not widely recommended due to the risk of sudden and severe hypotension.

b. General anaesthesia

It requires skilled ainvay management. control of pressor response to laryngoscopv and intubation, awareness of the potential drugs interactions and careful postoperative management.

6) Avoidance of certain drugs i.e.. Methoxyflurane, Ketamine and Methergin: reduction in dosage of Vasopressors when needed; prophylactic use of Ephedrine is not recommended and titration of muscle relaxant drugs.

7) Proper management in the I.C.U. in the postoperative period. Anticonvulsant and antihypertensive drugs to be continued as indicated. Good analgesia could be provided via the epidural route.

8) The successful management of these patients depends upon the team work and communications of obstetrician, mid-wife and anaesthcsiologisL all of whom have iniportant roles to play. While (lie majority of patients require minimal intervention, severe pre-ectamptic patients need expert care with excess to invasive monitoring and therapy in the intensive care units.

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