

MANAGEMENT OF POST PARTUM HAEMORRHAGE

Dr. NANA P NJOTANG

Senior lecturer, FMBS, University of Yaounde I

Central Maternity, Central Hospital Yaounde

Postgraduate Training in Reproductive Health Research

Faculty of Medicine, University of Yaoundé 2007

Plan

- Introduction.
- Vital statistics.
- Etiologies of PPH.
- Risk Factors to Specific Etiologies.
- Management of PPH.
- Conclusion.

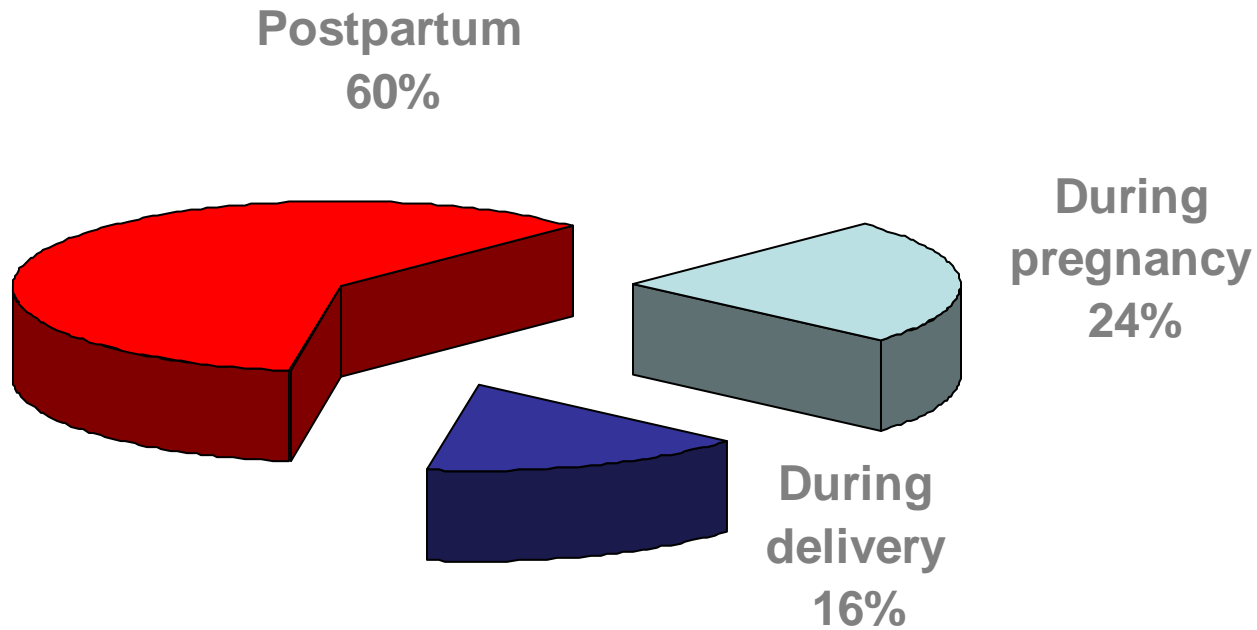
INTRODUCTION

- Maternal mortality, dramatic reduction, since blood transfusion.
- Haemorrhage remains prominent cause of maternal mortality.
- Delivery followed by $<500\text{ml}$, volume $>500\text{ml}$ after 3rd stage, constitutes PPH.
- Placenta delivery, AMTSL, maternal surface (Duncan), foetal surface (Baudelocque).
- Postpartum period: immediate, early and late.
- PPH divided into 1ary and 2ary (1-42 days).

Introduction-1

- Over half a million women die during pregnancy and childbirth each year
- 99% in developing countries
- 150,000 women bleed to death
- Postpartum haemorrhage is the major cause of maternal deaths
- Most deliveries are attended by non-skilled persons, often at home, when there are poorly functioning health systems
- Skilled care with a functional health system can make a difference

Vital Statistics.



WHO analysis of causes of maternal death: a systematic review

Lancet 367: 1066-1074, 2006

Country	Year	Maternal Death	MMR	Haemorrhage
DR Congo	97	143	510	16%
Egypt	200	84	585	30%
Senegal	202	87	690	22%
Tanzania	88	76	529	23%
South Africa	203	121	150	10%
Zambia	98	349	729	28%
Zimbabwe	201	92	695	19%
MCW Africa	201	55	334	33%

ETIOLOGY OF POSTPARTUM HAEMORRHAGE.

- PPH several predisposing factors, more than two may exist in the same patient.
- Causes may be summarised by four P's.
 1. Placental abnormalities: retention, placenta praevia, accreta, abruption.
 2. Passage (genital tract trauma): tears /lacerations.
 3. Porter (uterus): C/S, caesarean hysterectomy, uterine rupture, uterine atony, uterine inversion.
 4. Plasma: Coagulation defects / DIC.

Etiology of PPH: INSERM Study 1998

Etiology	Number of cases	Percent	
Atony	69	42%	
Placenta Retention	27	16%	
Uterine rupture+cervical tear	25	15%	
PP+Accreta	19	11%	
Abruptio placenta	18	10%	
Caesarean section	4	2.4%	
Others	3	1.8%	

RISK FACTORS: PLACENTAL ABNORMALITIES

- Previous uterine scar (C/S, Myomectomy, uterine perforation).
- Large placentas (multiple pregnancy, succenturate placenta, diabetes, Rhesus incompatibilities, molar pregnancy).
- IUD, chorioamnionitis.
- Poor management of 3rd stage labour.

RISK FACTORS: GENITAL TRACT TRAUMA

- Instrumental deliveries (forceps, vacuum extraction).
- Previous perineal tears, short perineum, vaginoplasty.
- Episiotomy done early in labour.
- Surgery / scar cervix.
- Poor conduct of delivery.
- Macrosomia >4000gms, shoulder dystocia, malposition, internal podalic versions.

RISK FACTORS: UTERINE RUPTURE

- Previous scarred uterus.
- Grandmultiparity.
- Use of oxytocics, misoprostol.
- Use of traditional oxytocics (honey).
- Previous induced abortions with perforation of uterus.
- Intra-uterine manipulations (internal podalic version, destructive deliveries).
- Foetal malformation (conjoint twins, hydrocephalus etc).
- Poor conduct of delivery (abdominal expression, shoulder dystocia).

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RISK FACTORS: UTERINE ATONY

- Over distension of uterus (multiple foetuses, hydramnios).
- Placental abruption (Couvelaire's uterus).
- Exhausted myometrium (precipitated or vigorous labour, prolonged labour, use of oxytocics, anaesthesia = halogenated agents, conduction anaesthesia).
- Past history of PPH.
- Myomatous uterus.
- Grandmultiparity, chorioamnionitis.
- Traditional practices e.g. hot water.

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RISK FACTORS: UTERINE INVERSION

- Less frequent, prevalence 1 in 2000-7000 deliveries.
- Poor management of 3rd stage of labour.
- Placenta accreta.
- Active management of 3rd stage of labour facilitates diagnosis and treatment.
- Divided into 4 stages, may be acute, sub-acute or chronic.

RISK FACTORS: COAGULATION DEFECTS

- Placental abruption.
- Intra-uterine death.
- Amniotic fluid embolism.
- Induced abortions.
- Chorioamnionitis.
- Massive blood transfusion.
- Eclampsia/ severe pre-eclampsia.
- Coagulation defects e.g. coagulation factor deficiency.
- Autoimmune thrombocytopenia.
- Drugs: anticoagulants.

MANAGEMENT OF PPH

Estimation of blood loss:

- Visual estimate, usually under estimate.
- Haemodynamic parameters (BP, pulse, CVP).
- Tilt test (orthostatic hypotension).
- Urine flow.

• Treatment:

- General measures: Set up IV-line, Fluid /blood replacement until urine flow varies 30-60ml/hr, Hct of 30%. Fresh blood and Ringer lactate solution preferably.

MANAGEMENT OF PPH (1)

- Etiologic measures. Treat the underlying cause.
- Preventive measures:
 - Type and cross match blood for high risk patients (P/H of PPH, grandmultiparity, placenta praevia, placental abruption, severe PET/eclampsia etc).
 - Active management of 3rd stage of labour.
 - Continue IV oxytocics infusion in induced or augmented labour.

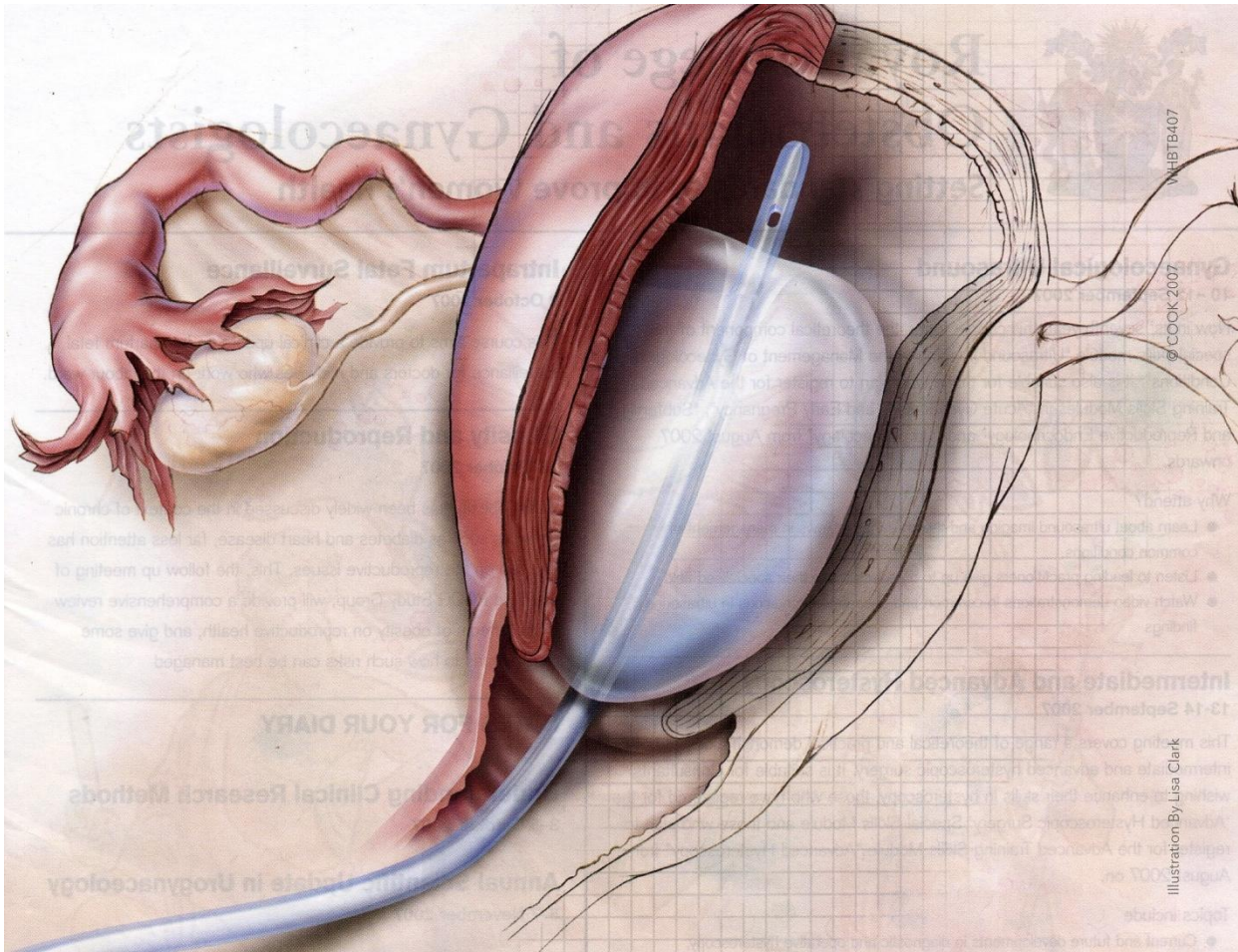
Uterine Atony

**Oxytocin 5 UI IV slowly
 10 UI intra-mural
 20 UI / 500 cc pass in an hour.**

➤ **Misoprostol IR (During uterine revision) 3 tabs of 200µg ?**

➤ **Sulprostone (Nalador) !!!**

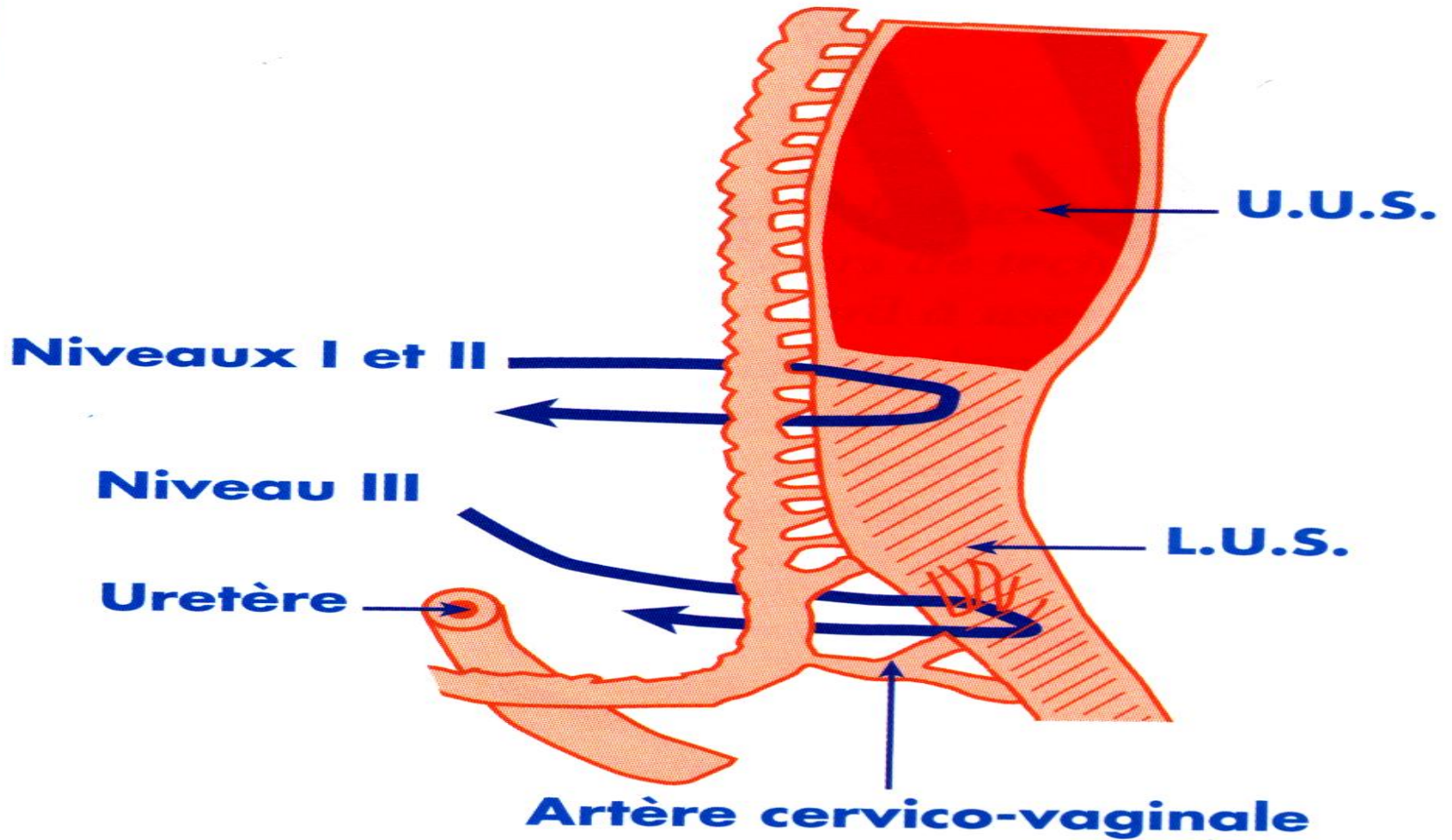
Uterine Atony-1



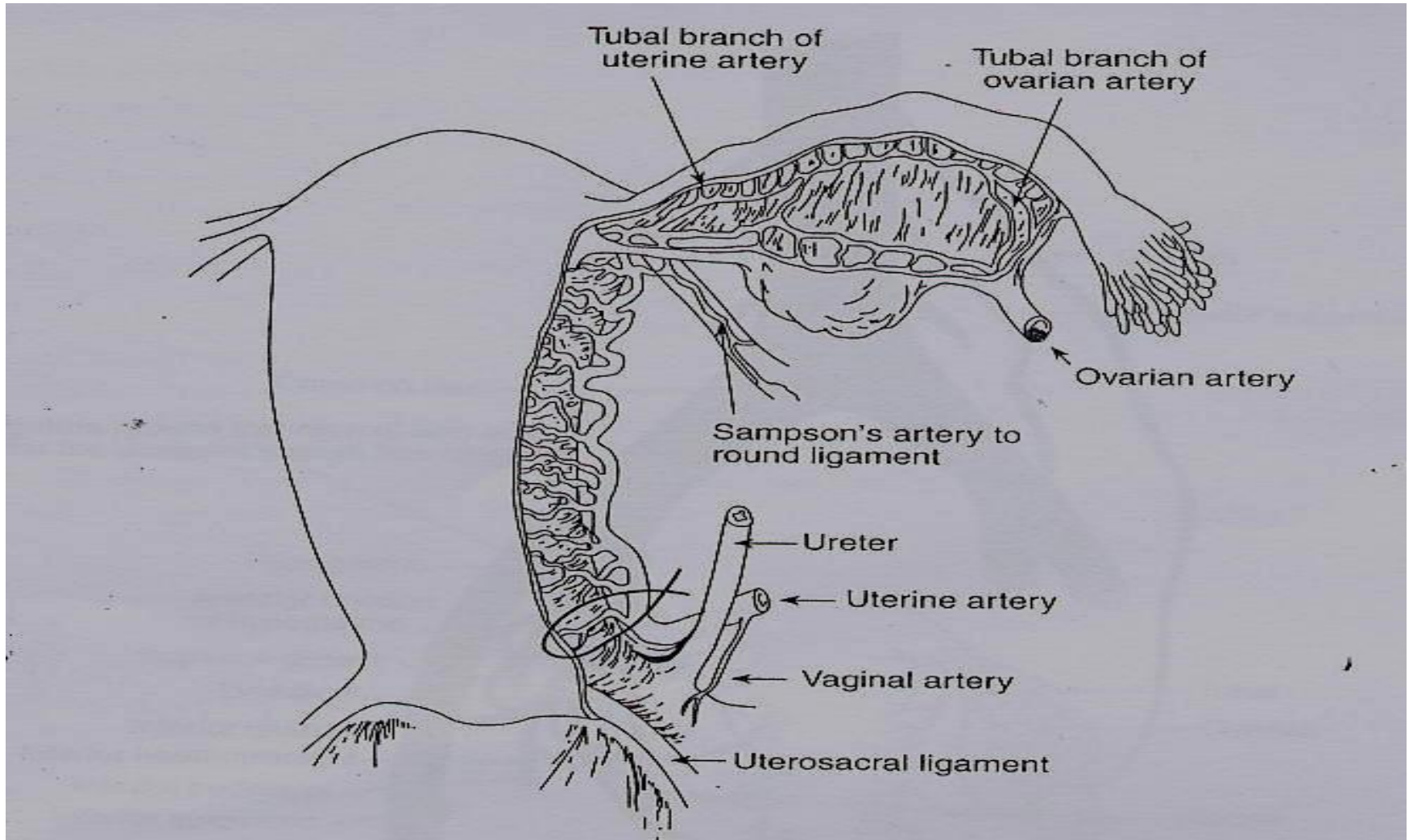
Surgical Management of PPH

- Surgical procedures range from conservative to radical surgery:
- Ligation of the Arterial supply to the uterus.
- Embolisation of the Artery.
- Sub-total hysterectomy.
- Total abdominal hysterectomy.
- N:B However, the choice of type of treatment will depend on the infrastructure, the competence of the team and the haemodynamic status of the patient.

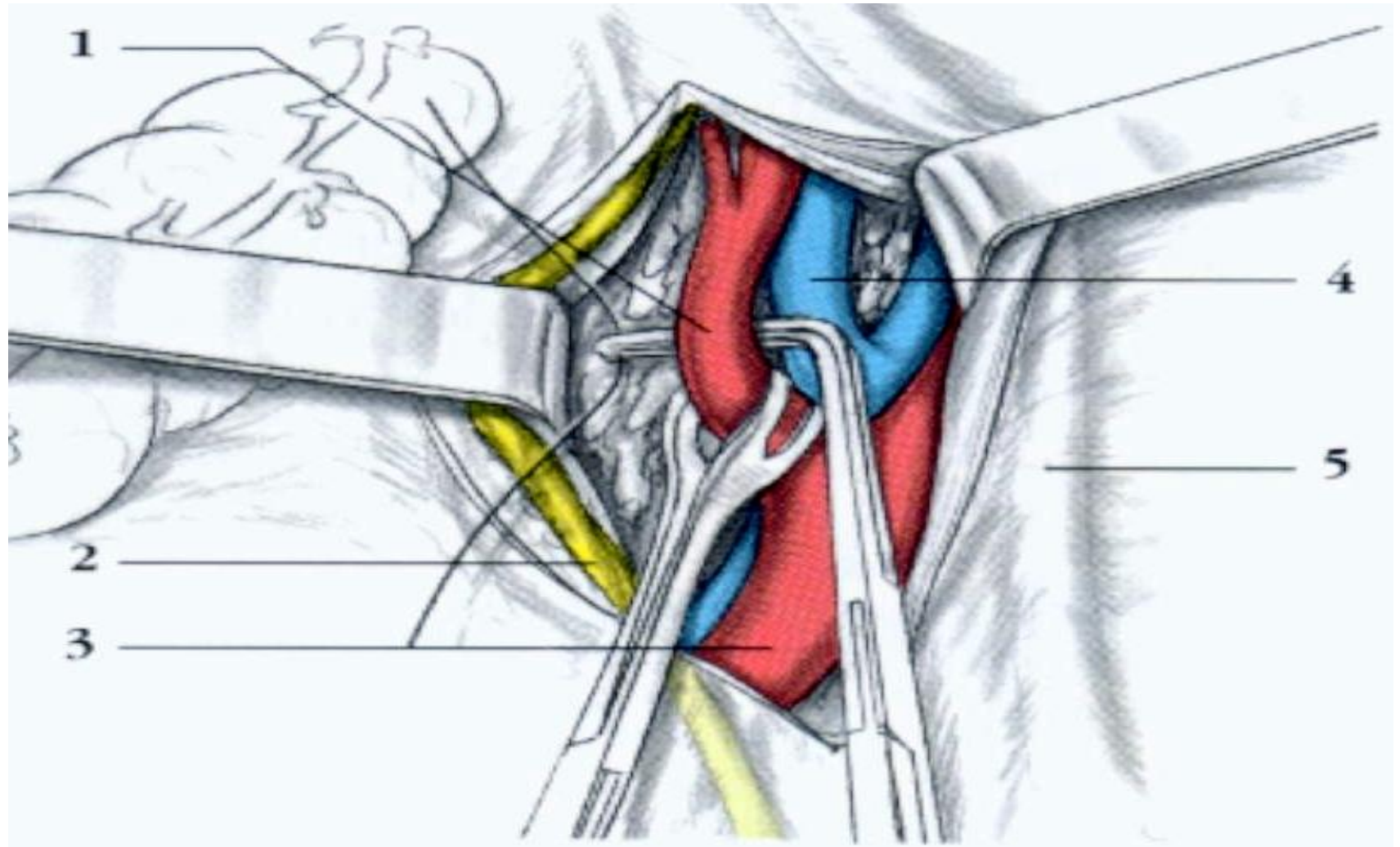
Uterine Atony-2



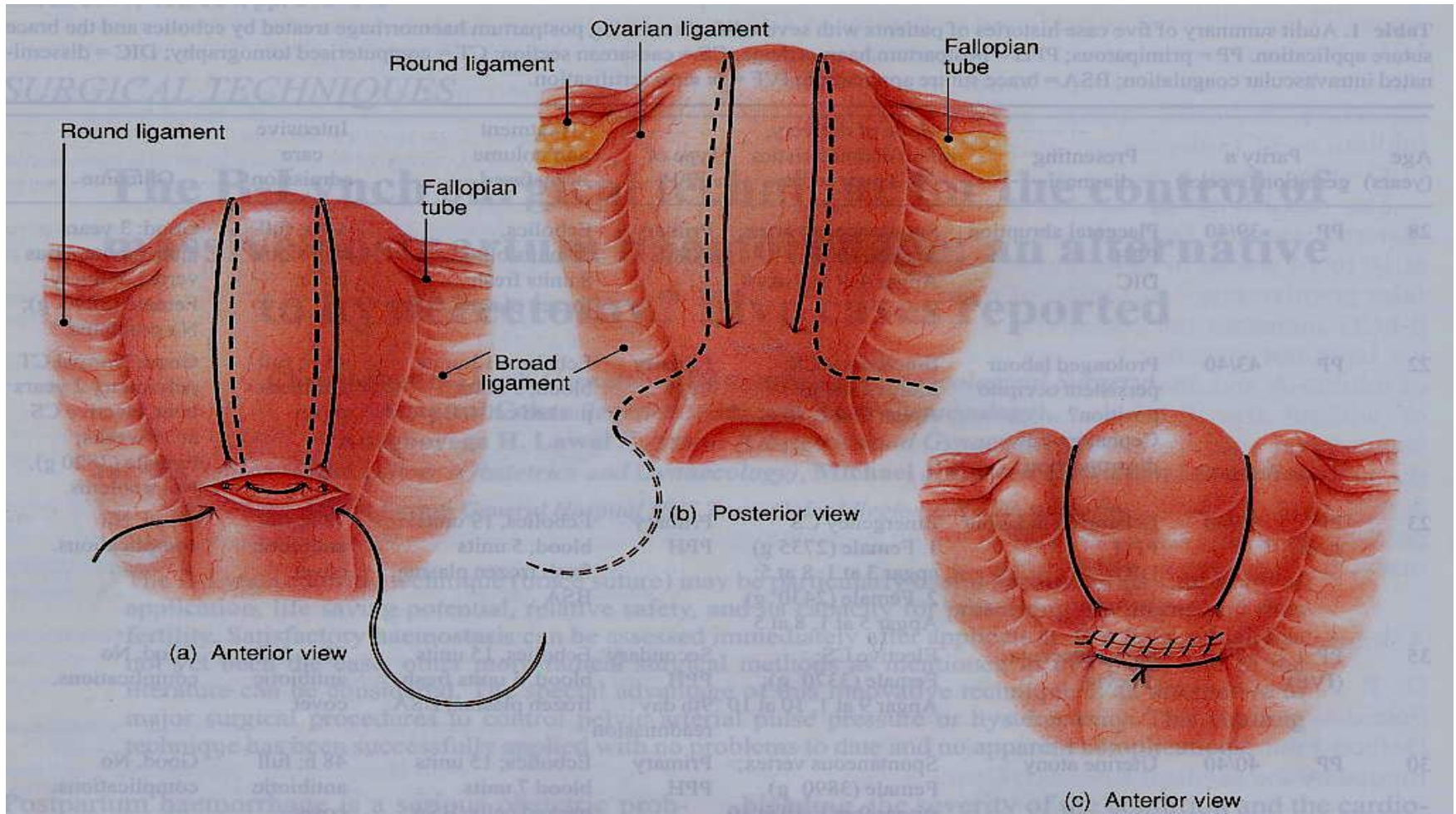
Uterine Atony, selective ligation



Ligation of the Hypogastric Artery

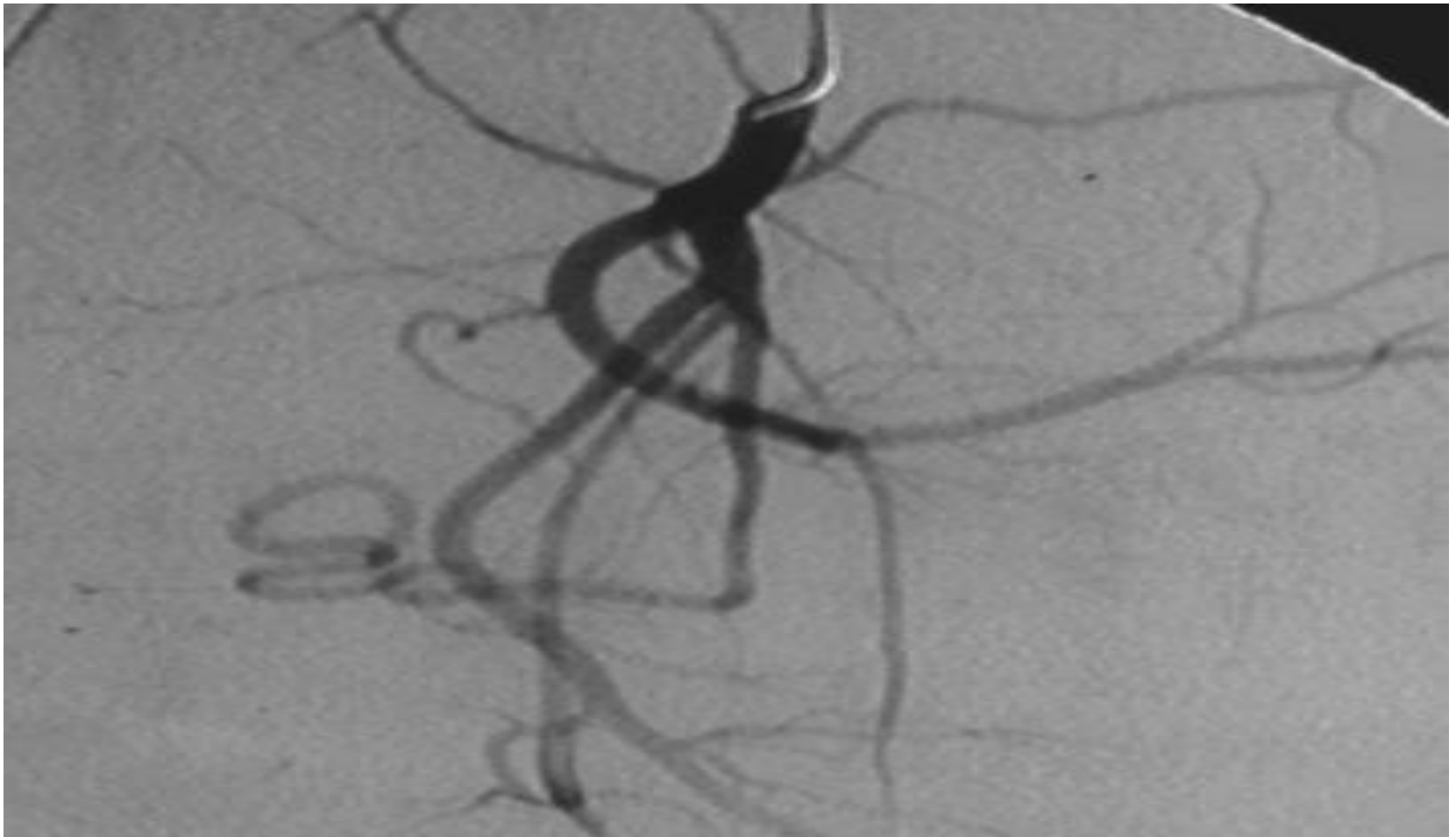


Uterine Compression Sutures

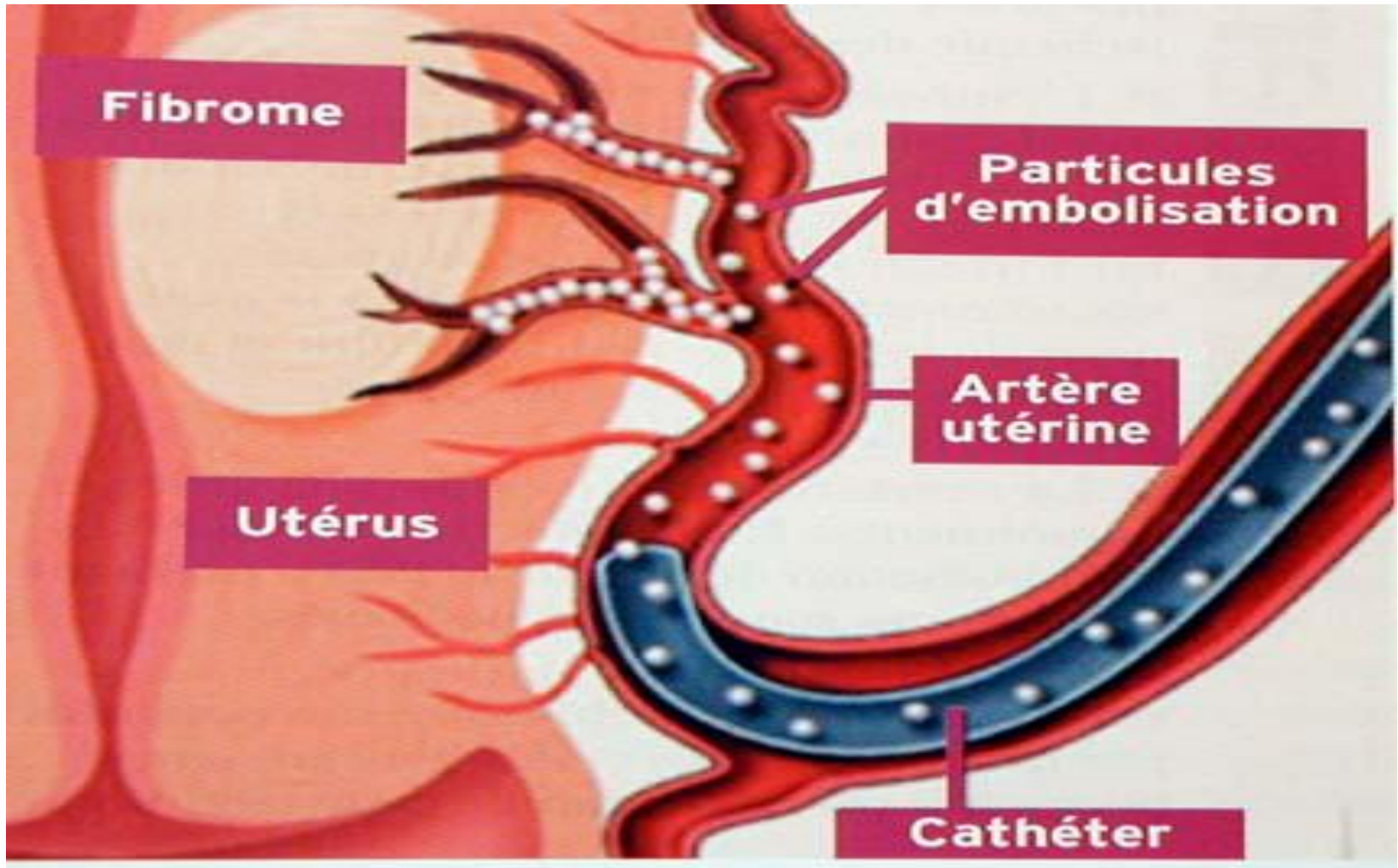


Hayman RG, Arulkumaran S, Steer J. Uterine compression sutures: surgical management of postpartum haemorrhage. *Obstet Gynecol.* 2002;99:502–6.

Embolisation of Hypogastric Artery



Technique of embolisation



DISSEMINATED INTRAVASCULAR COAGULATION

- Pregnancy causes increase in clotting factors (I, II, VII, VIII, IX, X).
- Plasminogen levels are increased.
- Plasmin activity during the antepartum is decreased.
- Various stresses incite conversion of plasminogen to plasmin, especially coagulation mechanism.
 - Extrinsic pathway, release of tissue thromboplastin (placenta, amniotic fluid, myometrium).

DISSEMINATED INTRAVASCULAR COAGULATION (1)

- Intrinsic pathway by collagen and other tissue to which plasma is exposed through loss of endothelial integrity (rupture, retroplacental haematoma).
- Direct activation of factor x by appropriate enzymes (protease), seen in some bacterial infection or neoplasm.

Plasminogen is activated to plasmin, lyses of fibrinogen, fibrin monomer and polymer, formation of fibrinogen-fibrin degradation products or split products.

DISSEMINATED INTRAVASCULAR COAGULATION (2)

- Degradation product, depending on size contribute to the defective haemostasis (delay fibrin polymerisation, prolong prothrombin time, impair clot retraction and stability).

The treatment of DIC is a combination of the following:

- Replacement of deficient factors especially fibrinogen.
- Injection of heparin (block further intravascular coagulation).
- Administration of epsilon amino caproic acid (block fibrinolysis).

CONCLUSION

- **PPH**
 - Obstetrical emergency;
 - May be catastrophic;
 - Grave consequence
 - Necessitates prompt action, involving nurses, hematologist, intensive care Doctor and obstetrician

THANK
YOU