Management of at Risk Neonate-1

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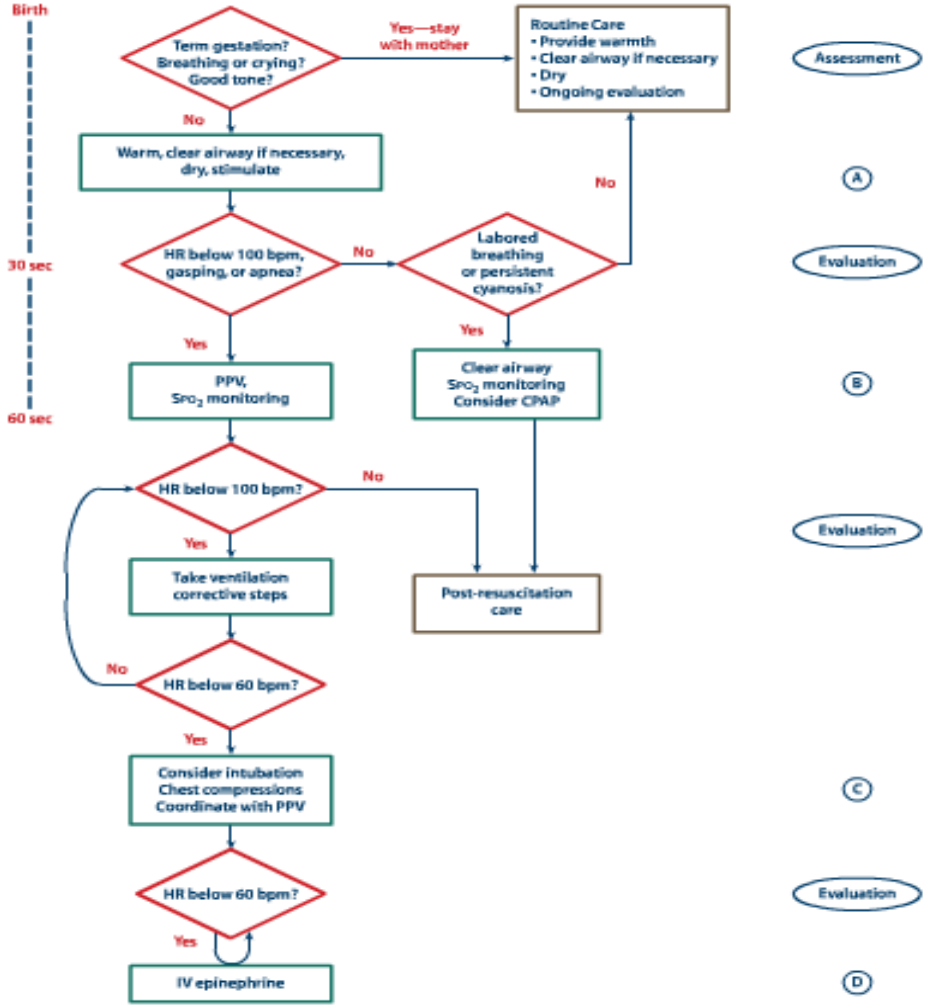
Mehr-1399

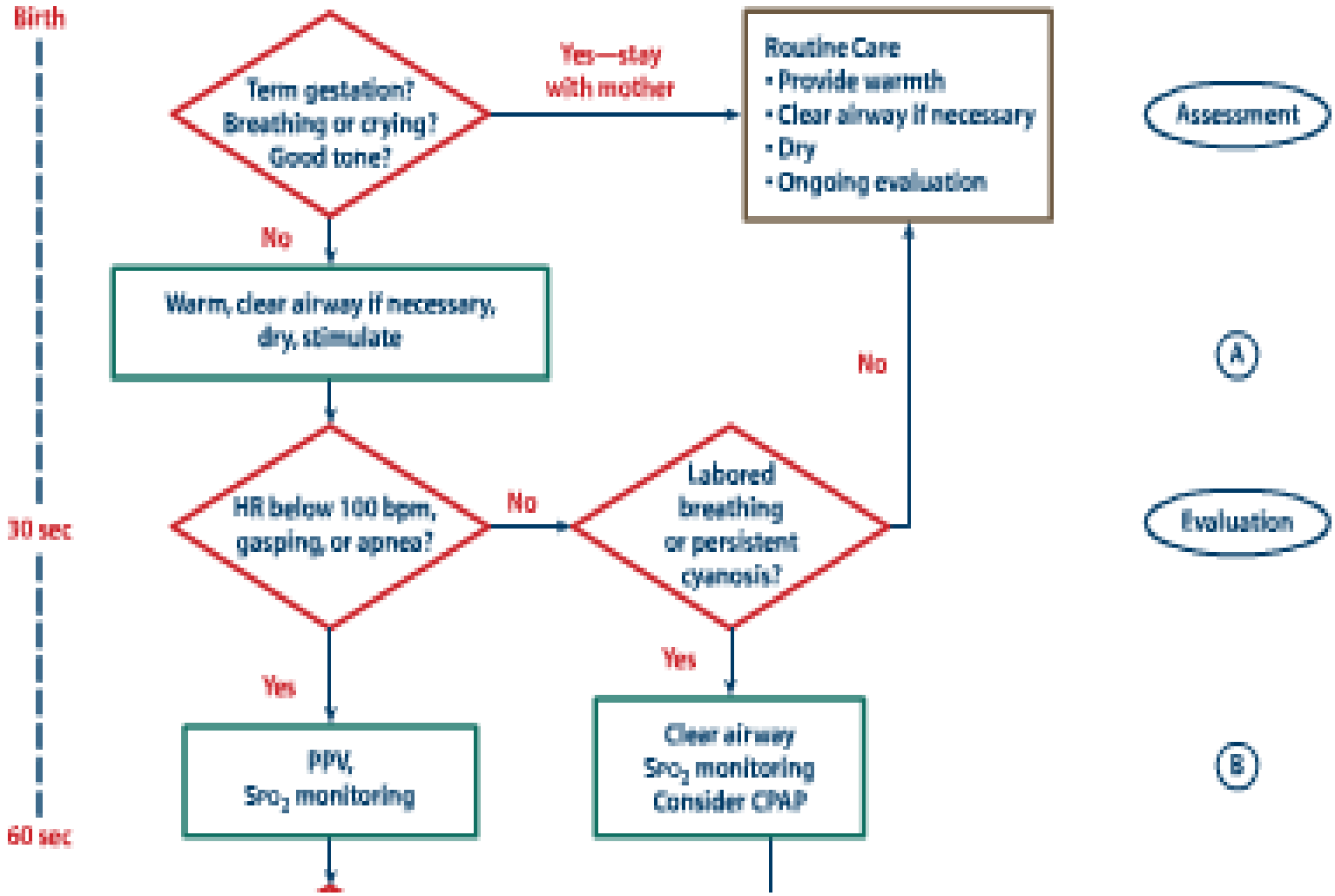
At Risk Neonate

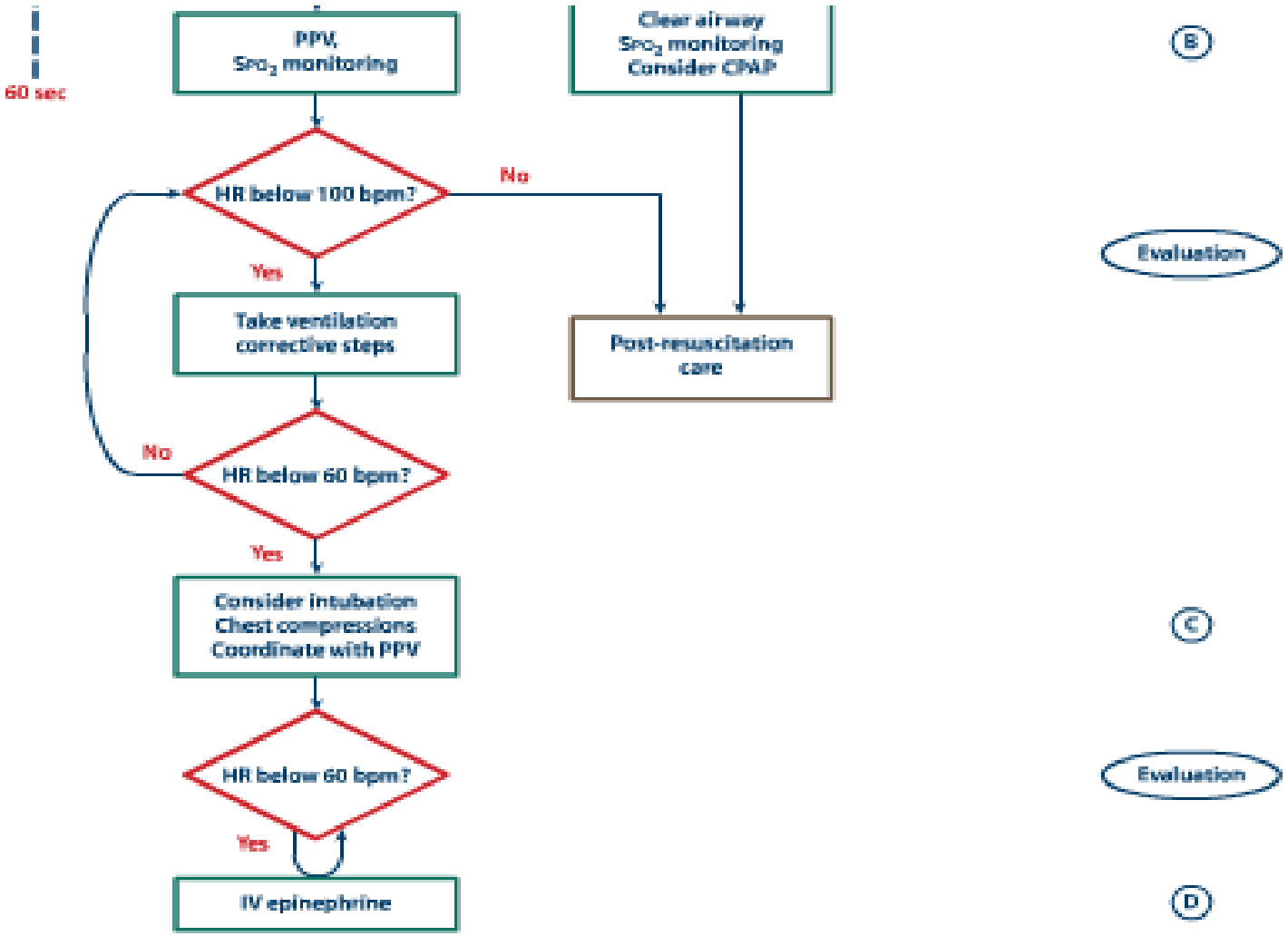
- CPR (NRP).
- Shock.
- Anemia.
- Polycytemia.
- Apnea.
- Hypo-hyperglycemia.
- Hypo-hypercalcemia.
- Metabolic acidosis.
- Temperature abnormality.
- Asphyxia.
- Fluid and electrolyte.
- NEC.
- RDS.
- Bleeding disorder.
- Cyanotic heart disease.
- Pulmonary air leak.
- Convulsion.
- Birth trauma

NRP

The Resuscitation Flow Diagram




Birth
30 sec
60 sec
Term gestation?
Breathing or crying?
Good tone?
Yes—stay with mother
Routine Care
• Provide warmth
• Clear airway if necessary
• Dry
• Ongoing evaluation
Assessment
No
Warm, clear airway if necessary, dry, stimulate
A
No
HR below 100 bpm, gasping, or apnea?
No
Labored breathing or persistent cyanosis?
Evaluation
Yes
Yes
PPV,
SpO2 monitoring
Clear airway
SpO2 monitoring
Consider CPAP
B


60 sec
PPV,
SpO2 monitoring
Clear airway
SpO2 monitoring
Consider CPAP
B
HR below 100 bpm?
No
Yes
Evaluation
Take ventilation
corrective steps
Post-resuscitation
care
No
HR below 60 bpm?
Yes
Consider intubation
Chest compressions
Coordinate with PPV
C
HR below 60 bpm?
Evaluation
Yes
IV epinephrine
D

Shock

Cloherty

- >90% of preterm infants <26 weeks'gestation have a mean arterial BP >30 mm Hg.

- More than 90% of term infants have a mean BP of >45 mm Hg immediately after birth with a rise to >50 mm Hg by the third postnatal day.

- Abnormal peripheral vasoregulation.
- Hypovolemia.
- Myocardial dysfunction.

1- volume:

- An infusion of **10 to 20 ml/kg isotonic saline** solution is used to treat suspected **hypovolemia**.

- **Blood cell transfusions or fresh frozen plasma** are recommended in cases of **blood loss or DIC.**

2- Correction of negative inotropic factors will improve cardiac output.

- **hypoxia**,
- **acidosis,**
- **hypoglycemia,**
- **other metabolic derangements.**
- **Hypocalcemia** frequently occurs in infants with circulatory failure, especially if they have received large amounts of volume resuscitation.

  calcium frequently produces a positive inotropic response. **Calcium gluconate 10% (100 mg/kg)** can be infused slowly if ionized calcium levels are low.

3- Medications:

A- Positive inotropic agents

I. Sympathomimetic amines:

a. **Dopamine** is a naturally occurring catecholamine.

At ***low doses*** **(0.5 to 2 mcg/kg/minute),** dopamine stimulates peripheral dopamine receptors (DA1 and DA2) and increases renal, mesenteric, and little coronary blood flow with effect on cardiac output.

In ***intermediate doses*** **(5 – 9 mcg/kg/minute),** dopamine has positive inotropic and chronotropic effects (beta1 and 2). . The increase in myocardial contractility depends in part on myocardial norepinephrine stores.

At ***High-dose*** **(10 – 20 mcg/kg/minute),** , dopamine stimulates alpha-1 and 2 adrenergic receptors and serotonin receptors, resulting in vasoconstriction and increased peripheral vascular resistance.

High-dose dopamine also increases venous return. In preterm infants, dopamine may stimulate the alpha receptors at lower doses Dopamine has been used at high infusion rates (>25 mcg/kg/minute) to normalize BP in preterm newborns without detrimental vasoconstrictive effects, probably due to the decreased cardiovascular sensitivity to sympathomimetic agents that is prevalent in these infants.

b. **Dobutamine** is a synthetic catecholamine with relatively cardioselective inotropic effects.

- In doses of 5 to 15 mcg/kg per minute,
- dobutamine increases cardiac output (alpha-1 receptors) with **little effect on heart rate**.
- Dobutamine can **decrease systemic vascular resistance** (SVR) (beta-receptors).
- Dobutamine is often used with dopamine to improve cardiac output in cases of decreased myocardial function as its inotropic effects, unlike those of dopamine, are independent of norepinephrine stores. However, because hypotension is a result of decreased SVR in the majority of nonasphyxiated newborns, dopamine remains the first line of pressor therapy.

C – **Epinephrine: (**Potent inotropic and chronotropic effects).

- It is ***not a firstline drug in newborns***; however, it may be effective in patients **who do not respond to dopamine.**
- Epinephrine may be helpful in conditions such as **sepsis** when low perfusion is due to peripheral vasodilatation.
- The starting dose is 0.05 to 0.1 mcg/kg/minute and can be increased (to 0.3mcg/kg/minute) rapidly as needed while dopamine infusion rates are decreased. (greater beta-2 and lower alpha receptors in these doses & leading to fall in SVR).
- Epinephrine is an effective adjunct therapy to dopamine because cardiac norepinephnne stores are readily depleted with prolonged and higher rate dopamine infusions.

II. **Milrinone** is a phosphodiesterase-III inhibitor that enhances intracellular cyclic adenosine monophosphate (cAMP) content preferentially in the myocardium

- leading to increased cardiac contractility.
- It improves diastolic myocardial function more readily than dobutamine.
- Also lowers pulmonary vascular resistance (PVR) and SVR by increasing cAMP levels in vascular smooth muscle **often necessitating the use of volume and dopamine to raise SVR**.

B- Vasopressor therapy:

1- **Dopamine:** (10 – 20 mcg/kg/min)

2- **norepinephrine or high dose epinephrine**

(NE preferred agent in shock associated with low SVR.)

3- Vasopressin:(0.0002 - 0.006 mcg/kg/min)

- therapeutic efficacy in the treatment of **vasodilatory shock** in children.
- Vasopressin is a hormone that is primarily involved in the postnatal regulation of fluid homeostasis but also plays an important role in maintaining vascular tone in the setting of hemodynamic instability.
- **Vasopressin deficiency may occur in catecholamine resistant hypotension** in the evolution of sepsis, and hence its reported effcacy in vasodilatory shock.
- Vasopressin is not routinely used to treat shock in the neonate but may be a therapeutic option to consider in the setting of abnormal peripheral vasoregulation as occurs in sepsis.
- An added beneficial effect may be its inhibitory action on NO-induced increases in the second messenger cyclic guanosine monophosphate (cGMP), a potent vasodilatory signal that predominates in the setting of sepsis from the increased endotoxin/inflammation-induced NO synthesis.

C- Corticosteroids

For refractory hypotension, **hydrocortisone** can be used at a dose of **1 mg/kg**. If efficacy is noted, the dose can be **repeated every 8 hours for 2 to 3 days,** especially if low serum cortisol levels are documented before hydrocortisone treatment.

- may be useful in **extremely premature infants with hypotension refractory** to volume expansion and vasopressors, but their usage has not been adequately tested in clinical trials.
- Hydrocortisone stabilizes BP through **multiple mechanisms**. It induces the expression of the cardiovascular adrenergic receptors that are downregulated by prolonged use of sympathomimetic agents and also inhibits catecholamine metabolism. Moreover, **some extremely preterm infants have adrenal insufficiency, especially in the setting of prolonged illness**. After hydrocortisone administration, there is a rapid increase in intracellular calcium availability, resulting in enhanced responsiveness to adrenergic agents. The **BP response is evident as early as 2 hours after hydrocortisone** treatment.

- High-dose steroids have been used in **sepsis,** but their efficacy remains controversial, perhaps because administration is initiated late in the clinical course after the cascade of inflammatory mediators has begun.

TYPICAL CLINICAL SCENARIOS OF SHOCK IN THE NEONATE AND THEIR MANAGEMENT

A. Very low birth weight (VLBW) neonate in the immediate postnatal period

- poor vasomotor tone, immature myocardium that is more sensitive to changes in afterload, and dysregulated NO production.

Recommended therapy is **dopamine and judicious use of volume if hypovolemia is suspected**. It is important not to give large volume infusions due to their association with increased risk of bronchopulmonary dysplasia reported in the premature infant.

B. Perinatal depression in preterm or full term neonate

Physiology involves release of endogenous catecholamines leading to normal or increased SVR clinically manifested by pallor, mottled appearance, and poor perfusion and myocardial dysfunction. The baby is likely to be **euvolemic** and may have associated **pulmonary hypertension**.

Recommended therapy is **dopamine with or without dobutamine** up to 10 mcg/kg/ minute.

Milrinone can be used to provide afterload reduction and inotropy without risk of further myocardial injury due to excess catecholamine exposure.

In cases with associated **pulmonary hypertension**, the use of **inhaled NO** is warranted for infants > 34 weeks' gestation.

Some infants may manifest **vasodilatory shock** and would benefit from **increased doses of dopamine rather than milrinone**. The patient's skin color and perfusion on physical examination can be used to guide therapy

C. Preterm neonate with PDA

Physiology includes ductal "steal" compromising vital organ perfusion and increase in left-to-right shunt with increased risk for pulmonary hemorrhage.

Recommended therapy includes **avoiding high dose dopamine (>10 mcg/kg/ minute)** as its use will further increase left to right shunting and reduce vital organ perfusion.

Use **dobutamine** **or** **milrinone** to enhance cardiac inotropy.

Target ventilation management to increase PVR by increasing positive end expiratory pressure (↑ **PEEP**), maintaining permissive hypercarbia, and avoiding hyperoxygenation.

D. Septic shock

Physiology involves relative hypovolemia, myocardial dysfunction, and peripheral vasodilation.

Therapy includes **volume resuscitation with crystalloid** (10-30 ml/kg) which should be repeated as needed an administration of **dopamine** 5 - 20 mcg/kg/minute with or without **epinephrine** 0.05 to 0.3 pg/kg/minute.

A cardiac **echocardiogram** can be obtained to evaluate cardiac function, volume status, and intracardiac shunting.

Consider extracorporeal membrane oxygenation (**ECMO**) in infants >34 weeks' gestation if they do not respond to these mterventrons.

E. Preterm neonates with "pressor-resistant" hypotension

A proportion of VLBW infants become dependent on medium to high doses of pressors (usually dopamine) beyond the first postnatal days. Etiologies include relative cortisol deficiency, adrenal insufficiency, and downregulation of adrenergic receptors.

Consider **low-dose hydrocortisone** (3 mg/kg/day for 3-5 days) after drawing serum cortisol level. Hydrocortisone may be preferable to equivalent steroid doses of dexamethasone due to the added mineralocorticoid effect of the former. Studies support the efficacy of hydrocortisone in raising BP within 2 hours of administration, yet the long-term **neurologic effects** of this treatment in the VLBW infant remain to be investigated. Additionally, due to one published report of possible increased incidence of **intestinal perforation** in infants treated with hydrocortisone that also received **indomethacin**, the concurrent use of these drugs cannot be recommended until larger trials are conducted.

Anemia & Polycytemia

Nelson Text

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Anemia in the Newborn:

Hemoglobin increases with advancing gestational age: at term, cord blood hemoglobin is **16.8 g/dL** (14-20 g/dL); hemoglobin levels in very low birthweight (VLBW) infants are 1-2 g/dL below those at term .

physiologic anemia

- 8-12 wk in term infants (hemoglobin, 11 g/dL)
- 6 wk in premature infants (7-10 g/dL).

Infants born by **cesarean section may have a lower hematocrit** (Hct) than do those born vaginally.

severe anemia with heart failure

Tx:

emergency exchange transfusion

to restore Hct and oxygen-carrying capacity

Acute blood loss:

usually results in severe distress at birth,

initially with a normal hemoglobin level,

no hepatosplenomegaly, and early onset of shock.

Chronic blood loss in utero:

- marked pallor,
- less distress,
- a low hemoglobin level with microcytic indices,
- and if severe, heart failure

guidelines for blood transfusion to preterm infants:

Hct ≤35/Hgb ≤11

Infants requiring **moderate or significant mechanical ventilation** (MAP >8 cm H_2O and Fio_2 >0.4)

Transfusion:

15 mL /kg PRBC* over period of 2-4 hr

Hct ≤30/Hgb ≤10

Infants requiring **minimal respiratory support** (any mechanical ventilation or endotracheal/nasal CPAP >=6 cm H_2O and Fio_2 ≤0.4)

Transfusion:

15 mL /kg PRBCs over period of 2-4 hr

Hct ≤25/Hgb ≤8

Infants **not requiring mechanical ventilation** but who are receiving *supplemental O_2 or CPAP with an Fio_2 ≤0.4* and in whom 1 or more of the following is present: ≤24 hr of tachycardia (HR > 180) or tachypnea (RR > 80)

An increased oxygen requirement from the previous 48 hr, defined as a ≥4-fold increase in nasal canula flow (i.e., 0.25 to 1 L/min) or an increase in nasal CPAP ≥20% from the previous 48 hr (i.e., 5 to 6 cm H_2O)

Weight gain <10 g/kg/day over the previous 4 days while receiving ≥100 kcal/kg/day

An increase in episodes of apnea and bradycardia (>9 episodes in a 24-hr period or ≥2 episodes in 24 hr requiring bag and mask ventilation) while receiving therapeutic doses of methylxanthines

Undergoing surgery

Transfusion:

20 mL /kg PRBCs over period of 2-4 hr

(divide into 2-10 mL/kg volumes if fluid sensitive)

Hct ≤20/Hgb ≤7

Asymptomatic and an absolute reticulocyte count <100 000 cells/Ml

Transfusion:

20 mL /kg PRBCs over period of 2-4 hr

(2-10 mL/kg volumes)

Asymptomatic full-term infants with a hemoglobin level of **10 g/dL** may be monitored, whereas symptomatic neonates born after abruptio placentae or with severe hemolytic disease of the newborn warrant immediate transfusion.

Preterm infants who have repeated episodes of apnea and bradycardia despite theophylline therapy and a hemoglobin level of **8 g/dL** or lower may benefit from RBC transfusion.

hyaline membrane disease or severe chronic lung disease may need hemoglobin levels of **12-14 g/dL** to improve oxygen delivery.

No transfusion is needed to replace blood removed for testing or for mild asymptomatic anemia.

Asymptomatic neonates with reticulocytopenia and hemoglobin levels of **7 g/dL** or lower may require transfusion; if a transfusion is not provided, close observation is essential.

Packed RBC transfusion (10-20 mL/kg) to raise the hemoglobin concentration;

2 mL/kg raises the hemoglobin level 0.5-1 g/dL.

Hemorrhage should be treated with whole blood if available; alternatively, fluid resuscitation is initiated and followed by packed RBC transfusion.

Hemolytic Disease of the Newborn (Erythroblastosis Fetalis)

Treatment.

The main goals of therapy are to

(1) prevent intrauterine or extrauterine death from severe anemia and hypoxia and

(2) avoid neurotoxicity from hyperbilirubinemia.

TREATMENT OF AN UNBORN INFANT

Survival of severely affected fetuses has been improved by the use of ultrasonographic and amniotic fluid analysis to identify the need for in utero transfusion. Intrauterine transfusion into the fetal peritoneal cavity is being replaced by direct intravascular (umbilical vein) transfusion of packed RBCs. **Hydrops or fetal anemia (Hct <30%) is an indication for umbilical vein transfusion in infants with pulmonary immaturity** .

Intravascular transfusion is facilitated by maternal and hence fetal sedation with diazepam and by fetal paralysis with pancuronium. Packed RBCs are given by slow-push infusion after cross matching with the mother's serum. The cells should be obtained from a CMV-negative donor and irradiated to kill lymphocytes to avoid graft vs host disease. Transfusions should achieve a **post-transfusion Hct of 45-55%** and can be **repeated every 3-5 wk**.

Indications for delivery include

1- pulmonary maturity,

2- fetal distress,

3- complications of PUBS,

4- 35-37 wk of gestation.

TREATMENT OF A LIVEBORN INFANT

The birth should be attended by a physician skilled in neonatal resuscitation.

Fresh,

low-titer,

group O,

irradiated Rh-negative blood cross-matched against maternal serum

should be immediately available.

.

If clinical signs of severe hemolytic anemia

(pallor, hepatosplenomegaly, edema, petechiae, or ascites)

are evident at birth:

immediate resuscitation

supportive therapy,

temperature stabilization,

monitoring before proceeding with exchange transfusion may save some severely affected infants.

Such therapy should include **correction of acidosis** with 1-2 mEq/kg of sodium bicarbonate; a small transfusion of compatible packed RBCs to correct anemia; volume expansion for hypotension, especially in those with hydrops; and provision of **assisted ventilation** for respiratory failure

EXCHANGE TRANSFUSION.

severe hemolysis:

- **Cord hemoglobin of 10 g/dL**
- **less and bilirubin of 5 mg/dL or more** but inconsistently predict the need for exchange transfusion.

Some physicians consider:

previous kernicterus

severe erythroblastosis in a sibling,

reticulocyte counts greater than 15%,

prematurity to be additional factors supporting a decision for early exchange transfusion.

Intrauterine, intravascular transfusions have decreased the need for exchange transfusion.

hemoglobin concentration,

Hct,

serum bilirubin level **should be measured at 4-6 hr intervals initially**, with extension to longer intervals if and as the rate of change diminishes. The decision to perform an exchange transfusion is based on the likelihood that the trend of bilirubin levels plotted against hours of age indicates that serum **bilirubin** will reach the levels indicated in Tables in text. Term infants with levels of **20** mg/dL or higher have an increased risk of kernicterus.

Ordinary **transfusions of compatible Rh-negative irradiated RBCs** may be necessary to **correct anemia at any stage of the disease up to 6-8 wk of age**, when the infant's own blood-forming mechanism may be expected to take over. **Weekly determinations of hemoglobin or Hct** should be done until a spontaneous rise has been demonstrated.

Careful monitoring of the serum bilirubin level is essential until a falling trend has been demonstrated in the absence of phototherapy . Even then, an occasional infant, particularly if premature, may experience an unpredicted significant rise in serum bilirubin as late as the 7th day of life. Attempts to predict the attainment of dangerously high levels of serum bilirubin based on observed levels **exceeding 6 mg/dL in the 1st 6 hr or 10 mg/dL in the 2nd 6 hr of life or on rates of rise exceeding 0.5-1.0 mg/dL/hr** can be unreliable.
Measurement of **unbound bilirubin** may be a more sensitive predictor of the risk associated with hyperbilirubinemia.

Blood

for exchange transfusion should be as fresh as possible. Heparin or citrate-phosphate-dextrose-adenine solution may be used as an anticoagulant. If the blood is obtained before delivery, it should be taken from a type O, Rh-negative donor with a low titer of anti-A and anti-B antibodies and should be compatible with the mother's serum by the indirect Coombs test. After delivery, blood should be obtained from an Rh-negative donor whose cells are compatible with both the infant's and the mother's serum; when possible, type O donor cells are generally used, but cells of the infant's ABO blood type may be used when the mother has the same type. A complete cross match, including an indirect Coombs test, should be performed before the 2nd and subsequent transfusions. Blood should be gradually warmed and maintained at a temperature between 35 and 37°C throughout the exchange transfusion. It should be kept well mixed by gentle squeezing or agitation of the bag to avoid sedimentation; otherwise, the use of supernatant serum with a low RBC count at the end of the exchange will leave the infant anemic. Whole blood or packed irradiated RBCs reconstituted with fresh frozen plasma to **an Hct of 40%** should be used. The infant's stomach should be emptied before transfusion to prevent aspiration, and body temperature should be maintained and vital signs monitored. A competent assistant should be present to help monitor, tally the volume of blood exchanged, and perform emergency procedures.

With strict aseptic technique, the **umbilical vein** is cannulated with a polyvinyl catheter to a distance **no greater than 7 cm** in a full-term infant(**2-4 cm**). When free flow of blood is obtained, the catheter is usually in a large hepatic vein or the inferior vena cava. Alternatively, the exchange may be performed through **peripheral arterial (drawn out) and venous (infused in) lines**. The exchange should be carried out over a **45-60 min** period, with aspiration of **20 mL** of infant blood alternating with infusion of 20 mL of donor blood. Smaller aliquots (5-10 mL) may be indicated for sick and premature infants. The goal should be an isovolumetric exchange of approximately two blood volumes of the infant (**2 × 85 mL/kg**).

Infants with acidosis and hypoxia from respiratory distress, sepsis, or shock may be further compromised by the significant acute acid load contained in citrated blood, which usually has a pH between 7 and 7.2. The subsequent metabolism of citrate may result in metabolic alkalosis later if citrated blood is used. Fresh heparinized blood avoids this problem.

During the exchange, blood pH and Pao_2 should be serially monitored because infants often become acidotic and hypoxic during exchange transfusions. Symptomatic hypoglycemia may occur before or during an exchange transfusion in moderately to severely affected infants; it may also occur 1-3 hr after exchange. Acute complications, noted in 5-10% of infants, include transient bradycardia with or without calcium infusion, cyanosis, transient vasospasm, thrombosis, apnea with bradycardia requiring resuscitation, and death. Infectious risks include CMV, HIV, and hepatitis. Necrotizing enterocolitis is a rare complication of exchange transfusion.

The risk of **death** from an exchange transfusion performed by an experienced physician is **0.3/100** procedures. However, with the decreasing use of this procedure because of the use of phototherapy and prevention of sensitization, the general level of physician competence is decreasing. Thus, it is best if this procedure is performed in experienced neonatal referral centers. After exchange transfusion, the **bilirubin level** must be determined at frequent intervals (**every 4-8 hr**) because bilirubin may rebound 40-50% within hours. **Repeated exchange transfusions should be carried out to keep the indirect fraction from exceeding the levels indicated in** Table (in text) for preterm infants and 20 mg/dL for term infants. Symptoms suggestive of kernicterus are mandatory indications for exchange transfusion at any time.

LATE COMPLICATIONS

Infants who have hemolytic disease or who have had an exchange or an intrauterine transfusion must be observed carefully for the development of anemia and cholestasis. **Late anemia may be hemolytic or hyporegenerative**. Treatment with supplemental iron, erythropoietin, or blood transfusion may be indicated. A mild graft vs host reaction may be manifested as diarrhea, rash, hepatitis, or eosinophilia. *Inspissated bile syndrome* refers to the rare occurrence of persistent icterus in association with significant elevations in direct and indirect bilirubin in infants with hemolytic disease. The cause is unclear, but the jaundice clears spontaneously within a few weeks or months. *Portal vein thrombosis* and *portal hypertension* may occur in children who have been subjected to exchange transfusion as newborn infants. It is probably associated with prolonged, traumatic, or septic umbilical vein catheterization.

Plethora

Plethora

A ruddy, deep red-purple appearance associated with a high Hct, is often due to polycythemia, **defined as a central Hct of 65% or higher**.

Peripheral (heelstick) Hct values are higher than central values, whereas Coulter counter results are lower than Hct values determined by microcentrifugation.
The incidence of neonatal polycythemia is increased at **high altitude** (5% in Denver vs 1.6% in Texas); in **postmature** (3%) vs term (1-2%) infants; in **small for gestational age** (8%) vs **large for gestational age** (3%) vs average for gestational age (1-2%) infants; during the **1st day** of life (peak, 2-3 hr); in the **recipient infant of a twin-twin transfusion**; after **delayed clamping of the umbilical cord**; in **infants of diabetic mothers**; in **trisomy 13, 18 or 21**; in **adrenogenital syndrome**; in **neonatal Graves disease**; in **hypothyroidism**; and in **Beckwith-Wiedemann syndrome**.
Infants of diabetic mothers and those with growth retardation may have been exposed to chronic fetal hypoxia, which stimulates erythropoietin production and increases RBC production.

Clinical manifestations

include **anorexia, lethargy, tachypnea, respiratory distress, feeding disturbances, hyperbilirubinemia, hypoglycemia, and thrombocytopenia.**

Severe complications include **seizures, pulmonary hypertension, necrotizing enterocolitis, and renal failure**.

Many affected infants are **asymptomatic**.

Hyperviscosity is present in most infants with central Hct values of 65% or higher and accounts for the symptoms of polycythemia.

Hyperviscosity determined at constant shear rates (e.g., 11.5 sec^{-1}) is present when whole blood viscosity is above 18 cycles/sec. Hyperviscosity is accentuated because neonatal RBCs have decreased deformability and filterability, which predisposes to stasis in the microcirculation

Treatment

symptomatic polycythemic newborns:

partial exchange transfusion (with normal saline).

asymptomatic infant:

The Hct level (without measurement of viscosity) at which to perform a partial exchange transfusion is unclear (**70 – 75**%?). Partial exchange will lower the Hct and viscosity and improve acute symptoms.

Blood volume for partial exchange transfusion:

(Desired hematocrite – patient hematocrite)

÷

patient hematocrite

×

patient blood volume

The long-term *prognosis:*

The long-term *prognosis* of polycythemic infants is unclear. Reported adverse outcomes include speech deficits, abnormal fine motor control, reduced IQ, school problems, and other neurologic abnormalities. It is thought that the underlying etiology (e.g., chronic intrauterine hypoxia) and hyperviscosity contribute to adverse outcomes. It is unclear whether partial exchange transfusion improves the long-term outcome.

Apnea

Cloherty. Nelson. Fanaroff

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Definition:

- >20 sec.

  or

- with:

  Oxygen desaturation & bradycardia.

50 – 75% = mixed (usually obstractive before centeral)

Mixed usually is more prolonged than centeral.

Causes:

1. **Prematurity**.
2. **Impaired oxygenation** (obstraction – vagal tone – pulm. Problems – muscle weakness - shock - anemia …).
3. **Inhibitory reflexes**.
4. **Infection** (sepsis - RSV – pertusis - other viral …).
5. **Metabolic disorders** (hypoglycemia – temp. instability – elect. imbalances – met. Acidosis - hyperammonia).
6. **CNS pathology** (meningitis - IVH – HIE – malformations – upper spinal cord trauma - Convulsion).
7. **Medications** (opiates – benzodiazepines – PG E1 – mg sulfate)
8. **GER** ?

Infection:

Clinic:

- Feeding intolerance,
- lethargy,
- temperature instability

Dx:

- **Complete blood count,**
- **cultures, if appropriate**

Impaired oxygenation:

Clinic:

- Cyanosis,
- tachypnea,
- respiratory distress

Dx:

- **Continuous oxygen monitoring,**
- **arterial blood gas measurement,**
- **Chest x-ray examination**

Metabolic disorders:

Clinic:

- Jitteriness,
- poor feeding,
- lethargy,
- CNS depression,
- irritability

Dx:

- **Glucose,**
- **calcium,**
- **electrolytes,**
- **ammonia**

Drugs:

Clinic:

- hypotonia,
- Maternal history,
- CNS depression,

Dx:

- **Magnesium,**
- **Screen for toxin**
- **Substances in urine**

Temperature instability:

Lethargy

Dx:

- **Monitor temperature of patient**
- **and environment**

Intracranial pathology:

Clinic:

Abnormal neurologic examination,

seizures

Dx:

- **Cranial ultrasonographic examination**

Treatment:

- Monitoring./
- Gentel skin stimulation./
- NPO./
- Gentel suction./
- Normal position of neck./
- Prone position.

- **Xanthine therapy**
  - amino-theophylin 5-8 mg/kg and then 1-3 mg/kg every 6-12 hrs or
  - caffeine 20 mg/kg and after 24 hr as 5-10 mg/kg every 24 hr. po or IV over 30 min).

- **Doxapram ?**(potent respiratory stimulant)

- **KMC.**
- **Supplemental oxygen.**
- **CPAP** (4 – 6 cm Hg) or **Nasal Cannula** (high flow) or **sNIPPV**.
- **Packed cell transfusion.** (if Hct<25%)
- **Inspired low concentration of Co2.**
- **Mechanical ventilation.**

Hypoglycemia

- **Low Blood glucose: <40 (47) mg/dl**
- **Severe Hypoglycemia: <25 (32) mg/d**

<u>During normal transition after birth:</u>

- **1 to 2 hours of life: 30 mg/dL**
- **3 to 4 hours of age: >45 mg/dL,**
- **Then: 65 to 70 mg/dL by.**

Hypoglycemia

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Hypoglycemia

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Management of Hypoglycemia

1. Feeding: breast milk or formula, 40% dextrose gel

Asymptomatic infants with early glucose levels in the 30s (mg/dL) will respond to feeding (breast milk or formula).

A follow-up **blood glucose 1 hour after** the start of the feeding.

If glucose not rise, IV glucose infusions are required.

Feeding of **glucose water is not recommended**.

IV therapy

Indications

i. Inability to tolerate oral feeding

Ii Persistent symptoms of hypoglycemia after feeding

Iii Oral feedings do not maintain normal glucose levels.

Iv Severe hypoglycemia

Urgent treatment

200 mg/kg of glucose over 1 minute; (2 mL/kg of D10W) infused intravenously.

Continuing therapy

glucose Infusion **6-8 mg/kg/min.**

IDM

Diabetic mothers have a high incidence of :

- polyhydramnios,
- preeclampsia,
- pyelonephritis,
- preterm labor,
- chronic hypertension;

- their **fetal mortality** rate, which is high at all gestational ages, especially after 32 wk, is greater than that of nondiabetic mothers

Problems in IDM

- hypoglycemia,
- Hypocalcemia,
- hypothermia,
- polycythemia,
- cardiac failure, congenital anomalies
- transient tachypnea, RDS,
- or cerebral edema from birth trauma or asphyxia,
- Hyperbilirubinemia,

Tx (intensive observation and care)

- **Asymptomatic infants:**
  screening glucose **test within 30 min of the 1st feeding**
  and then
  prior to each other feeding **(Q2-3hr)**

- **if clinically well and normoglycemic:**
  oral or gavage feeding with breast milk
  or formula should be started as soon as possible
  (within 1 hr after birth) and continued **Q2-3hr**

If any question arises about an infant's ability

to tolerate oral feeding:

peripheral intravenous glucose infusion

4-8 mg/kg/min.

- Historically, providers have administered **hydrocortisone, 10 mg/kg/day** intravenously in two divided doses, if it is difficult to maintain glucose values in the normal range despite 12 to 15 mg of glucose per kilogram per minute. Hydrocortisone reduces peripheral glucose utilization, increases gluconeogenesis, and increases the effects of glucagon. The hydrocortisone will usually result in stable and adequate glucose levels, and it can then be rapidly tapered over the course of a few days. Before administering hydrocortisone, providers might consider drawing a cortisol level. We do not use hydrocortisone routinely for hypoglycemia.

- **Diazoxide (8 to 15 mg/kg/day** in divided doses every 8 to 12 hours) may be given orally for infants who are persistently hyperinsulinemic. This drug inhibits insulin release by acting as a specific ATP-sensitive potassium channel agonist in normal pancreatic beta cells and decreases insulin release. It can take up to 5 days for a positive effect to be seen. Side effects include fluid retention, and coadministration with a diuretic such as hydrochlorothiazide may be considered.

- **Octreotide (5 to 20 µg/kg/day** subcutaneously or intravenously divided every 6 to 8 hours). A long-acting somatostatin analog that inhibits insulin secretion. It can be used when diazoxide does not successfully control the glucose level. Tachyphylaxis can develop.

- **Glucagon (0.2 mg/kg** intramuscularly, subcutaneous (SC), or IV, maximum 1.0 mg) is rarely used. It may be given to hypoglycemic infants with good glycogen stores, but it is only a temporizing measure to mobilize glucose for 2 to 3 hours in an emergency until IV glucose can be given. The glucose level will often fall after the effects of glucagon have worn off, and it remains important to obtain IV access to adequately treat these infants. For IDMs, the dose is 0.3 mg/kg (maximum dose is 1.0 mg)

- If medical treatment does not control the blood glucose level, consider a 18F-fluoro-L-DOPA positron emissiontomography **(PET) scan** to identify focal lesions in the pancreas and consider surgical treatment by **subtotal pancreatectomy**. Referral to a subspecialty center with experience in these procedures should be considered if a genetic defect of glucose control is suspected or confirmed.