Pregnancy and treatment of vascular disease:

- 12-80% of maternal death during pregnancy and puerperium are due to cerebrovascular disease
- Physiological changes during pregnancy:

1. Blood changes (plasma volume increases by 40-50%,, erythrocyte volume increases by 25% -decrease in Hb (physiological anaemia of pregnancy)
2. Cardiovascular changes: increase in heart rate and stroke volume -**60%** increase in **cardiac output** this occurs during the first 2 trimesters. **Decrease in peripheral vascular resistance** (Estrogens and progesterone lead to vasodilatation) and **10% decrease in arterial blood pressure**. In the third trimester compression of IVC and aorta by uterus in supine position leads to decrease in placental blood flow which has implications for women positioning for surgery
3. Respiratory: increase in RR and TV – 69-**70%** increase in alveolar ventilation. Respiratory alkalosis balance the mild metabolic acidosis occurring during pregnancy
4. Coagulation: **increase in fibrinogen, II, VII, VIII, X**, increase in platelet aggregation and **decrease in t-plasminogen activator** and protein S leads to **hypercoagulable state.**

- Pharmacological consideration:

1. **Anticonvulsants**: all anticonvulsants are established human teratogens; however the risk of seizures, with associated maternal and fetal hypoxia justifies the use of anticonvulsants. **Phenytoin (**growth retardation, mental retardation and microcephaly), **Phenobarbital (**craniofacial anomalies, limb abnormalities and psychomotor delay), **carbamazepine (**craniofacial abnormalities, fingernail hypoplasia, cardiac defects and spina bifida). **Tegretol is thought to be the safest** of all to use during pregnancy. In general anticonvulsants should be used only if the risk of seizures is high, frequent check of the level due to altered metabolism, use single agent if possible and patients should be given folic acid and vit k to oppose the interaction of anticonvulsant and the metabolism of folic acid and vit. K.
2. **Anticoagulants**: **warfarin can cross the placental barrier** and cause congenital anomalies in **30%** (facial abnormalities, growth retardation, hypoplasia of limbs and digits, spontaneous abortion) , hence its use during pregnancy should be avoided. **Heparin does not cross the placenta** and does not cause congenital syndromes. Fractionated and LMW heparin should be used during pregnancy particularly in the first trimester (PE, artificial valve etc..).
3. **Antiplatlet**s: low dose aspirin is safe in the second and third trimester , no data on safety during the first trimester
4. **Mannito**l can **cross the placenta** and cause fetal dehydration and its use should be restricted to life threatening increase in ICP
5. **Nimodipine**: experimental evidence of congenital malformation in animals, no human studies. **It is released in milk and breast feeding** should be avoided while mother on nimodipine.
6. **Antihypertensive**s: should be used with caution and hypotension should be avoided (uterine hypoperfusion and fetal compromise).

- Some cerebrovascular conditions:

1. **SAH** should be treated the same way as in non pregnant. CT head and cerebral angio can be done safely **radiation exposure of the fetus include a maximum dose of 0.5 rem** (roentgen equivalent man). The maximum dose to the fetus during head **CT can is <0.05 rem** and during angio **< 0.1 rem**. The decision to have **vaginal delivery or CS should be dictated by obstetric causes** and not by SAH. There are no data on the safety of MRI scan and probably should be avoided during the first trimester. One should consider eclampsia and preeclampsia in the deferential diagnosis of SAH. **The risk of rupture of aneurysms during pregnancy has not been firmly established, but most authors believe that the risk is slightly increased.**
2. **AVM**: pregnancy does not increase the risk of AVM rupture. The management should be similar to no pregnant woman . Surgical intervention should be dictated by neurosurgical rather than obstetric criteria and CS vs. vaginal delivery should be dictated by obstetric criteria.
3. **Ischemic stroke**: the estimated incidence is 0.004-**02%,** 60-80% arterial, 20-40% venous thrombosis. The causes of arterial occlusion and venous thrombosis (look page 2428 Yuman's). **Venous thrombosis** carries mortality **of 33%,** however the neurological outcome in survivors is better than in those with arterial infarctions (60% recover without neurological deficit).
4. **Metastatic choriocarcinoma** is a cause of ICH in pregnant women. This is the most common malignant tumour associated with pregnancy. Trophoblastic tissue has the ability to penetrate and destroy blood vessels. This tumour can cause mycotic aneurysms in the distribution of MCA. Treatment follows the usual guidelines fro ICH. Mycotic aneurysms can be clipped or excised.
5. **Postpartum cerebral vasospasm** is a rare complication of pregnancy and usually occurs in the first 3 weeks after delivery. The cause is not known and it is thought to be a form of eclampsia. Treatment is similar to vasospasm from SAH (triple H and nimodipine).
6. **Carotico-cavernous fistula** is reported to occur during pregnancy due to hemodynamic changes. These fistulas resolve spontaneously **in 60%** of cases.
7. **Pituitary apoplexy**: severe shock at the time of delivery can cause pituitary infarction and pan hypopituitarism (Sheehan's syndrome).Pituitary gland enlarges during pregnancy due to stimulation of prolactin secreting cells. Treatment is by hormone replacement