Anaesthetic considerations in high-risk pregnancy

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In tertiary care centres, 30 to 40 per cent of pregnancies qualify for the label "high-risk." These previous or current obstetric problems, and associated medical disorders (Table I), adversely affect fetal or maternal outcome, and may lead to increased perinatal or maternal mortality. This presentation focuses on pre-eclampsia, diabetes mellitus, breech presentation and premature labour, as examples of high-risk obstetrics. Due to space restrictions, the written summary is limited to pre-eclampsia and diabetes mellitus. To assist the reader, a full listing of suggested information sources for breech presentation and premature labour is also included.

Pre-eclampsia

The hypertensive disorders of pregnancy remain major contributors to maternal and perinatal mortality. Pre-eclampsia is a syndrome characterized by the development of hypertension, and proteinuria, oedema, or both, after the 20th week of gestation (or sometimes earlier in the presence of trophoblastic disease).^{1,2}

Definitions

Hypertension implies a systolic pressure ≥ 140 mmHg (or an increase ≥ 30 mmHg) and a diastolic pressure of at least 90 mmHg or an increase ≥ 15 mmHg, on at least two occasions more than 6 hours apart.³ Proteinuria means $> 0.3 \text{ g}\cdot\text{L}^{-1}$ in a 24 h specimen, or a concentration $\geq 1 \text{ g}\cdot\text{L}^{-1}$ in at least two random specimens, more than 6 h apart.

In severe pre-eclampsia hypertension equals or exceeds 160/110 mmHg, and one or more of the complications listed in Table II occurs.

Eclampsia, diagnosed by the occurrence of seizures, usually follows rapidly progressive pre-eclampsia, and is characterized by headache, epigastric pain, increased CNS excitability and haemoconcentration, but may occur abruptly in a patient

with minimal hypertension and apparently "mild" pre-eclampsia.

Superimposed pre-eclampsia refers to the occurrence of the syndrome in women with pre-existent hypertension.

Aetiology and pathophysiology

Despite intensive research, the cause of pre-eclampsia remains unknown, and the lack, until recently, of a suitable animal "model" has hampered its elucidation. Cavanagh *et al.*⁴ recently produced changes consistent with pre-eclampsia in pregnant baboons, by the chronic occlusion of subrenal aortic blood flow. This, and earlier studies, suggest that sustained uteroplacental ischaemia plays an important role in the development of pre-eclampsia.^{4,5} Since the syndrome occurs most frequently in first pregnancies, has an apparently familial incidence, and may recur in later pregnancies especially when a change of partner is involved, an immune mechanism may also be implicated.^{1,6}

The hypertension in pre-eclampsia reflects increased vascular sensitivity to endogenous peptides (e.g., angiotensin) and catecholamines.² Moutquin *et al.*⁷ identified systolic blood pressures 7–22 torr higher than normal controls ($p < 0.001$), during early pregnancy in nulliparae who later developed pre-eclampsia. Diastolic pressures were consistently higher from the first antenatal visit, at 9–12 weeks, in future pre-eclamptics. Evidence suggests deficient prostacyclin (PGI_2) production in maternal,⁸ fetal^{9–11} and placental¹¹ vasculature in pre-eclampsia. Since PGI_2 possesses vasodilator properties, this deficiency may, in part, explain the generalised vasospasm of pre-eclampsia.

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TABLE I Classification of high-risk pregnancies

Previous obstetric problems**Prenatal**

- Maternal – Recurrent abortions
 - Incompetent cervix
 - Isoimmune (usually Rh) sensitization
 - Intrauterine fetal death
 - Abruptio placentae
 - Premature labour
 - Infertility
- Fetal – IUGR
 - Fetal anomaly/disorder
 - Pregnancy induced hypertension

Intrapartum

- Delivery complications
- Stillbirth

Postnatal

- Thromboembolism
- Neonatal death

Current obstetric problems**Maternal – Advanced maternal age**

- Uterine anomalies
- Premature rupture of membranes
- Premature labour
- Pregnancy induced hypertension
- Placenta praevia
- Herpes

Fetal – Multiple gestation

- Breech presentation
- IUGR
- Fetal anomalies

Associated medical disorders

- Diabetes mellitus
- Chronic hypertension
- Renal disease
- Thyroid disease
- Cardiac disease (usually Class III & IV)
- Haematologic disorders
- Neurologic disease
- Collagen/vascular disorders
- Respiratory disease
- Other medical conditions

After: Cohen AW, Mennuti MT, Gabbe SG, Liston RM. High risk obstetrics. The three-year experience of four subspecialists. *J Reprod Med* 1985; 30: 472–7.

Normal pregnancy reduces the circulatory and sympathoadrenal responses to postural change,^{12,13} but patients with pregnancy-induced hypertension demonstrate increased release of adrenal catecholamines, and increased forearm vasoconstriction, in response to tilt.^{13,14}

Measurements of plasma volume in normal and pre-eclamptic pregnancies, show less pregnancy-

TABLE II Indicators of severe pre-eclampsia

- Proteinuria ≥ 5 g in 24 h (3+ to 4+)
- Oliguria < 400 ml/24 h
- Cerebral or visual disturbances
- Epigastric pain
- Pulmonary oedema
- HELLP syndrome
(Haemolysis, ELevated liver enzymes, Low Platelets)*

*Weinstein, L. Pre-eclampsia/eclampsia with hemolysis, elevated liver enzymes, and thrombocytopenia. *Obstet Gynecol* 1985; 66: 657–60.

induced volume expansion in the latter.^{15,16} The average plasma volume in pre-eclamptic women is nine per cent below expected values, and may be as much as 30–40 per cent below normal in severe pre-eclampsia.¹⁵ Some studies have demonstrated inadequate volume expansion before clinical evidence of pre-eclampsia, suggesting hypovolemia as a causative factor.^{14–17} Others believe decreased volumes result from the vasoconstriction of the disease process.² Two therapeutic “schools” presently exist: those who favour expansion of the intravascular volume in the prevention and treatment of pre-eclampsia,¹⁸ and others who recommend vasodilator therapy.² However, plasma volume expansion remains controversial, and increases the risk of pulmonary oedema in the absence of vasodilation.^{1,2}

The glomerular filtration rate decreases in pre-eclampsia, by up to 25 per cent, in contrast to a 30–50 per cent increase in normal pregnancy.¹ In part, this reduction results from swelling of the glomerular endothelium.² Elevated plasma levels of uric acid occur, but differ significantly from non-hypertensive pregnancies only in the week before delivery.¹⁹ Sodium excretion is also decreased. The fibrin deposition on the glomerular basement membrane leads to increased permeability and proteinuria.¹

Central venous pressure (CVP) in pre-eclampsia reflects the severity of the disease and varies inversely with the level of hypertension.^{1,20} Most studies of cardiac output and pulmonary capillary wedge pressure (PCWP) have been confounded by lack of appropriate controls, administration of parenteral fluids, and vasoactive therapy.^{2,21} Newsome and Bramwell²¹ reported hyperdynamic ventricular function, and poor correlation between CVP and PCWP, in severe pre-eclampsics studied

before vasodilation or magnesium therapy. Reviewing their experience with Swan-Ganz catheterization, Clark *et al.*²² noted simple hypovolaemia, with low PCWP, moderately increased systemic vascular resistance (SVR), and hyperdynamic left ventricular function in most pre-eclampsics. These patients respond to fluid therapy alone. Less frequently, there is evidence of impending left ventricular failure – high PCWP, markedly elevated SVR and depressed ventricular function – which requires treatment with fluid restriction and after-load reduction.

In addition to vasospasm, hypovolemia and frequent oliguria, pre-eclamptic women have increased vascular permeability and consequent loss of intravascular fluid and protein into interstitial tissues.¹ Mean colloid osmotic pressure (COP) is lower in pre-eclampsia, both antepartum (18 torr) and postpartum (14 torr), than in normal pregnancy (22 and 17 torr respectively).²³

Coagulation abnormalities are well documented in pre-eclampsia, usually, but not exclusively, in patients with severe disease. Thrombocytopenia occurs in 15–30 per cent of cases,^{24,25} and a prolonged bleeding time may exist in thrombocytopenic or non-thrombocytopenic cases.²⁵ The thrombocytopenia appears to reflect increased platelet consumption.²⁶ Prostacyclin (PGI₂) deficiency may underlie this excessive consumption,²⁷ and interesting recent work suggests that a combination of antiplatelet agents (dipyridamole and aspirin) can prevent pre-eclampsia, if commenced early in pregnancy.^{2,28}

Isolated thrombocytopenia, without other abnormal coagulation parameters, but with evidence of haemolysis and abnormal liver enzymes, characterises the HELLP syndrome, a severe uncommon variant. Rarely, thrombocytopenia results from DIC.¹ Although thrombin, and partial thromboplastin, times may be variably prolonged in pre-eclampsia, the coagulation screen (excluding platelets) is most frequently normal.²⁵

Eclamptic seizures have been attributed to:

- platelet thrombi which obstruct the cerebral microcirculation;
- intense cerebral vasoconstriction, or;
- hypertensive encephalopathy.^{1,2}

Although their occurrence may correlate with the degree of hypertension, seizures may also occur in women with mild blood pressure elevation.^{1,2}

Other organ systems involved in pre-eclampsia include the liver and lungs. In the HELLP syndrome, patients experience epigastric pain, nausea and vomiting, significant elevation of serum transaminases (SGOT and SGPT), and hyperbilirubinaemia (mostly indirect bilirubin, resulting from haemolysis – microangiopathic haemolytic anaemia).

Pulmonary oedema may complicate severe pre-eclampsia. Usually, this results from altered hydrostatic-oncotic forces in the pulmonary vasculature, with elevated PCWP and a low COP. Other patients demonstrate increased pulmonary capillary permeability in the face of normal pressures, or left ventricular failure.²⁹

Current obstetric management

Definitive treatment involves stabilization, followed by delivery. When pre-eclampsia occurs early in the third trimester, stabilization usually implies bed rest, with or without antihypertensive therapy. Mothers who respond to this regime then proceed to induction and delivery at term. Hypertension which fails to respond within 24 to 48 h calls for urgent delivery, irrespective of fetal maturity. Severe pre-eclampsia, with increasing CNS excitability, worsening proteinuria, oliguria, and haematologic or hepatic complications, demands urgent stabilization and expedited delivery.

ANTIHYPERTENSIVE THERAPY

Data from animal studies suggest that the non-autoregulated maximal uterine blood flow at term, may not apply to pre-eclamptic women.^{1,2} Theoretical concern about decreased uteroplacental perfusion and consequent adverse fetal effects, from antihypertensive drugs, has not been justified by clinical experience, although a precipitous drop in blood pressure should be avoided since it may cause fetal distress.

In labour, increments of IV hydralazine (5–10 mg, Q20–30 min) are the preferred treatment for severe hypertension.² Cardiac index and heart rate increase significantly, while systemic vascular resistance and mean arterial pressure show a maximal decrease in 15–30 min, but there is a wide variation in response.³⁰ Correction of hypovolemia before hydralazine avoids severe hypotension, and results in less dramatic reduction of mean arterial pressure. Cotton³⁰ believes patients with an already hyper-

dynamic myocardial function, especially if refractory to hydralazine, may be more appropriate candidates for afterload reduction with beta-blockers. Alternatively, diazoxide can be used in mothers refractory to hydralazine, but must be given in 30 mg "minibolus" doses, since maternal circulatory collapse and death have followed 300 mg.²

Nitroglycerine or sodium nitroprusside provide effective attenuation of the pressor response associated with general anaesthesia.

Antihypertensive medications for long-term control of pre-eclampsia include methyldopa, beta-receptor antagonists, and mixed alpha-beta-receptor antagonists (e.g., labetalol).² The role of calcium channel blockers in pregnancy induced hypertension also appears promising.³¹ Most obstetricians eschew diuretics, believing they may exacerbate hypovolemia and intensify pre-eclampsia.

ANTICONVULSANTS

Magnesium sulphate remains the drug of choice in North America, for the prevention and management of eclamptic seizures.² Although its mode of action is poorly understood, it decreases CNS excitability, probably by specific, rather than generalised CNS depression. At the neuromuscular junction, magnesium inhibits presynaptic acetylcholine release, decreases end-plate acetylcholine responsiveness, and diminishes excitability of the muscle fibre.³² Magnesium potentiates non-depolarizing block, and depolarising block, by factors of four and two respectively.

Therapeutic anticonvulsant levels are 3–4 mmol·L⁻¹. Loss of the patellar reflex occurs at 5 mmol·L⁻¹, respiratory arrest at 6–8 mmol·L⁻¹, and asystole at 12 mmol·L⁻¹.¹ Magnesium is excreted via the kidney, and high plasma levels may occur in renal insufficiency. The IV dosage schedule (4 g loading dose, 2 g·h⁻¹ maintenance), although usually favoured, has no therapeutic advantage over IM administration.³

Neonatal depression occurs only with severe maternal hypermagnesaemia. Intravenous calcium gluconate decreases the maternal and neonatal toxic effects, and should be given when magnesium may be implicated in prolonged apnoea following muscle relaxants.

Anaesthetic management

Successful patient care requires a multidisciplinary

team approach, involving obstetrician, anaesthetist and neonatologist. Before anaesthetic intervention, moderate and severe pre-eclampsics need stabilisation, appropriate hydration, magnesium therapy for increased CNS excitability, and vasodilator therapy, if indicated. An accurate intake-output chart and indwelling catheter are mandatory. In mild and moderate pre-eclampsia, a platelet count and bleeding time, in addition to routine investigations, usually suffice to exclude coagulopathy. Severe pre-eclampsics, however, require a full coagulation screen, liver function tests, serum electrolytes, and a peripheral blood smear if any of these prove abnormal.

Before regional anaesthesia the depleted intravascular volume must be corrected. Joyce *et al.*²⁰ related volume of preload to diastolic pressure and CVP. Severe pre-eclampsics, with high diastolic pressures (greater than 110 torr) required 750 ml of Plasmanate and 2 L of crystalloid to achieve an acceptable CVP, while those with diastolic values between 90 and 110 torr needed only 1 L of lactated Ringer's solution. However, this aggressive hydration led to unacceptably high CVP values following delivery, with enhanced risk of pulmonary oedema.³³ Joyce and Loon³³ therefore recommended small volumes of 25 per cent albumin (75–100 g) in severe disease, and less in mild pre-eclampsia. They also reduced the infused volume of lactated Ringer's solution.

In moderate and severe disease, especially in the presence of oliguria, crystalloid replacement should be guided by CVP values. Injudicious prehydration may lower COP, precipitating pulmonary oedema,²⁹ and caution must be exercised in crystalloid replacement.³⁴ Although CVP monitoring provides useful information in assessing the response to prehydration, CVP has no correlation with PCWP in severe pre-eclampsia.^{21,29}

Concern about the potential complications of pulmonary artery catheters,^{22,35} and questions about the usefulness of the data obtained, have limited their application in obstetrics. However, they provide valuable information in severe pre-eclampsia and eclampsia with oliguria refractory to fluid challenge, pulmonary oedema and other signs of cardiac decompensation, and mothers with co-existent Class III and IV heart disease.^{21,22,29} Parturients may be at greater risk of non-bacterial thrombotic endocarditis (NBTE), and Chestnut *et*

*al.*³⁶ recommend serial echocardiography if the pulmonary artery catheter remains *in situ* for longer than 48 h. A sudden change in neurologic status, apparent embolic phenomena, or the development of coagulopathy, should suggest the possibility of NBTE.

Severe pre-eclampsia calls for insertion of an arterial line, for continuous pressure monitoring and serial blood sampling.

ANALGESIA IN LABOUR

Despite the wealth of accumulated clinical experience,³⁷⁻³⁹ and several publications which attest to the safety and beneficial effects of epidural analgesia in pre-eclampsia,⁴⁰⁻⁴³ one may still read: "epidural block should be avoided, since in pre-eclampsia it is associated with sudden and marked falls of blood pressure and on occasion with vascular collapse".² Pritchard *et al.*³ have, for years, avoided conduction anaesthesia in severe pre-eclampsia and eclampsia, because of their concern for sudden severe hypotension, and their anxiety about dangers of pressor agents and the vigorous overinfusion of crystalloid to correct hypotension. These legitimate but increasingly isolationist views nonetheless pinpoint the principal pitfalls which await the unwary anaesthetist – hypotension with consequent fetal distress, pulmonary,²⁹ laryngeal,⁴⁴ and, possibly, cerebral oedema,³ and an irate obstetrician!

Carefully conducted epidural analgesia has no adverse effect on intervillous blood flow,^{45,46} cardiac output,^{21,47} or Apgar scores,⁴³ and effectively reduces maternal circulating catecholamines⁴⁸ and arterial pressure. Epidural block is *not*, however, a substitute for antihypertensive and anticonvulsant therapy.

I consider bupivacaine, 0.125–0.25 per cent, the local anaesthetic of choice, and the dose must be carefully titrated to patient response. Fetal monitoring, by scalp electrode, should continue throughout performance and maintenance of the block. Epinephrine containing solutions must be avoided, and hypotension treated with additional crystalloid or minimal increments of IV ephedrine (5–10 mg), in view of the increased maternal sensitivity to catecholamines. Thrombocytopenia below 100,000· μL^{-1} , an abnormal bleeding time (> 10 min), or abnormal PTT contraindicate regional anaesthesia.

ANAESTHESIA FOR CAESAREAN SECTION

General anaesthesia (GA) for Caesarean section, although the rule in some centres,³ carries significant risks in pre-eclampsia:¹ aspiration of gastric contents, laryngeal oedema with difficult intubation,⁴⁴ adverse hypertensive response to laryngoscopy and tracheal intubation,⁴² pulmonary oedema from increased left ventricular afterload,⁴⁹ significant impairment of intervillous blood flow,⁵⁰ prolonged apnoea from magnesium therapy, and extubation hypertension.⁴² However, coagulopathy, severe fetal distress, abruptio placentae and unanticipated technical difficulties, may sometimes dictate GA.⁵¹

Hood *et al.*⁵² documented successful prophylactic use of IV nitroglycerin (NTG) (200 $\mu\text{g}\cdot\text{mL}^{-1}$) to attenuate the hypertensive response to tracheal intubation in severe pre-eclampsia. All patients had intra-arterial cannulae, and were treated with MgSO_4 prior to anaesthesia. After preoxygenation, mothers received thiopentone 5 $\text{mg}\cdot\text{kg}^{-1}$, and succinylcholine 1.2 $\text{mg}\cdot\text{kg}^{-1}$, in a rapid sequence induction. No volatile inhalation agents were administered (although the addition of halothane 0.5 per cent, for example, might have further stabilised intraoperative pressures, without adverse fetal effect).

Writer *et al.*⁵³ expressed concern about loss of cerebral autoregulation, and the potential for a brisk intracranial pressure rise following NTG, if blood pressure should increase abruptly. However, Hood's experience confirms the safe clinical application of this technique. No adverse maternal or neonatal effects resulted.

Alternatives to NTG include sodium nitroprusside (SNP – more controllable, and safe in short-term use) and trimethaphan. Trimethaphan, however, may prolong succinylcholine apnoea, cause histamine release and reduce cardiac output.

Epidural anaesthesia is the preferred technique in Caesarean section, when no contraindications exist. Hodgkinson *et al.*⁴² compared GA and epidural block in severely pre-eclamptic mothers having Caesarean section. All patients had an indwelling arterial line, and pulmonary artery catheter. During tracheal intubation and extubation, mothers receiving general anaesthetics experienced marked rises in mean arterial pressure and PCWP. Mothers having epidural block, in contrast, had stable

pressures, and only one experienced transient systolic hypotension.

In severe pre-eclampsia, CVP measurement and an intra-arterial line facilitate patient monitoring during epidural anaesthesia. Preload volume should be adjusted to the CVP response, to secure a normal CVP before induction.¹ Bupivacaine, 0.5 per cent, because of its slower onset, remains the preferred local anaesthetic, and should be given in 5 ml increments to achieve a T₄ level. Three per cent chloroprocaine represents an alternative for emergency Caesarean section, especially in mothers with an epidural catheter *in situ* and already prehydrated. Marx *et al.*⁵⁴ confirmed the safety of 2-chloroprocaine for emergency Caesarean section in fetal distress. Mothers in labour, with adequate prehydration, experience less hypotension with regional anaesthesia.

The anaesthetist must exercise continued vigilance over fluid intake during and immediately following Caesarean section, and resist the temptation to overtransfuse.³⁴ In the presence of a normal CVP, and stable arterial pressure, mothers require low maintenance volumes of crystalloid, and a figure of 75 ml·h⁻¹, or less, has been suggested.⁵⁵ Aggressive fluid therapy increases the retained interstitial fluid, and sets the stage for postpartum pulmonary oedema when PCWP rises with mobilization of this fluid in the presence of continuing oliguria.³⁴ The onset of diuresis, usually 24–48 h following delivery, signals improvement, but proteinuria and oedema may persist up to one week postpartum.³

Diabetes mellitus

Diabetes is the commonest medical problem encountered in pregnancy.⁵⁶ Although the obstetric care and anaesthetic management of diabetic mothers have advanced considerably in recent years, perinatal mortality and morbidity still significantly exceed that of the non-diabetic mother.^{56,57}

Glucose homeostasis in pregnancy

Pregnancy represents a “diabetogenic” stress, which becomes more marked in the latter half of gestation.⁵⁸ Coincident with the production of metabolically active steroids and peptides by the placenta, and the increasing demands of the fetus, the fasting state is marked by greater diversion to fat

metabolism (accelerated starvation), with increased plasma concentrations of free fatty acids (FFA) and ketones. Plasma glucose levels fall more rapidly, since the beta-cells of the maternal islets show hyperplasia, increased insulin secretion and greater sensitivity to smaller doses of glucose.

The postprandial state, also, is modified. Glucose ingestion results in greater and more prolonged elevation of plasma glucose, owing to the contra-insulin effects of hPL, progesterone and cortisol. Thus, greater amounts of glucose reach the fetus.⁵⁸ Increases in amino acids are less marked and more short lived. Basal insulin levels rise higher than in the non-pregnant female, especially late in pregnancy, and a meal effects a two- to three-fold increase in insulin production, in an attempt to counter hormonal insulin resistance.

The net result of these changes in normal pregnancy is an overall lowering of plasma glucose, enhanced fat breakdown and a carbohydrate induced triglyceridaemia, which supports maternal metabolism while glucose is diverted to the fetus.⁵⁹

The metabolic disturbance in insulin dependent type 1 diabetes (IDDM)

In early pregnancy, Type I diabetics more frequently experience hypoglycaemia and difficult glucoregulation.⁵⁹ Most patients require larger amounts of long- or intermediate-acting insulin to achieve effective homeostasis in later pregnancy, but tolerate lower plasma glucose levels. In addition, they may need supplements of short-acting insulin with meals, because of contra-insulin effects and the more prolonged elevation of postprandial glucose levels. Some centres employ portable insulin pumps to achieve tight control in later pregnancy.⁶⁰

Diabetic ketoacidosis exerts a major influence on perinatal morbidity, and the principle of diabetic management is increased caloric and carbohydrate intake (under careful insulin control) to offset accelerated starvation and the tendency to ketosis. Four factors increase the risk of ketoacidosis:

- 1 the relative insulin deficiency
- 2 excess contra-insulin hormone
- 3 fasting (accelerated starvation) and
- 4 dehydration.⁵⁷

Effect of diabetes on the fetus

Diabetes increases the incidence of major fetal

anomalies, prematurity, pre-eclampsia, polyhydramnios, respiratory distress syndrome (RDS), macrosomia and neonatal hypoglycaemia.⁵⁷ Macrosomia appears to result from increased fetal insulin secretion in response to the exaggerated delivery of glucose, and other substrates, across the placenta.⁵⁸ Poor diabetic control in the first trimester may account for small for gestational age (SGA) infants, and the increased incidence of birth defects. Women with first trimester elevation of glycosylated haemoglobin (A_{1c} greater than five per cent), indicating inadequate diabetic control, have a sevenfold increase in the incidence of congenital fetal defects.⁶¹

Neonatal hypoglycaemia, hyperbilirubinaemia and hypocalcaemia occur more frequently.

Classification

White's classification of diabetic pregnant women,⁶² although less frequently used, forms a basis for understanding the severity of the disease. Classifications B through D describe increasing duration of diabetes (with expected increase in complications). Classes F, T, R and H refer to the presence of the complications nephropathy, post-renal transplant, proliferative retinopathy, and coronary artery disease.

Principles of management

Metabolic control appears to be the principal determinant of pregnancy outcome. Total caloric intake is maintained at $30 \text{ kcal} \cdot \text{kg}^{-1}$ ideal body weight in the first trimester, then increased to $38 \text{ kcal} \cdot \text{kg}^{-1}$ (with 45 per cent carbohydrate) in the second and third trimesters.⁵⁸ Insulin treatment is adjusted, as described earlier, to avoid fasting and postprandial hyperglycaemia, as well as hypoglycaemia. Glucose oxidase strips and portable reflectance colorimeters, enable reliable control in most patients. Subjects monitor their capillary blood sugar before each meal, and at bedtimes, as well as 1 h after meals one day per week. The goal is to achieve tight glucose control ($3.5\text{--}5.5 \text{ mMol} \cdot \text{L}^{-1}$ fasting; less than $7.8 \text{ mMol} \cdot \text{L}^{-1}$ post-prandial), and mean plasma values of $4.4\text{--}6 \text{ mMol} \cdot \text{L}^{-1}$.^{57,58} Sequential measurement of haemoglobin A_{1c} provides an indicator of long-term control. Maternal insulin requirements fall abruptly following delivery, but may decline in or before labour. A rapid diminution in requirement close to term, implies fetop-

lacial compromise, and the need for early delivery.

Management of labour and delivery

The objective, when feasible, is to deliver the fetus vaginally at term (from 38 weeks on). Fetal monitoring in later pregnancy includes fetal movement counting, twice weekly (or more frequent) non-stress tests, and planning scores. The phosphatidylglycerol (PG) assay has replaced the L/S ratio as an index of fetal maturity, and when PG exceeds one per cent there is minimal risk of RDS.⁶³

Very strict control of the maternal blood glucose in labour ($5.5\text{--}6.5 \text{ mMol} \cdot \text{L}^{-1}$) avoids hypoglycaemia, which may interfere with the progress of labour, and hyperglycaemia, which may promote increased fetal insulin production, fetal distress and neonatal hypoglycaemia. In addition, maternal (and consequently fetal) hyperglycaemia accentuates fetal metabolic acidosis, especially that secondary to hypoxia.⁵⁷

Maternal euglycaemia can best be achieved in labour by continuous glucose infusion (D10W, commencing at $50 \text{ ml} \cdot \text{h}^{-1}$, and increasing, as necessary, to $150 \text{ ml} \cdot \text{h}^{-1}$, i.e., $5\text{--}7.5 \text{ g} \cdot \text{h}^{-1}$), monitoring of plasma glucose Q1-2H, and low dose insulin infusion (0 to $2 \text{ units} \cdot \text{h}^{-1}$). Usually, very little insulin is required and severe postpartum maternal hypoglycaemia may occur from larger doses.

Anaesthetic management

Good anaesthetic care requires careful patient evaluation, assessment of complications such as peripheral neuropathy, and an understanding of the pathophysiology of diabetic pregnancy. Insulin dependent diabetics need team care, with informed involvement and good communication by all parties.

Diabetic mothers frequently have impaired uteroplacental blood flow and may have abnormal O_2 transport, with impaired O_2 release at tissue level in the presence of high haemoglobin A_{1c} levels.⁵⁷ Brouillard *et al.*⁶⁴ reported decreased buffering capacity (and an impaired response to an acid load) in the infants of diabetic mothers. Their haemoglobin also has enhanced O_2 affinity, making them more susceptible to hypoxia.

In labour, lumbar epidural block is the analgesic technique of choice, but requires scrupulous attention to detail since diabetic mothers appear more susceptible to hypotension.⁶⁵ A second IV infusion

should be established for administration of the non-dextrose containing preload. Epidural or spinal anaesthesia is also preferred for delivery, since both avoid the possibility of fetal/neonatal depression.

Datta and Brown⁶⁵ noted more severe acidosis in the infants of mothers having spinal anaesthesia for Caesarean section, than in a comparable group of mothers receiving general anaesthetics. Subsequently, they reported a 60 per cent incidence of neonatal acidosis (pH 7.20 or less) following epidural anaesthesia.⁶⁶ They postulated fetal compromise both from maternal hypotension and diabetes during regional anaesthesia. These mothers received D5W with lactated Ringer's solution as a preload. Datta *et al.*⁶⁷ repeated this work with three modifications:

- rigid diabetic control (maternal glucose 4.5–5.5 mMol·L⁻¹)
- prehydration with dextrose-free solutions, and
- "aggressive" management of maternal hypotension, with prompt IV ephedrine whenever a fall from baseline pressure occurred.

They observed no significant differences between spinal anaesthesia and GA groups, thus confirming the three important principles underlying anaesthetic care of the diabetic parturient. The fetal acidosis in their earlier cases probably resulted from maternal dextrose preloading, with resultant fetal lactic acidemia (see Ref. 57 for discussion).

In view of the abrupt fall, following delivery, in maternal insulin requirements, which may remain at zero for 48 h,⁶⁷ maternal glucose levels require careful monitoring in the early postpartum period.

Care of the neonate

Infants of diabetic mothers require immediate admission to a special care facility, usually for 24 h. Immediate investigations include blood gases (umbilical cord sample), blood glucose, and serum electrolytes to exclude neonatal acidosis, hyper- or hypo-glycaemia and hypocalcaemia. Elevated intra-partum maternal glucose levels promote fetal hyperinsulinaemia, and neonatal hypoglycaemia at 1 to 2 h of age. Investigations are therefore repeated at 30 min intervals, for 2 h, to monitor progress. Subsequent monitoring is dictated by clinical findings, and intensive observation may be necessary for 48–72 h.

"Diabetic" neonates also need careful observation for evidence of grunting, retraction and

tachypnoea, indicative of RDS. The incidence of RDS has declined significantly since the advent of amniocentesis and PG estimation. Some neonates show haemoconcentration, which, in its most severe form, may precipitate renal vein thrombosis.⁵⁷

With the specialist team approach outlined above, the perinatal mortality of diabetic pregnancies has declined, in some centres, to that of the general population, but it remains unacceptably high in areas which lack special facilities.⁵⁸

Summary

The pathophysiology, and principles of obstetric and anaesthetic management, of two high-risk situations have been reviewed. Information sources related to two additional problems are provided. The encouraging decline witnessed in perinatal morbidity and mortality in recent years, stems from multidisciplinary team care in tertiary centres. The obstetric anaesthetist who understands the pathophysiology of high-risk pregnancy, and the principles which underlie patient management, becomes an irreplaceable member of such perinatal care teams.

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