**EXAMINATION, ADAPTATION, CARE OF THE NEWBORN**

Learning Objectives

1. **Key Areas of Newborn** Examination

 - Understand the importance and timing of neonatal examination.

 - Learn a systematic approach to physical examination.

 - Identify common neonatal conditions during examination.

2. Provide an overview of neonatal adaptation.

 **Key Points**: - Definition of neonatal adaptation

 - Importance of understanding this process for clinical practice

3. **Key Areas of Newborn Care**

NEWBORN

Period from birth - 28 days of life

**1. Importance of Neonatal Examination**

**-Early detection** of congenital anomalies or health issues.

- Establish a baseline for the newborn's health.

- **Guide immediate and long-term care plans**.

“The early years last a lifetime”

Timing of Neonatal Examination

* Immediately after birth: Assess APGAR score and stabilize.
* Within 24 hours: Conduct a detailed physical examination.
* Before discharge: Ensure newborn readiness for home care.

INTRODUCTION

* Monitoring of neonates is the keynote to their successful outcome.
* Accurate nursing observation is a vital factor in the survival and future development of newborn.
* The initial physical examination should be performed as soon as after the birth.
* • All newborns should be thoroughly examined in the first 24-48 hrs of age.
* The basic tools of assessment are the human senses of vision, hearing, touch and smell.
* Examination of newborn entails investigation into the history using different techniques namely inspection, palpation, percussion and auscultation.
* The newborn assessment database includes information gathered from the history, reviewing mother’s record, head to toe examination for physical and neurological characteristics and is used to establish nursing priorities, which guide nursing diagnosis and nursing interventions.

PURPOSE OF EXAMINATION

The overall purposes of new born examination are to:

* Identify the physical and neurological characteristics of new born.
* Identify and record evidence of common neonatal problems and congenital anomalies.
* Provide a basis for identification of needs and plan nursing care of new born.

SPECIFIC INSTRUCTIONS

To perform thorough skilled examination of newborn, the following specific instructions should be kept in mind:

1. Observation should be made when newborn is quiet and awake.

1. Ensure adequate light in examination room.
2. The temperature of the examination room is maintained at 28 +/- 2 degree C. avoid draft and chills in the examination room.
3. Wash your hands till elbow for 3 minutes before and after handling the newborn.

SYSTEMATIC ASSESSMENT OF NEWBORN

* Neonatal assessment is done systematically from birth till discharge of neonate.
* Examination of newborn soon after birth is done very quickly. Examination at birth includes assessment of certain important parameters, to evaluate the adjustment of newborn to these life processes.

On the basis of time of performing, assessment is of three types:

* 1. Immediate assessment of newborn
2. Transitional assessment during period of reactivity
* 3. Periodic assessment.

PURPOSES

The purposes of first examination at birth are:

* 1. To ensure the patency of orifices and spontaneous breathing.
* 2. To identify life threatening congenital malformations and birth injuries.
* 3. To classify the new born according to weight and gestational age.

Immediate response of newborn to extra uterine life can be determined by:

* Apgar score at one, five and ten minutes.
* Birth weight
* Length
* Axillary temperature
* Patency of orifices – anal patency, esophageal atresia.

A detailed examination of newborn is performed after 24 hrs of birth.

FIRST DAY

New born can tolerate much handling after first day, as they recover from labour stress.

• Examination of newborn within first 24 hrs include information about physiological establishment and future physiological changes that the newborn might undergo.

• Therefore, a thorough assessment that identifies normal and abnormal findings , facilitates planning of care by nurses.

PUPROSES

The purposes of first day examination are to:

* Identify any congenital anomaly missed out at
* birth.
* Assess feeding behavior.
* Ensure passage of urine and stool.
* Perform thorough head to toe examination.
* Record measurements.

**IMMEDIATE ASSESSMENT OF NEW BORN**

For assessment of baby immediately after birth, APGAR scoring is done.

* APGAR scoring is a quantitative method of assessing infant’s respiratory , circulatory and neurological status.
* APGAR scoring is done at 1 min & 5 minutes after birth.
* Maximum APGAR score is 10 & the score of more than 7 is considered satisfactory & indicates absence of difficulty in adjusting to extra uterine life.
* Score 4-6 : Moderate distress
* 0-3 : Severe distress

Immediate newborn assessment includes:

* APGAR scoring
* Recording of birth weight
* Umbilical cord is examined for presence of 2 umbilical arteries and 1 vein.
* Orifice counting & checking their patency.
* Mouth is checked for cleft palate and lip.
* Ears and nose
* Anus is checked for imperforation or malformation.
* Urethra is checked for hypospadias or epispadias.
* Any visible lesions on back or front

**Examination at 24 hrs: Assess**

**Ask**

* Breastfeeding
Activity of the baby o Any other problems\*

**Check**

* Weigh the baby o Temperature

**Record**

* *Passage of meconium up to 24 hrs and urine up to 48 hrs of life is usually normal*

ROUTINE EXAMINATION

Detailed examination on routine basis is not required.

• But till the time, the new born remains in the hospital the new born should be observed for feeding behavior and maintenance of temperature, jaundice, seizures and any superficial infections.

• The mother should be enquired about the behaviour of the new born eg; feeding problems, passage of urine and stool, vomiting.

PURPOSE OF ROUTINE EXAMINATION

• To assess the feeding behavior.
• To detect any superficial infection.
• To assess the temperature maintenance.

• To identify any feeding problem.

ON DISCHARGE

Before the new born is sent home, a detailed examination is necessary. The purposes are:

-  To identify any anomaly and birth injury which might have got missed out at earlier examination.

-  To assess any other problem.

-  To educate the mother about care of new born at home.

-  To record baseline data for future comparison.

-  To refer the newborn, if needed.

STEPS OF EXAMINING THE NEW BORN

* Place the newborn on a flat surface at a comfortable height to yourself.
* The examiner’s hands must be dry and warm, as cold hands startle the new born. Warm up your hands by drying and rubbing.
* The examiner’s nail should be short and free of nail polish.
* Handle newborn gently.
* Don’t expose the newborn unnecessarily. Redress after completion of examination.
* Proceed systematically.
* The sequence in which the various features of the examination are assessed is a matter of personal preference.
* Generally, the nurse begins by performing examination of those areas that require newborn to be in quiet state. Eg, counting respiratory rate.
* Measure head and chest circumference and length at same time to compare the results.
* Involve parents during newborn examination, by swaddling, holding, keeping the baby clean.
* Avoid performing a detailed assessment just before or after feeding.
* The findings should be recorded promptly, accurately and systematically.
* Collect required articles, ensure proper functioning and that they are accessible.

**Assess:**

Listen for

* **Grunting, Cry, Heart sounds**

Feel for

* Any abnormal swelling: Caput, cephalhematoma
* Palpable femoral pulses
* Dislocation of hip
* Capillary refill time ( CRT)
* Confirm the findings of inspection
* Palpate the abdomen
* Feel for testes in male baby

Variable. - Range of normal values

Heart rate. - 120-160 at rest (100-180) bpm

Respiratory rate - 40-60 breaths per minute

Temperature - 36.5 – 37.5 OC

Lenght - 48-52 cm

Head circumference - 32-38 cm

Wheight - 2500 – 4000 g

Term of gestation - 37 – 42 weeks of gestation

\*Note – these variants of normal apply to term babies born after 37 weeks’ gestation

Tips for Effective Examination

- Ensure a warm, calm environment.

- Use gentle techniques to avoid distress.

- Document all findings systematically.

**Head to toe examination**

Neonatal Reflexes

* Palmar and plantar grasp reflex
* Moro reflex (startle reflex)
* Rooting reflex
* Sucking reflex
* Babinski reflex
* Step reflex
* Tonik neck reflex (fencing reflex)
* Reflex name
* Reflex responses
* Reflex disappears
* <https://www.youtube.com/watch?v=rHYk1sYsge0> (video)

**2. Overview of neonatal adaptation**

* The transition from a fetus to a newborn is the most complex physiologic adaptation that occurs in human experience. Prior to medicalization of delivery, the transition had to occur quickly for survival of the newborn. All organ systems are involved at some level, but the major immediate adaptations are the establishment of air breathing concurrently with changes in pressures and flows within the cardiovascular system.
* Other essential adaptations are striking changes in endocrine function, substrate metabolism, and thermogenesis ([Box 1](https://pmc.ncbi.nlm.nih.gov/articles/PMC3504352/)). Hospital based deliveries increase the difficulties for transition for many fetuses because of the frequent use of Cesarean sections, deliveries prior to the onset of labor, rapid clamping of the cord, and the anesthetics and analgesics associated with these hospital deliveries. The net result is the frequent need to assist the newborn with the birth transition. Preterm deliveries cause particular difficulties for transition and expose the preterm infant to lung injury from mechanical ventilation. These components of the fetal to neonatal transition will be reviewed for preterm and term deliveries.

Box 1. Essential components for a normal neonatal transition.

* Clearance of fetal lung fluid
* Surfactant secretion, and breathing
* Transition of fetal to neonatal circulation
* Decrease in pulmonary vascular resistance and increased pulmonary blood flow
* Endocrine support of the transition

B. Endocrine adaptions to Birth
1. Cortisol

* Cortisol is the major regulatory hormone for terminal maturation of the fetus and for neonatal adaption at birth ([1](https://pmc.ncbi.nlm.nih.gov/articles/PMC3504352/)). The “cortisol surge” is initiated with the switch from maternal-transplacental derived corticosteroids to the ability of the fetal adrenal to synthesize and release cortisol under fetal hypothalamic control. Fetal cortisol levels in the human are low (5–10ug/ml) relative to normal cortisol levels until about 30 weeks gestation.
* Cortisol levels progressively increase to about 20ug/ml by about 36 weeks gestation and increase further to about 45ug/ml prior to labor at term.
* Cortisol increases further during labor to peak at high levels of about 200ug/ml several hours after term delivery. The increase in fetal cortisol throughout late gestation supports multiple physiologic changes that facilitate normal neonatal adaption. For example over the final weeks of gestation, the conversion of T4 to T3 increases, catecholamine release by the adrenal and other chromaffin tissues increases, glucose metabolic pathways in the liver mature, gut digestive capacity increases (enzyme induction), β-adrenergic receptor density increases in many tissues including the heart and the lungs, and the surfactant system in the lungs is induced to mature
* Cortisol in association with increasing thyroid hormones activates the sodium pump that clears fetal lung fluid at birth. These cortisol-modulated changes are normally a progressive process of preparation for birth as the cortisol levels rise prior to birth then peak soon after delivery. This normal increase in cortisol supports an integrated transition following birth.
* Cesarean section without labor at term blunts the postnatal rise in cortisol, and the cortisol responses to preterm birth also are attenuated because of unresponsiveness and immaturity of the adrenal gland ([3](https://pmc.ncbi.nlm.nih.gov/articles/PMC3504352/)). A particularly stressful delivery can uncover a “functional” adrenal insufficiency if the adrenal gland cannot respond to the increased stress. The very preterm infant may have low cortisol levels around birth with symptoms such as low blood pressure that are responsive to cortisol treatment. In contrast antenatal exposure to chorioamnionitis may increase fetal cortisol levels prior to delivery ([4](https://pmc.ncbi.nlm.nih.gov/articles/PMC3504352/)).

2. Catecholamines

* The term human fetus can release catecholamines (norepinephrine, epinephrine, and dopamine) from adrenal medullary and other sympathetic tissues in response to fetal stresses of various sorts, as evaluated by catecholamine values in cord blood.
* The preterm fetus has higher cord catecholamine levels than the term fetus, and cesarean delivery is associated with lower cord catecholamine levels.
* The catecholamine surge is primarily responsible for the increase in blood pressure following birth, adaption of energy metabolism with support of the primary substrates for metabolism after birth – glucose and fatty acids, and for initiating thermogenesis from brown fat. The preterm secretes more catecholamines because the organ systems are less responsive – higher concentration thresholds for response and lower responses. Cesarean section of the unlabored fetus depresses catecholamine release. Catecholamine release at birth can be viewed as the “gas” that drives the adaptive responses. However, fetal exposure to cortisol is the “carburetor” that is the potent regulator of the responses of the newborn to catecholamines.

3. Thyroid Hormones

* The thyroid axis matures in late gestation in parallel to the increase in cortisol with increased thyroid simulating hormone (TSH), T3 and T4 levels, and decreased rT3 levels as term approaches.
* Following term birth, TSH quickly peaks and decreases, and T3 and T4 increase in response primarily to the increased cortisol, to cord clamping and to the cold stimulus of birth.
* Acute ablation of thyroid function at birth did not greatly alter thermogenesis or cardiovascular adaptation in experimental animals. However, inhibition of thyroid function more chronically prior to birth did interfere with postnatal cardiovascular adaptation and thermogenesis in newborn lambs.
* These results demonstrate a supportive and preparative role for thyroid hormones for birth rather than as acute modulators of endocrine adaptation to birth. For example, fetal infusions of T3 and cortisol can activate the Na+, K+, ATPase that helps clear fetal lung fluid after birth. Term infants with congenital hypothyroidism generally do not have abnormalities of early neonatal adaptation that are evident in the controlled environment of hospital deliveries. Very preterm infants have a blunted thyroid functional transition from fetal to newborn life with very low levels of plasma T3 and T4relative to term infants. The effects of the depressed thyroid function on the early postnatal transition in the preterm are unclear but probably contribute to the depressed adaptive behavior of the preterm.

C. Metabolic Adaptations
1. Energy Metabolism

* Fetal energy needs are supported primarily by the transplacental transfer of glucose to the fetus. Although the fetal liver is capable of gluconeogenesis from early gestation, gluconeogenesis is minimal during normal fetal homeostasis.
* Rather as term approaches glucose and other substrates are being stored as glycogen and fat in anticipation of birth in the high insulin and low glycogen fetal environment. With delivery and cord clamping, the maternal glucose supply is removed, and plasma glucose levels normally fall over the early hours after birth.
* The glucose and free fatty acid levels are accompanied by a fall in insulin, and increase in glycogen, the normal glucose homeostatic hormones. However, the large catecholamine release and increase in cortisol are probably the major acute regulators of plasma glucose and free fatty acid levels in the immediate newborn period.
* Cortisol and catecholamine responses to preterm birth are dysregulated with less cortisol and more catecholamine release. The preterm also has minimal glycogen and fat stores. Therefore, the availability of energy substrates during the birth transition will be severely challenging for the preterm. This aspect of adaptation in the immediate newborn period is treated routinely with glucose infusion to prevent hypoglycemia. However, the integrated effects of the endocrine abnormalities and responses to glucose infusions have not been well described in extremely low birth weight infants.

2. Thermoregulation

* Fetal body temperature is about 0.5°C above the maternal temperature. Although the fetus produces heat from metabolism, that heat is effectively dissipated across the placenta and fetal membranes.
* At birth the sympathetic release resulting from the redundant stimuli of increased oxygenation, ventilation, cord occlusion and a cold stimulus to the skin activates thermogenesis by brown adipose tissue. This thermogenic response potential has developed during late gestation by an increase in brown adipose tissue around the kidney and in the intrascapular areas of the back to become about 1% of fetal weight at term.
* Brown adipose tissue generates heat by uncoupling oxidative metabolism from ATP synthesis in the mitochondria, with the release of heat.
* This uncoupling is mediated by the mitochondrial membrane protein uncoupling protein 1 (UCP1) which is activated by norepinephrine released by the sympathetic innervation of brown adipose tissue. UCP1 levels increase in the brown adipose tissue during late gestation in response to a local conversion of T4 to T3 and to induction of UCP1 synthesis in response to the increasing cortisol levels in the fetal plasma as term approaches. Thus the same hormones that modulate the fetal preparation for birth and the transition period are central to thermogenesis by brown adipose tissue. The term infant also can generate some heat by shivering thermogenesis, which is an increase in non-purposeful skeletal muscle activity signaled by cutaneous nerve endings via central motor neurons. Shivering thermogenesis seems to be of secondary importance to the newborn human. The preterm human is at a major disadvantage for thermoregulation following birth as brown adipose tissue has not developed in quantity or response potential for a cold stress.

D. Cardiovascular Adaptations

* Profound changes in the cardiovascular system occur after delivery in response to removal of the low resistance placenta as the source of fetal gas exchange and nutrition. Much of our knowledge regarding cardiovascular adaptation after birth is based on studies in animals, particularly the sheep. The major changes are an increase in the cardiac output and transition of fetal circulation to an adult type of circulation. Increased cardiac output is required to provide for increases in basal metabolism, work of breathing, and thermogenesis. In the close-to-term fetus, the combined ventricular output is about 450 mL/kg/min, with the right ventricular output accounting for 2/3rd of the cardiac output and the left ventricle ejecting 1/3rd of the cardiac output. Soon after birth, the circulation changes from “parallel” to “series”, where the right ventricular output equals the left ventricular output. The cardiac output nearly doubles after birth to about 400 mL/kg/min (for the right and the left ventricle). This marked increase in cardiac output parallels closely the rise in oxygen consumption. The organs experiencing increased blood flow after birth are the lungs, heart, kidney and the gastrointestinal tract. Although the precise mechanisms mediating increased cardiac output after birth are not known, the increase in cortisol and vasoactive hormones, that include catecholamines, the rennin-angiotensin system, vasopressin and thyroid hormone contribute to support of blood pressure and cardiovascular function.
* In the fetus, the relatively well-oxygenated blood from the placenta is delivered via the umbilical cord and ductus venous. This ductus venous blood enters the right atrium from the inferior vena cava and is directed preferentially to the left atrium by the foramen ovule and subsequently delivered preferentially to the brain and the coronary circulation by the fetal left ventricle. The right ventricle is the predominant ventricle in the fetus, and most of the right ventricular output goes to the descending aorta via the ductus arteriosus since very little blood enters the pulmonary circulation. With birth and removal of the low resistance placenta, blood flow increases to the pulmonary circulation. Shortly after birth functional closure of ductus arteriosus begins. The mechanisms contributing to the high pulmonary vascular resistance in the fetal lung are primarily the low oxygen tension and low pulmonary blood flow which suppresses the synthesis and release of nitric oxide (NO) and prostaglandin I2 from the pulmonary endothelium. Fetal exposure to hypoxia will increase the already high pulmonary vascular resistance and hyperoxia will decrease pulmonary vascular resistance and increase fetal pulmonary blood flow. Experimentally, ventilation of the fetal lung without changing oxygenation will decrease pulmonary vascular resistance and increase pulmonary blood flow by 400%. With delivery, ventilation, and oxygenation, NO and PGI2 increase with a rapid fall in pulmonary vascular resistance. The use of supplemental oxygen for the initiation of ventilation will cause pulmonary vascular resistance to decrease more rapidly with the resultant more rapid increase in pulmonary blood flow ([24](https://pmc.ncbi.nlm.nih.gov/articles/PMC3504352/)). However, there is no benefit in systemic oxygenation, and the pulmonary vessels subsequently become more refractory to dilation by NO or acetylcholine.
* The cardiovascular transition at birth also is modulated by corticosteroids. Exposure of fetal sheep to betamethasone increased fetal pulmonary blood flow but did not alter postnatal pulmonary vasodilation in preterm sheep. Heart function after preterm birth is improved by antenatal exposure to corticosteroids.
* The fetal and newborn blood pressures increase, as does cardiac output and left ventricular contractibility. These effects are partially explained by an increase in beta-receptor signaling to an increase in cyclic AMP. Similarly adrenalectomy ablates the increase in blood pressure that normally occurs at birth.
* Thus, although there are specific mediators such as NO and PGI2 that facilitate cardiovascular transition, the consistent theme is that the same mediators – corticosteroids and catecholines also facilitate this transition.
* The normal oxygen saturation of fetal blood in the left atrium is about 65%. During labor the human fetus tolerates oxygen saturations as low as 30% without developing acidosis. After birth, the pre-ductal saturation in normal term infants gradually increases to about 90% at 5 minutes of age. This knowledge is important to avoid unnecessary administration of supplemental oxygen during resuscitation.

E. Lung Adaptations
1. Fetal Lung Fluid

* The most essential adaptation to birth is the initiation of breathing, but the airspaces of the fetal lung are filled with fetal lung fluid. What is fetal lung fluid and how is it cleared from the airspaces? Fetal lung fluid is secreted by the airway epithelium as a filtrate of the interstitial fluid of the lung by the active transport of chloride. Consequently the chloride content of fetal lung fluid is high and protein content is very low. The production rate is high, although direct measurements are not available for the human fetus. The volume of lung fluid of the fetal sheep increases from mid gestation and the secretion rate increases to about 4ml/kg/hr by late gestation. Production and maintenance of the normal volume of fetal lung fluid is essential for normal lung growth. The electrochemical gradient for the production of fetal lung fluid is substantial and can over-distend the airspaces. This behavior of the production of fetal lung fluid is used to advantage to obstruct the trachea, which will distend the hypoplastic lungs of fetuses with diaphragmatic hernia.
* The endocrine adaptations that begin before delivery are critical to fluid clearance. Cortisol, thyroid hormones and catecholamines all increase and shut down the active chloride mediated secretion of fetal lung fluid and activate the basal Na+, K+, ATPase of type II cells on the airway epithelium. Sodium in fetal lung fluid enters the apical surfaces of type II cells and is pumped into the interstitium with water and other electrolytes following passively, thus removing fluid from the airways. In preterm fetal sheep, infusion of cortisol and T3 will activate the sodium pump, which normally occurs at term. The components of fetal lung fluid then are cleared directly into the vasculature or via lymphatics from the lung interstitium over many hours.
* This clearance of a large volume of airspace fluid is remarkably efficient normally. The essential contribution of activation of Na+ transport was demonstrated by respiratory distress in animals from amiloride inhibition of the Na+, K+, and ATPase. Mice with defective Na+ transporters will die following delivery because of failure to clear fetal lung fluid. The frequent clinical scenario where retained lung fluid contributes to poor respiratory adaptation is the operative delivery of infants who were not in labor. These infants do not increase their oxygen saturations as quickly as vaginally delivered term infants, and there is an increased incidence of transient tachypnea of the newborn and other respiratory morbidities. In experimental studies in sheep, the increased volume of fetal lung fluid interferes with respiratory adaptation, and vaginal delivery facilitates adaptation relative to operative delivery at equivalent volumes of fetal lung fluid.
* Transient tachypnea of the newborn is most frequent in late preterm infants. This syndrome is thought to directly result from ineffective clearance of fetal lung fluid because of inadequate Na+transport, either because of decreased numbers of transporters or lack of activation. Preterm infants also have decreased Na+ transport, and late preterm infants with transient tachypnea of the newborn have low amounts of surfactant. Thus, the infant with transient tachypnea of the newborn has immaturity of Na+ transport and a tendency for surfactant deficiency while the infant with RDS has more severe surfactant deficiency that also includes immature Na+ transport. These two diseases probably are, in fact, a continuum of these two abnormalities from mild to severe.
* A hypothetical calculation may help the clinician to understand why lung fluid can compromise neonatal adaptation. If the 3 kg term infant has about 30 ml/kg of fetal lung fluid in the airspaces at Cesarean delivery without labor and that infant is intubated, then no fluid can passively drain from the lungs. Assuming that the blood volume of this infant is 80 ml/kg and the hematocrit is 50 %, then the plasma volume is 40ml/kg. The fetal lung fluid will move from the airspace to the lung interstitium initially interfering with lung mechanics and gas exchange. This fluid then will be transferred to the plasma, which if this occurred acutely would expand plasma volume from 40 ml/kg to 70 ml/kg. This transfer occurs over hours in reality. Nevertheless, the fetal lung fluid volume that must be accommodated during neonatal adaptation is added stress for the newborn.

2. Breathing at Birth

* The essential component to neonatal adaptation to birth is the maintenance of adequate respiratory effort. The stimuli changing the fetal breathing pattern virtually instantaneously to continuous breathing remain incompletely defined and probably are redundant as are the stimuli for other adaptations to birth. Most of the information about fetal breathing and it’s transition after birth is from quite old studies using fetal sheep models, with some verification in the human fetus. The fetal state in utero can be classified into REM sleep and quiet sleep with no clear periods of wakefulness. During REM sleep, the fetus has irregular breathing activity characterized by long inspiratory and expiratory times with movement of variable volumes of fetal lung fluid (mixed with amniotic fluid) into and out of the lung. Fetal breathing, swallowing and licking activities are confined to REM sleep, with minimal movements during quiet sleep. Fetal hypoxia abolishes fetal breathing while high fetal PO2 values stimulate fetal breathing. With birth, the fetal sheep will not breathe consistently until the cord is clamped. This observation has generated the hypothesis that breathing is suppressed by a placentally derived substance except in the REM state. Fetal sheep given prostaglandin E2 infusions stop breathing, and treatment with prostaglandin synthetase inhibitors such as indomethacin cause continuous fetal breathing. The net effect is that the normal fetal to neonatal transition results in the rapid onset of vigorous breathing because of the combined stimuli of cord clamping (and the probable removal of rapidly catabolized prostaglandins that suppress breathing), diffuse tactile and cold stimuli that act centrally, and changes in PCO2 and PO2 levels in the blood. The newborn will not initiate breathing if hypoxia is severe. Remarkably, in the absence of hypoxia, virtually all term infants will effectively initiate breathing. The majority of very preterm infants also will successfully initiate breathing if given opportunity.

3. Surfactant and Lung Adaptation

* The adequate development of the fetal lung to support gas exchange is the essential adaptation in preparation for birth. During the last third of gestation the fetal lung septates into about 4 million distal saccules (respiratory bronchioles and alveolar ducts) derived from the 17 generations of airways by about 32 weeks and then further separates to form alveoli. In parallel the lung parenchymal tissue mass decreases relative to body weight such that the potential gas volume of the airways and alveoli increase greatly.
* Concurrently from about 22 weeks gestational age surfactant lipid and the lipophilic proteins SP-B and SP-C begin to be synthesized and aggregated into lamellar bodies in the maturing type II cells. The lamellar bodies are the storage and secretory packets for the essential biophysically active components of surfactant. As the lung matures, more and more of the lamellar bodies are released into fetal lung fluid and subsequently mix with amniotic fluid or are swallowed. By term type II cells in the fetal lung contain much more surfactant than does the adult lung, and this large pool of surfactant is poised for release prior to and at delivery.

Key Points

1. The transition from fetal to extrauterine life is the summation of multiple rapid organ adaptations that often have redundant mediators.
2. The primary mediators that both prepare the fetus for birth and support the multi-organ transitions are cortisol and catecholamines.
3. Lung adaptation requires the coordinated clearance of fetal lung fluid, surfactant secretion, and the onset of consistent breathing.
4. Cardiovascular transition requires striking changes in blood flow, pressures and pulmonary vasodilation.
5. Abnormalities in adaptation are frequent following preterm birth or delivery by cesarean section at term.

3. **Newborn care**

* **Importance of Newborn Care**
	+ Newborns are vulnerable and require special attention for survival and well-being.
	+ The first 28 days of life (neonatal period) are critical for the infant's health.
	+ Medical professionals play a key role in ensuring proper care.

**Key Areas of Newborn Care**

* Immediate care after birth
* Assessment and monitoring
* Nutrition and feeding
* Preventive healthcare
* Recognizing and managing common newborn conditions
* Family and parental support

**Immediate Care After Birth**

* **Initial Steps**:
	+ Clear the airways if necessary.
	+ Dry the baby and stimulate breathing (if not crying).
	+ Apgar Score assessment at 1 and 5 minutes.
* **Thermal Protection**:
	+ Keep the baby warm (skin-to-skin contact).
	+ Avoid cold stress.
* **Cord Care**:
	+ Clamp and cut the umbilical cord.

**Neonatal Assessment**

* **Physical Exam**:
	+ Head-to-toe examination.
	+ Check for abnormalities (e.g., congenital defects).
* **Vital Signs**:
	+ Heart rate, respiratory rate, temperature, and blood pressure.
* **Neurological Assessment**:
	+ Muscle tone, reflexes, and response to stimuli.

**Apgar Scoring**

* **Purpose**: Quickly assess the newborn's health at 1 and 5 minutes.
* **Components**:
	+ **A**ppearance (skin color)
	+ **P**ulse (heart rate)
	+ **G**rimace (reflex response)
	+ **A**ctivity (muscle tone)
	+ **R**espiration (breathing)
* **Scoring**: 0-2 for each component (max score of 10).

**Nutrition and Feeding**

* **Breastfeeding**:
	+ Recommended as the primary source of nutrition.
	+ Colostrum (first milk) is rich in antibodies and nutrients.
* **Formula Feeding**:
	+ For cases where breastfeeding is not possible.
	+ Ensuring proper formula preparation and hygiene.
* **Feeding Frequency**:
	+ Typically every 2-3 hours in the first few weeks.

**Preventive Healthcare**

* **Vaccinations**:
	+ Hepatitis B vaccine (first dose within 24 hours).
	+ BCG vaccine (for specific country, Including RM)
* **Vitamin K**:
	+ Administered to prevent bleeding disorders.
* **Eye Prophylaxis**:
	+ Prevention of neonatal conjunctivitis (e.g., erythromycin or silver nitrate).
* **Newborn Screening**:
	+ Screening for genetic disorders, hearing loss, and metabolic diseases.

**Managing Common Newborn Conditions**

* **Jaundice**:
	+ Monitoring bilirubin levels.
	+ Phototherapy if necessary.
* **Hypoglycemia**:
	+ Risk in low-birth-weight or diabetic mothers’ babies.
	+ Early detection and feeding.
* **Infections**:
	+ Signs of neonatal sepsis (fever, lethargy, poor feeding)

**Common Birth Injuries**

* **Brachial Plexus Injury**:
	+ Due to difficult delivery (e.g., shoulder dystocia).
* **Cephalohematoma**:
	+ Accumulation of blood between the skull and periosteum.
* **Fractures**:
	+ Clavicular fractures are common in difficult deliveries.

Parental Support and Education

* **Importance of Bonding**:
	+ Encourage skin-to-skin contact and breastfeeding.
* **Parental Education**:
	+ Educate parents on infant care, including feeding, diapering, and safe sleep practices.
	+ Discuss signs of illness and when to seek medical attention.

**Safe Sleep Practices**

* **Sudden Infant Death Syndrome (SIDS) Prevention**:
	+ Place the baby on their back to sleep.
	+ Use a firm mattress and avoid soft bedding.
	+ Room-sharing without bed-sharing.

**Discharge and Follow-Up Care**

* **Before Discharge**:
	+ Ensure the baby has adequate feeding.
	+ Assess for jaundice and other health concerns.
* **Follow-Up Appointments**:
	+ Schedule a visit with the pediatrician within the first week.

**Special Considerations**

* **Preterm or Low Birth Weight Infants**:
	+ Special care required, including monitoring for temperature regulation, feeding issues, and developmental concerns.
* **Multiple Births**:
	+ Increased monitoring due to potential complications.

**Conclusion**

* **Summary**:
	+ Effective newborn care is essential for the survival and well-being of the infant.
	+ Early intervention can prevent complications and ensure healthy development.
* **Final Message**:
	+ Pediatricians and healthcare providers play a crucial role in the health of newborns and families.