Eclampsia and Preeclampsia

High blood pressure in pregnancy is classified to include four main conditions: preeclampsia-eclampsia, gestational hypertension, chronic hypertension and preeclampsia superimposed on chronic hypertension.

- ➔ We've established thus far that during normal placental development, the trophoblasts invade the arterioles of the uterine endometrium forming lacunae or 'holes' to remodel maternal arterioles into large blood vessels with low resistance to allow more blood flow and to decrease the high resistance of arterioles through uncertain mechanisms.
- ➔ But in pregnancy-induced hypertension, things -for unknown reasons- don't really go according to plan.
- ➔ It involves a decrease in placental blood flow, leading to an ischemic placenta that releases inflammatory substances (TNF, IL-6) that disturb endothelial cells throughout the body, the endothelial cells will release less nitric oxide, and blood flow into the kidney, brain, liver, and heart be severely decreased. And the vasoconstriction from the dysfunctional endothelial cells incapable of releasing nitric oxide will lead to hypertension. (Keep in mind that aldosterone and estrogens are also released, leading to more water retention in pregnancy, and with less blood flow to the placenta the blood is further trapped in the circulation).

Gestational Hypertension, Preeclampsia and Eclampsia

These are commonly used terms to refer to hypertension in pregnancy, and are falsely used interchangeably.

- Preeclampsia is different from conventional gestational hypertension is that it includes proteinuria as well due to effect on renal vasculature.
- Eclampsia includes all features of preeclampsia, but the hydrostatic blood pressure becomes so intense that it leads to cerebral edema resulting in convulsions.

Preeclampsia or Toxemia of Pregnancy:

Initiated by placental hypoperfusion, an ischemic placenta releases substances that disrupt endothelial cells throughout the body, less NO released leads to a rapid rise in arterial blood pressure.

- ❏ And is often characterized by excess salt and water retention, development of edema and hypoperfusion of vital organs increasing the risk of acute renal failure (due to reduced renal blood flow from low NO and GFR).
- ❏ It also affects the fetus as it frequently results in intrauterine growth restriction and prematurity (recall that it was started by decreased blood flow to placenta).
- ❏ Delivery of fetus is curative.

Eclampsia:

- ❏ It's basically the convulsive stage of preeclampsia being characterized by vascular spasm throughout the body unattributed to other causes; Clonic seizures in the mother followed by coma.
- ❏ The exact mechanism remains unclear but it has been attributed to both increased blood coagulability and fibrin deposition in the cerebral vessels.
- ❏ This greatly spastic condition puts the kidney and the liver at a vulnerable position leading to malfunction and a generalized toxic condition of the body. Usually happens shortly before birth of the baby.
- ❏ Optimal use of rapid acting vasodilators to reduce arterial pressure followed by immediate termination of the pregnancy -by c-section if necessary- reduces mortality rates.

Treatment:

- Hydralazine: A vasodilator, the proposed mechanism of action is that it acts on receptors on endothelial cells to cause release of nitric oxide.
- Labetalol: A cardioselective beta-blocker, decreases cardiac output with minimal systemic effects.
- Nifedipine: Calcium channel blockers are sometimes used, they block calcium entry into SMCs leading to blood vessel relaxation and thereby restoring blood flow to vital organs