# Hypertensive Disorders of Pregnancy

**Hypertension**:

- One measurement of diastolic BP of 110 mmHg or more; or
- Two consecutive measurements of diastolic BP of ≥ 90 mmHg 4 hours or more apart.

**proteinuria**:

- One 24-h urine collection with a total protein excretion of 300 mg or more; or
- Two random clean-catch urine specimen with a 2+ or more on reagent strip

## Pre-eclampsia

- hypertension + proteinuria after 20th week of gestation in a previously normotensive women & resolving completely by the 6th postpartum week.

- **Eclampsia:** tonic-clonic convulsion with established pre-eclampsia, in the absence of any other neurological or metabolic cause.

- **Pregnancy-induced hypertension (non-proteinuric gestational HT):** arising in the second half of pregnancy without proteinuria
- **Chronic hypertension:** prior to, in the first half of, or persisting more than 6 weeks after pregnancy.
- superimposed pre-eclampsia: in the presence of chronic hypertension is usually associated with a worsening of the hypertension & the development, or worsening of, proteinuria.

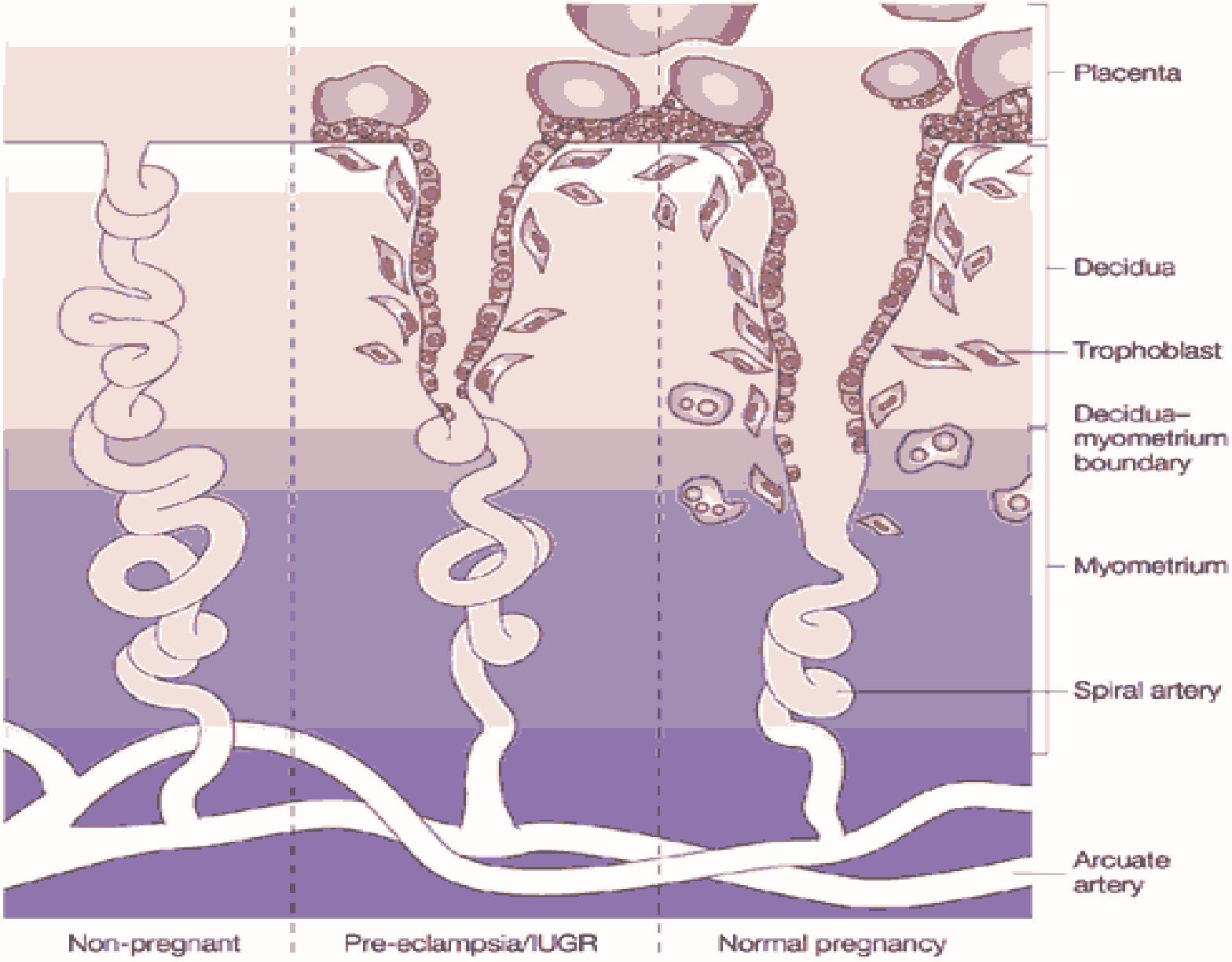
Incidence: 3 % of pregnancies

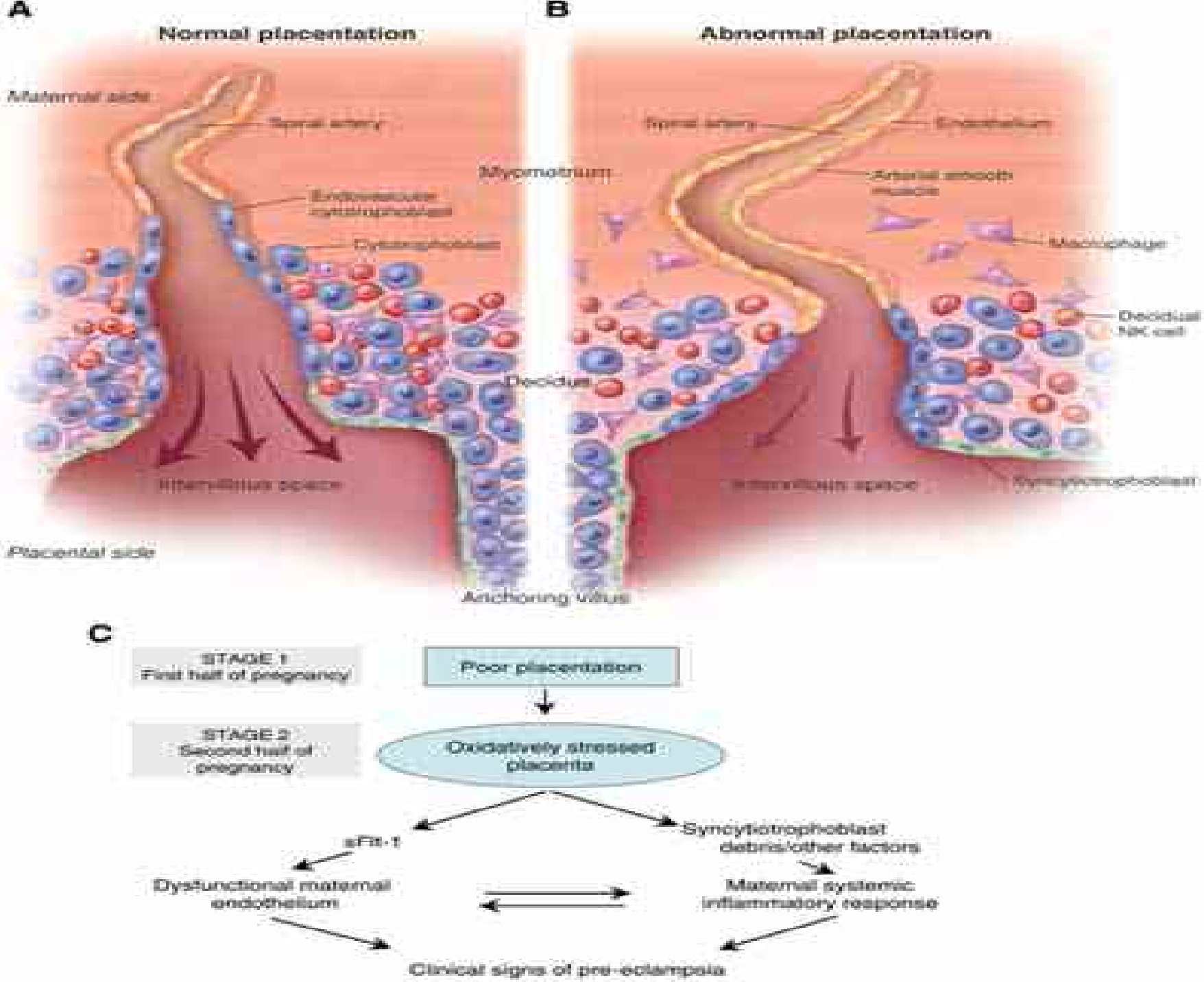
Risk Factors:

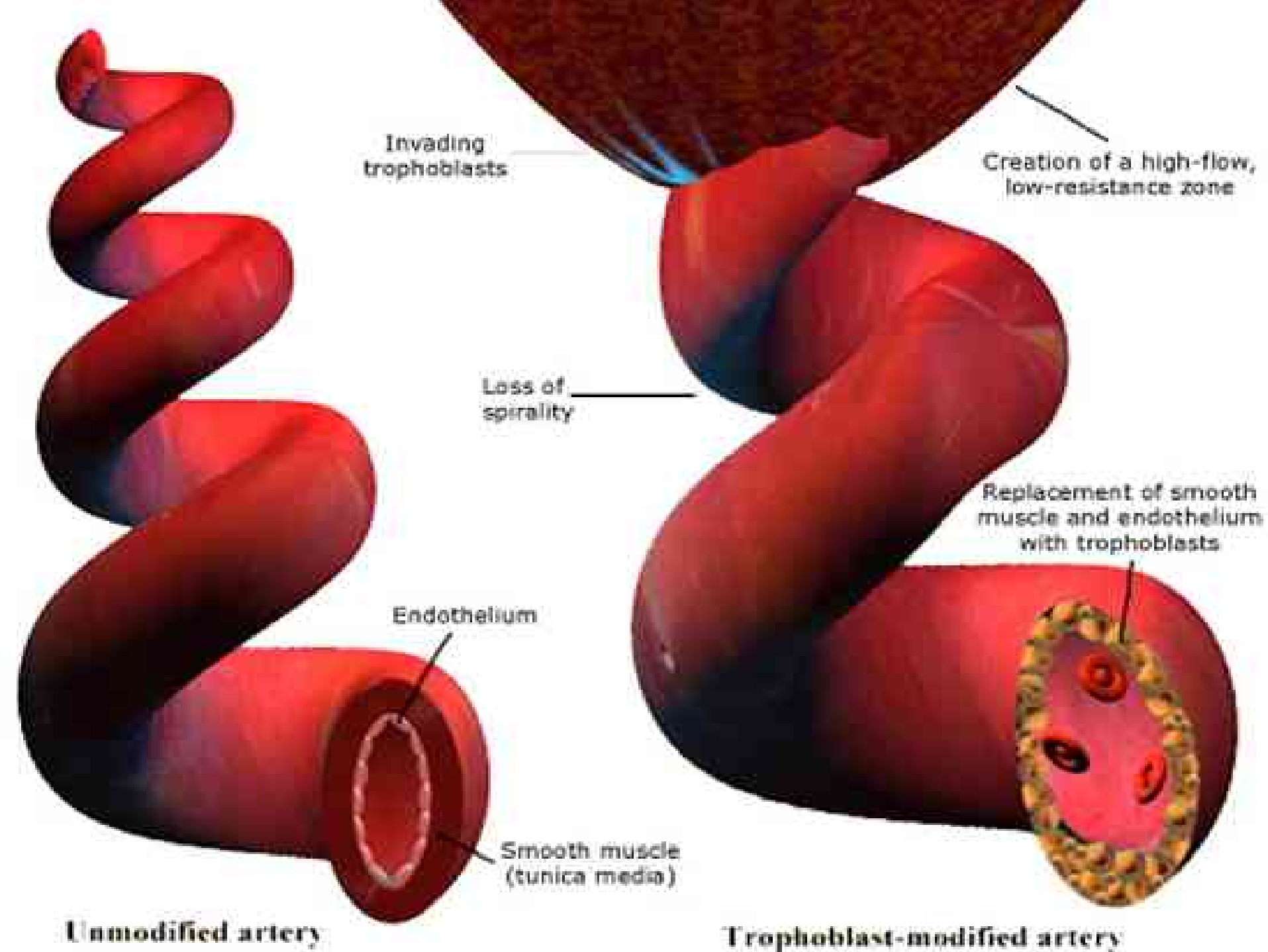
- Antiphospholipid syndrome
- Previuos Hx of PE
- Family Hx : 3-4 fold increase risk
- conditions in which the placenta is enlarged
- pre-existing hypertension or renal disease.
- pre-existing vascular disease (as in diabetes or autoimmune vasculitis
- Raised BMI
- Age over 40
- Raised diastolic BP > 80 mmHg

# Aetiology:

- normal pregnancy: the cytotrophoblast invade the spiral arteries. dilatation of the spiral artery & increased intervillous blood flow.
- pre-eclampsia: trophoblast invasion is patchy & the spiral arteries retain their muscular walls
- Impaired perfusion & ischaemia result in production of reactive oxygen species & a condition of oxydative stress
- Placenta release certain factors (adhesion molecules, von-willebrand factor) into the maternal circulation which target the vascular endothelium & cause dysfunction.


Placenta
Decidua
Trophoblast
Decidua–
myometrium
boundary
Myometrium
Spiral artery
Arcuate
artery
Non-pregnant
Pre-eclampsia/IUGR
Normal pregnancy


A
Normal placentation
B
Abnormal placentation
Myometrium
Decidua
Macrophage
Placental side
Anchoring villus
C
STAGE 1
First half of pregnancy
Poor placentation
STAGE 2
Second half of pregnancy
Oxidatively stressed placenta
sFlt-1
Syncytiotrophoblast debris/other factors
Dysfunctional maternal endothelium
Maternal systemic inflammatory response
Clinical signs of pre-eclampsia


Invading trophoblasts
Creation of a high-flow, low-resistance zone
Loss of spirality
Replacement of smooth muscle and endothelium with trophoblasts
Endothelium
Smooth muscle (tunica media)
Unmodified artery
Trophoblast-modified artery

Normal pregnancy:

- peripheral vasodilatation is accomplished through a reduced vascular sensitivity to vasoconstrictors such as angiotensin.

In pre-eclampsia the insensitivity to vasoconstrictors is lost.

- Vasospasm & endothelial cell dysfunction, with subsequent platelet activation & micro-aggregate formation.

- Cardiovascular:

Generalized vasospasm

Increased peripheral resistance

- Haematological

Platelet activation & depletion

Coagulopathy

Decreased plasma volume

Increased blood viscosity

- Renal

Proteinuria

Decreased glomerular filtration rate

Decreased urate excretion

- Hepatic

Periportal necrosis

Subcapsular haematoma

- Central nervous system

Cerebral oedema

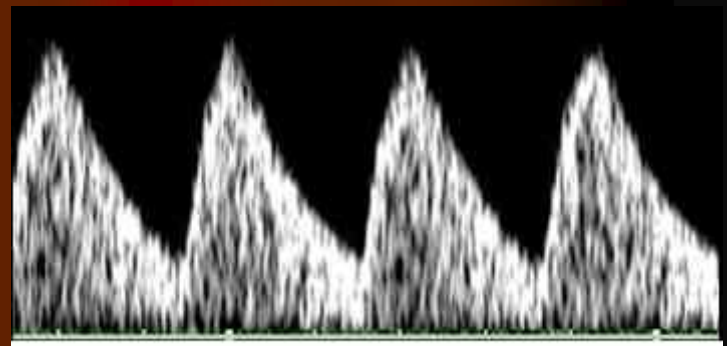
Cerebral haemorrhage

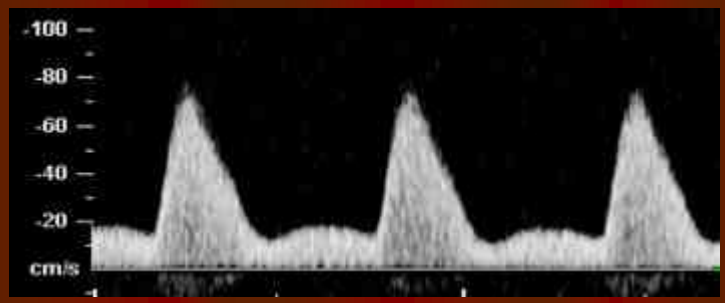
## Symptoms of pre-eclampsia:

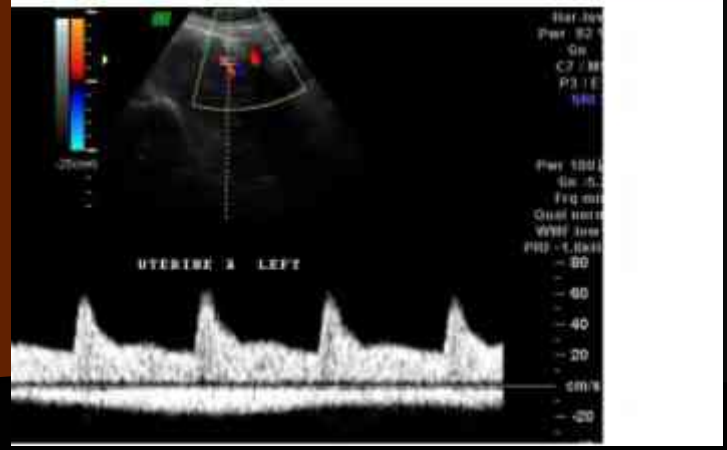
## Signs of pre-eclampsia:

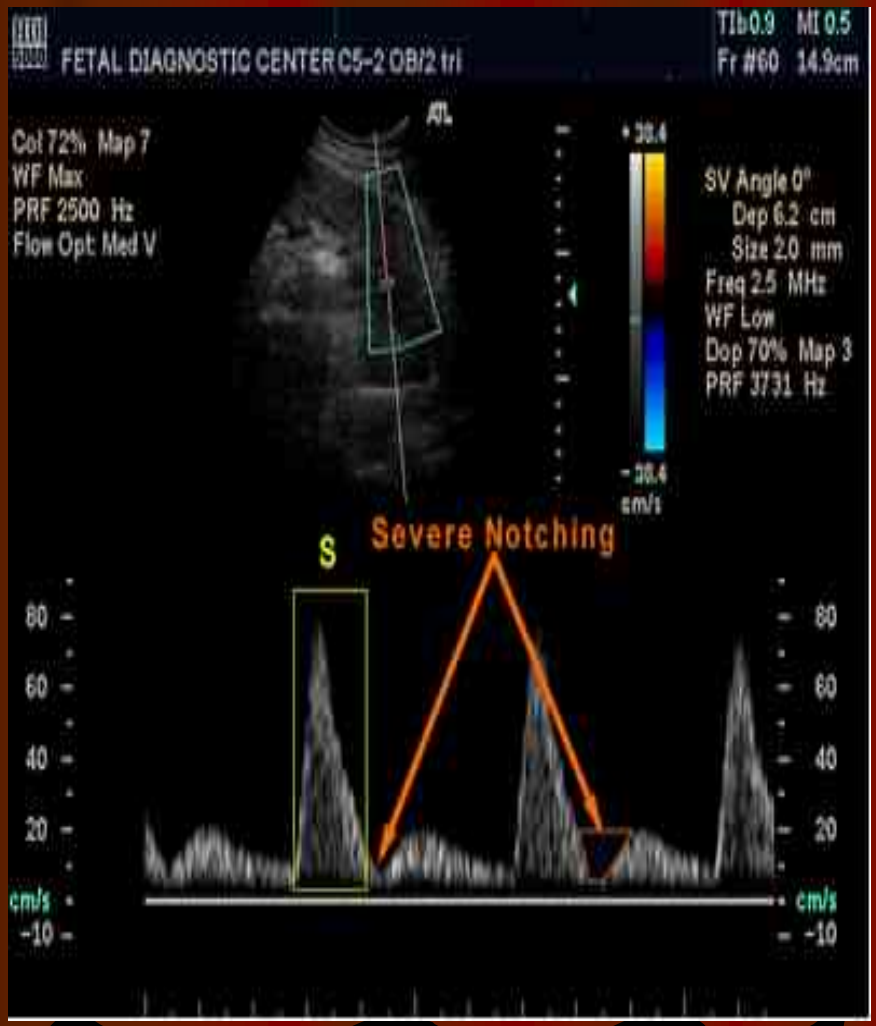
# Screening tests:

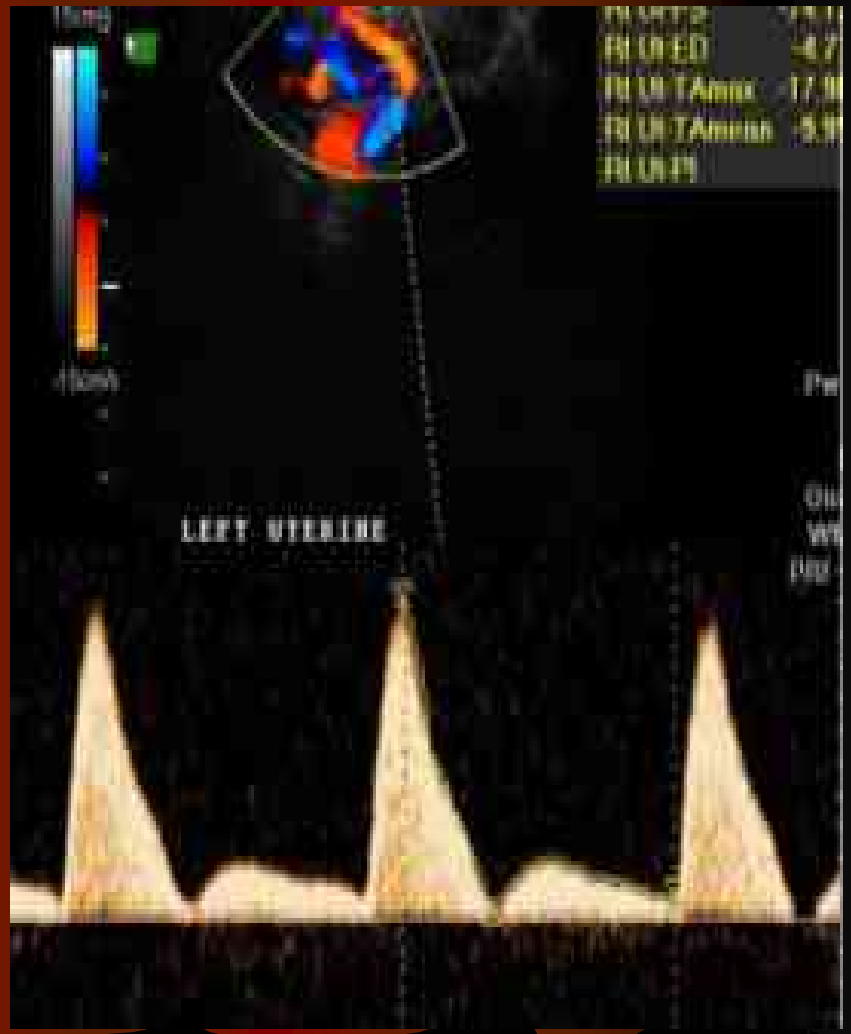
- Doppler ultrasound of the uterine artery waveform analysis
- a characteristic 'notch' can be seen in the waveform pattern.






FETAL DIAGNOSTIC CENTER C5-2 OB/2 tri
TIb 0.9 MI 0.5
Fr #60 14.9cm
Col 72% Map 7
WF Max
PRF 2500 Hz
Flow Opt: Med V
ATL
+ 38.4
- 38.4
cm/s
SV Angle 0°
Dep 6.2 cm
Size 2.0 mm
Freq 2.5 MHz
WF Low
Dop 70% Map 3
PRF 3731 Hz
S
Severe Notching
80
60
40
20
cm/s
-10


LEFT UTERINE

# Management:

- Urinalysis by dipstick
- 24-hour urine collection ( total protein & creatinine clearance)
- Full blood count
- Blood chemistry ( renal function, protein concentration)
- Plasma urate concentration
- Liver function
- Coagulation profile
- Ultrasound assessment:
  - Fetal size
  - amniotic fluid volume
  - maternal & fetal Doppler

- Mild PE: out patient
- Severe PE: admission

Criteria of severe pre-eclampsia are:

- BP of ≥ 160 mmHg systolic or ≥ 110 mmHg diastolic on at least two occasions at least 6 h apart with patient at rest.
- Proteinuria of ≥ 5 g per 24 h.
- Oliguria ( ≤ 400 ml in 24 h ).
- Cerebral or visual disturbance.
- Epigastric pain.
- Pulmonary oedema or cyanosis.
- Impaired liver function.
- Thrombocytopenia

- The mainstay of treatment of PE is by ending the pregnancy by delivering the fetus & the placenta.

Antihypertensives:

- Labetolol: alpha & beta- blocking agent
- Methyldopa: centrally acting antihypertensive agent, takes up to 24 hours to take effect.
- Nifedipine: calcium channel blocker with a rapid onset of action.
- Hydralazine: arterial vasodilator.

# Management of labour & delivery

- expectant management should be continued to 37-38 weeks gestation

Indication for preterm delivery are:

- severe uncontrolled hypertension ( ≥ 160/110 mmHg)
- haemolysis with thrombocytopenia & elevated ALT
- progressive symptoms (headache, visual disturbance, epigastric pain)
- pulmonary oedema
- renal compromise with oliguria
- eclampsia
- fetal distress

- The mode of delivery is determined by gestational age, the state of the cervix & fetal condition
- Most severe cases delivered by C/S
- Prolonged pushing should be avoided
- ergometrine should not be used in the active management of the third stage because of the risk of dangerous hypertension
- Fluid management is important in severe PE:1 litre Ringer lactate / 12 h).
- use of diuretics should be confined to women with pulmonary oedema

# Postnatal councelling

- The risk of recurrence is increased with increased severity of PE.
- increased risk of death from cardiovascular disease in the future particularly in those who remain hypertensive in the puerperium.