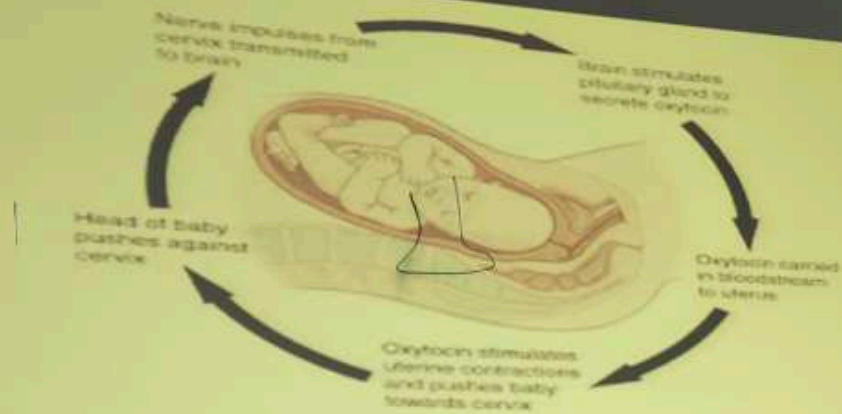


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Physiology of Labour

Dr Shabnum Sibtain



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The process that initiates labour is poorly understood.



The onset of labour occurs when the factors that inhibit contractions and maintain a closed cervix diminish and are overtaken by the actions of factors that do the opposite

Labour is the process by which the fetus and the placenta and membranes are expelled through the birth canal.

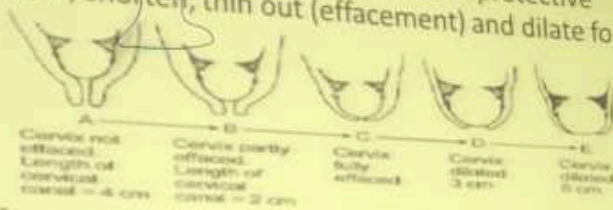
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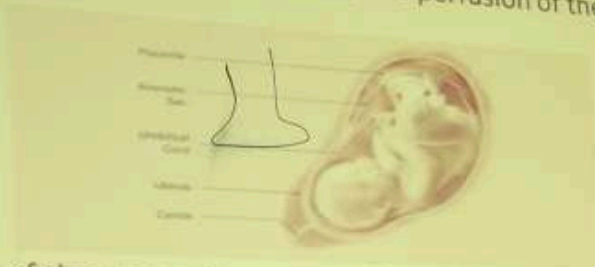
There are a number of important elements

The cervix, which is initially long, firm, and closed, with a protective mucus plug, must soften, shorten, thin out (effacement) and dilate for labour to progress.



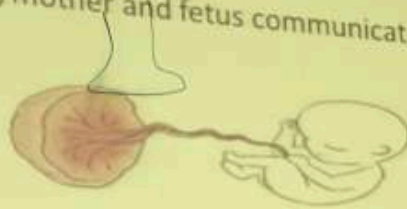
The uterus change from a state of relaxation to an active state of regular, strong, frequent contractions to facilitate the fetus through the birth canal.

Each contraction must be followed by a resting phase in order to maintain placental blood flow and adequate perfusion of the fetus.



The pressure of the presenting part on the pelvic floor muscles as the fetus descends from the midpelvis to the pelvic outlet produces a maternal urge to push, enhanced further by stretching of the perineum.

Throughout gestation, mother and fetus communicate with each other via the placenta.



This communication includes the placental production, metabolism and/or distribution of hormones.



fetal adrenal gland

Towards the end of pregnancy, the developing fetal adrenal glands produce increasing amounts of cortisol. This steroid acts to mature foetal organ systems, especially the lungs.

The maturing fetal adrenal also produces increasing amounts of dehydroepiandrosterone (DHEA), which is an important precursor to oestrogen production in the placenta.

Oestriol, the dominant oestrogen in pregnancy, increases in the mother's circulation and promotes the uterine activation phase .

Oestrogen effects

An increase in uterine gap junctions.

Gap junctions are intercellular connections that enhance the spread of electrical signals.

This essentially 'wires up' the uterus for effective coordinated contractions in labour.

Under the influence of oestrogen and prostaglandins, gap junctions increase substantially up to labour onset.



Progesterone effects

Progesterone maintains uterine relaxation
By suppressing prostaglandin production,
Inhibiting communication between myometrial cells
Preventing oxytocin release.



Prior to labour, there is a reduction in progesterone receptors and an increase in the concentration of oestrogen relative to progesterone.

Oxytocin



Uterine oxytocin receptor numbers rise from mid pregnancy to term, and are even higher in early labour.

Strong contractions with oxytocin drive a positive feedback cycle, whereby uterine sensations trigger oxytocin release from the maternal brain, causing stronger contractions and more oxytocin release.

Other physiological processes

Reduction in quiescence

Prostaglandins and inflammation.



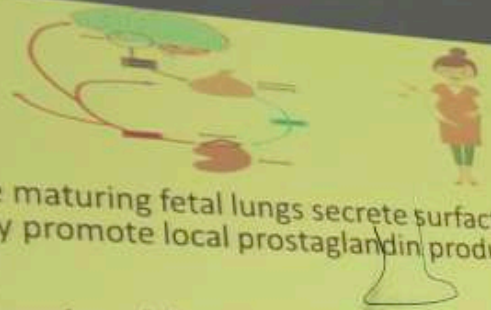
There is an inflammation in local fetal and maternal tissues even before labour onset.

Pro-inflammatory chemicals, including cytokines and interleukins, promote the production of prostaglandins, which help with collagen degradation and cervical ripening.

Prostaglandins in the uterus promote contractility by increasing oxytocin receptors and gap junctions and also increasing placental production of the Corticotropin Releasing Hormone (CRH).



The production of CRH by the placenta increases in concentration towards term and potentiates the action of prostaglandins and oxytocin on myometrial contractility.



The maturing fetal lungs secrete surfactants into amniotic fluid, which may promote local prostaglandin production and uterine contractility.

Loosening of the tissue interface between the fetal membranes and the uterine wall results in raised levels of fetal fibronectin in the week or so prior to labour onset.

The fetal pituitary secretes oxytocin and the fetal adrenal gland produces cortisol, which stimulates the conversion of progesterone to oestrogen.



In pregnancy, large amounts of CRH are made by the placenta.

CRH stimulates fetal adrenal DHEA production and subsequent placental oestrogen production, promotes inflammation, and has direct uterine pro-contractile effects.

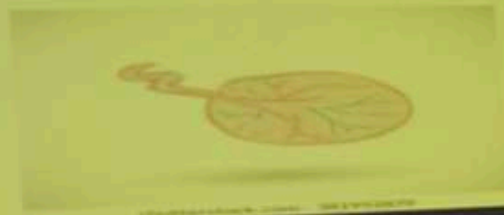


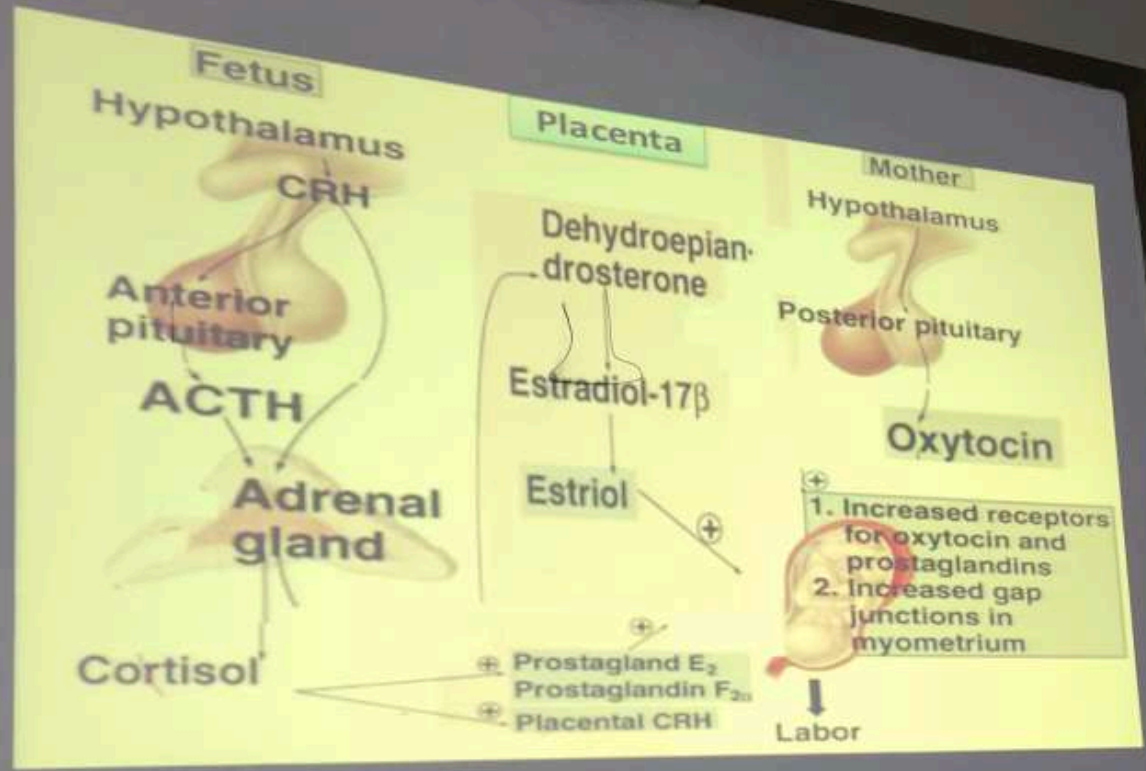


In pregnancy, large amounts of CRH are made by the placenta.

CRH stimulates fetal adrenal DHEA production and subsequent placental oestrogen production, promotes inflammation, and has direct uterine pro-contractile effects.

Both CRH and prostaglandins are also involved in positive feedback loops that drive labour .







Pregnancy



Late pregnancy



Labour/birth



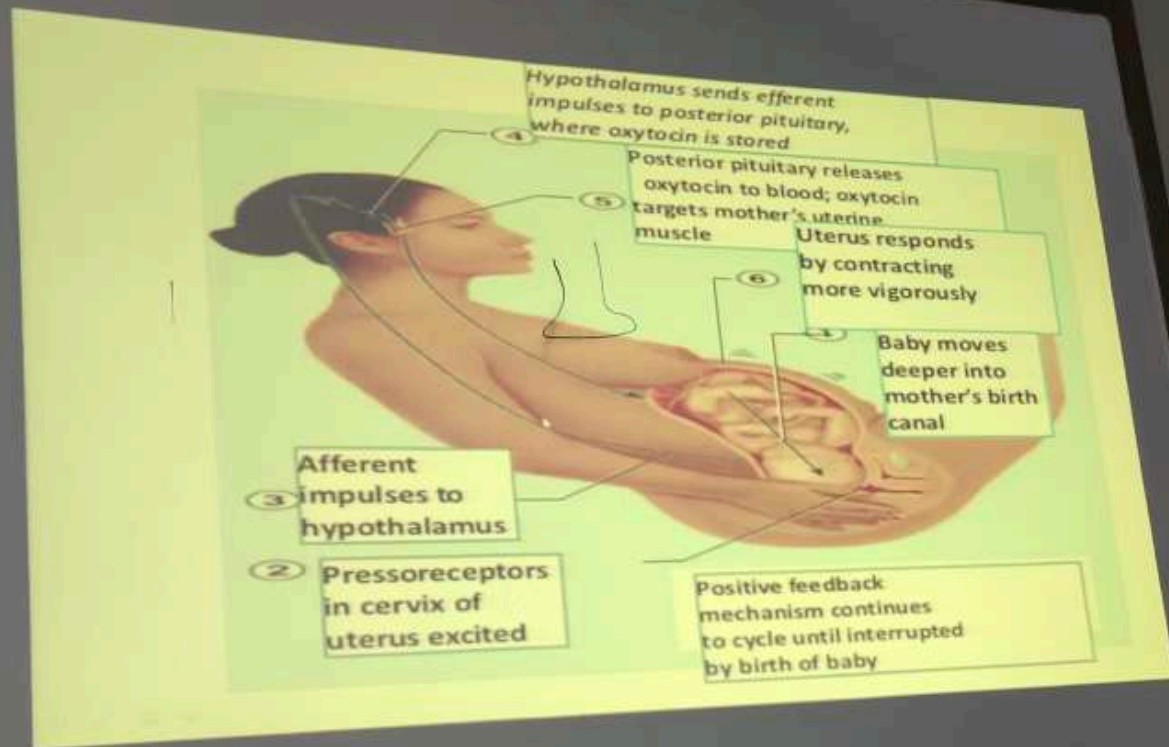
Postpartum

Phase 0
Quiescence
 ↑ Progesterone
 ↑ CRH (Corticotropic Releasing Hormone)

Phase 1
Activation
 ↓ Progesterone
 ↑ Estrogen
 ↑ Oxytocin receptors
 ↑ Gap junctions
 ↑ Inflammation
 ↑ Prostaglandins
 ↑ Cervical ripening
 ↑ CRH

Phase 2
Stimulation
 ↑ Oxytocin
 ↑ Prostaglandins
 ↑ CRH

Phase 3
Involution
 ↓ Progesterone
 ↓ Estrogen
 ↑ Oxytocin (breastfeeding)



Impact of late pregnancy/labour initiation priming on the fetus/neonate

In addition to these fetal pre-labour preparations, the processes of physiological labour and birth further optimize the fetal-to-newborn transition.

The so-called 'stress of being born' involves a late-labour surge of adrenaline and noradrenaline, which maximizes newborn adaptations, including the respiratory transition.

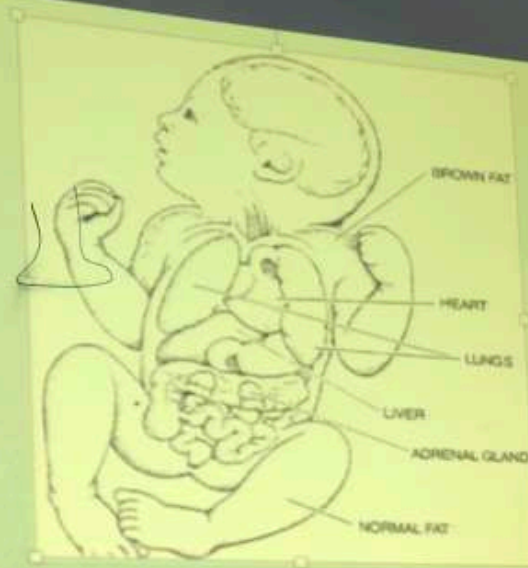
IMPROVES BREATHING
INCREASES LUNG SURFACTANT
INCREASES LUNG-LIQUID ABSORPTION
IMPROVES LUNG COMPLIANCE
DILATES BRONCHIOLES

PROTECTS HEART AND BRAIN
INCREASES BLOOD FLOW TO VITAL ORGANS

MOBILIZES FUEL
BREAKS DOWN NORMAL FAT INTO FATTY ACIDS
BREAKS DOWN GLYCOGEN (IN LIVER) TO GLUCOSE
STIMULATES NEW PRODUCTION OF GLUCOSE BY LIVER

FACILITATES BONDING
DILATES PUPILS
APPEARS TO INCREASE ALERTNESS

INITIATES THERMOREGULATION
BURNS BROWN FAT



Lagercrantz, H. and T. A. Slotkin (1986). "The "stress" of being born." *Sci Am* 254 (4): 100-7 Used with permission...

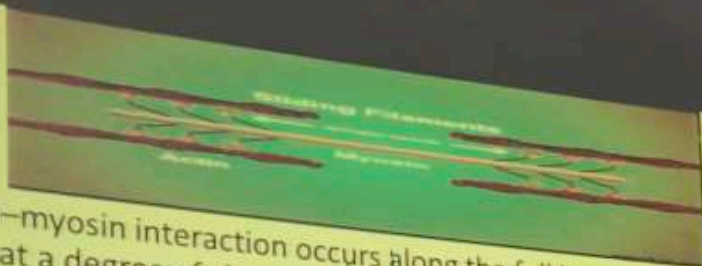
The uterus



Myometrial cells of the uterus contain filaments of actin and myosin, which interact and bring about contractions in response to an increase in intracellular calcium.

Prostaglandins and oxytocin increase intracellular free calcium ions, whereas beta-adrenergic compounds and calcium-channel blockers do the opposite.

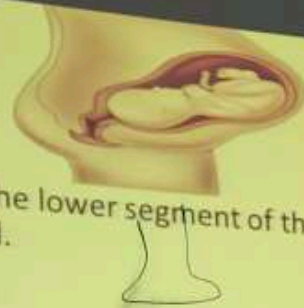
Separation of the actin and myosin filaments brings about relaxation of the myocyte



Actin-myosin interaction occurs along the full length of the filaments so that a degree of shortening occurs with each successive interaction.

This progressive shortening of the uterine smooth muscle cells is called retraction and occurs in the cells of the upper part of the uterus.

The result of this retraction process is the development of the thicker, actively contracting 'upper segment'.

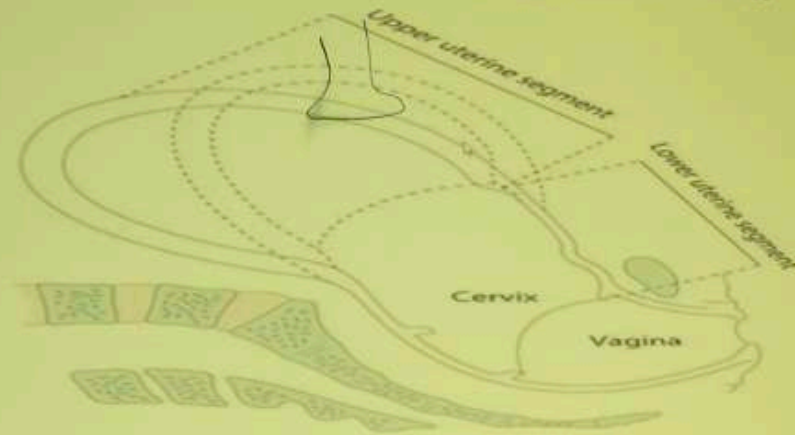


At the same time, the lower segment of the uterus becomes thinner and more stretched.

This results in the cervix being 'taken up' (effacement) into the lower segment of the uterus so forming a continuum with the lower uterine segment.

The cervix effaces and then dilates, and the fetus descends in response to this directional force

The dotted lines indicate the position assumed by the uterus during contraction.



PHYSIOLOGY OF THE UTERINE CONTRACTION

The contraction wave starts normally in the most proximal part and spreads distally (fundal dominance).

Because of the relative amounts of contractile protein (actomyosin) in the upper, middle and lower segments of the uterus a coordinated contraction wave is achieved.

That is, the contraction wave in the fundus starts some 12-15 seconds before that in the lower segment, but because of the greater strength and duration of the former, the peak of the two contraction waves coincide.



WHAT CHANGES TO BRIDGE THIS GAB? CONTRACTION AND THICKENING OF THE BILLY MUSCLES, AND EXPANSION OF THE UTERUS EXPANDING BLASTOCYST CENTER SOURCE

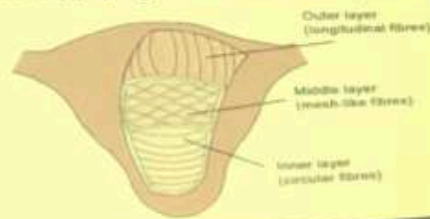
EVERY CONTRACTION EVER

The onset of labour is marked physiologically by the commencement of retraction which is a phenomenon unique to uterine muscle in labour and which results in permanent shortening of the fibres.

Most of the growth of the uterus in pregnancy is due to proliferation of the myometrium with muscle cells increasing both in size and number.

The uterus muscle fibres

- The muscle fibres of the uterus in late pregnancy and in labour are arranged in three indefinite overlapping layers.
 - (1) Outer longitudinal fibres which form a relatively thin layer.
 - (2) A thick mass of middle interlacing fibres which run spirally downwards and inwards to form an interdigitating network perforated by the spiral arterioles and vessels supplying the decidua and placenta.
 - (3) Inner circular fibres



It is essential that the myocytes of the uterus contract in a coordinated way.



Individual myometrial cells are laid down in a mesh of collagen.

There is cell-to-cell communication by means of gap junctions, which facilitate the passage of various products of metabolism and electrical current between cells.

The musculature is most abundant in the upper uterine segment and the fibres thin out as they approach the cervix.

Thus, in normal labour the upper segment is actively contracting and retracting, while the lower segment is more elastic and relatively passive.

Uterine contractions are involuntary in nature and there is relatively little extrauterine neuronal control.

Labour contractions

The frequency of contractions may vary during labour and with parity.

In the majority of labour, they occur at intervals of 2–4 minutes and are described in terms of the frequency within a 10-minute period (i.e. 2 in 10 increasing to 4–5 in 10 in advanced labour).

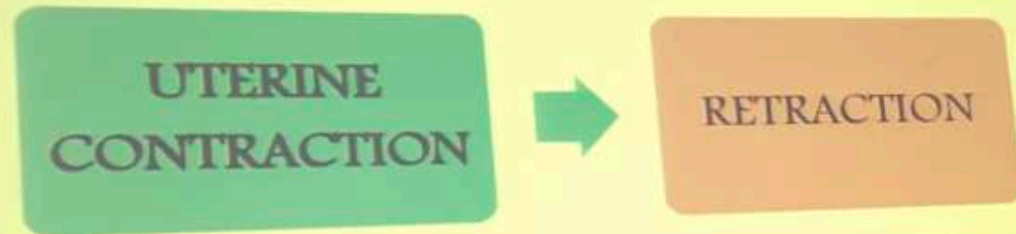
Their duration also varies during labour, from 30 to 60 seconds or occasionally longer.

The frequency of contractions can be recorded on a Cardiotocography (CTG) using a pressure transducer.

The intensity or amplitude of the intrauterine pressure generated with each contraction averages between 30 and 60 mmHg.

PHYSIOLOGY OF NORMAL LABOUR

Physiology of normal labour complete into two steps

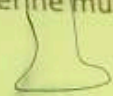


Retraction

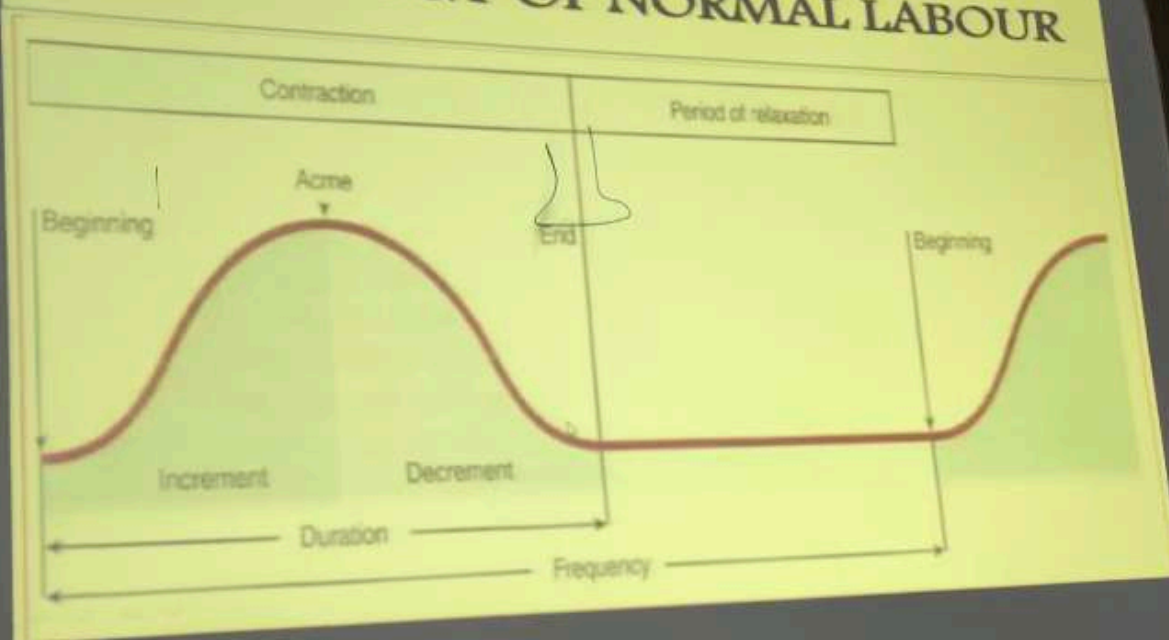
Permanent shortening of uterine muscles.

It results in

- Formation of lower uterine segment
- Reduce surface area of uterus hence favouring placental separation
- Effective haemostasis after separation of placenta.



PHYSIOLOGY OF NORMAL LABOUR



The cervix

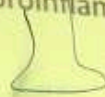


The cervix contains myocytes and fibroblasts separated by a 'ground substance' made up of extracellular matrix molecules.

Interactions between collagen, fibronectin and dermatan sulphate (a proteoglycan) during the earlier stages of pregnancy keep the cervix firm and closed.

Under the influence of prostaglandins, and other humoral mediators, there is an increase in proteolytic activity and a reduction in collagen and elastin.

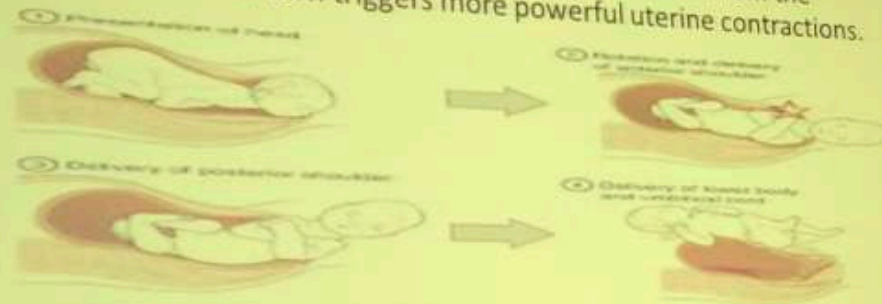
Interleukins bring about a proinflammatory change with a significant invasion by neutrophils.



Dermatan sulphate is replaced by the more hydrophilic hyaluronic acid, which results in an increase in water content of the cervix.

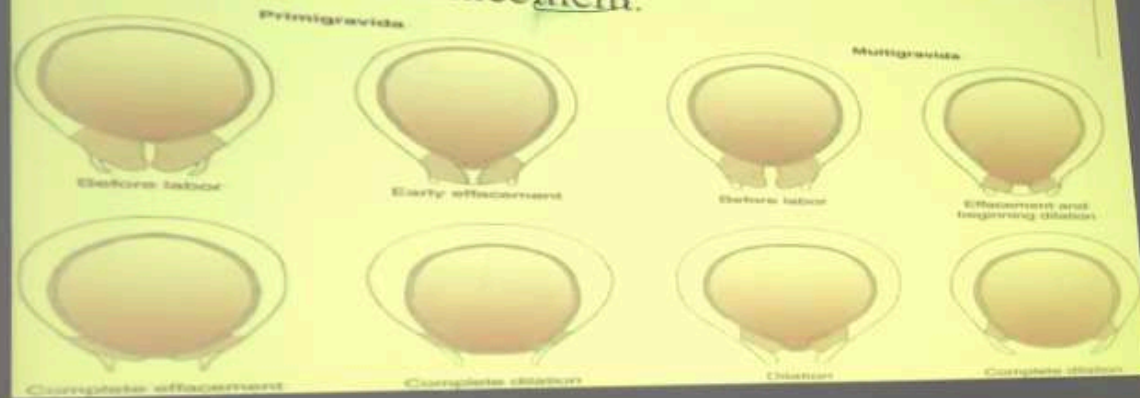
This causes cervical softening or 'ripening', so that contractions, when they begin, can bring about the processes of effacement and dilatation.

- Cervical stretching induces reflexive uterine contractions that dilate and efface the cervix further
- In addition, cervical dilation boosts oxytocin secretion from the pituitary, which in turn triggers more powerful uterine contractions.



DEFINITION OF NORMAL LABOUR

Labour is defined as the presence of regular uterine contractions with progressive cervical dilatation and effacement.



PHYSIOLOGY OF NORMAL LABOUR

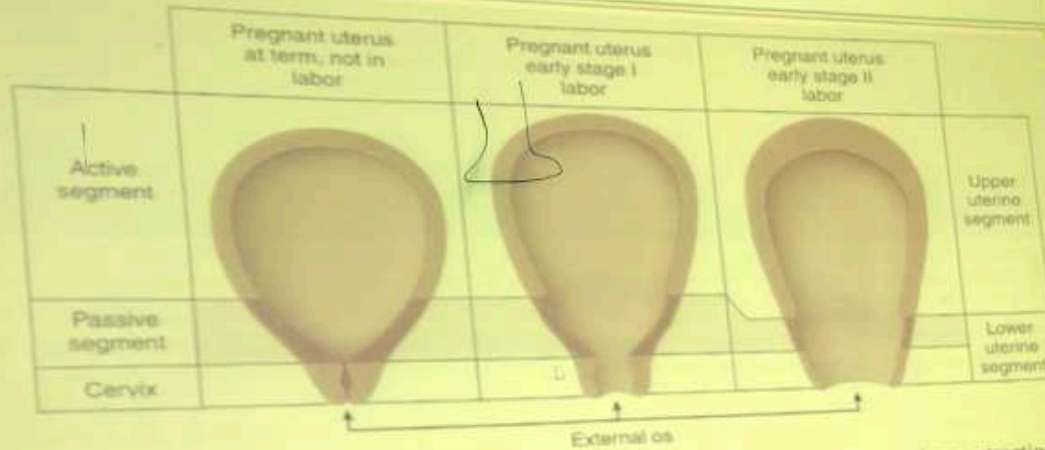


FIGURE 8.1. Mechanism of effacement, dilation, and labor. With continuing uterine contractions, the upper uterus (active segment) thickens, the lower uterine segment (passive segment) thins, and the cervix dilates. In this way, the fetus is moved downward, into and through the vaginal canal.

CLINICAL SIGNIFICANCE OF BENIGN EPITHELIAL LESIONS

LESION	RELATIVE RISK (ABSOLUTE LIFETIME RISK)
NON - PROLIFERATIVE BREAST CHANGES	1(3%)
PROLIFERATIVE DISEASE WITHOUT ATYPIA	1.5-2(5 - 7%)
PROLIFERATIVE DISEASE WITH ATYPIA	4-5(13 - 17%)
CARCINOMA IN SITU	8-10(25 - 30%)