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1 Saslow et al. Screening Guidelines for the Prevention and Early Detection of Cervical Cancer. *Am J Clin Pathol* 2012.
2 **cobas** HPV package insert.

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Original Review

Newborn Transition

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CEU

The transition from intrauterine to extrauterine life is a complex adaptation. Although, in a sense, the entire time in utero is in preparation for this transition, there are many specific anatomic and physiologic changes that take place in the weeks and days leading up to labor that facilitate a healthy transition. Some, including increasing pulmonary vasculature and blood flow, are part of an ongoing process of maturation. Others, such as a reversal in the lung from secreting fluid to absorbing fluid and the secretion of pulmonary surfactant, are associated with the hormonal milieu that occurs when spontaneous labor is impending. Interventions such as elective cesarean birth or induction of labor may interfere with this preparation for birth. Postnatal interventions such as immediate clamping of the umbilical cord and oropharyngeal suction may also compromise the normal process of newborn transition. This article reviews the physiology of the fetal to newborn transition and explores interventions that may facilitate or hinder the optimal process.
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Keywords: infant, cardiopulmonary adaptation, extrauterine life, newborn, placental circulation/physiology, transition

INTRODUCTION

A newborn's first breath initiates a cascade of changes that allow the newborn to make the transition from dependence on the placenta and maternal metabolism in utero to life as a separate individual. These changes are preceded by a series of physiologic events prior to spontaneous labor and can be either hindered or helped by numerous intrapartum or postpartum events. An appreciation for factors that facilitate physiologic transition and ways in which it can go awry is important for anyone involved in the care of newborns. Although the processes for fetus to term newborn and fetus to preterm newborn are the same, factors such as immaturity, inadequate fat, and increased surface area increase the chance of complications in the preterm newborn, such as respiratory distress syndrome from surfactant deficiency, hypothermia, and hypoglycemia. This article focuses on term newborns and describes the fetal to newborn transition. Clinical interventions that may facilitate or interfere with this transition in the term newborn are reviewed.

FETAL CIRCULATION

A review of fetal circulation is integral to an understanding of newborn transition. The fetus is dependent on the mother for oxygenation and removal of carbon dioxide, nutrition, and excretion of waste. The fetal lungs are not necessary for gas exchange, and there is no benefit for the entire blood volume to pass through pulmonary circulation. Fetal shunts allow for maximal flow of the most highly oxygenated blood to perfuse the heart and brain and largely bypass the lungs.[1] In addition, there are several fetal vessels that are vasoreactive and either dilate or constrict in response to changing oxygen levels and direct the oxygenated blood to where it is most essential. These shunts, vasoreactive vessels, and the resulting blood flows are described in Table 1. They are also presented in diagrammatic form in Figure 1. Fetal circulation has the capacity in times of stress to shunt blood away from less critical organ systems such as the kidneys and the gut to the heart, brain, and adrenal glands, that is, those organs critical for survival.[2]

The placenta is the interface between the fetus and the mother, and the vast majority of fetal blood flow is directed toward the placenta via the umbilical arteries. After the exchange of gases, nutrients, and wastes, the blood returns to the fetus via the umbilical vein (UV), with an oxygen saturation of about 80%. Because of the characteristics of fetal hemoglobin that facilitate transfer of oxygen from the mother to the fetus, this correlates with a pO_2 between 30% and 35%.[3]

As diagrammed in Figure 1, approximately 50% of the blood from the umbilical vein traverses the fetal liver.[1] The oxygenated blood mixes with desaturated blood from the portal veins and further with desaturated blood from the inferior vena cava, which is returning venous blood from the lower body. On entering the right atrium, the oxygen saturation has decreased to about 67%, or a pO_2 of 25.[4] Because of flow dynamics and the anatomic location of the foramen ovale, on entering the right atrium, this relatively oxygenated blood from the inferior vena cava crosses the foramen ovale to the left atrium. The left atrium also receives the blood returning from the pulmonary veins. The blood in the left atrium is pumped to the left ventricle and out the ascending aorta to perfuse the coronary arteries and brain with an oxygen saturation of 62% ($pO_2 = 20$). Meanwhile, the blood returning to the heart via the superior vena cava (SVC) also enters the right atrium. Because of the same flow dynamics that directed the return from the inferior vena cava through the foramen ovale, the blood from the SVC flows into the right ventricle and to the pulmonary arteries. The pulmonary arteries (PAs) are vasoconstricted because of physiologic fetal hypoxia causing high resistance, whereas the ductus arteriosus is dilated due to the same physiologic hypoxia and offers little resistance to flow. Most of the right ventricular output flows across the ductus arteriosus (DA) to the descending arch of the aorta, whereas only 10% to 12% perfuses the lungs. As pulmonary

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Quick Points

- In the majority of cases, spontaneous labor occurs at the time of optimal maturation of the fetus.
- Recent research provides evidence that birth without labor leads to decreased absorption of lung fluid.
- Newborn transition is a complex cascade of events that most healthy, term newborns are able to complete without difficulty.
- Routine interventions such as early cord clamping, separating the newborn from the mother, and suctioning of the newborn airway can hinder physiologic transition.
- Delayed cord clamping and skin-to-skin contact provide support to the normal newborn transition process.

vasculature further develops toward term, the amount of blood flow to the lungs increases but continues to be less than the flow through the ductus arteriosus.[2-4]

FETAL CORTISOL

Cortisol plays a multisystem role in preparing the fetus to make a successful transition to extrauterine life. Fetal cortisol levels increase by 50% between 35 weeks' gestation and term as the fetal adrenal gland develops the capability to synthesize and release cortisol. Cortisol levels quadruple during term labor and the first hours after birth. The late gestation increase in cortisol affects fetal and neonatal thyroid metabolism, catecholamine release, glucose metabolism, the digestive capability of the gut, maturation of surfactant production, the switch from secreting to absorbing lung fluid, and increased β-adrenergic receptors.[5]

Prior to term the lung epithelium secretes fluid, which contributes to amniotic fluid. Lung fluid secretion is accomplished by chloride secretion and the relative inactivity

Table 1. Fetal Shunts and Vasculature Involved in Fetal to Neonatal Transition

Shunt/Vessels	Status in Utero	Response to First Breaths	Adult Vestige
UMBILICAL VESSELS			
Umbilical vein (UV)	Carries blood from placenta to fetus. Nonmuscular walls, not vasoactive in response to oxygenation.	Collapses as blood flow from the placenta ceases.	Ligamentum teres
Umbilical arteries (UAs)	Carry blood from descending aorta to placenta. Muscular, vasoactive in response to oxygenation. Dilated in hypoxemic environment.	Vasoconstrict with increasing oxygenation.	Medial umbilical ligaments
FETAL SHUNTS			
Ductus venosus (DV)	Shunts 50% of flow from UV directly to inferior vena cava. Nonmuscular walls, not vasoactive in response to oxygenation.	Collapses as blood flow from the placenta ceases.	Ligamentum venosum
Foramen ovale (FO)	"Flap door" between right and left atria. Allows interatrial flow when pressure is higher in the right atrium than the left atrium.	Functionally closes when systemic pressure exceeds pulmonary pressure. Anatomically closes by approximately 30 months of age.	Interatrial septum
Ductus arteriosus (DA)	Shunts blood from PA to aorta. Muscular, vasoactive in response to oxygenation. Dilated in hypoxemic environment and under influence of prostaglandin E_2.	Functionally closes within several hours of birth as oxygenation increases and prostaglandin E_2 levels plummet with loss of the placenta. Negligible flow by 4 days. Permanent closure takes place at 1- 3 months.	Ligamentum arteriosum
OTHER VASOREACTIVE ARTERIES			
Pulmonary arteries (PAs)	Muscular, vasoactive in response to oxygenation. Constricted in hypoxemic environment.	Dilate quickly after birth in response to rising oxygen levels.	

Sources: Moore et al[2]; Blackburn[12]; Fineman & Clyman.[1]

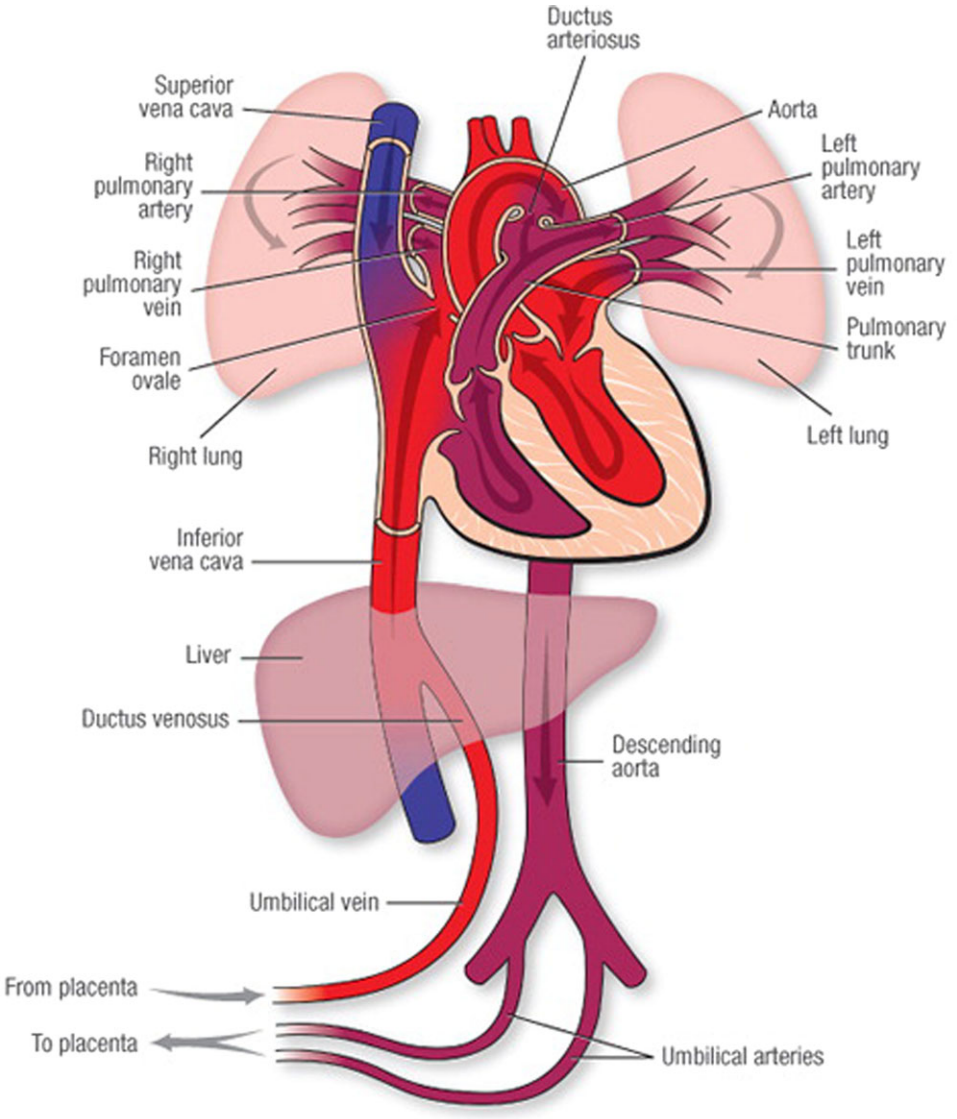

Figure 1. Fetal Circulation

Source: http://www.heart.org/HEARTORG/Conditions/CongenitalHeartDefects/SymptomsDiagnosisofCongenitalHeartDefects/Fetal-Circulation_UCM_315674_Article.jsp

of sodium (Na^+) reabsorption. Prior to birth, under the influence of cortisol, endogenous catecholamines, and thyroid hormones, the secretion of chloride and resultant fluid shuts down, and Na^+ absorption begins to occur, removing fluid from the airways.[5-7] Increasing levels of cortisol, catecholamines, and surfactant at term facilitate and increase sodium-specific channels (epithelial sodium channels) that are responsible for transporting sodium from the lumen of the alveoli into the alveolar epithelial cells. The sodium then is excreted into the interstitial space by active transport.[6,7] Figure 2 diagrams in detail the movement of Na^+, and consequently fluid, from the alveoli to the interstitium. This shift from secretion of fluid to absorption of fluid begins prior to the onset of spontaneous labor and increases during labor. The decrease in lung fluid enhances the adaptation to air breathing. Sheep studies have shown that lung fluid was 45% less in mature fetuses in labor than in fetuses that did not experience labor.[8]

IMMEDIATE PHYSIOLOGIC TASKS OF THE NEWBORN

The newborn must accomplish major changes within the first minutes after birth. These include: 1) initiate and maintain regular respirations, 2) convert from fluid-filled to air-filled lungs, and (3) redirect blood flow toward the fetal lungs and close the fetal shunts. These events are closely intertwined but are dependent on the critical first breaths.

Initiation of Respiration

Numerous factors lead to the initiation of respirations, including mild asphyxia; temperature changes; and tactile, light noise, and proprioceptive stimuli greater than ever before experienced. There is also thoracic recoil with the birth of the torso. Temperature change and the normal physiologic asphyxia experienced at birth are significant stimuli. In the

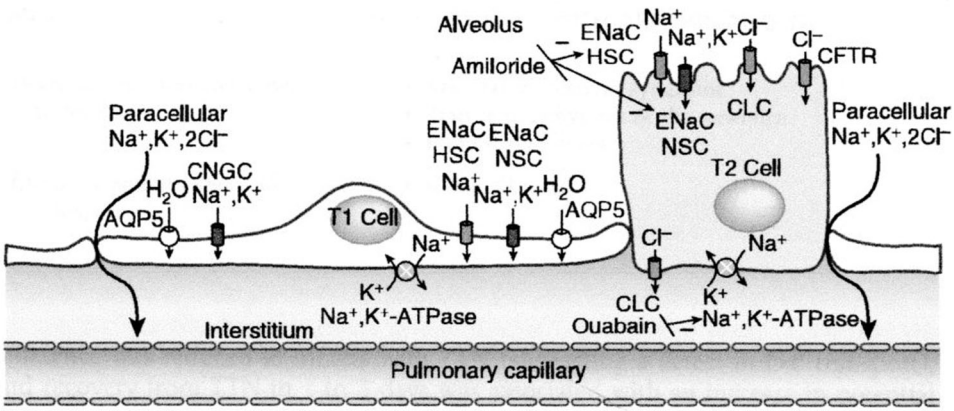

Figure 2. Epithelial Sodium Absorption in the Fetal Lung Near Birth

Na^+ enters the cell through the apical surface of both alveolar type i (T-I) and alveolar type ii (T-II) cells via amiloride-sensitive epithelial sodium channels (ENaCs), both highly selective channels (HSCs) and nonselective channels (NSCs), and via cyclic nucleotide gated channels (CNGCs, only in T-I cells). Electroneutrality is conserved with Cl^- movement through the cystic fibrosis transmembrane conductance regulator (CFTR) or through chloride channels (CLCs) or paracellularly through tight junctions. The increase in cell Na stimulates Na, K^+-ATPase activity on the basolateral aspect of the cell membrane, which drives out 3 Na^+ ions in exchange for 2 K^+ ions (a process that can be blocked by the cardiac gltcoside ouabain). When the net ion movement is from the apical surface to the interstitium and osmotic gradient is created, directing water in the same direction via either aquaporins or diffusion.

Source: Reprinted from Keene et al[7] with permission from Elsevier.

healthy fetus the pH ranges from 7.40 to 7.43 in the umbilical vein down to 7.36 to 7.39 in the descending aorta. The corresponding pCO_2 levels are 38 to 42 in the umbilical vein and 43 to 48 in the descending aorta.[1] Immediately after birth the umbilical venous pH has decreased to 7.32 to 7.35, and the umbilical arterial pH has decreased even further, to 7.24 to 7.28. The UV pCO_2 is 38 to 44, but the umbilical arterial pCO_2 has increased to 49 to 53.[9] Elevated CO_2 levels are a powerful respiratory stimulant, much more so than lowered oxygen levels. (Try holding your breath as long as you can. It is the increased CO_2 that leads to the overwhelming need to begin breathing!) Physiologic asphyxia stimulates respiration, but acute excess asphyxia will depress respirations. The different responses to slight versus profound asphyxia can be demonstrated by the occurrence of primary and secondary apnea. The fetus/newborn responds to asphyxial stress by rapid breathing. If this breathing is not successful in reversing the asphyxia, a period of primary apnea ensues. During primary apnea, the fetus/newborn responds to stimuli by initiating respirations. Gasping follows the period of primary apnea. If the gasping is ineffective, such as if it occurs prior to birth, the fetus then enters secondary apnea, which is not only associated with apnea and bradycardia, but also a profound decrease in blood pressure. Newborns in secondary apnea will not respond to stimulation and require resuscitation to initiate respiration.[10]

Convert From Fluid-Filled to Gas-Filled Lungs

Both clearing the lungs of fluid and maintaining the inflation of the alveoli are essential to successful fetal-newborn transition. As discussed above, the fluid has already decreased prior to spontaneous labor. Sodium absorption and therefore fluid absorption accelerate even more after birth in response to the increased catecholamines associated with birth and the new presence of oxygen and surfactant,[7] which leads to an additional 38% decrease in lung fluid over the first 6 hours after a normal vaginal birth.[8] The "vaginal squeeze" also contributes to emptying the lung of fluid, whereas lymphatic absorption plays a lesser role.[8]

Role of Surfactant

During the middle of the second trimester of pregnancy, the developing terminal sacs (destined to become the alveoli) are lined with cuboidal epithelium. By 20 weeks' gestation, the pulmonary epithelium of the terminal sacs begins to differentiate into type II cells (pneumonocytes) that retain their cuboidal shape and type I cells that transform into squamous epithelium, flattening and covering large areas of the terminal sac. Between 20 to 24 weeks' gestation the type II cells begin to develop lamellar bodies, which are responsible for surfactant production. During the last month of pregnancy the type I cells, although fewer in number than type II cells, continue to thin, covering approximately 93% of the alveolar surface[11] (Figure 2). Surfactants are lipoproteins produced by the type II cells that allow the alveoli to remain inflated during exhalation. Surfactant production is increased during the last month of gestation, especially as labor approaches. The alveolar stretch associated with the initiation of breathing and the catecholamine release at birth further stimulate secretion of surfactant. Surfactant arranges into a film spread over the alveolar surface, having a polar side oriented toward the alveolar membrane and a nonpolar side oriented toward the air within the alveolus. With expiration, these molecules compress, preventing the development of an air/liquid interface, thereby decreasing surface tension and preventing collapse of the alveolus.[12]

In addition to reducing surface tension and maintaining patency of the alveoli, surfactant has other important roles

Table 2. Physiologic Preductal SpO_2 After Birth	
1 min	60%-65%
2 min	65%-70%
3 min	70%-75%
4 min	75%-80%
5 min	80%-85%
10 min	85%-95%

Source: Kattwinkel et al.[13]

in pulmonary function. The protein components of surfactant are critical components of the nonspecific immune functions within the lung, which include maintaining a barrier to pathogens, recognizing pathogens, modulating chemotaxins, and facilitating phagocytosis.[12] Premature newborns have less surfactant than term newborns, usually in direct proportion to the degree of prematurity. The surfactant deficiency places them at increased risk for the atelectasis of respiratory distress syndrome and pneumonia.

Vascular Changes and Closure of Shunts

Following the first extrauterine breaths, the newborn's oxygen level is higher than it was in utero. The physiologic hypoxia that maintained constriction of the pulmonary arteries and dilation of the umbilical arteries becomes reversed. Quickly, the pulmonary arteries dilate, decreasing resistance and increasing blood flow to the lungs, further increasing oxygenation. Oxygenation continues to increase gradually during the first 10 minutes after birth[13] (Table 2). The increased pulmonary blood flow leads to lower pressure in the right atrium and ventricle and increased flow to the left atrium via the pulmonary veins. The increasing oxygen levels and the stretching of the umbilical cord that occurs during the birth[14] also cause the vasoactive umbilical arteries to constrict, cutting off flow to the low-resistance circuit of the placenta. The ductus arteriosus constricts more gradually in response to oxygen than do the umbilical arteries. As pulmonary resistance decreases and systemic resistance increases, the pressure in the 2 circuits equalizes, and blood flow through the ductus arteriosus functionally ceases.

Dilation of the pulmonary arteries opens a vascular space that had been a potential space in the fetus, which now must be well perfused. Flow from the newborn to the placenta ceases because of vasoconstriction of the umbilical arteries, but the umbilical vein remains patent, allowing continued blood flow from the placenta to the newborn. A transfusion of blood from the placenta occurs with the continued physiologic contractions of the uterus. In the original research by Yao et al, the newborn was held approximately 10 cm lower than the introitus, and newborn total blood volume increased from 70 mL/kg at birth to approximately 90 to 95 mL/kg with a 3-minute delay in clamping the cord.[15] With the current practice of placing a newborn skin to skin on the mother's abdomen or chest, full placental transfusion requires approximately 5 minutes.[16,17] The increased vascular volume resulting both from the placental transfusion, as presented in Figure 3, and the absorption of any remaining lung fluid allows for perfusion of the pulmonary vasculature without the need for the newborn to shunt blood away from the less critical organs and extremities.

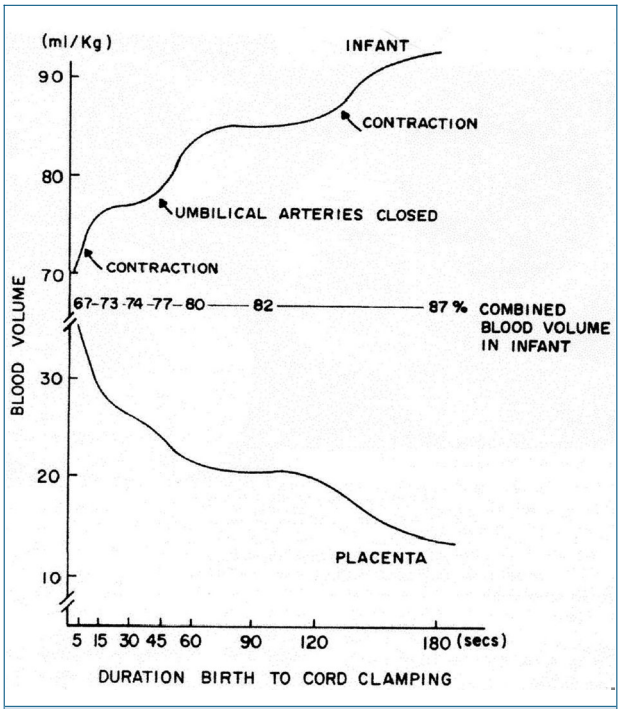

Figure 3. Placental Transfusion

Source: Smith and Nelson.[48]

FURTHER ADAPTATIONS TO EXTRAUTERINE LIFE

Thermoregulation

In utero, fetal temperature is usually about 0.5°C warmer than the mother because of heat generated by the uterus, placenta, and fetal metabolism.[18] Heat generated by fetal metabolism is transferred to the mother primarily via placental circulation, with a smaller amount dissipated via the amniotic fluid and uterine wall. Brown adipose tissue (BAT) is apparent by 29 weeks' gestation and continues to increase throughout the third trimester. By term, a newborn has about 30 g of BAT deposits, located between the scapulae at the nape of the neck, in the axillae, in the mediastinum, and surrounding the kidneys and adrenal glands. In comparison to white adipose tissue, BAT has high concentrations of mitochondria, a richer blood supply, numerous lipid inclusions, greater glycogen stores, and more extensive sympathetic nerve supply, all of which facilitate rapid metabolism, heat production, and transfer of heat outward.[12] Metabolism of BAT is suppressed prior to birth.[18]

At birth the newborn leaves a fluid-filled thermal bubble and enters into a cooler, drier environment. Immediately the newborn begins to lose heat via conduction, convection, evaporation, and radiation, described in Table 3. Convective, radiant, and evaporative heat loss are also associated with concurrent fluid loss. Although this fluid loss may not compromise a term newborn, it can lead to significant fluid balance concerns in the preterm newborn. Cold receptors in the skin and spinal column and hypothalamic thermal receptors are stimulated, further activating the sympathetic

Table 3. Mechanisms of Newborn Heat Loss

Mechanism	Process	How to Minimize Heat Loss
Conduction	Transfer of heat to or from newborn to a cooler solid surface through direct contact	Place newborn skin to skin with mother
		If separation is necessary, place on a prewarmed surface
Convection	Transfer of heat from newborn to the surrounding air	Maintain ambient temperature of 72°F
		Minimize air currents
Evaporation	Heat loss due to energy exchange during evaporation of liquid	Dry newborn thoroughly following birth
		Maintain adequate relative humidity
Radiation	Transfer of energy to or from newborn to a surface not in direct contact, such as walls, windows, heat lamps	Maintain distance between newborn and cold exterior walls

Adapted from: Smith and Nelson[48]; Blackburn.[12]

nervous system that had been stimulated by labor.[18] This increased sympathetic nervous system activity releases norepinephrine within the BAT, producing heat via nonshivering thermogenesis. Brown adipose tissue deposits are reduced in preterm and growth-restricted newborns, placing them at higher risk for cold stress and hypothermia. Decreases in newborn skin temperature can stimulate a 100% increase in heat production.[19]

Thermoregulation is a balance of heat production and heat loss. In addition to heat production by nonshivering thermogenesis, the newborn has the ability to produce heat by increasing activity and, to a limited degree, by shivering. Heat loss has 2 components: an internal heat gradient from the core to the periphery of the newborn and an external heat gradient from the newborn to the environment. The internal heat gradient is affected by the degree of subcutaneous fat and peripheral vasodilatation or constriction, whereas the external gradient is more influenced by external forces, with newborn control limited to changing the surface area through flexion. Newborns who are not protected from heat loss produce more heat with activity and nonshivering thermogenesis, consuming more oxygen and energy. Because of increased metabolic activity, neonatal cold stress can lead to hypoglycemia and occasionally hypoxia. Conversely, oxygen consumption also increases with higher body temperature. The *neutral thermal environment* is the environmental temperature at which basal metabolic demands are lowest. Minimizing metabolic demands is critical for the preterm or low-birth-weight newborn; it also facilitates normal transition in the term, normally grown newborn.

Nutrition

The fetus depends on the placenta for the transfer of nutrients from the maternal circulation, but metabolism of these nutrients is regulated by the fetus. Glucose is the major energy source and is transported from maternal to fetal circulation in the placenta via facilitated diffusion. Between 30% and 40% of this glucose is utilized by the placental tissue itself.[20] The rest enters fetal circulation and stimulates insulin secretion by the fetal pancreas. Insulin not only induces lipogenesis, but also releases insulin-like growth factor produced by the fetal liver. Birth weight is correlated to levels of insulin-like growth factors.[21] In addition to meeting metabolic needs, large amounts of glucose are converted to glycogen and stored in the liver. Glycogen is also stored within the cardiac and skeletal muscle. At birth, the stream of nutrients from the mother ceases, and the newborn must independently maintain normal glucose levels. Newborn blood glucose initially decreases, reaching a nadir about one hour after birth. Insulin production decreases with the lower blood glucose, and glycogenolysis (release of glucose from glycogen) is stimulated.[22] Hepatic glycogen is mostly depleted by 10 hours after birth, and newborn glucose production switches from glycogenolysis to gluconeogenesis, the production of glucose from lactate and amino acids.[23] Lipolysis provides another energy source. Initiation of feeding usually stabilizes the term neonate's glucose levels.[12]

Initiation of Breastfeeding

Human milk provides the ideal nutrition for newborns and infants, and the American Academy of Pediatrics recommends exclusive breastfeeding for the first 6 months of life, beginning within the first hour after birth.[24] Colostrum contains antibodies, is not irritating, is easily swallowed, and enhances meconium passage.[12] Initiation of feeding stimulates production of the hormones that are responsible for postnatal gut development.

Excretion of Waste

Fluid and electrolyte balance and excretion of wastes maintained by the placenta in utero must also be assumed by the newborn after birth. Renal blood flow and glomerular filtration rate (GFR) increase at birth but remain lower in newborns than in older infants, primarily because of residual elevations of renal vascular resistance continuing from the fetal state.[25] Newborns are able to dilute their urine similarly to adults but do not concentrate urine as effectively. Because of the lower GFR, a newborn is unable to manage large water loads.[12] Many newborns void soon after birth, and 95% do so within 24 hours.[26] Within 12 to 24 hours the newborn diureses, mobilizing extracellular fluid and decreasing that compartment from 40% at birth to 30%. This fluid loss

accounts for much of the weight loss that occurs over the first 5 days.[27]

Although some fetuses pass meconium in utero, for the most part intestinal motility is quiescent prior to birth. Gut motility is related to increasing maturity and gastrointestinal hormones, especially motilin, which increases intestinal motor activity.[28] More than half of term newborns will pass meconium within the first 12 hours, 94% within 24 hours, and 99.8% will have passed meconium by 48 hours after birth.

FACTORS AND INTERVENTIONS THAT FACILITATE NORMAL NEWBORN TRANSITION

Awaiting Spontaneous Labor

The onset of labor is related to a complex interaction of maternal, fetal, placental, cervical, and uterine processes. Awaiting spontaneous labor allows these interactions to proceed optimally, allowing for optimal maturation of the fetal/newborn lung. Fetal cortisol levels more than double between 35 weeks' gestation and term, increasing further during labor.[5] For the vast majority of fetuses, spontaneous labor occurs at the prime time of lung function, having reversed the flow of lung fluid from secretion to absorption and producing adequate surfactant to prevent atelectasis and respiratory distress syndrome.

Delayed Cord Clamping

The procedure of tying and clamping the umbilical cord soon after birth was introduced in the 1600s, and steadily grew in popularity. Despite widespread adoption of the practice, there remained voices criticizing early cord clamping as having a deleterious effect on the newborn, and there have never been well-controlled studies that demonstrate a benefit to early or immediate cord clamping.[29] Rather, there has been an increasing body of evidence that supports benefits of delayed cord clamping. The primary effect of delayed cord clamping is expansion of the blood volume by 20 to 30 mL/kg.[15,17] This placental transfusion offers benefits beyond the immediate volume expansion that facilitates cardiopulmonary transition; it also provides additional red cell volume that contains high iron levels (thereby decreasing iron deficiency anemia) and large numbers of hematopoietic stems cells.[17] In situations when a delay in clamping the cord is not feasible, milking the cord from the introitus toward the umbilicus 5 times will provide 70% of the physiologic gain that would have occurred if cord clamping were delayed for 5 minutes.[16] Administration of uterotonics hastens but does not increase the final volume of the placental transfusion.[17]

Skin-to-Skin Contact

Placing the newborn in skin-to-skin contact on the maternal chest confers benefits to both the newborn and the mother. The most immediate benefit is optimal thermoregulation. A review of the research on SSC has validated SSC effectiveness in aiding newborn thermoregulation.[30] Three studies included in this review, with 168 participants total, were analyzed for infant temperature at 90 minutes of life.[31-33] Infant temperatures among SSC newborns were 0.4°C to 0.5°C higher in the Christiansson 1992 and 1995 studies but 0.1°C lower in the Villalon 1993 study. A larger study of 204 by Carfoot et al in 2005 found temperatures among SSC infants to be 0.15°C higher ($P < .02$) than among infants with cot care at 60 minutes of life.[34] Marin also found slightly higher temperatures (0.07°C) among SSC infants at 1, 5, and 120 minutes of life in a study of 274 term newborns.[35]

Newborns who are placed in skin-to-skin contact following birth cry less,[31] have more stable blood glucose levels,[30] and demonstrate an organized progression of behaviors that culminate in breastfeeding.[36] Placement of the newborn prone and flexed between the breasts safely allows the newborn to locate the areola and self-initiate breastfeeding.[36] Early skin-to-skin contact has been associated with higher rates of breastfeeding 1 and 4 months following birth.[30]

INTERVENTIONS THAT MAY INTERFERE WITH NORMAL TRANSITION

It is clear that elective cesarean birth without labor, prematurity, and early cord clamping interfere with optimal fetal to neonatal transition. Other interventions that were implemented without adequate study are also being shown not to have benefit and probably cause harm. Such interventions include routine suctioning of the airway and liberal oxygen administration.

Elective Cesarean Birth

Newborns who are born at 37 weeks' gestation or later by elective repeat cesarean birth experience almost twice the incidence of respiratory distress syndrome (2.1% vs 1.4%, $P = .0003$) and transient tachypnea of the newborn (4.1% vs 1.9%, $P = .0001$) when compared with those born vaginally after a prior cesarean birth.[37] Elective cesarean birth prior to the onset of labor is associated with more retained lung fluid, surfactant deficiency, and pulmonary hypertension.[38] Because of these increased risks, some authors have recommended administering antenatal corticosteroids to women prior to elective cesarean birth, even at term.[6,37,39]

Oropharyngeal Suctioning

Oropharyngeal suctioning has been considered a routine intervention in many delivery units for decades. Although it is often performed with a soft rubber bulb syringe, a suction catheter may also be used. A classic study in 1971 by Cordero and Hon[40] found that as many as 15% of term neonates will develop severe arrhythmias, including profound bradycardia, in response to nasopharyngeal suction. Despite this study, suctioning remained prevalent. In 1992 Estol et al provided evidence that oro-nasal-pharyngeal suction provided no benefit for respiratory mechanics.[41] Subsequent research has failed to find any benefit to routine suctioning whether following vaginal birth or cesarean birth.[42-44] Two studies found that newborns who were suctioned took longer to reach optimal oxygen saturation than those who were not suctioned.[42,43]

Gastric Suctioning

Gastric suctioning is often used to decrease gastric contents at birth, decreasing the risk of aspiration. Widström et al evaluated the effect of gastric suction on healthy term neonates. No benefits were found, initiation of breastfeeding behavior was disrupted, and newborns responded to suctioning by swatting at the catheter and retching. A larger study by Kiremitci et al of 310 term newborns randomly assigned to gastric suction or no gastric suction, once again finding no benefit to suctioning and possibly adverse effects related to spikes in systolic blood pressure.[45]

The most recent guidelines for neonatal resuscitation recommend that suctioning, including via bulb syringe, be limited to newborns with obvious obstruction or those who require positive pressure ventilation.[13,46]

Oxygen Administration

Prior to 2005, supplemental oxygen was recommended for newborns with central cyanosis and 100% oxygen for newborns requiring positive pressure ventilation. By 2005 the guidelines still recommended supplemental oxygen for cyanosis but acknowledged potential concerns about possible adverse effects of 100% oxygen on respiratory physiology, cerebral circulation, and potential tissue damage.[47] Incorporating the body of research on the effects of oxygen, the most recent guidelines, published in 2010, recommend administering supplemental oxygen only if the oxygenation parameters in Table 2 are not met and that positive pressure ventilation may be initiated with either room air or a blended air/oxygen mixture.[13]

CONCLUSION

The transition from fetal to neonatal life is an intricate and complex process that most term newborns are able to negotiate without difficulty. Unfortunately, many obstetric and neonatal practices can interfere with the physiologic process, placing the newborn at risk for suboptimal outcomes. Those responsible for the care of pregnant women and newborns can facilitate the successful transition by avoiding interventions that have not been found to confer benefit to the newborn.

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CONFLICT OF INTEREST

The authors have no conflicts of interest to disclose.

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