

DEVIATIONS IN GROWTH PATTERNS

Deviations in intrauterine growth patterns not only increase the risk of morbidity and mortality in the early newborn period, but may also have long-term implications for altered growth and development and for altered CNS function and learning disabilities in childhood.

This general plan of care is designed to facilitate optimal nursing management of the infant with deviations in intrauterine growth and is to be used in conjunction with the CPs: The Neonate at Two Hours to Two Days of Age, and The Preterm Infant, as appropriate. Growth deviations are classified as SGA, intrauterine growth retardation/restriction (IUGR), and LGA.

SGA/IUGR: Any newborn whose birth weight falls at or below the 10th percentile on classification charts, considering local factors (e.g., ethnicity, altitude).

LGA/Macrosomic: Any newborn whose birth weight is at or above the 90th percentile on classification charts, considering local population at any week in gestation (with special attention to determining appropriate gestational age), or who at birth weighs more than 4000 g (8 lb 13 oz).

NEONATAL ASSESSMENT DATA BASE

SGA Infant

Activity/Rest

Activity level may be excessive, with vigorous cry/hungry suck attributable to chronic intrauterine hypoxia.

MATERNAL FACTORS

Excessive/strenuous exercise program

Circulation

MATERNAL FACTORS

Resides at high altitude

Heart/lung disease; bleeding, severe anemia or sickle cell anemia; chronic hypertension or PIH

Elimination

Abdomen may appear scaphoid or concave.

MATERNAL FACTORS

Pyelonephritis, chronic renal disease

Food/Fluid

All body parts may be below expected size for gestational age but in proportion/symmetrical to each other (suggests a chronic or prolonged problem throughout gestation).

Disproportionate weight as compared to length and head circumference (appears long and thin with normal head circumference) suggests episodic vascular insufficiency in third trimester.

Sunken abdomen; absence of subcutaneous tissue.

Decreased muscle mass, especially in the cheeks, buttocks, and thighs.

May demonstrate metabolic instability associated with hypoglycemia/hypocalcemia.

MATERNAL FACTORS

Small stature

Malnutrition/poor nutritional intake (chronic or during third trimester); history of eating disorders

Advanced diabetes mellitus (class D or above); PKU

Neurosensory

Skull suture and fontanels appear widened; bulging of fontanels caused by inadequate bone growth may be evident.

Small head with protruding forehead, sunken nasal bridge, short upturned nose, thin upper lip, receding chin (indicative of fetal alcohol syndrome [FAS]).

Muscle tone may appear tight with flexion of upper and lower extremities, minor joint/limb abnormalities, and restricted movement (suggests FAS).

Wide-eyed appearance (associated with chronic hypoxia in utero).
Chromosomal syndromes.

Respiration

Signs of respiratory distress may be present (especially in presence of meconium aspiration syndrome [MAS], polycythemia, or infection).

Mucus may be green-tinged.

MATERNAL FACTORS

Heavy smoker

Safety

Dry, cracked, and peeling skin present, with loose skin fold; sparse scalp hair.

Meconium staining may be evident with greenish stains on fingernails and at base of umbilical cord.

Umbilical cord may have single artery and/or be thin, slightly yellow, dull, dry.

Congenital anomalies/malformations or infection may be present.

MATERNAL FACTORS

Irradiation and use of medications with teratogenic side effects (e.g., antimetabolites, anticonvulsants, trimethadione)

Collagen disease; maternal infections such as rubella, syphilis, cytomegalovirus, toxoplasmosis; uterine tumors

Sexuality

Females tend to be smaller than males at birth.

MATERNAL FACTORS

Adolescent or advanced maternal age (younger than age 16 or older than age 40)

Primiparity, grand multiparity

Placenta previa/separation, insufficiency, infarction, fibrosis, thrombosis, hemangioma, abnormal cord insertion and single umbilical artery with vascular anastomoses (twin-to-twin)

Chromosomal abnormalities, chronic intrauterine infections, congenital anomalies, multifetal pregnancy, inborn errors of metabolism

Social Interaction

MATERNAL FACTORS

Low socioeconomic class

Other child(ren) at home with history of FTT

May have previous or current involvement with Department of Social Services

Teaching/Learning

May be premature (and/or member of multifetus pregnancy)

MATERNAL FACTORS

Poor/incomplete formal education

Alcoholism, drug abuse

Lack of prenatal care

LGA Infant

Activity/Rest

Difficulty maintaining quiet, alert state; slower to arouse

Circulation

Skin color ruddy (associated with polycythemia), jaundice (indicative of hyperbilirubinemia)

May have congenital anomalies such as transposition of the great vessels, Beckwith-Wiedemann syndrome, or erythroblastosis fetalis

Food/Fluid

Macrosomia; excess fat deposits and reddened complexion; increased body size proportional (except in infant of diabetic mother [IDM], whose weight may appear disproportionately large for length)

May demonstrate metabolic instability associated with hypoglycemia/hypocalcemia, may have feeding problems

Weight may be 4000 g (8 lb 13 oz) or more (dependent on gestational age)

MATERNAL FACTORS

Inappropriate/overnutrition, excessive pregravid weight and/or weight gain >35 lb

Large stature

Diabetes mellitus (class A, B, or C)

Neurosensory

Large amount of scalp hair

May display hypotonia/hypertonia; decreased reflex functioning

Respiration

Signs of respiratory distress may be present if stress of delivery has induced meconium aspiration/asphyxiation, if delivered by cesarean section, or if infection present.

Safety

Birth injury(s) may be present, e.g., bruising, caput succedaneum, cephalhematomas; facial/phrenic nerve paralysis, brachial palsy; fractured clavicles, intracranial bleeding/depressed skull fracture(s); bulging fontanel indicative of neurological problems, depressed fontanel suggestive of dehydration.

Intrapartal/delivery events may reveal fetal distress, meconium-stained amniotic fluid, oligohydramnios, late/variable decelerations, scalp pH levels 7.20, resuscitative measures.

Evidence of congenital malformations may involve the heart, CNS, kidney, lungs, GI tract.

Long, hard nails extending beyond ends of toes and fingers.

Absence of vernix caseosa/lanugo; desquamation or epidermis.

Sexuality

Higher incidence in males

MATERNAL FACTORS

Birth of previous LGA infant

Cesarean birth because of cephalopelvic disproportion or oxytocin-induced labor related to diabetes/fetal distress/prolonged pregnancy

Multiparity

Social Interactions

Slow to orient to maternal face/voice (generally improves within 48 hr)

Teaching/Learning

May be preterm/postterm by clinical assessment

May be postterm (42 wk or more) because of postconceptional bleeding, leading to a miscalculation of dates/prolonged pregnancy associated with menstrual cycle longer than 28 days.

DIAGNOSTIC STUDIES

Dextrostix Glucose Estimations: Less than 40 mg/dl in LGA infant or 25 mg/dl in SGA infant during first 3 days indicates hypoglycemia.

Serum Glucose: Verifies Dextrostix value <40 mg/dl in LGA infant, 25 mg/dl in SGA infant.

Chest X-Ray and ABGs: Help determine cause/severity of respiratory distress if present, e.g., pneumonia, MAS, RDS.

CBC: May reveal central venous Hct elevated above 65%; central venous Hb 20 g/dl associated with polycythemia/hyperviscosity.

WBC Count: May be elevated or depressed.

Platelet Count: May be depressed if mother was pre-eclamptic or if infant was born with a congenital viral infection.

Coagulation Studies (prothrombin time [PT], partial thromboplastin time [PTT], fibrinogen, fibrin split products [FSP]): May indicate DIC, especially in the presence of polycythemia or asphyxia.

Blood Type for ABO Group, Rh Factor, and Crossmatch: Plasma exchange may be necessary if Rh incompatibility exists.

Serum Electrolytes (including ionized calcium): Assesses for hypocalcemia (level 7 mg/dl [3.5 mEq/L] or less in first 3 days of life); inappropriate antidiuretic hormone secretion, and instability related to metabolic complications.

Bilirubin: May be elevated secondary to polycythemia and resorption of bleeding associated with birth injury, e.g., intracranial hemorrhage, cephalhematoma.

Urinalysis on/after Second Voided Specimen, Including Specific Gravity and Sugar/Acetone: Assesses homeostasis, renal involvement.

Bacterial and Viral Cultures: Rule out/diagnose infectious process.

Electrocardiography (ECG), Echocardiography, Ultrasonography, Angiography, and Genetic Studies: As appropriate with suspected FAS, congenital defects, and/or complications.

NURSING PRIORITIES

1. Maintain physiological homeostasis.
2. Prevent and/or treat complications.
3. Identify/minimize effects of birth trauma.
4. Provide family with appropriate information/strategies for meeting short- and long-term needs associated with growth deviation.

NURSING DIAGNOSIS:

May Be Related To:

Possibly Evidenced By:

**DESIRED OUTCOMES/EVALUATION
CRITERIA—NEONATE WILL:**

GAS EXCHANGE, impaired

Alveolar capillary membrane changes (decreased surfactant levels, retained pulmonary fluid, meconium aspiration), altered oxygen supply (diaphragmatic paralysis/phrenic nerve paralysis, increased intracranial pressure)

Restlessness/irritability; inability to move secretions, tachypnea, cyanosis, hypoxia

Display spontaneous, unassisted regular respiratory effort with rate of 30–50/min; and ABGs WNL.

Be free of apnea and complications of hypoxia/lung disease.

ACTIONS/INTERVENTIONS

RATIONALE

Independent

Review history for abnormal prenatal growth patterns and/or reduced amounts of amniotic fluid, as detected by ultrasonography/fundal changes.

Low-birth-weight infant or infant with IUGR suffers chronic intrauterine asphyxia, resulting in hypoxia/malnutrition. Fetal contribution to the amniotic pool is reduced in the stressed infant. Macrosomia can be related to maternal diabetes, prolonged pregnancy, heredity, and inappropriate nutrition. Macrosomia in IDM results from excess release of growth hormone (thyroid stimulation), increasing the number of cells and/or organ size throughout the body.

Note type of delivery and intraparturient events indicative of hypoxia.

Infant with chronic hypoxia will be more susceptible to acidosis/respiratory depression/persistent fetal circulation (PFC) after delivery. Cesarean birth increases risk of excess mucus because thoracic compression by the birth canal does not occur as in a vaginal delivery.

Note time/onset of breathing and Apgar scores. Observe ensuing respiratory patterns.

The infant with intraparturient asphyxia may present with a delayed onset of respirations and altered respiratory pattern. Apgar scores aid in evaluation of the degree of depression or asphyxia of the newborn at birth and are directly correlated with serum pH/degree of infant acidosis.

Assess respiratory rate, depth, effort. Observe and report signs and symptoms of respiratory distress, distinguishing from symptoms associated with polycythemia.

Infant with altered growth is more susceptible to respiratory distress associated with chronic asphyxia in SGA infant, inadequate surfactant levels in IDM, perinatal asphyxia, aspiration of meconium or amniotic fluid, and PFC. Diminished lung compliance may occur as a result of polycythemia.

Auscultate breath sounds regularly.

Presence of crackles/rhonchi reflect respiratory congestion and need for intervention.

Suction nasopharynx/endotracheal tube as needed, after first providing supplemental oxygen.

Ensures patency of airway, removes excess mucus. Supplemental oxygen reduces hypoxic effect of procedure.

Auscultate apical pulse; note presence of cyanosis.

Tachypnea, bradycardia, and cyanosis may occur in response to altered oxygen levels.

Prevent iatrogenic complications associated with cold stress, metabolic imbalance, and caloric insufficiency.

Such complications increase metabolic demands and oxygen needs.

Ensure availability of resources in the event complications occur.

Equipment for oxygenation, suction, intubation, assisted ventilation, resuscitation, and chest tube placement must be readily available in the event of severe/prolonged respiratory distress.

Collaborative

Monitor transcutaneous oxygen/pulse oximeter readings.

Identifies therapy needs/effectiveness.

Monitor laboratory studies, as indicated:

Serum pH;	Detects possible metabolic acidosis occurring from inadequate oxygen intake/respiratory acidosis and anaerobic metabolism with acid end products. Note: Normal pH values range from 7.35–7.44; HCO ₃ is 19–22 mEq/L (bicarbonate reflects buffering capacity of the blood).
ABGs;	Indicate degree of hypoxia/hypercapnia, as well as therapy needs/effectiveness. Note: Normal newborn arterial PO ₂ ranges from 50–70 mm Hg, arterial PCO ₂ from 35–45 mm Hg.
Hct.	Polycythemia, which occurs in 50% of SGA infants related to excess RBC production in response to chronic intrauterine hypoxia, typically increases capillary/venous Hct levels to greater than 60%, with resultant respiratory distress associated with diminished lung compliance.
Administer warm, humidified oxygen; provide assisted ventilation, as indicated.	Corrects/prevents hypoxia, hypercapnia, and respiratory acid-base imbalances.
Review chest x-rays.	May confirm meconium aspiration pneumonia, common in SGA or postterm infant, or RDS, in IDM.
Provide chest physiotherapy, as indicated.	Percussion and postural drainage promote mobilization of secretions, enhancing airway patency and gas exchange, especially in the presence of MAS. Note: Contraindicated in preterm infant.
Administer medications as indicated:	
Sodium bicarbonate;	Corrects metabolic imbalances/acidosis resulting from prolonged respiratory acidosis.
Xanthine derivatives, e.g., aminophylline, theophylline;	Sympathomimetic bronchodilators may be useful in treating apnea of prematurity.
Tolazoline HCl (Priscoline);	Potent vasodilator that relaxes smooth muscle to maximize circulatory effort/oxygenation in cases of meconium aspiration/PFC.
Dopamine.	May be required to counteract hypotensive effect of Priscoline administration.

NURSING DIAGNOSIS:

May Be Related To:

Possibly Evidenced By:

DESIRED OUTCOMES/EVALUATION CRITERIA—NEONATE WILL:

PARENT(S) WILL:

NUTRITION: altered, less than body requirements

Decreased nutritional stores, increased insulin production and/or hyperplasia of the pancreatic beta cells

Weight deviation from expected; decreased muscle mass/fat stores, electrolyte imbalance

Ingest and digest adequate nutrients for weight gain (or to prevent weight loss of 2% or more).

Display serum glucose >40 mg/dl, and other associated laboratory studies WNL.

Identify/treat/prevent short- and long-term complications of malnutrition.

ACTIONS/INTERVENTIONS

RATIONALE

Independent

Compare weight in relation to gestational age and size. Document on growth chart. Weigh daily.

Identifies presence, degree, and risk of altered growth pattern. LGA infant with excess extracellular fluid experiences a postdelivery diuresis, resulting in a loss of up to 15% of birth weight. SGA infant may have already lost weight in utero or may suffer from reduced fat/glycogen stores.

Maintain thermoneutral environment, including use of incubator/radiant warmer, as indicated. Monitor heat controls, temperature of infant and environment frequently, noting hypothermia/hyperthermia.

The SGA infant does not have adequate adipose tissue for insulation and has a large body surface area compared to body weight. Brown adipose tissue stores may be inadequate to maintain thermoregulation. Both hypothermia and hyperthermia increase metabolic demands, necessitating increased intake in an already compromised infant. Note: Thermal instability may be iatrogenically induced by improperly operated heating equipment used to maintain thermogenesis in dysmature infant. (Refer to CP: The Preterm Infant; ND: Thermoregulation, ineffective.)

Initiate early and frequent feedings and advance as tolerated.

Assists in maintaining fluid/electrolyte balance and meeting caloric needs to support metabolic process. Helps prevent hypoglycemia associated with inadequate body stores in SGA infant or continued pancreatic secretion of insulin in IDM.

Assess tolerance to feedings. Note stool color, consistency, and frequency; presence of reducing substances; abdominal girth; vomiting, and gastric residuals.

Advances in amount and caloric composition of feedings are dependent on tolerance. Evidence of reducing substances (increased glucose) in stool suggests inability to digest formula because of obstruction and/or disease process present. Note: Poor sucking reflex and recurrent vomiting or persistent regurgitation may be seen in FAS, requiring further evaluation/intervention. (Refer to CP: The Infant of an Addicted Mother.)

Monitor intake and output. Calculate daily caloric and electrolyte consumption.

Provides information about actual intake in relation to estimated needs for use in readjustment of dietary prescription.

Assess hydration level, noting fontanelles, skin turgor, urine specific gravity, condition of mucous membranes, and weight fluctuations.

Increased metabolic demands of the SGA infant may increase fluid requirements. Hyperglycemic states may produce diuresis in the infant. IV administration of fluids may be required to meet increased demands but must be conscientiously managed to avoid fluid excess.

Monitor Dextrostix levels immediately after birth and routinely until serum glucose is stabilized.

Hypoglycemia may occur after birth because of limited glucose stores in the SGA infant and from hyperplasia/continued release of insulin in the LGA infant. Symptoms in the SGA infant usually appear between 24 and 72 hr of age, but may begin as early as 3 hr or as late as 7 days; the IDM may be symptomatic within 1–3 hr of birth. Hyperglycemia may result from inappropriate infusion of solutions containing glucose.

Observe for signs of hypoglycemia, e.g., tachypnea and irregular respirations, apnea, lethargy, flaccidity, cyanosis, temperature fluctuations, diaphoresis, poor feeding, jitteriness, high-pitched cry, tremors, eye rolling, seizure activity.

Note signs of hypocalcemia, e.g., neuromuscular irritability (tremors, twitching, seizing, clonus), hypotonia, vomiting, high-pitched cry, cyanosis, apnea, and cardiac dysrhythmias with prolonged Q-T interval.

Discuss long-term complications of malnutrition in SGA infant and obesity in LGA infant; review importance of protein during phase II of brain growth.

Collaborative

Monitor laboratory studies as indicated:

Serum glucose;

Calcium;

Sodium, potassium, chloride, phosphorus, and magnesium;

BUN, creatinine, serum/urine osmolality, urine electrolytes;

Triglyceride/cholesterol levels, and liver function tests.

Establish intravascular access as indicated.

Because glucose is a major source of fuel for the brain, deficits may cause permanent CNS damage. Hypoglycemia significantly increases morbidity and mortality rates, and severity of long-term effects is dependent on duration of such episodes. Untreated symptomatic infants have a higher incidence of neurological abnormalities and/or lower mean IQ later in childhood than treated infants.

Peak incidence of early hypocalcemia is during first 48–72 hr of life and is often related to temporary abdominal distension, neonatal hypoparathyroidism in premature infant, or prenatal asphyxia in SGA infant or IDM. Late hypocalcemia (at 6–10 days) may be caused by ingestion of milk formulas containing a higher ratio of phosphorus to calcium.

Chronic protein deficiencies in the SGA infant make child prone to learning difficulties and cerebral dysfunction, characterized by short attention span, poor fine-motor coordination, hyperactivity behavior problems, and speech defects. Excess fat cells in the LGA/macrosomic infant may create lifelong problems associated with obesity (e.g., diabetes, cardiovascular disease, stroke). Adequate protein during hyperplasia/hypertrophy phase of brain growth during the first 6 mo of life helps to overcome insults that occurred in early gestational period of brain development.

Hypoglycemia may occur as early as 1–3 hr after birth. In the SGA infant, glycogen reserves are quickly depleted and gluconeogenesis is inadequate because of reduced stores of muscle protein and fat, while the IDM has decreased ability to release glucagon and catecholamines needed to stimulate glucagon breakdown/facilitate release of glucose. Frequency of screening is dependent on this risk group, i.e., IDM, intraparturial asphyxia, or preterm infants. Levels 7 mg/dl require further evaluation/intervention.

Electrolyte instability may be a consequence of deviations in growth, inadequate placental transfer, or altered maternal mineral balance. Note: Hypomagnesemia/hyperphosphatemia are usually associated with hypocalcemia.

Detects altered kidney function associated with reduced nutrient stores and fluid levels resulting from acute malnutrition/asphyxia.

Useful in determining nutritional deficits and therapy needs/effectiveness.

IV access allows for fluid and electrolyte administration. Intra-arterial access allows for ease in obtaining samples for monitoring laboratory values.

Administer glucose-containing solutions/parenteral nutrition.

Provide electrolyte supplementation as indicated, e.g., 10% calcium gluconate.

The severely compromised infant with deviations in intrauterine growth may be unable to consume adequate fluids and nutrients via an enteral route. IV glucose (i.e., infusion of D₅W) may be required when serum glucose cannot be maintained at or above 40 mg/dl, or seizure activity occurs.

Metabolic instability in the SGA/LGA infant may necessitate supplements to maintain homeostasis, especially calcium, sodium, and occasionally magnesium.

NURSING DIAGNOSIS:

Risk Factors May Include:

Possibly Evidenced By:

DESIRED OUTCOMES/EVALUATION CRITERIA—NEONATE WILL:

TISSUE PERFUSION, risk for altered

Interruption of arterial or venous blood flow (hyperviscosity associated with polycythemia)

[Not applicable; presence of signs/symptoms establishes an *actual* diagnosis]

Maintain normal vital signs, with adequate peripheral pulses and Hct WNL.

Be free of complications associated with polycythemia.

ACTIONS/INTERVENTIONS

RATIONALE

Independent

Note risk factors for/presence of polycythemia.

Maternal fetal or infant transfusion (during gestation or at birth), twin gestation (twin-to-twin transfusion), chronic fetal distress associated with maternal PIH, placenta previa; chromosomal anomalies, endocrine disorders; and residing at altitudes over 5000 ft place the infant at risk for developing increased blood volume. Polycythemia resulting from increased erythropoietin production in response to chronic intrauterine hypoxia may occur in both the SGA/LGA infant. In many cases, infants of diabetic mothers are polycythemic. Although the pathophysiology of the hyperviscosity is not fully understood, it may be related to decreased extracellular fluid volume or increased bone marrow stimulation associated with hypoxia.

Monitor temperature, intake/output, and urine specific gravity. Note skin turgor, condition of mucous membranes, and fontanel.

Prevention or correction of dehydration decreases the risks of hyperviscosity.

Observe skin color for ruddiness or pallor. Note presence of hyperthermia, respiratory distress, hypertension or hypotension, tachycardia, decreased pulses, oliguria, hematuria, or altered neurological findings.

Collaborative

Monitor central/peripheral Hct/Hb and bilirubin. Send blood for type, crossmatch, and Rh. (Refer to CP: Newborn: Hyperbilirubinemia.)

Establish intravascular access, preferably through umbilical catheterization.

Prepare for/assist with exchange transfusion as indicated.

Monitor for complications of procedure, including transfusion reactions, overload (CHF), catheter complications, cardiac dysrhythmias, hypovolemia, anemia, and hepatitis.

Helps detect/promotes prompt intervention to prevent possible complications of polycythemia, e.g., myocardial, cerebral, and renal ischemia; cardiopulmonary congestion; hyperbilirubinemia; thromboembolism, and convulsions.

Indicates degree of polycythemia/hyperviscosity. Hyperbilirubinemia often results from polycythemia (central Hct is >65%; Hb is 22 g/dl) as excess RBCs break down.

Provides for fluid administration to correct hyperviscosity and exchange transfusion, if necessary.

Fresh frozen plasma, 5% albumin or 9% saline, replaces infant’s blood in equal amounts, thereby diluting infant’s remaining blood volume to prevent/correct polycythemia and hyperviscosity. Usually 10% of the infant’s blood volume is removed/exchanged at one time.

Provides for early detection/intervention.

NURSING DIAGNOSIS:

Risk Factors May Include:

Possibly Evidenced By:

DESIRED OUTCOMES/EVALUATION CRITERIA—NEONATE WILL:

INJURY, risk for

Altered growth patterns, delayed CNS/neurological development, abnormal blood profile, immature immune response

[Not applicable; presence of signs/symptoms establishes an *actual* diagnosis]

Be free of complications.

ACTIONS/INTERVENTIONS

RATIONALE

Independent

Inspect all LGA infants for birth injuries. Note bulging/tense fontanel; muscle spasticity, twitching, or flaccidity; high-pitched, weak, constant cry; tremors or seizure activity; changes in pupil size/reaction; or asymmetrical chest movement.

Because of disproportion between fetal size and maternal pelvis, LGA infants have a high incidence of birth injuries/trauma, such as IICP/CNS damage, cervical/brachial plexus palsy, fractured clavicle/humerus, diaphragmatic paralysis, and cephalhematoma.

Observe invasive sites, urine/stool, NG drainage, and pulmonary secretions for signs of bleeding. Note petechiae/bruising, changes in responsiveness/activity level or muscle tone, nystagmus, opisthotonic posturing, or convulsions.

Measure occipital frontal circumference, as indicated.

Monitor vital signs. Observe peripheral capillary refill and color and temperature of skin.

Assess home situation for abusive relationships.

Monitor for signs and symptoms of infection (e.g., changes in temperature, color, muscle tone and activity, feeding tolerance, cardiopulmonary status; or presence of petechiae, rash, jaundice).

Discuss with parents the potential of conditions that may have long-term sequelae, such as birth injury or fetal malnutrition hypoxia (significant role in late or subnormal CNS functioning), which may result in convulsions or altered reflex responses.

Collaborative

Monitor laboratory studies as indicated:

CBC with differential;

Coagulation studies: PTT/activated PTT (APTT), PT, fibrinogen levels, FSP, platelets; Blood type, crossmatch, and Rh factor; Bacterial viral cultures and sensitivities.

Provide supplemental oxygen, CPAP, and/or mechanical ventilation, as needed. (Refer to ND: Gas Exchange, impaired.)

Administer blood products, albumin, and coagulants, as necessary.

Administer anticonvulsants (e.g., phenobarbital), as indicated.

Assist with diagnostic studies, as indicated, e.g., lumbar puncture, CT, or ultrasound scan.

Refer family to appropriate community resources/support groups.

Early recognition promotes timely intervention. Septicemia, congenital syphilis, cytomegalic inclusion disease, and rubella may result in DIC, resulting in pulmonary, cerebral, or IVH.

Hydrocephalus may develop following IVH.

Hypotension, bradycardia, apnea, hypothermia, delayed capillary refill, and pallor reflect developing shock related to blood loss, requiring prompt assessment/intervention.

Maternal abuse during pregnancy is a significant risk factor for development of SGA infant. Releasing infant into potentially unsafe environment increases risk of abuse/neglect and FTT.

Poor resistance to infection, cracks in epidermis, and possible exposure to infectious agents in utero place the SGA/postterm infant at risk for infection, which may be life-threatening, especially if not detected in its earliest phases. (Refer to CP: The Preterm Infant; ND: Infection, risk for.)

Enhances early identification and promotes optimal management of long-term effects of injury/condition.

Dropping Hct may reflect hemorrhage; a shift of the differential may suggest infection.

Alterations in clotting times, presence of FSP reflect developing coagulopathies.

Blood replacement may be required.

Verifies presence, etiology, severity of infectious process.

Aids in correcting hypoxia, acidemia, and hypotension; reduces risk of IVH/increased ICP.

Replaces losses/enhances circulating volume; may help control bleeding.

May be necessary to quiet cerebrum and reduce electrical stimuli that precipitate seizure activity.

Bloody spinal fluid indicates IVH. Scans are useful in identifying site/extent of cerebral hemorrhage.

Provides support/guidance for dealing with possible long-term effects of condition.

NURSING DIAGNOSIS:**INFANT BEHAVIOR, risk for disorganized****May Be Related To:**

Functional limitations related to growth deviations (restricting neonate's opportunity to seek out, recognize, and interpret stimuli), electrolyte imbalance, and psychological stress, immaturity or CNS damage (low threshold for stress), low energy reserves, poor organizational ability, limited ability to control environment

Possibly Evidenced By:

[Not applicable; presence of signs/symptoms establishes an *actual* diagnosis]

DESIRED OUTCOMES/EVALUATION CRITERIA—NEONATE WILL:

Exhibit organized behaviors that allow the achievement of optimal potential for growth and development as evidenced by modulation of physiological, motor, state, and attentional-interactive functioning.

PARENT(S)/CAREGIVER(S) WILL:

Recognize individual infant cues.

Identify appropriate responses (including environmental modifications) to infant cues.

Verbalize readiness to assume caregiving independently.

ACTIONS/INTERVENTIONS**RATIONALE**

Independent

Determine infant's capacity for stimulation, noting behavioral responses involving gross and fine motor movement, presence of irritability, restlessness, crying, eye contact, and facial expressions.

Allows provision of stimuli consistent with infant's capabilities and with cues reflecting sensory overload.

Provide stimuli consistent with chronological and developmental capabilities.

Size of the infant with deviations in growth may be a false indicator of capacity for stimulation.

Monitor environmental stimulation. Minimize/remove inappropriate or hazardous stimuli.

Control of stressors can reduce infant's energy demands, aiding in maintenance of homeostasis (promotes rest, weight gain).

Provide parents with information regarding the newborn's capabilities/needs.

Parents who are informed of their newborn's capabilities can provide appropriate stimulation to optimize the newborn's development.

Encourage parental involvement in identification of coping strategies/deficits of infant, including control of behavioral state and temperament.

Fosters continued support of the infant's capacities in the acute care setting and transition to home. Identifies role for parents in providing coregulation to the infant.

Promote ongoing assessment of neurodevelopmental gains/deficits. Plan future interventions accordingly.

Prevents/minimizes complications and optimizes growth and development.

Collaborative

Suggest/refer to early stimulation program if available/indicated.

Optimizes infant development/interactional skills (affective, cognitive, social).

Refer for long-term neurodevelopmental assessment and intervention as indicated.

Sequelae of morbidity associated with deviations in intrauterine growth may affect long-term coping.

(Refer to CP: The Preterm Infant; ND: Infant Behavior, risk for disorganized.)

NURSING DIAGNOSIS:

KNOWLEDGE deficit [Learning Need], parent(s), regarding condition, prognosis, and treatment needs

May Be Related To:

Lack of exposure, misinterpretations, unfamiliarity with resources

Possibly Evidenced By:

Verbalization of problem, misconception, request for information

DESIRED OUTCOMES/EVALUATION

Identify newborn's short- and long-term needs.

CRITERIA—PARENT(S) WILL:

List available resources.

Participate in discharge planning.

ACTIONS/INTERVENTIONS

RATIONALE

Independent

Provide appropriate anticipatory guidance regarding implications of growth deviations.

Malnutrition in SGA infant may result in subnormal CNS/intellectual development as well as hearing/speech deficits. LGA infant is susceptible to malnutrition problems associated with obesity. (Refer to ND: Nutrition: altered, less than body requirements.)

Review short- and long-term needs for care of the SGA/LGA infant. Assist with follow-up in discharge planning.

Provides information for parents to understand need for ongoing physical, neurodevelopmental, and psychosocial assessment needed for long-term follow-up. (Refer to CP: The Parents of a Child with Special Needs; NDs: Knowledge deficit [Learning Need]; Family Coping: ineffective, risk for compromised.)

Identify community/governmental resources as appropriate.

Presence of specific needs such as feeding problems, resolving complications, or congenital anomalies require ongoing monitoring and problem solving to foster optimal growth and development.