Preeclampsia/Eclampsia: An Insight into the Dilemma of Treatment by the Anesthesiologist

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Abstract- A complicated and controversial subject in obstetrics i.e., toxemia of pregnancy is looked upon, both from an anesthesiological and obstetrical point of view. As pre-eclampsia and eclampsia involve immediate treatment and obstetric considerations, the choice between epidural and general anesthesia becomes necessary when cesarean section is contemplated. Apart from the pathophysiology of the vessel spasm as it is induced by preeclampsia, the therapeutic managements of fluid administration, the drugs of choice to treat hypertension as well as the technical aspects of anesthesia are reviewed.

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Introduction

Preeclampsia is defined as the association of pregnancy-induced hypertension with proteinuria of greater than or equal to 300 mg/24 h after 20 weeks of gestation (1). It is a severe complication of pregnancy leading to fetal morbidity and mortality and has been reported to complicate 4-7 % of all pregnancies (2). The American college of obstetricians and gynecologists characterized preeclampsia as the development of hypertension with proteinuria, edema or both (the traditional triad) induced by pregnancy after the 20th week of gestation (1,2). Controversies in regard to definition stem from the early signs and symptoms with which diagnosis is derived. Because it was recognized that albuminuria and hypertension could precede the onset of fits, the term preeclampsia was coined although this nomenclature is now criticized on the grounds that only a small proportion of patients subsequently develop eclampsia (3). In the UK, 38% of convulsions occur before the diagnosis of proteinuric preeclampsia is made and 44% occur in the postpartum period. Although edema traditionally is included defining preeclampsia, many authors believe that edema, even of the hands and the face is a common finding in pregnant women. Its presence should not validate the diagnosis of preeclampsia any more than its absence should preclude the diagnosis as there is no statistical correlation between hypertension and edema (1).

Redman and Jeffery proposed a revised definition of preeclampsia which is based on absolute blood pressure (b.p) levels and a rise from the base line in the first half of pregnancy. Proteinuria is not a necessity (4), and massive proteniuria identifies patients in the advanced disease (5). If proteinuria is present, immediate hospitalization is mandatory.

Regard to anesthetic which may become necessary in eclampsia it is believed that epidural anesthesia in women with severe preeclampsia is detrimental to both mother and fetus due to a possible profound hypotension. Other studies however demonstrated that epidural anesthesia in such women has a favorable effect on maternal hemodynamics (2). Critical insight regarding the understanding of the controversies of anesthesia and obstetric management is even more problematic and differences widen as you explore the mystery further.

Controversies

Discrepancies exist among physicians as to the lack of intravascular volume related to eclampsia. Although intravascular volume may be decreased, as a result of a compensatory mechanism to counteract hypertension the vessels are not under filled (6). Despite edema and hypertension, intravascular volume is a prominent feature of the disease (1,7). However in terms of capacity, the intravascular compartment in eclampsia is not usually under filled (7). If no hemorrhage is present

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others state that preeclampsia/eclampsia is characterized not only by vascular constriction but also by a remarkable under filling of the constricted vascular compartment (8). There is a consensus about the changes in the extra cellular compartment as fluids are shifted (1,4). However as far as the intravascular compartment is concerned, opinions differ and conflicting views are published.

While most agree that preeclampsia is diagnosed if blood pressure is above 140/90 mm Hg, and proteinuria or persistent edema or both are present (2). Other authors diagnose preeclampsia only if the diastolic blood pressure is greater than 90 mm Hg or has risen by 20 mm Hg compared to the pressure measured in early pregnancy (7). Definition of hypertension also remains controversial. In preeclampsia Page considers a rise of 20 mm Hg mean arterial blood pressure (MABP) to be significant even if previous blood pressure measurement is not available. A MABP of 105 mm Hg or greater is considered abnormal (9). In one center , a parturient has mild preeclampsia when she presents with the following: a blood pressure of 140/90 mm Hg on two occasions 6 h or more apart or a rise of 30 mm Hg systolic or 15 mm Hg diastolic from mid-trimester values, proteinuria (+) on two consecutive urine specimens and significant non-dependent edema; and labeled as severe preeclampsia if the blood pressure is persistently above 160/110 mm Hg and proteinuria above 5g/24 h (+ + +) and if the patient has symptoms of headache, blurring of vision, epigastric pain and oliguria (10). However, according to the Seventh Report of the Joint National Committee on Prevention Detection, Evaluation, and Treatment of High Blood Pressure, individuals with a systolic blood pressure of 120-139 mm Hg, and/or a diastolic blood pressure of 80-89 mm Hg should be considered as pre-hypertensive (11). This suggests that the diagnostic threshold of 140/90 mmHg may be too high for any population and particularly high for young females of childbearing, potentially leading to underestimation of hypertensive pregnancy disorders (12).

In the standard edition of Dieckman's 'The toxemia of pregnancy', there is a graph illustrating that 22% of eclamptic women developed convulsions without ever having a systolic blood pressure (SBP) above 140 mm Hg , and 12% of that group died of eclampsia (13). The graph not only negates the significance of blood pressure as a factor determining the severity of the disease but lays an emphasis on the SBP not referring to the diastolic blood pressure (DBP) which of course needs the same attention. Fisher *et al*. retrospectively compared the clinical signs of mild and severe preeclampsia of 176 cases and combined them with anatomical findings. He found that only 55% of the patients had a renal lesion which is typical of preeclampsia, while 4.5% had no lesion. The renal lesion consisted of the remaining 40% showed nephrosclerosis, chronic glomerulonephritis, chronic interstitial nephritis, pyelonephritis and a cluster of other renal diseases all of which promoted or at best simulated the clinical picture of preeclampsia (14). These findings suggest that the diagnostic data do not help much to support the final diagnosis of mild or severe preeclampsia and a high percentage of cases are falsely diagnosed as mild or severe preeclampsia. The study gives no rational explanation why such a high percentage of false diagnosis can occur in physicians who not only are renowned but also have taken great effort in their work.

Proteinuria is another subject of considerable debate and widespread controversy. To diagnose preeclampsia the terminology requires the presence of acute hypertension in late pregnancy together with proteinuria and facial, digital or generalized edema (14). Thus a person may have preeclampsia in the absence of proteinuria, but Sheehan and Lynch rarely found renal lesions unless the patients had proteinuria and the terminology does not require proteinuria to be an indispensable factor to diagnose preeclampsia. In a clinically valid assumption, proteinuria is usually a late sign in the disease and roughly 10% of eclamptic women do not have it before the onset of convulsions. The presence of proteinuria however is a warning sign and therefore should be take seriously.

Clinicians make major decisions based on the degree of proteinuria in such patients. In a systematic review by Thangaratinam and colleagues (15), they suggest that proteinuria is a poor predictor of either maternal or fetal complications in women with preeclampsia. Logically a test has its value when it is used to intervene because the outcome is no longer considered as the test itself influences the outcome. However there were some almost a decade back who nurtured this feeling that proteinuria not only strengthens the diagnosis but it also indicates a poor prognosis for the mother and the baby than when it is absent (3).

Edema as a reliable diagnostic sign is ignored by many and is not included in the criteria formulated by the committee on terminology and in Nelson's classification (13).

The International Society for the Study of Hypertension (ISSH) in its definition of preeclampsia does not include edema because it may be detected in

80% of normotensive pregnant women, most of whom are healthy. More recently, 32% of a series of eclamptic patients were noted to have no edema (3).

One may fail to identify this classical sign in preeclampsia and in an impending preeclampsia it is hidden in the HELLP, a variant of severe preeclampsia (2, 14). When the triad of symptoms i.e laboratory evidence of hemolysis, elevated liver enzymes (SGOT) and thrombocytopenia is encountered, a full blown picture of severe preeclampsia should be anticipated within the nest hours, days or even weeks (14).

Although there is improvement in the armamentarium of diagnostic tools, the syndrome requires special attention because cases with non typical signs of preeclampsia might be diagnosed as something other than preeclampsia.

The data on serum hematocrit are contradictory. While clinical improvement is characterized by hemodilution, a rise in hematocrit indicates worsening. Dieckman supports these findings; however others suggest that a fall in hematocrit is not a significant finding as it occurs most often with delivery (8). How should one tackle hematocrit? Assuming that the intravascular compartment is insufficiently filled and the capillary walls show a marked permeability, any additional attempts of infusion with electrolyte solution would bring down the raised hematocrit however at the same time would expand the extravascular compartment and end up in deterioration.

Controversies in the management of preeclampsia

Any treatment of preeclampsia still is empirical. The obstetricians refrain from reducing the blood pressure on the assumption that hypertension increases uterine perfusion. However, if the DBP is prevented from falling below 90 mm Hg, uterine perfusion will not decrease, on the contrary will increase. Also, antihypertensive drugs do not only decrease the peripheral resistance, they also help in relieving the uterine vasoconstriction which in turn increases uterine perfusion.

A conventional approach in the reduction of severe hypertension is the use of hydralazine (2,16), which is considered to be superior to sodium nitroprusside as it increases utero-placental blood flow (6). Hydralazine although it still has a good reputation in the therapeutic regime some look at it with skepticism. Serious fetal bradycardia secondary to a reduction in utero-placental perfusion can occur if blood pressure is dropped rapidly and decreased by hydralazine (1). Tachycardia with hydralazine also is a common and troublesome feature (5,17), propranolol often is advocated to correct tachycardia but propanolol causes fetal bradycardia and is usually not recommended in the treatment of toxemia of pregnancy (5). A satisfactory response to hydralazine administration is a decrease in DBP to 90 mm Hg (1,7), but it is worth to remember that lowering DBP below this level can result in decreased utero-placental perfusion. Redman citing the side effects even suggests that hydralazine can mimic eclampsia, a notion which is supported by others (17). With onset of action between 15-20 minutes after 20 mg hydralazine, the effect may be delayed for 30-40 minutes when the drug is given by drip. This delay can be dangerous to patients with malignant hypertension (17). Also are conflicting reports about its effects on cerebral blood flow. Since hydralazine is metabolized in the liver by acetylation and the enzyme level of N-acetyltransferase has dropped to 5% in this population, an exaggerated response may occur (13). Magnesium sulfate is another controversial drug used in the management of eclampsia. This drug seems to be the favorite for the American obstetricians because of its superb anticonvulsant effect. Now where more potent and safer drugs are available there seems to be little advantage in magnesium sulfate other than the experience and confidence obstetricians have with its use (5). Due to the effects of magnesium sulfate to decrease venous capacitance which in turn lead to a decreased cardiac preload and cardiac output the drug is contraindicated in cases with an already existing decreased cardiac output as it would result in inducing hypotension further (18). Magnesium sulfate administered parenterally is a valuable anticonvulsant commonly employed (1,9) most favored in the U.S. in spite of the fact that some patients developed eclamptic convulsions while receiving the standard intramuscular regimen as recommended by Pritchard (7).

The prophylactic use of magnesium halves the risk of developing eclampsia and that diazepam should be avoided as it is known to cause neonatal withdrawal syndrome (19,20). Again the routine 24 administration of maintenance dose of magnesium sulfate after a loading dose to all patients with preeclampsia has not been properly subjected to scientific scrutiny (8,21).

Others stated that ultra short protocol of 14 g magnesium sulfate given 4 g intravenously and 10 g intramuscularly was effective as an anticonvulsant in 92.6% of eclamptic patients in their study area (22).

Other authors suggest that there is little evidence to support the use of magnesium in the treatment of toxemia. It appears that this is a subject of speculations which still has to be looked into.

Diazoxide is another drug that has aroused considerable controversy and aired concern among obstetricians. Although it is advocated by some others do not foster its usage (1). Although it has a rapid onset of action and a fast reduction in elevated blood pressure (18) it is feared that it causes a dangerous hypotension. When used in mini boluses such falls can be prevented and a dose of 150 mg by slow i.v. infusion does not result in a dramatic blood pressure decline (18). Others still consider it a poor option in preeclampsia and it is recommended only if the blood pressure cannot be controlled with hydralazine. Aggressive treatment consists of diazoxide 300 mg i.v. along with 40-80 mg furosemide when the diastolic blood pressure is 120 mm Hg or greater (5). Diazoxide 300 mg was first used by Finesty, Morris and Norman near term, however Pritchard states that the use of diuretics/and or diazoxide is contraindicated in the management of preeclampsia. Nimodipine is also cited as an effective, easily controllable antihypertensive agent in patients with preeclampsia but its widespread use is still limited (23).

The use of fluids in preeclampsia/eclampsia

The type and the quantity of fluids have been dealt with in numerous articles but the question still remains unsolved. Volume expansion although advocated in patients with severe preeclampsia on the rational that the intravascular compartment is contracted, however there is no evidence to support the notion that volume expansion in the presence of an increased after load results in normalization of cardiac output and peripheral resistance (1). Although vigorous volume expansion has been implicated to result in cerebro-vascular insults, pulmonary edema and renal failure, it remains to be decided whether careful expansion would also result in similar complications, As in severe preeclampsia fluid shifts to the extravascualr compartment while the intravascular compartment is contracted, at the same time it is argued that these patients are hypovolemic. Looking in to the physiological changes it is evident that normal pregnancy is associated with a 1500 ml increase in volume which is not found in patients with pregnancy induced hypertension (PIH). In this regard patients with PIH are hypovolemic. But it we take the increased peripheral resistance into consideration the question remains as to whether these patients really are hypovolemic. Those who believe that the intravascular compartment is contracted but not under filled would not give fluids on the assumption that this would lead to pulmonary edema. On the other hand that these patients have a deficiency in volume, vigorous fluid therapy may be advocated as volume deficit may trigger vasospasm, hypertension and a variety of other functional derangements (7).

Fluid replacement against central venous pressure (CVP) measurement is cautioned since it leads, in most cases to over hydration of the patient. The reasons are obvious. Assuming that the normal CVP in severe preeclampsia is -1 cm of H_2O any rise may cause a patient to develop congestive heart failure if the fluids are administered with the normal logical intention of raising the CVP to 6-8 cm of H_2O which is the normal CVP in normally hydrated and fluid loaded patients. Large volumes of crystalloids are thought by certain authors to further expand the already expanded extracellular fluid volume, produce more edema and may worsen brain edema (24).

Albumin given i.v. would shift fluid into the constricted intravascular compartment and possibly precipitate circulatory overload and pulmonary edema. In this regard it should be kept in mind that fluids preferably should be infused along with a vasodilator.

Volume expansion and verapamil therapy effectively reduces maternal blood pressure in preeclampsia without adversely affecting utero-placental or umbilical artery resistance (25).

Oliguria is another complication which is contradictory as far as treatment is concerned. Diuretics, such as furosemide in the antepartum patient with PIH are contraindicated in the presence of cardiac failure and pulmonary edema (2,26). Since some patients develop oliguria on the basis of an intravascular volume depletion and systemic vasospasm oliguria is treated by volume expansion. But some patients develop oliguria because of renal hypo-perfusion as the result of renal vasospasm. This would not respond to volume expansion but to the administration of hydralazine together with cautious fluid administration. Such a differentiation between different causes of oliguria is undoubtedly of scientific interest but clinically it does not help in avoiding this complication. In countries where antenatal care and public awareness is below the standard, usually fulminant cases of preeclampsia reach the hospitals and it is there where anesthesiologists and obstetricians have to initiate a clear cut therapeutic regime.

Controversies in anesthetic management in patients with preeclampsia/eclampsia

Preeclampsia/eclampsia, being a complex disease taxes the expertise of the most experienced anesthetist, who has to focus on blood pressure stabilization,

optimization of fluid status and prevention of seizures (27).

The suitable anesthetic technique for patients with preeclampsia and or eclampsia is a challenge because both general anesthesia and an epidural block are fraught with pitfalls. Opinions are controversial regarding the use of an epidural in preeclamptic or eclamptic patients undergoing caesarean section. The standard textbook, "Williams Obstetrics" in 1985 recommended avoiding regional anesthesia in preeclamptic patients because of concern for sudden severe hypotension (26).

The proponents of an epidural block emphasize that placental perfusion which is significantly reduced in severe preeclamptic toxemia is normalized by an epidural block. Furthermore, an epidural results in a significant reduction in maternal catecholamine levels (6,17-19), which makes the epidural technique more favorable. Others have underlined these achievements with epidural block stating that it results in maximum analgesia, elimination of pain, anxiety and excitement, protection from pulmonary edema, relief of fetal asphyxia and little or no effect on the heart, lungs, kidneys and the liver. Regional anesthesia including spinal, epidural and combined spinal/epidural is believed to be beneficial in caesarean deliveries in these hypertensive patients. In the preeclamptic patient with acceptable coagulation and platelet count > 75×10 g/l, spinal anesthesia is safe, provided that fluid balance has been adjusted and effective vasodilator therapy commenced before cesarean section. Preoperative intravenous fluids should be restricted to 10 ml/kg in the absence of hemorrhage, since there is a risk of pulmonary edema in patients given excessive fluids, after regression of the block (28).

To achieve these goals the lowest possible effective dose of the local anesthetic together with adequate hydration should be used. However apprehension can not be eliminated in the awake patients in whom a regional technique is employed. Also, the decrease in peripheral resistance and protection from pulmonary edema which are expected goals of the local technique may not be achieved since preloading with a crystalloid solution of 1500-2000 ml might precipitate pulmonary edema in these susceptible patients (16,18). And thirdly the improvement of placental circulation which is attributed to an adequate blood pressure which of course can not always be guaranteed especially in the hands of an unskilled. Although foolproof there is no guarantee with this technique and a close monitoring of vital parameters is necessary. Although a drop in blood pressure after an epidural block is a frequent finding, still some think that patients with preeclampsia are less prone to develop hypotension an impression which is not substantiated by clinical studies. Since blood loss is approximately halved with an epidural, however the body's responses to hemorrhage can be significantly impaired (25). Some authors also doubt the efficacy of regional anesthesia stating that it is contraindicated in severe preeclampsia or eclampsia because sympathetic blockade may lead to pooling of blood with ensuing hypotension and further impairment of regional perfusion in patients who already have a reduced plasma volume (29).

This fear is based on the fact that the contracted intravascular compartment in these patients is extremely sensitive to vasodilatation which of course is a common finding in epidural anesthesia (1). Some prefer regional block in severe toxemia, but generally inhalational anesthesia is employed for cesarean section and hysterectomy (30,31). For regional anesthesia, esters such as 2 chlorpromazine (1-2%) is recommended than amides such as lidocaine or bupivacaine which are detoxified in the liver and excreted by the kidney (31). In our personal experience the amide group of local anesthetics which was used in a large number of preeclamptics showed no side effects (32,33). Most anesthesiologists also agree that spinal anesthesia is as good as epidural anesthesia in the management of these high risk patients provided there are no contraindications to its use (34). A report from India suggests that ideal anesthesia for eclampsia remains unknown but that general anesthesia can produce favorable outcomes with lower perinatal mortality (35).

The use of vasopressors in regional anesthesia should be avoided with lumbar epidural block in preeclamptic patients (20). Contrary others speculate that the epinephrine which is absorbed form the peridural space seems to exhibit a beta adrenergic agonistic effect and does not worsen preexisting hypertension in preeclamptic patients (35). Preeclamptics do not exhibit more cardiovascular responses to conventional doses of vasopressors than normal parturients under spinal anesthesia (36). In the same context, it is stated that severe preeclamptics may exhibit less hypotension during spinal anesthesia than healthy parturients (37). Many warn against the use of an epidural block in preeclamptic patients because splanchnic blockade may cause a dramatic fall in blood pressure which has to be corrected by pressor agents and crystalloids with ensuing iatrogenic ailments (1).

However pressor agents if given cautiously under

close surveillance do not cause much of a problem. Ephedrine in a dose as small as 2.5 mg is recommended to correct hypotension but higher doses should be avoided because these patients are sensitive to the pressor effects of vasopressors. Hypotension should be treated according to similar guidelines as in normal parturient, with the additional recommendation that vasopressors should not be given while the MABP remains above 100 mmHg. In preeclamptics with a non-reassuring fetal heart rate trace, spinal anesthesia has been associated with acceptable pulse and blood pressure changes, although it may be associated with a greater neonatal umbilical arterial base deficit and lower pH than general anesthesia (38).

General anesthesia although recommended by some is not advocated by others. General anesthesia with thiopental sodium, nitrous oxide and oxygen has been recommended for cesarean section in preeclamptics (1), but induction and endotracheal intubation are likely to precipitate a rise in blood pressure with pulmonary edema and cerebrovascular rupture (6). Maternal stress produces fetal asphyxia which is likely due to uterine vasoconstriction stemming from the release of maternal catecholamines (39,40).

Some authors do not advocate thiopental on the grounds that these patients suffer from hepatic dysfunction. But even in the most severe cases of preeclampsia, hepatic dysfunction is rarely that severe to cause problem (6). Both depolarizing and non-depolarizing neuromuscular blocking drugs (NMBD) are incriminated to have an additive effect in the presence of magnesium administration. Again this can only be stated with surety for suxamethonium where the effect is enhanced and prolonged by hypermagnesaemia (6,9). Additionally, another author states that non-depolarizing NMBD are be totally avoided in patients receiving magnesium sulfate (41). While Ecken Hoff and Vandam question the value of atropine and advise that it should be avoided, they however advocate a full dose of suxamethonium for induction. Due to the intense vagomimetic effect of suxamethonium, it in general is advisable not to use a maximum dose (9).

Problems evolving during the induction of general anesthesia in preeclamptics

To avoid unwanted complications during the induction period such as tachycardia, marked rises in blood pressure and episodes of hypoxia along with the detrimental sequelae, most authors suggest rapid and smooth induction (40). But despite the innumerable techniques for smooth induction, many a time it becomes a challenging task.

The circulatory responses to direct laryngoscopy and tracheal intubation and the associated rise in noradrenaline plasma level suggest an increased sympathetic activity (41-43).

The effects of these changes i.e., tachycardia and hypertension are minimal on healthy woman but in patients with preexisting hypertension, these changes would bring about a further devastation of circulation.

Any agent that rapidly decreases blood pressure without adversely affecting an already compromised fetus would be useful during laryngoscopy and induction. To minimize such circulatory responses, numerous drugs and techniques are advocated but unfortunately none satisfies the demands or assures absolute safety. Opioid drugs attenuate the hemodynamic and catecholamine stress response to tracheal intubation, and the use of alfentanil 7.5-10 μg/kg in preeclampsia has been well described as part of a general anesthetic technique (44-46). Alfentanil in dose of 10 μg/kg has been used before induction of anesthesia for elective cesarean delivery which attenuated the maternal stress response but at the cost of early neonatal depression (44).

Establishment of an endotracheal airway in obstetric patients is always conducted in lighter planes of anesthesia. This is the main reason why marked cardiovascular changes are elicited. Also awareness under general anesthesia may be possible and has been reported in the annals of medicine (15). Fiberoptic laryngoscopy is preferred technique by some as it is accompanied by a reduced response. But the fact remains that fiberoptic intubation takes longer which of course is a drawback in cesarean section (47).

Likewise beta blockade with practolol 10 mg i.v. before induction failed to prevent tachycardia during induction (39). The use of topical lidocaine, volatile agents, i.v. lidocaine, ganglion blockers, beta blockers and vasodilators have been shown to obtund the response associated with intubation, none however has earned wide spread approval (1,39-41,46,48).

Nifedipine, a calcium channel blocking drug appears to be of value to minimize the stress responses during intubation because of its fast onset of action, its potency and negligible toxicity. Nonetheless it causes tachycardia which of course is a disadvantage. This effect is ignored by some in the light of its advantageous property of reducing after load of the left ventricle (40,46,47). Nitroglycerine is also effective and propagated by some. It lags behind other drugs in potency to control the maternal responses during

intubation in preeclamptic patients. Hydralazine in small doses of 5-10 mg i.v. before induction is advocated to counteract and blunt the rapid spikes of blood pressure during laryngoscopy and intubation (1,6). But its efficacy is questionable because of the tachycardia that it induces together with its slow onset of action (5,18). Nevertheless it still is a favorite drug for many anesthesiologists. Propranolol 1-5 mg i.v. can block the reflex tachycardia, but it can cause fetal bradycardia as it crosses the placenta. The beta-1-selective compound esmolol makes a better choice in situations such as tracheal intubations where beta receptor blockade is desired. Even in patients with renal failure esmolol can be safely given either in increments or as infusion (41).

Discussion

Having reviewed a great deal of attention, one thing becomes clear. None of the available techniques do guarantee absolute safety. Obstetricians and anesthesiologists alike have ample reasons for concern while undertaking the challenging task of the management. It would be better to think before using a method that yields the best results. There is no single remedy for this disease but a collective approach is necessary.

Obviously such a collective approach shows the best results and is a panacea for this state of emergency. It has been suggested that the surgeon should not push the anesthesiologist into a certain technique. It is the general attitude that the surgeons do not foster regional techniques and many times these techniques are felt as the better noir by most obstetricians. Persuading an anesthesiologist to go for a technique in which he is not at home would rather result in serious drawbacks that is not of advantage for the patient. No technique or model is for everyone and empirical experience should be kept abreast with the latest developments. A scrupulous technique coupled with adequate monitoring such as central venous pressure, intra arterial pressure, neuromuscular transmission and if circumstances permit the use of a Swan-Ganz catheter for pulmonary capillary wedge pressure measurement would be useful. Although delivery is associated with a resolution of the hypertension (45), it is sometimes coupled with fatal complications.

While inducing anesthesia in these high risk patients, the objective should be to prevent perioperative complications. To achieve this desired goal, the following points should be kept in mind:

1. Ketamine should be avoided because of its propensity to cause hypertension and tachycardia (41).

2. Midazolam has been incriminated to produce neonatal depression and in toxemic patients, the fetus is compromised. Therefore any drug inducing depression would further imperil the fetus (41).

3. A competitive muscle relaxant such as flaxedil should be avoided because of its vagolytic activity leading to dangerous tachycardia (41).

A dose of 1 microgram of fentanyl administrated 15 minutes before cesarean delivery dose not depress the newborn (42). Thiopental sodium in small but adequate doses, followed by suxamethonium is helpful to complete endotracheal intubation but this hurdle is not very easy to overcome because of the dangers of tachycardia and hypertension frequently encountered during laryngoscopy and tracheal intubation. These effects can be overcome or blunted with prior administration of fentanyl 50-100 micrograms, lidocaine 100 mg, hydralazine 2.5-5 or labetalol in 20 mg increments. Alfentanil in a dose of 10 μg/kg before induction can equally be effective in blunting the spikes in blood pressure during laryngoscopy (46). Halothane in small concentrations of 0.5-0.75% 3-5 minutes before induction of anesthesia had been helpful and a rise in blood pressure had been minimal and many a time eliminated (44).

But halothane is no longer used except in some parts of the developing world owing to its hepatic toxicity and because it adversely affects the green zone. Some authors prefer 10 mg nifedipine (sublingual) before induction when hydralazine is not available (47,48).

Further more fluids should be infused against CVP measurement. However it should not be raised above 5 cm H_2O. Colloids are preferred because they maintain a normal colloid pressure. In the presence of marked hemo-concentration, careful search for pulmonary edema has to be made (49,50). Fluid administration is conservative in order not to produce pulmonary edema (1). In oliguric patients, 10-20 mg of furosemide during the course of the operation would be useful providing the CVP is not low and intravascular volume is not depleted (46). Regarding muscle relaxants, atracurium would be a better choice because its elimination is not via the kidneys. As mentioned, muscle relaxants should be titrated using nerve-muscle stimulator because these patients have been given magnesium sulfate which enhances the sensitivity of both depolarizing and non-depolarizing agents.

We are of the opinion that once the fetus is delivered, there is enough relaxation of the abdominal wall to suture the different layers of musculature without

additional relaxation with muscle relaxation, however this hypothesis needs a multicenter study to be fully validated (33,52). Therefore relaxants can be avoided because they are not needed. Also it is to be remembered that relaxants are no substitute for inadequate anesthesia. N_2O in concentration of 40-50% can be safely used along with oxygen because it does not cause changes in the uterine tone or contraction. Narcotics can be freely used after the baby is delivered but it would be better to avoid a narcotic such as pethidine that causes tachycardia or induces vomiting. Dangerous falls in blood pressure can again compromise utero-placental circulation resulting in shunting of blood and sudden fetal distress (24), which demands immediate correction with fluids, left uterus displacement and pure oxygen.

The patient should not be extubated unless one is 100% sure that the tidal volume is optimal, skeletal muscle relaxation has returned to normal, there is no sign of a raised intracranial pressure or pulmonary congestion. Lidocaine 1mg/kg i.v. would allow the patient to tolerate the endotracheal tube. If there is some suspicion that the parameters for extubation are not fulfilled, the patient should be sent to the intensive care unit and kept under close surveillance (32,33,50).

In summary, spinal anesthesia has been considered an option in patients with severe preeclampsia ever since the first trial was published (34). Nevertheless, hypotension and placental under-perfusion remain at risk (49), and spinal anesthesia may be associated with more neonatal acidosis than general anesthesia (37).

1. Epidural anesthesia can be performed but patient should be hydrated prior to the block so that the CVP is maintained between 3 and 4 cm H_2O or until a PCWP reaches 5-12 mmHg.
2. Combined spinal-epidural anesthesia has been successfully used for cesarean delivery in patients with severe preeclampsia.
3. In emergency cesarean section, general anesthesia is the better choice because of the lowest incidence of hypotension (52,53).
4. In regard to the drugs to be used in hypertensive episodes, dihydralazine is administered intravenously either as 2.5 boluses or as a continuing infusion. If there is no immediate fetal or maternal indication for delivery, oral alpha methyldopa and/or nifedipine are used for blood pressure control.

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