Life-Threatening Complications in Obstetrical Patients

羅良明　醫師

Relevant Physiology of Pregnancy

- Cardiovascular Changes
- Coagulation
- Effect of Delivery
- Uteroplacental Effects
- Anatomic Changes

Physiologic Changes That Affect Resuscitation on Pregnancy

- Uteroplacental vasoconstriction in response to hypercarbia and hypoxia
- Increased minute ventilation and decreased lung residual volume
- Normal PCO_2 of 30 to 32 mm Hg with physiologic "hyperventilation"
- Expanded plasma volume and physiologic anemia
- Hypercoagulability: increased levels of multiple coagulation factors and fibrinogen
- Lack of autoregulation of uterine blood flow in response to changing maternal blood pressures
- Obstruction of venous return in the supine position after 20 weeks' pregnancy
- Uterine vasoconstriction in response to α-adrenergic stimulation and maternal hypovolemia
- Impaired fetal carbon dioxide excretion with maternal hypercarbia
- Generalized smooth muscle relaxation and increased abdominal pressure with risk of aspiration.

Anatomic and Physiologic Effects of Pregnancy Affecting Closed-Chest Cardiopulmonary Resuscitation

- Increased blood volume
- Increase cardiac output
- Decreased peripheral vascular resistance, except in toxemic syndromes
- Enlarging uterus and supine position
  - Decreased compliance for artificial ventilation
  - Decreased compliance for thoracic compression
  - Uterine compression of aorta and inferior vena cava
    - » Decreased venous return
    - » Aortolilac occlusion with decreased renal and uterine arterial flow
- Increased oxygen consumption
- Increased rate of acid metabolite production

Principles of Basic and Advanced Life Support

- Early Cardiopulmonary Resuscitation
- Early Defibrillation
- Early Establishment of Airway and Ventilation
- Medications to Restore Circulation and Coronary Perfusion Pressure
- Discovery and Treatment of Reversible Causes of Cardiac Arrest
- Perimortem Cesarean Section

Potentially Reversible Caused of Cardiopulmonary Arrest in Pregnancy

Cause	Setting	Intervention
Pulmonary embolus	Late pregnancy Peripartum Hypoxia,cyanosis,respiratory disease	Oxygen, CPR Consideration of thrombolytics
Amniotic fluid embolus	Labor Uterine evacuation or procedure	Inotropic support Coagulation factor replacement
Hypovolemia	External blood loss Suspected abruptio placentae GI bleeding; ruptured liver, spleen, or other bleeding source	Fluid bolus:500-1000 mL NS or RL Coagulation factor support
Anaphylaxis	Anesthetic or IV pharmacologic agents Radiographic dye procedure	Fluid bolus:500-1000 mL NS or RL IV epinephrine
Tension pneumothorax	Intubation, positive-pressure ventilation	Chest decompression
Pericardial tamponade	Chronic renal failure, TB	Fluid bolus, 500 mL NS or RL Needle aspiration of pericardium
Sepsis	Infection: pyelonephritis; pelvic, septic embolic	Oxygen, CPR, bicarbonate
Primary respiratory failure	Magnesium toxicity; pulmonary infection or emboli or congestive heart failure	Positive-pressure ventilatory support
	Seizure, eclampsia, intracranial bleeding	Suctioning, hyperfentilation

Methods of Monitoring

- Oxygen Saturation Monitoring
- Hemodynamic Monitoring of Cardiac Output
- Invasive Pressure Monitoring
- Capnometry

Indications for Pulmonary Artery Catheterization

1. Hypovolemic shock that is unresponsive to initial volume resuscitation attempts
2. Septic shock when vasopressor therapy is needed
3. Pregnancy-induced hypertension complicated by unresponsive oliguria
4. Ineffective intravenous antihypertensive therapy
5. Adult respiratory distress syndrome requiring ventilatory support
6. Cardiac disease, class 3 or 4, in labor or requiring surgery
7. Anaphylactoid syndrome of pregnancy (amniotic fluid embolism)
8. Isolated primary or secondary pulmonary hypertension in labor or during surgery (selective application)
9. Pulmonary edema, from any etiology, that is unresponsive to initial therapy

(ACOG, 1992)

Normal Central Hemodynamic Parameters in Healthy Nonpregnant and Pregnant Patients

	Nonpregnant	Pregnant
Cardiac output (L/min)	4.3 ± 0.9	6.2 ± 1.0
Heart rate (beats/min)	71 ± 10	83 ± 10
Systemic vascular resistance (dyne × cm × sec^{-5})	1530 ± 520	1210 ± 266
Pulmonary vascular resistance (dyne × cm × sec^{-5})	119 ± 47	78 ± 22
Colloid oncotic pressure (mm Hg)	20.8 ± 1.0	18.0 ± 1.5
Colloid oncotic pressure-pulmonary capillary wedge pressure (mm Hg)	14.5 ± 2.5	10.5 ± 2.7
Mean arterial pressure (mm Hg)	86.4 ± 7.5	90.3 ± 5.8
Pulmonary capillary wedge pressure (mm Hg)	6.3 ± 2.1	7.5 ± 1.8
Central venous pressure (mm Hg)	3.7 ± 2.6	3.6 ± 2.5
Left ventricular stroke work index (gm × m × m^{-2})	41 ± 8	48 ± 6

Estimated Fluid and Blood losses based on patient's initial presentation

	Class I	Class II	Class III	ClassIV
Blood loss,ml	Up to 750	750-1500	1500-2000	>2000
Blood loss,%BV	Up to 15%	15-30%	30-40%	>40%
Pulse rate	<100	>100	>120	>140
Blood pressure	Normal	Normal	Decreased	Decreased
Pulse pressure, mmHg	Normal or increased	Decreased	Decreased	Decreased
Respiratory rate	14-20	20-30	30-40	>35
Urine output(ml/h)	>30	20-30	5-15	Negligible
CNS/mental status	Slightly anxious	Mildly anxious	Anxious and confused	Confused and lethargic
Fluid replacement, 3:1rule	Crystalloid	Crystalloid	Crystalloid and blood	Crystalloid and blood

Clinical Staging of Hemorrhagic Shock By Volume of Blood Loss

Severity of Shock	Findings	Blood Loss (%)	Volume (mL) *
None	None	Up to 20	Up to 900
Mild	Tachycardia (<100 beats/min) Mild hypotension Peripheral vasoconstriction	20-25	1200-1500
Moderate	Tachycardia (100-120 beats/min) Hypotension (80-100 mm Hg) Restlessness Oliguria	30-35	1800-2100
Severe	Tachycardia (>120 beats/min) Hypotension (<60 mm Hg) Altered consciousness Anuria	>35	>2400

Main Conditions Causing Critical Illness in Pregnancy

- **Preeclampsia and its variants**
  - **Eclampsia***
  - **HELLP**
- **Thrombotic thrombocytopenic purpura**
- **Acute fatty liver**
- **Hemorrhage***
- **Sepsis***
- **Thromboembolism***
- **Cardiac problems***
  - **Arrythmias**
  - **Cardiomyopathy**
- **Neurologic problems***
- **Trauma**
- **Metabolic**
- **Anaphylactoid syndrome of pregnancy***
- **Others**

Litigation in Obstetrics

A perfect baby is the expectation of the parents. A perfect outcome is the mission of obstetricians.

產科醫師今日與明日面臨的挑戰

- 肩難產的預防、 胎兒體重的預估
- 子宮破裂
- 羊水栓塞；懷孕羊水過敏性休克反應
- *HELLP syndrome*；the “great masquerader” *孕產婦可怕的變臉殺手*
- 胎兒肢體缺損的產前診斷
- 植入性胎盤的產前診斷與處理
- 胎兒心臟超音波為產前診斷最困難的一部分，尤其是診斷心房心室中隔缺損，罕見複雜性心臟疾病或血管結構異常心臟疾病
- 罕見遺傳基因或代謝疾病及染色體異常之產前診斷
- 神經管缺損疾病及腦部異常之產前診斷
- 胎兒異常之正確產前診斷

肩難產

- 產科生產最恐怖併發症之一。
- **Unexpected** 無法預期它的發生，**Unpredictable** 無法預測它的產程進展結果，**Unpreventable**無法預防(無法準確預估體重)， **Unacceptable** (病人及醫護人員)無法接受。

肩難產

- 發生率 0.2 - 1.4%
- Greater shoulder-to-head and chest-to-head disproportions. The mean head-to-body delivery time in normal births was 24 seconds compared with 79 seconds in those with shoulder dystocia.
- Maternal consequences: PPH usually from uterine atony, vaginal, cervical lacerations and puerperal infection.
- Fetal consequences (25% fetal injuries): transient (66%) or persistent (1.55%) brachialplexus palsies, clavicular fracture(38%), humeral fractures (17%), neonatal death (1/285 victims).

OB-GYN AND THE LAW
Can we anticipate shoulder dystocia (one of obstetrician's greatest fears) ?

- Problems in detecting fetal macrosomia. Brachial nerve palsy : an unavoidable injure?
- US EFW not accurate enough to diagnose macrosomia effectively : The absolute error of EFW formulas ranges 7-10% within 1 week of delivery,may be as high as 15% (sensitivity 28-65%, PPV 70%). Thus, when calculate on a 4000-g infant, the margin of error is 600g.
- No perfect model for prediction dystocia.
- C/S of the macrosomic fetus does not guarantee the absence of birth injury, including brachial nerve palsy.

Should dystocia (ACOG 1997, 2000 EBM)

- Most cases cannot be predicted or prevented because there are no accurate methods to identify which fetuses will develop this complication.
- US measurements to estimate macrosomia have limited accuracy.
- Planned C/S based on suspected macrosomia is not a reasonable strategy.
- Planned C/S may be reasonable for the nondiabetic with an EFBW >5000 g (4500 g Taiwan) or DM >4500 g (4000 g)

Uterine rupture
子宮破裂

- **Incidence 0.035% (1/2808; 42/117685, AJOG 2003; 189 : 1042-6)**
- 危險因素：
- **Previous cesarean section (OR=6.0, 95% CI 3.2-11.4); uterine rupture rates 24.5/1000 in VBAC with PGs-induced labor, while lower rates 5.2/1,000 and 7.71/1,000 with spontaneous labor and without PGs.**
- **Malpresentation (OR = 5.4, 95% CI 2.7- 10.5)**
- **Second-stage dystocia (OR=13.7, 95% CI 6.4-29.3)**

Uterine rupture

- Risk of uterine rupture 0.8%-1.1% with one prior cesarean section.
- 9.7% labor induced with misoprostol (Aslan H et al.)
- Accident of uterine rupture on unscarred uterus following labor induction with misoprostol occurs in 0.1% to 1% and only rare cases have been attributed to the use of misoprostol.
- Clinicians should be particularly careful in patients with prior cesarean section or a weakened uterus.
- ACOG committee opinion: The risk of uterine rupture during VBAC attempts is substantially increased with the use of various prostaglandins, and their use for this propose is discouraged.

Uterine Rupture: Are CTG useful in the diagnosis

- ✓ CTG is of limited or no vlaue with silent uterine rupture, which is difficult to diagnose. Warning signal are often absent and the rupture is clinically suspected only it has occurred.
- ✓ The classic description of a decrease in uterine tone or the cessation of labor at the time of uterine rupture has been refuted by intrauterine pressure monitoring data.
- ✓ The most common single warning signal: abnormal FHR patter.

羊水栓塞

(舊名稱Amniotic fluid embolism)；

懷孕羊水過敏性休克反應

（新名稱anaphylactoid syndrome of pregnancy: 急性生產前後缺氧, 心肺功能衰竭及DIC瀰漫性凝血出血病變）

- Unexpected 無法預期它的發生，Unpredictable 無法預測它的病程進度，Unpreventable無法預防， Unacceptable (病人及醫護人員)無法接受。
- 發生率極低，約8,000到83,000次生產當中才有一名羊水栓塞的病例。
- 死亡率極高，文獻報導有高達86% ，約佔母親生產5-18% 。
- 台灣每年約有二十萬生產人次，母親死亡率6-8/100,000婦女因懷孕生產發生死亡，每年平均二十至三十人間，死於羊水栓塞的病人估計大約為三至六人。

羊水栓塞急救處理原則

- 左側躺輕頭低腳高姿勢低於100%高濃度氧氣
- 維持動脈血氣PO_2>60mmHg, 血紅素氧氣飽和度90%以上
- 使用升壓藥物，如ephedrine，dopamine，dobutamine，維持收縮壓在90mmHg以上
- 使用高劑量類固醇hydrocortisone 500mg IV q 6 hours
- 最後一步救命絕招，體外心肺循環機extracorporeal membrane oxygenation and intra-aortic balloon counterpulsation. Anaphylactic reaction induced by AFE may be transient and resolved within few hrs. Cardiopulmonary bypass plays a major role in saving the lives of patients by obtaining a few critical hours to maintain the CV status (replacement of a dysfunctional cardiopulmonary system).

HELLP Syndrome : the “great masquerader”

can be a challenge to diagnose because the signs and symptoms of preeclampsia may appear late in its disease course or not at all and because the patient’s presentation can resemble other disorders. An accurate diagnosis is essential. Imitators of the “great masquerader”, HELLP syndrome include acute fatty liver of pregnancy (AFLP), thrombotic thrombocytopenic purpura (TTP), and hemolytic uremic syndrome (HUS)

HELLP syndrome診斷具有挑戰性，因為妊娠毒血症的症狀，在疾病中晚期才顯現出來，有些病例甚至病發前沒有任何警訊。病人表現出來的症狀呈現變化多端具有“孕產婦可怕變臉殺手”稱號。

植入性胎盤

產科出血主要原因，以植入性胎盤之產前診斷與處置是產科醫師面臨的最大臨床挑戰工作。由於近年來台灣地區婦女接受人工流產人數日漸增多及超高的剖腹生產率，因此吾人預期懷孕婦女合併植入性胎盤之病例數也會日漸增加。

Color Doppler signs suggestive of placenta accreta

- Dilated vascular channels with diffuse lacunar flow
- Irregular vascular lakes with focal lacunar flow
- Hypervascularity linking placenta to bladder
- Dilated vascular channels with pulsatile venous flow over cervix
- Poor vascularity at sites of loss of hypoechoic zone

Source: modified by Haratz-Rubinstein et al.-(Contemporary OB/GYN April 2002) from Chou MM et al. Ultrasound Obstet gynecol 2000 ; 15 : 28-35

Robert Resnik Managing placenta accreta Contecporary OB/GYN Nov. 2001 CME Credit

我們必須強調超音波是一種「影像」檢查，它不是萬能的檢查工具，必有其限制及盲點存在。譬如一些罕見先天代謝異常及遺傳基因疾病如無法合併其它器官型態異常，超音波產前診斷有其困難度，其次我們也必須指出胎兒異常的出現有其發生時間順序，例如：懷孕初期檢查一切正常，卻在懷孕中期時突然發現水腦出現，家屬往往會質疑是否延誤診斷。

The incidence of IUFD after reassuring biophysical profile

(8 or more) is 0.726/1000.

Parents must not expect the perfect baby and obstetricians must accept that the CTG does not always accurately reflect fetal condition.

Catastrophic intrapartum events that may proceed too rapidly for actionto succeed in avoiding serious sequelae

- Sudden large placental abruption.
- Massive feto-maternal hemorrhage.
- Complete cord occlusion.
- Prolapsed cord
- Ruptured uterus
- Ruptured vasa previa
- Sudden development of a terminal fetal bradycardia with little preceding CTG warning.

HELLP Syndrome: The State of the Art

Jason K. Baxter, MD, and Louis Weinstein, MD

Preeclampsia/eclampsia has been recognized for centuries and continues to plague both the patient and the obstetrician. A severe variant, the syndrome of hemolysis, elevated liver enzymes, and low plateletes (HELLP), has been recognized for 50 years. Although much new data has been elucidated about the conditions, only several observations have withstood the test of time. These are the uniqueness of the disease to humans, the progressive nature of the disease, and the fact that delivery is the sole therapy.

(SOURCE : Obstetrical and Gynecological Survey, pp838-845,Volume 59, Number 12 ,2004)

HELLP Syndrome孕產婦可怕變臉殺手

- Peak LDH>peak SGOP>SGPT
- SGOP>2000 IU/L or LDH>3000 IU/L mark patients at high risk for maternal death (FFP or plasmapheresis may be lifesaving in such cases)
- The lowest platelet count and the peak SGOT and LDH values occurred within 72-96 hours after delivery.
- The normal time course for resolution of lab abnormalities is about 4 days, and delayed recovery need to be evaluated for additional causes
- Although HELLP syndrome takes and indolent course (病程進展緩慢), in some patients, the disease may progress with astonishing and frightening rapidity, and culminate in maternal death. (病程進展惡化快速驚人，造成孕產婦死亡)

Pathogenesis

The HELLP syndrome probably represents a severe form of preeclampsia, but this relationship remains controversial. As many as 15 to 20 percent of patients do not have antecedent hypertension or proteinuria, leading some experts to believe that HELLP is a separate disorder from preeclampsia.

Fetuses of some affected women have long-chain 3-hydroxyacyl-coenzyme A dehydrogenase deficiency (LCHAD), which is more commonly a major cause of acute fatty liver of pregnancy. An association with medium-chain acyl-CoA dehydrogenase deficiency was also reported.

Corticosteroids

Initial observational studies and small randomized trials suggested use corticosteroids may be associated with more rapid improvement in laboratory and clinical parameters. These findings were not supported by a subsequent large, well-designed randomized, double-blind, placebo-controlled clinical trial evaluating the use of dexamethasone to improve maternal outcome in patients with HELLP syndrome.

Corticosteroids do no appear to be of value in treatment of women with HELLP syndrome other than for acceleration of fetal lung maturity. Future use for antepartum or postpartum therapy of HELLP syndrome should be considered experimental.