Occasional Review

Obstetrical anaesthesia for patients with the syndrome of haemolysis, elevated liver enzymes and low platelets

Edward T. Crosby BSc MD FRCPC

The syndrome of haemolysis, elevated liver enzymes and low platelets (HELLP Syndrome) is a consequence of severe preeclampsia/eclampsia. The clinical course is characterized by an unusual presentation with abdominal pain, and manifestations of inadequate haemostasis and excessive bleeding are common. Maternal and perinatal morbidity and mortality are high. We report our experience with 33 patients over a five-year period. The mean gestational age (GA) of the pregnancies was 34 ± 2.8 wk including 11 patients who delivered 12 neonates of less than 34 wk GA. The most common presenting complaints were right upper quadrant or epigastric pain in 25 patients (76%) and nausea or vomiting in 14 patients (42%). Diagnosis was missed or delayed in 12 patients (36%). Thirty-one patients (94%) were delivered by Caesarean section and a deteriorating maternal condition was the most common indication for operative delivery. Twenty-three patients received general anaesthesia, eight received epidural anaesthesia and there were no complications related to the anaesthetic. There was clinical evidence of abnormal haemostasis: seven patients had excessive blood loss at Caesarean section, two had postpartum haemorrhage, three developed DIC and four developed wound haematoma. The average decrease in haemoglobin concentration was $32 \text{ g} \cdot \text{L}^{-1}$ and twelve patients (36%) received blood transfusions. There was one stillbirth. There were no neonatal deaths but morbidity was prominent and related primarily to prematu-

rity. Delayed or missed diagnosis is common in HELLP syndrome and a premature delivery by Caesarean section is usual. HELLP syndrome represents a relative contraindication to regional anaesthesia and epidural anaesthesia should be restricted to patients without clinical evidence of bleeding, a platelet count $> 100 \times 10^9 \cdot \text{L}^{-1}$, and a normal bleeding time. A paediatrician should attend the delivery to provide care for the premature, often growth-retarded neonate. Following delivery the mother should be closely monitored for the development of haemorrhagic complications or eclampsia.

La prééclampsie sévère et l'éclampsie peuvent entraîner un syndrome incluant hémolyse, élévation des enzymes hépatiques et thrombocytopénie; c'est le « HELLP syndrome ». Il se présente souvent par des douleurs abdominales accompagnées de signes de coagulopathie; la morbidité et la mortalité maternelles et néonatales sont considérables. Durant les cinq dernières années, nous avons participé au traitement de 33 victimes de ce syndrome. Elles en étaient en moyenne à $34 \pm 2,8$ semaines de grossesse et 25 d'entre elles (76%) se présentèrent avec de la douleur à l'épigastre ou à l'hypochondre droit tandis que 14 (42%) avaient des nausées et/ou des vomissements. Trente et une (94%) patientes accouchèrent par césarienne, précipitée habituellement par une détérioration de leur état. Lors de la césarienne, on fit 23 à patientes une anesthésie générale et à huit, une péridurale. Il n'y eut aucune complication d'ordre anesthésique mais sept patientes perdirent beaucoup de sang lors de la césarienne, deux saignèrent en post-partum, trois développèrent une CIVD et quatre, un hématome. La concentration de l'hémoglobine dans le sang baissa en moyenne de $31 \text{ g} \cdot \text{L}^{-1}$ et douze patientes (36%) reçurent une transfusion sanguine. A part une mortinaissance, il n'y eut pas de décès en période néonatale toutefois, la morbidité découlant surtout de la prématurité était significative. Avec le HELLP, il est fréquent que le diagnostic soit posé tardivement et qu'une césarienne s'impose rapidement. Le HELLP constitue une contrindication relative à l'anesthésie régionale et on ne devrait

Key words

ANAESTHESIA: obstetric;

COMPLICATIONS: preeclampsia;

SYNDROMES: HELLP.

From the Department of Anaesthesia, Ottawa General Hospital, University of Ottawa, 501 Smyth Road, Ottawa, Ontario, Canada K1H 8L6.

Address correspondence to: Dr. Edward Crosby.

Accepted for publication 7th November, 1990.

réserver l'anesthésie péridurale qu'aux cas où rien ne suggère une coagulopathie, où le décompte plaquettaire est $>100 \times 10^9 \cdot L^{-1}$ et où le temps de saignement est normal. Lors de l'accouchement, un pédiatre devrait être présent pour s'occuper du prématuré et/ou du nouveau-né avec retard de croissance. En postopératoire, on devrait être à l'affût de complications d'ordre hémorragique et de l'éclampsie.

The acronym HELLP, coined by Weinstein, describes a syndrome of (H) haemolysis, (EL) elevated liver enzymes and (LP) low platelets, which appears to be a variant of severe preeclampsia/eclampsia.¹ The syndrome was first described as a clinical entity by Pritchard, who, in 1954, reported three cases, of whom only one survived.² The incidence of this syndrome among patients with severe preeclampsia/eclampsia varies from 4–12%.³ The common clinical features of HELLP syndrome are shown in Table I. Abdominal pain and upper gastrointestinal symptoms including nausea and vomiting are the predominant presenting complaints and, for this reason, diagnosis is often delayed.^{3–5} Maternal and perinatal morbidity and mortality are increased in pregnancies complicated by HELLP syndrome. The anaesthetic management of patients with HELLP syndrome is not well described. We report our experience with 33 patients with the syndrome, who presented for anaesthetic care over a five-year period.

Methods

The charts of all patients with the clinical diagnosis of HELLP Syndrome, presenting to our institution between April 1984 and August 1989, who received care from the Department of Anaesthesia, were retrieved and analyzed. The charts of the infants were identified and also

TABLE I Anaesthesia and HELLP Syndrome

Essential

thrombocytopaenia (platelet count $<100 \times 10^9 \cdot L^{-1}$)
elevated SGOT, SGPT
abnormal blood smear: haemolysis

Associated

abdominal pain
nausea and vomiting
headache and neurological dysfunction
renal insufficiency
intrauterine growth retardation
intrauterine fetal death
placental abruption
disseminated intravascular coagulation
premature delivery
postpartum haemorrhage

TABLE II Outline for data retrieval

1 Maternal demographics

age
obstetrical/medical history

2 Clinical presentation

Blood pressure
– at admission
– peak
– time to resolution of hypertension
– antihypertensive medications at discharge
Symptoms
– headache
– visual disturbance
– neurological dysfunction
– abdominal pain
– nausea and/or vomiting

3 Laboratory evaluation

Haemoglobin concentration
– at admission
– lowest
– change (admission – lowest)
Platelet count
– at admission
– lowest
– time to resolution of thrombocytopaenia
PT, PTT, fibrin split products
– at admission
– any abnormal value recorded
Biochemistry: (SGOT, LDH, uric acid, BUN, creatinine)
– at admission
– time to normalization

4 Delivery

– mode of delivery
– indications for operative delivery
– complications and morbidity

5 Anaesthesia

– type of anaesthesia
– invasive haemodynamic monitoring
– complications and morbidity

6 Haemorrhagic complications

– events and incidence of events
– treatment provided
– morbidity

7 Transfusion therapy

– blood components
– indications
– response

8 Neonate

– condition at birth
– morbidity and mortality

reviewed. Data were retrieved from the maternal and neonatal charts and the anaesthetic records using the outline presented in Table II. Statistical analysis of the data was carried out with the Chi-square test (with Yates correction) or the Fisher exact test. Statistical significance was assumed when $P < 0.05$.

Results

Demographic data

Thirty-three patients with HELLP syndrome received anaesthetic care during the study period. This represented 0.3% of the total obstetrical population delivered during the period and 15% of the preeclamptic/eclamptic group. There were no maternal deaths. The average age of the patients was 26.5 yr (± 4.2 yr), 17 were primigravida, the remaining 16 multigravida. Of the 16 multigravid patients, nine had had previous abortions, seven spontaneous, two therapeutic. One patient had previously delivered a stillborn at 28 wk gestation. Only six of the 16 multigravid

patients had previously delivered a liveborn, term infant. The mean gestational age was 34 wk (± 2.8 range 27–39 wk) and 11 patients were less than 34 wk gestational age. One patient had a history of chronic hypertension and no patient had experienced toxemia during a previous pregnancy. The signs, symptoms and laboratory indices at the time of admission are summarized in Table III. Blood pressures were highly variable and there was no correlation between blood pressure and severity of thrombocytopenia nor did the degree of thrombocytopenia predict intraoperative or postoperative bleeding complications. Epigastric and right upper quadrant (RUQ) pain were prominent presenting complaints and in six patients (18%), the diagnosis of preeclampsia complicated by HELLP syndrome was delayed. The patients were diagnosed initially as having oesophagitis (three patients), peptic ulcer disease (one patient) or nonspecific gastrointestinal pain (two patients). One patient convulsed before delivery and one complained of visual impairment and was found to have bilateral retinal detachments.

TABLE IIIa HELLP Syndrome – Clinical Presentation

Blood pressure mmHg	Mean $\pm$ SD	Range
Systolic	161 $\pm$ 21	(120–220)
Diastolic	101 $\pm$ 11	(79–125)
Mean	122 $\pm$ 13	(95–150)
Symptoms	n	%
Headache	6	18
Visual disturbances	2	6
RUQ pain	25	76
Nausea/vomiting	14	42
Eclampsia	1	3
Oedema – dependent	16	48
– generalized	2	6

Coagulation abnormalities

Fibrin degradation products (FDP) were noted in the serum of four patients (13%) before delivery and an additional 12 patients (36%) after delivery. In seven of the 16 patients who tested positively for FDP, the levels were at the lowest limits detectable by our laboratory. However, in three patients postpartum there was clinical evidence of disseminated intravascular coagulation and in these three patients FDP's were markedly elevated.

Caesarean section and anaesthesia

Thirty-one patients (94%) were delivered by Caesarean section, 23 because of the maternal condition and eight for fetal welfare. In three patients Caesarean section followed

TABLE IIIb HELLP Syndrome – Laboratory Indices at Presentation

	n	Mean $\pm$ SD	Range	Abnormal patch %
Haemoglobin g $\cdot$ L ⁻¹	33	128 $\pm$ 11	(110–163)	
Platelet count (PLT) $\times 10^9 \cdot$ L ⁻¹	33	108 $\pm$ 55	(23–234)	
Lowest PLT $\times 10^9 \cdot$ L ⁻¹	33	50 $\pm$ 30	(15–146)	
SGOT U $\cdot$ L ⁻¹	33	250 $\pm$ 165		100
LDH U $\cdot$ L ⁻¹	33	542 $\pm$ 237		97
Urate μ mol $\cdot$ L ⁻¹	30	378 $\pm$ 130		33
Creatinine μ mol $\cdot$ L ⁻¹	27	85 $\pm$ 32		7
BUN mmol $\cdot$ L ⁻¹	27	4.75 $\pm$ 1.5		0
FDP	27			15
PT	26			12
PTT	20			12

failed induction of labour. Two patients were delivered by forceps. Twenty-three patients were administered general anaesthesia and eight patients received epidural anaesthesia for Caesarean section. The two patients delivered with forceps were provided with local infiltration for delivery. One epidural catheter had been placed in a referring institution and the remaining seven were placed in our institution. At the time of catheter insertion, the diagnosis of HELLP syndrome had not been made in six of these seven patients. The average platelet count was higher in the patients who received epidural anaesthesia compared with those who were given general anaesthesia, $140 (\pm 46) \times 10^9 \cdot L^{-1}$ vs $96 (\pm 53) \times 10^9 \cdot L^{-1}$, respectively. Abnormal bleeding was remarked upon in seven of the patients (23%) undergoing Caesarean section, wound haematoma developed in four (13%) and two patients (7%) had postpartum haemorrhage. In two patients a placental abruption was seen at Caesarean section. There was no difference in the platelet counts in those patients with clinical evidence of abnormal haemostasis compared with those without.

In one patient an arterial line had been inserted prior to induction of anaesthesia and in one patient a central line was inserted preoperatively to monitor fluid therapy. No other invasive monitors were used. Twelve patients (52%) received *iv* lidocaine or fentanyl or both to blunt the hypertensive response to tracheal intubation. All patients underwent rapid sequence induction of anaesthesia, with cricoid pressure, no intubation was deemed to be difficult and the tracheas of all patients were extubated in the operating room at the end of the surgery. There were no complications related to the anaesthetic in any patient.

Transfusion therapy

Eight patients (26%) were transfused platelets (6–8 units). In four patients there was either no increase or a decrease in subsequent platelet count (PCT) and in the other four there was an increase in PCT averaging $44 \times 10^9 \cdot L^{-1}$. Twelve patients (36%) received transfusions of packed red cells perioperatively, ten in the postpartum period for low haemoglobin concentration ($83 \pm 10 \text{ g} \cdot L^{-1}$, range 66–96 $\text{g} \cdot L^{-1}$), one during surgery and one in the recovery room because of intraoperative blood loss. Two to four units were transfused to each patient, with the mean transfusion requirement being 2.9 units. One patient was transfused two units of FFP intraoperatively because of persistent oozing. There was an average $32 \pm 14 \text{ g} \cdot L^{-1}$ decrease in haemoglobin concentration perioperatively. There was no difference in the decrease in haemoglobin concentration between patients undergoing Caesarean section ($32 \pm 14 \text{ g} \cdot L^{-1}$) and those who delivered vaginally ($33 \pm 5 \text{ g} \cdot L^{-1}$).

TABLE IV Neonatal demographic features

	<i>Mean ± SD</i>		<i>Range</i>	
Weight	2014 ± 738 g		830–3620 g	
Platelet count	(n = 24) 232 ± 73 10 ⁹ · L ⁻¹		145–378 10 ⁹ · L ⁻¹	
Apgar scores				
	<7		≥7	
	<i>n</i>	%	<i>n</i>	%
One minute	10	30	23	70
Five minutes	1	3	32	97
Morbidity				
	<i>n</i>	%		
RDS	7	21		
BPD	2	6		
IUGR	6	18		

Key: RDS – respiratory distress syndrome, BPD – bronchopulmonary dysplasia, IUGR – intrauterine growth retardation.

Neonates

Thirty-four neonates were delivered. There was one stillbirth at 36 wk gestation (a recognized intrauterine death) and one patient delivered twins at 34 wk gestation. Three infants, at 27, 28 and 34 wk gestational ages, were transferred to a regional neonatal intensive care facility for specialized care. Their charts were retrieved to allow inclusion of their data in this review. The demographic features of the neonates is presented in Table IV. Only one neonate had a platelet count of less than $100 \times 10^9 \cdot L^{-1}$. There were no neonatal deaths and neonatal morbidity is outlined in Table IV. The morbidity was related primarily to prematurity rather than to the maternal condition at the time of birth.

Postpartum course

After delivery, blood pressure returned to normal, in most patients, by the third or fourth postpartum day (Figure 1). Six patients (18%) were discharged on antihypertensive therapy. The platelet count continued to decrease to a nadir at 24–36 hr after delivery and then began to increase to normal. The time required for the platelet count to return to levels above $100 \times 10^9 \cdot L^{-1}$ ranged from one to nine days, with the majority of patients having achieved such levels by the third to fourth day (Figure 2). Three patients developed postpartum DIC, were treated with blood component infusions and the coagulopathy resolved. The elevated liver enzyme concentrations began to decrease within 24 hr of delivery and had returned to

Number of Patients

FIGURE 1 Time to resolution of hypertension. This figure shows the number of patients ($n = 33$) who remained hypertensive at the start of each postpartum day. Six patients were discharged home on antihypertensive therapy.

normal in most patients before discharge from hospital. One patient was found to have retinal detachments before delivery and these were resolving at the time of discharge and had resolved at follow-up in the ophthalmology clinic.

Discussion

Patients with preeclampsia exhibit a spectrum of pathophysiological changes and one manifestation of the disease is HELLP syndrome characterized by haemolysis, severe thrombocytopenia and abnormal liver function. The decrease in circulating platelets is secondary to an increased rate of consumption.¹ Platelets adhere to collagen, exposed at the sites of damaged vascular endothelium. A microangiopathic haemolytic anaemia is present to some degree in all patients with HELLP syndrome and examination of the peripheral blood film is likely to reveal the presence of burr cells, schistocytes and polychromasia. Intravascular deposition of fibrin in the sinusoids of the liver results in obstruction of blood flow, liver distention, right upper quadrant or epigastric pain and elevation of liver enzymes. If the intrahepatic pressure exceeds the ability of Glisson's capsule to distend, liver rupture occurs and this has been reported in patients with HELLP syndrome.⁶

The incidence of HELLP syndrome in pregnancies complicated by preeclampsia varies from 4–12% and in our study was 15%. Our centre is a regional referral centre for obstetrics and 50% of our HELLP patients were referred from other centres, possibly accounting for our higher incidence. Maternal mortality was as high as 66%

Number of Patients

FIGURE 2 Time to resolution of thrombocytopenia. This figure shows the number of patients ($n = 33$) whose platelet counts remained below $100 \times 10^9 \cdot L^{-1}$ at the start of each postpartum day.

in early reports and Sibai, reporting on 112 patients with HELLP syndrome, recorded two deaths (1.8% maternal mortality).^{2,3} There were no maternal deaths in our series. Reported perinatal mortality rates have been high and Sibai reported a rate of 367 per 1000 deliveries with 22 stillbirths and 16 neonatal deaths.³ With one stillbirth in our series, and no neonatal deaths, our corrected perinatal mortality was 29 per 1000 deliveries. In Sibai's series 41% percent of the infants were born at <30 wk gestational age compared with 5.8% of the infants in our group and this probably accounts for our lower mortality.

Delayed or missed diagnosis has been a common occurrence in reports of HELLP syndrome.³⁻⁵ This has been attributed to the unusual features of presentation, with impressive abdominal pain, nausea and vomiting. As well, the classical signs of preeclampsia (hypertension, proteinuria and oedema) may be unimpressive. In our series, six patients (18%) who presented with epigastric or RUQ pain were initially diagnosed as having a condition other than preeclampsia with HELLP syndrome (missed diagnosis). Six of seven patients who received epidural anaesthesia were not diagnosed at the time that the epidural catheter was placed (delayed diagnosis). This has obvious implications for the anaesthetist engaged in obstetrical anaesthesia practice in that patients may present for anaesthetic care without a correct diagnosis having been made.

Sibai reported an incidence of DIC of 38%; 21% of his patients demonstrated prolonged prothrombin and partial thromboplastin times compared with 9% and 12%, respectively, in our series.³ Twenty per cent of the patients in Sibai's series had placental abruption and 19%

had *in utero* fetal death and the high incidence of coagulopathy may have been influenced by these abnormalities. There was only one *in utero* death (3%) and two placental abruptions (6%) in our series.

The infusion of platelet concentrates into thrombocytopenic patients without clinical evidence of bleeding is controversial.⁶ The pathophysiological process in HELLP syndrome is one of accelerated platelet consumption and the transfused platelets are likely to be consumed as quickly as native platelets, providing no therapeutic advantage. Also, there is some concern that the consumptive process may be accelerated and DIC may result.⁶ In eight patients who were transfused platelet concentrates preoperatively, the platelet count was either the same or decreased in four and increased somewhat in the other four. There appears to be little reason to delay operation to provide prophylactic platelet transfusions in patients with HELLP syndrome in the absence of clinical bleeding. However, because of the high, although variable, reported incidence of DIC, the availability of cross-matched blood, platelet concentrates and plasma should be assured before taking the patient to the operating room.

Although none of our patients who received epidural anaesthesia experienced a recognized complication, Sibai reported one patient with persistent bleeding into the epidural space after epidural anaesthesia.³ The patients in our series who received epidural anaesthesia had higher platelet counts at the time of operation than the patients who received general anaesthesia. Bleeding times (BT) were not performed routinely on the patients in our series. Prolonged BT is a common laboratory abnormality in preeclampsia, even in patients with normal platelet counts, and does not seem to predict abnormal haemostasis or excess bleeding in otherwise uncomplicated preeclamptic pregnancies.⁷ However, the BT appears to have its greatest utility in evaluation of a patient with active bleeding, with a well-documented history of bleeding or with a clinical condition that predisposes the patient to excess blood loss.⁸ A bleeding time is recommended in the patient with HELLP syndrome, before the insertion of an epidural catheter.

Invasive monitoring was little utilized in the management of the patients in our series. HELLP Syndrome, by itself, is not an indication for invasive monitoring and it is the severity of the underlying preeclampsia that will determine the need. An arterial line is useful in the parturient in whom repeated blood sampling is required, who is receiving potent vasodilators for control of blood pressure or in those patients whose blood pressure is controlled poorly. Preeclamptic women have smaller plasma volumes than normal pregnant women but this appears to be especially important in women with severe disease or with growth retarded fetuses (IUGR).⁹ Use of

invasive central monitoring should reflect this and so, in women with mild to moderate preeclampsia and normally growing fetuses, there is little indication for invasive central monitoring. However, in severe preeclampsia, and especially if complicated by oliguria, pulmonary oedema or underlying cardiac pathology, a role for central venous monitoring has been established. The pulmonary artery catheter should be used to guide therapy of women with severe unresponsive hypertension, oliguria resistant to fluid therapy with appropriate CVP or signs and symptoms of pulmonary oedema.¹⁰

Recommendations for anaesthetic management

Because of the high incidence of missed or delayed diagnosis of HELLP syndrome, the anaesthetist should have a high index of suspicion and recognize that a parturient presenting with abdominal pain, nausea and vomiting may have HELLP syndrome. For patients with known or suspected HELLP syndrome presenting for Caesarean section, preoperative assessment must include a recent complete blood count with platelet count, liver function tests, a prothrombin and partial thromboplastin time and fibrin degradation products. Blood components including crossmatched red cells, platelet concentrates and plasma should be immediately available.

Premature delivery is usual in HELLP syndrome. The pregnancies are complicated by intrauterine growth, retardation and placental abruption. The presence of a neonatologist/paediatrician to care for the neonate and equipment and drugs to resuscitate the neonate are mandatory.

The anaesthetic management of the patient with HELLP syndrome is modelled on that for the underlying preeclamptic condition.^{9,11,12} An assessment of intravascular volume and the adequacy of blood pressure control, as well as the need for invasive haemodynamic monitoring should be made. The intravascular volume depletion usually correlates with the severity of the hypertension. Invasive monitoring should be restricted to those patients outlined above. The diagnosis of HELLP syndrome is a relative, if not absolute, contraindication to the use of epidural anaesthesia. If the clinical history and examination is negative for evidence of abnormal haemostasis and both the platelet count and the bleeding times are within the normal range, then an epidural block may be offered. Because the platelet count often continues to decrease postpartum and bleeding complications are common in patients with HELLP Syndrome, it is prudent to remove the epidural catheter as soon as possible after delivery. Induction of general anaesthesia should follow intravascular volume replacement and pay heed to both the need for airway protection with cricoid pressure as well as the marked lability of blood pressure and exaggerated haem-

odynamic response to intubation in the preeclamptic patient.

Finally, the patient should be monitored to detect the development of postpartum haemorrhagic complications, DIC and eclampsia. In most patients, platelet count, blood pressure and liver function should return to normal by the fourth postoperative day.

Provision must be made for both the care of the mother and a premature, often growth-retarded, infant. Maternal and perinatal morbidity is likely to be higher than normal and the challenge to the perinatal team (obstetrician, anaesthetist and paediatrician) is to cooperate in order to optimize care and reduce both mortality and morbidity.

Acknowledgements

The author would like to thank Dr. R.D. Elliott for reviewing the manuscript and offering useful criticism and acknowledge the assistance of Health Information Analysts Lori Greenberg and Pauline Saumure as well as the secretarial skills of Sylvie Paquette in the preparation of this manuscript.

References

- 1 Weinstein L. Syndrome of hemolysis, elevated liver enzymes, and low platelet count: a severe consequence of hypertension in pregnancy. *Am J Obstet Gynecol* 1989; 142: 159–67.
- 2 Pritchard JA, Weisman R, Ratnoff OD, Vosburgh GJ. Intravascular hemolysis, thrombocytopenia and other hematologic abnormalities associated with severe toxemia of pregnancy. *N Engl J Med* 1954; 250: 89.
- 3 Sibai BM, Taslimi MM, El-Nazer A, Amon E, Mabie BC, Ryan GM. Maternal-perinatal outcome associated with the syndrome of hemolysis, elevated liver enzymes, and low platelets in severe preeclampsia-eclampsia. *Am J Obstet Gynecol* 1986; 155: 501–9.
- 4 Thiagarajah S, Bourgeois FJ, Harbert GM, Caudle MR. Thrombocytopenia in preeclampsia: associated abnormalities and management principles. *Am J Obstet Gynecol* 1984; 150: 1–7.
- 5 Weinstein L. Preeclampsia/eclampsia with hemolysis, elevated liver enzymes, and thrombocytopenia. *Obstet Gynecol* 1985; 66: 657–60.
- 6 Olan P, Maltau JM, Aabyholm T. HELLP Syndrome – a serious complication of hypertension in pregnancy. *Acta Obstet Gynecol Scand* 1984; 63: 727–9.
- 7 Ramanathan J, Sibai BM, Vu T, Chauhan D. Correlation between bleeding time and platelet counts in women with preeclampsia undergoing cesarean section. *Anesthesiology* 1989; 71: 188–91.
- 8 Lind SE. Prolonged bleeding time. *Am J Med* 1984; 77: 305–12.
- 9 Norris MC. Preeclampsia. In: Kirby RR, Brown DL (Eds.). *Problems in Anesthesia*, Hood DD (Guest Ed.). *Anesthesia for Obstetrics and Gynecology*, Philadelphia: J.B. Lippincott Company, 1989; 3: 90–111.
- 10 Clark SL, Cotton DB. Clinical indications for pulmonary artery catheterization in the patient with severe preeclampsia. *Am J Obstet Gynecol* 1988; 158: 453–8.
- 11 Morison DH. Anaesthesia and preeclampsia. *Can J Anaesth* 1987; 34: 415–22.
- 12 Marx GF, Bassell CM. Anesthetic considerations in hypertensive disorders of pregnancy. In: Gallagher TJ (Ed.). *Advances in Anesthesia*, Volume 1. Chicago: Year Book Medical Publishers, 1984; 237–87.