Table 4.1: Stages of labor	
First stage	Onset of true labor pains to complete cervical dilatation
Second stage	Complete cervical dilatation to delivery of the fetus or fetuses
Third stage	Separation and expulsion of the placenta and the membranes

First Stage

The stage of opening of the passages marked by cervical effacement and dilatation. In primigravida, effacement occurs first followed by dilatation. In multigravida, both effacement and dilatation occur simultaneously. The first stage is divided into two phases:

1. ***Latent phase:*** Mainly involves cervical dilatation up to 3–4 cm. It lasts for about 6–8 hrs in nullipara and 4–6 hrs in multipara.

2. ***Active phase:*** Extends from 4 cm of dilatation (WHO) to complete cervical dilatation. The average duration is about 5 hrs in nullipara and 3 hrs in multipara. The mean rate of cervical dilatation is 1.2 cm/hr in nullipara and 1.5 cm/hr in multipara, minimal acceptable progress rate is 1 cm/hr. The pattern of cervical dilatation is a sigmoid curve and that of fetal descent is a hyperbolic curve (Fig. 4.9).

The first stage is also divided into the following phases:

1. Acceleration phase (3–4 cm of cervical dilatation)
2. Phase of maximum slope (4–9 cm of cervical dilatation)
3. Deceleration phase (9–10 cm of cervical dilatation)

First stage is charactertized by

1. *Uterine contractions:* Contractions of labor are regular and increase in duration and frequency. In active labor, these contractions last for 30–90 sec and the frequency is at least 3 in 10 mins. These contractions have a fundal dominance along with a rise in intra-amniotic pressure. In the first stage of labor, the peak intrauterine pressure is 40–50 mm Hg and in the second stage it is 80–100 mm Hg (resting tone is about 6–10 mm Hg).

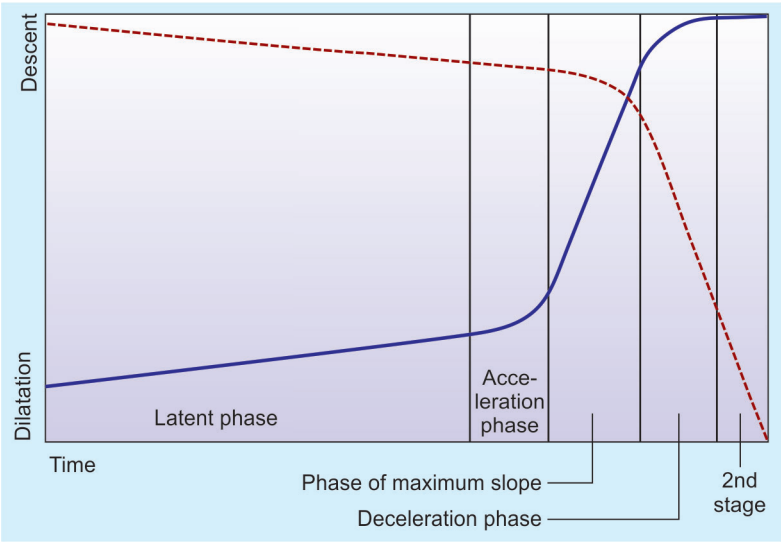

Fig. 4.9: Phases of labor and descent of head

5 Abnormal Uterine Action

• Saroj Singh

Over last quarter of century the incidence of cesarean section due to dystocia has increased. Abnormal uterine actions are one of the important causes of dystocia.[1] Overall labor abnormalities occur in about 25% of nulliparous women and 10% of multiparous women.

PHYSIOLOGY OF NORMAL UTERINE CONTRACTIONS

The pacemaker of uterine contractions lies in the cornua of uterus near fallopian tubes with left being dominant and leads to commencement of waves of uterine contractions and retractions, that passes downwards towards the lower segment and become progressively less intense. Though the lower segment needs to be passive and relaxed in order to efface and dilate to facilitate normal labor (Fig. 5.1).

- During normal labor the uterus contracts every 3–4 minutes and each contraction increase the intrauterine.
- Pressure 25–75 mm Hg above a baseline of 5–20 mm Hg.
- Interval gradually shortens and intensity increases.
- Associated with cervical dilatation
- Associated with discomfort in back and abdomen not relived by sedation.

In contrast, Braxton Hicks contractions, are contractions that may start around 26 weeks gestation and are sometimes called "false labor", should be infrequent, irregular and involve only mild cramping.[2]

In the second stage of labor the uterine work is complmented by maternal expulsive efforts that become an important part of the power required to achieve vaginal delivery.

Any deviation of normal uterine contraction and or coupled with inadequate cervical dilatation and descent of presenting fetal part affecting the course and outcome of labor is defined as abnormal uterine action.

Dystocia literally means difficult labor and is characterized by abnormally slow labor progress. It may be a result of abnormal uterine action.[3]

Classification

1. ***Over-efficient uterine action***
   - *Precipitate labor:* In absence of obstruction
   - *Excessive contraction and retraction:* In presence of obstruction.
2. ***Inefficent uterine action***
   - Hypotonic inertia
   - Hypertonic inertia
     - Colicky uterus
     - Hyperactive lower uterine segment
     - Constriction (contraction) ring

postpartum. There must be no evidence of pre-eclampsia.

UNCLASSIFIED HYPERTENSIVE DISORDERS

Cases who do not have enough informations for classifications.

Etiology

Hypertensive disorders due to pregnancy are more likely to devlop in women who:

1. Are exposed to chorionic villi for the first time.
2. Are exposed to excessive chorionic villi, as in twins or hydatidiform mole.
3. Have preexisting vascular disease.
4. Generally predisposed to hypertension developing during pregnancy.

According to Sibai (2003), currently plausible potential causes include the following:

1. Abnormal trophoblastic invasion of uterine vessels.
2. Immunological intolerance between maternal and fetoplacental tissues.
3. Maternal maladaptation to cardiovascular or inflammatory changes of normal pregnancy.
4. Dietary deficiencies.
5. Genetic influence.

1. ***Abnormal trophoblastic invasion:*** In pre-eclampsia there is incomplete trophobslastic invasion. Early pre-eclamptic changes include endothelial damage, exudation of plasma constituents into vessel wall, proliferation of myometrial cells, medial necrosis, lipid accumulation in myometrial cells and macrophages termed 'Atherosis'. Obstruction of spiral arteriolar lumen by atherosis impair placental blood flow. These changes cause diminished placental perfusion leading to "pre-clampsia syndrome".
2. ***Immunological factors:*** There are strong evidences to support the theory that pre-eclampsia is immune mediated. Risk of pre-eclampsia is enhanced in circumstances where formation of blocking antibody to placental antigenic sites might be impaired as in first pregnancy where effective immunization by previous pregnancy is lacking or number of antigenic sites provided by placenta is more than antibodies as in multiple pregnancies.

   In early 2nd trimester, women destined to develop PET have lower proportion of helper T cell. These helper T lymphocytes secreate specific cytokines that promote implantation and their dysfunction may favor pre-eclampsia. In women with anticardiolipin antibodies placental abnormalities and pre-eclampsia develop more commonly.
3. ***The vasculopathy and inflammatory changes:*** Pre-eclampsia is considered a disease due to an extreme state of activated leukocyte in maternal circulation. Cytokine such as tumor necrosis factor (TNF) and interleukins may contribute to oxidative stress associated with PET. Oxidative stress is characterized by reactive oxygen species and free radicals the lead to formation of self propagating lipid peroxides. These in turn, generate highly toxic radicals that injure endothelial cells, modify their nitric oxide production and interfere with prostaglandin balance. Other consequences of oxidative stress include production of lipid laden macrophage foam cell seen in atherosis. Activation of microvascular coagulation seen as thrombocytopenia and increased capillary permeability seen as edema and proteinuria.
4. ***Nutritional factors:*** A number of dietary deficiencies or excess have been blamed as the cause of eclampsia. Blood pressure in women is affected by a number of dietary influences including minerals and vitamins. Various elements such as zinc, magnesium and calcium are found to be useful in prevention of pre-eclampsia in some studies. Some studies proved that diet rich

2. Cardiac failure.
3. Massive edema not relieved by rest and producing discomfort to patient.

Antihypertensives: The use of antihypertensive drugs is to prolong pregnancy or modify perinatal outcomes in pregnancies complicated by various types and severities of hypertensive disorder has been of considerable interest. The objective of antihypertensive treatment is to prevent intracranial bleeding and left ventricular failure. Some investigators believe that antihypertensive treatment is useful in avoiding selective cerebral arterial vasospasm that causes eclamptic seizure. Anti-hypertensive has limited value in controlling BP due to pre-eclampsia.

The unavoidable indication for its use:

1. Persistent rise of blood pressure especially when diastolic pressure is >110 mm Hg and associated with protenuria.
2. In severe pre-eclampsia to bring down the blood pressure during continued pregnancy or during induction of labor.

   The commonly used oral drugs are:

   a. *Methyl dopa:* Central peripheral antiadrenergic action dose—250–500 TDS/QID.
   b. *Labetalol:* α and β blocker (adrenoceptor blocker) dose—250 mg TDS/QID.
   c. *Nifedipine:* Calcium channel blocker dose—10–20 mg BD.
   d. *Hydralazine:* Vascular, smooth muscle relaxant—10–25 mg BD.

In hypertensive crisis: Any of the following drug can be used till blood pressure comes down to <110 mm Hg.

1. *Labetalol:* 200 mg in 200 ml of normal saline at the rate of 20 mg/hr to be doubled every ½ hr.
2. *Hydralazine:* 5 mg IV bolus to be followed by infusion of 25 mg in 200 ml normal saline, rate is 2.5 mg/hr to be doubled every 30 min.
3. *Nitroglycerin:* 5 µg/min IV or sodium nitroprusside—0.25–5 µg/kg/min

Once the blood pressure is under control, oral therapy can be continued.

Monitoring of patient: The effect of treatment is evaluated by recording:

- *Blood pressure:* At least every 6 hourly.
- Daily observation of weight gain and edema.
- Fluid intake and urinary output charting.
- Daily urinary protein estimation.
- Once a week blood examination for:
  - Hematocrit, platelet count.
  - Serum uric acid estimation.
  - Creatinine level.
  - Liver function test.
- *Ophthalmoscopic examination:* On admission and if necessary repeated.
- Fetal well-being assessment

Definitive treatment of pre-eclampsia is termination of pregnancy. The role of antihypertensive drugs is only to continue pregnancy, if possible without affecting the maternal prognosis till fetal maturity.

Criteria for Delivering Patients with Severe Pre-eclampsia

1. Blood pressure persistently 160/100 or greater despite treatment.
2. Urine output <400 ml/24 hr.
3. Platelet counts <50,000/mm^3
4. Progressive increase in serum creatinine.
5. LDH >1000 IV/L.
6. Repetitive late deceleration with poor variability.
7. Severe IUGR with oligohydramnios
8. Decreased fetal movement.
9. Reversed umbilical diastolic blood flow.

Method of Termination of Pregnancy

1. Induction of labor.
2. Cesarean section.