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Polycythemia in the Newborn

Juan I. Remon, MD,*
Aarti Raghavan, MD,*
Akhil Maheshwari, MD*

Abstract
Neonatal polycythemia, defined as a venous hematocrit ≥65% (0.65), is a common problem in newborns. Infants born postterm or small for gestational age, infants of diabetic mothers, recipient twins in twin-to-twin transfusion syndrome, and those who have chromosomal abnormalities are at higher risk. Although the cause of polycythemia is often multifactorial, most cases can be classified as having active (increased fetal erythropoiesis) or passive (erythrocyte transfusion) polycythemia. By increasing blood viscosity, polycythemia can impair microcirculatory flow in end organs and can present with neurologic, cardiopulmonary, gastrointestinal, and metabolic symptoms. In this article, we review the pathophysiology, clinical presentation, diagnosis, and management of polycythemia in the newborn.

Objectives After completing this article, readers should be able to:
1. List the causes of polycythemia and hyperviscosity in the neonate.
2. Review the signs, symptoms, and diagnostic criteria for polycythemia and hyperviscosity.
3. Discuss the treatment of polycythemia and hyperviscosity in the neonate.

Introduction
Polycythemia (or more accurately, erythrocythemia), an abnormal elevation of the circulating red blood cell (RBC) mass, is seen frequently in newborns. Although neonatal polycythemia usually represents a normal fetal adaptation to hypoxemia rather than a true hematopoietic defect, the abnormal increase in hematocrit increases the risk of hyperviscosity, microcirculatory hypoperfusion, and multisystem organ dysfunction. In this article, we review the definition, pathophysiology, clinical presentation, and management of polycythemia in the newborn.

Definition
In healthy term infants, the hematocrit and hemoglobin concentrations in venous blood obtained at birth are 50.2±6.9% (0.5±0.07) (mean ± standard deviation) and 15.9±1.86 g/dL (159±18.6 g/L), respectively. (1) Polycythemia is defined in newborns as a venous hematocrit greater than 65% (0.65) or a hemoglobin value greater than 22 g/dL (220 g/L). (2)(3)(4) Based on this definition, the incidence of polycythemia in healthy newborns has been reported to be 0.4% to 5%. (5)(6)(7) Increased incidence is seen in certain high-risk groups such as postterm neonates, small-for-gestational age infants, infants of diabetic mothers, identical twins who share the same placenta and develop twin-to-twin transfusion, and infants who have chromosomal abnormalities. (7)(8)(9)

Pathophysiology
Although the cause of polycythemia is often multifactorial, most patients can be classified as having active (increased fetal erythropoiesis) or passive (erythrocyte transfusion) polycythemia (Table 1). (5)(7)
Increased fetal erythropoiesis is frequently seen in conditions associated with hypoxia:

- Placental insufficiency due to preeclampsia, maternal chronic hypertension, chronic or recurrent placental abruption, maternal cyanotic congenital heart disease, postdate pregnancy, maternal smoking and heavy alcohol intake.
- Endocrine abnormalities such as congenital thyrotoxicosis or maternal diabetes with poor glycemic control.
- Genetic disorders such as trisomy 13, trisomy 18, trisomy 21, and Beckwith-Wiedemann syndrome.

Erythrocyte Transfusion

- Placental-fetal transfusion with delayed cord clamping, relative positioning of the delivered infant in relation to the maternal introitus before cord clamping, perinatal asphyxia, oxytocin administration.
- Twin-to-twin transfusion syndrome.

Increased fetal erythropoiesis is frequently seen in conditions associated with hypoxia:

- Placental insufficiency due to preeclampsia, primary renovascular disease, chronic or recurrent placental abruption, maternal cyanotic congenital heart disease, postdate pregnancy, maternal smoking, and maternal heavy alcohol intake. (10)(11)(12) The severity of hematopoietic dysfunction in these conditions appears to be proportional to the degree of placental insufficiency and fetal growth restriction. (8) Whereas mild placental dysfunction and consequent tissue hypoxia are associated with increased erythropoietin concentrations and polycythemia, more severe placental vasculopathy may cause erythropoietin resistance and anemia. (13)(14)

- Endocrine abnormalities associated with increased fetal oxygen consumption, such as congenital thyrotoxicosis or maternal diabetes with poor glycemic control. (13)(14) Thyrotoxicosis is presumed to increase erythropoiesis through a direct effect on marrow progenitor cells, increased erythropoietin expression, and the indirect effects of intracellular growth restriction. (15)(16) In diabetic mothers who have poor glycemic control, maternal hyperglycemia is proposed to increase fetal erythropoiesis through fetal hyperinsulinemia, tissue hypoxia, and increased erythropoietin concentrations. (17)(18) Although plasma leptin concentrations are frequently elevated in infants of diabetic mothers, the actual concentrations do not correlate with the severity of polycythemia, and current data do not support a causative role of leptin in this process. (18)

- Genetic disorders, such as trisomy 13, trisomy 18, trisomy 21, and Beckwith-Wiedemann syndrome. The incidence of polycythemia in infants who have Down syndrome is 15% to 33%. (19)(20)(21) Although the cause of polycythemia in Down syndrome is not known, high cord blood erythropoietin concentrations in affected infants has led to the speculation that intrauterine hypoxemia may play a role. (9) Among neonates who have trisomy 13 and trisomy 18, the prevalence of polycythemia has been estimated as 8% and 17%, respectively. (22)

Erythrocyte transfusion polycythemia can result from placental-fetal transfusion. Delayed cord clamping allows for delivery of an increased blood volume to the infant. When cord clamping is delayed more than 3 minutes after birth, blood volume may increase by as much as 30%. (23) Hutton and Hassan (24) analyzed data from seven randomized studies and showed that neonates in the late-clamping group had a higher incidence of asymptomatic polycythemia with a benign course (relative risk [RR], 3.82; 95% confidence interval [CI], 1.11 to 13.21). However, a more recent meta-analysis showed that delayed cord clamping did not cause a clinically significant change in hematocrit at 1 and 4 hours of age. (25)

Placental-fetal transfusion is likely influenced by gravity and the relative position of the delivered infant in relation to the maternal introitus before cord clamping; raising or lowering the baby by 15 to 20 cm or more with the cord intact appears to influence placental transfusion. (26) No randomized studies have examined this issue to date. Placental transfusion is augmented in infants who have perinatal asphyxia, which causes an active shift of the blood volume from the placenta to the fetus. (27) Oxytocin administration to the mother can also increase the volume of placental transfusion to the newborn. (28)

Twin-to-twin transfusion syndrome due to a vascular communication occurs in approximately 10% of monzygotic twin pregnancies. (29)

**Polycythemia and Hyperviscosity**

Polycythemia remains an area of interest due to its potential effects on the viscosity of blood and its flow properties in the microcirculation. (5)(7)(8) Viscosity is a measure of the resistance of a fluid that is being deformed by either shear stress or tensile stress. (30) More simply, viscosity refers to the “thickness” of a fluid and is a measure of the fluid’s internal resistance to flow.
Hyperviscosity is arbitrarily defined as a viscosity measurement of greater than 14.6 centipoise detected in a viscometer at a shear rate of 11.5/sec. (5)(7)(30) Viscosity rises linearly with increasing hematocrit until the hematocrit reaches 60% (0.6) but increases exponentially when hematocrit equals or exceeds 70% (0.7). (5)(31)(32) Although the terms polycythemia and hyperviscosity are often used interchangeably, they are not equivalent and show only modest concordance in clinical cohorts. (5)(30) Furthermore, blood viscosity can also rise with an increase in plasma proteins, platelets, leukocytes, and endothelial factors. (30)(32)

Hyperviscosity occurs in polycythemia due to the presence of an abnormally large number of circulating erythrocytes; the plasma viscosity in the newborn is almost always normal. (5)(7) According to Poiseuille’s law, flow velocities in the circulation are determined by the resistance to flow, which varies with the viscosity of the blood and inversely with the fourth power of the radius of the blood vessel. This relationship can be expressed by the equation $R = \frac{8\eta L}{\pi r^4}$, where $R$ represents the resistance to blood flow, $\eta$ is the viscosity, $L$ is the length of the vessel, and $r$ is the radius of the vessel. (33)/(34) Because resistance is affected by viscosity as well as the caliber of the blood vessel, the effects of polycythemia on blood flow patterns are usually most pronounced in the microcirculation. (34) In these small vessels, non-newtonian mechanisms such as rouleaux formation and increased erythrocyte-endothelial interaction are also active and further contribute to the altered flow. (32)

Polycythemia and hyperviscosity are associated with decreased blood flow to the brain, heart, lung, intestines, and carcass. (5)(7)(35)(36) Although renal blood flow is not affected, renal plasma flow and glomerular filtration rate are often diminished. (35) Hyperviscosity can also reduce pulmonary blood flow that, in turn, can cause systemic hypoxia. (36) In contrast to the effects of polycythemia on the kidney and lungs, reduced cerebral blood flow in polycythemia likely represents a vascular response to the increased arterial oxygen content (related to increased hemoglobin concentrations) rather than hyperviscosity. (7)(37) Changes in blood flow may also alter the delivery of substrates (such as glucose) to organs that are dependent on plasma flow. (7)(38)(39)

**Clinical Findings**

The symptom complex associated with polycythemia is frequently described by the term “hyperviscosity syndrome,” although it is important to remember that only 47% of infants who have polycythemia exhibit hyperviscosity, and only 24% of infants who have hyperviscosity have a diagnosis of polycythemia. (6)(7)(40) Whereas most patients who have polycythemia remain asymptomatic, characteristic clinical features may be recognized as early as 1 to 2 hours after birth as the hematocrit peaks with normal postnatal fluid shifts. (3) In some infants who have high borderline hematocrits, symptoms may be delayed until the second to third postnatal day, when excessive depletion of the extracellular fluid may lead to hemoconcentration and hyperviscosity. Infants who have no symptoms by 48 to 72 hours of age are likely to remain asymptomatic. (3)(41)

Clinical features associated with neonatal polycythemia are generally nonspecific and include ruddy complexion, irritability, jitteriness, tremors, feeding difficulties, lethargy, apnea, cyanosis, respiratory distress, and seizures. (7) Neurologic symptoms occur in approximately 60% of affected patients. (5)(7)(42) The cause of these symptoms is uncertain, but reduced cerebral blood flow and altered tissue metabolism likely play important roles. Neurologic signs may also be related to metabolic changes such as hypoglycemia and hypocalcemia. Hypoglycemia is the most common metabolic derangement and is observed in 12% to 40% of infants who have polycythemia. Hypocalcemia is found in 1% to 11% of neonates who have polycythemia, possibly related to elevated concentrations of calcitonin gene-related peptide (CGRP) in affected infants. (43) The pathophysiology of increased CGRP concentrations is not clear; CGRP may play a role in the normal postnatal circulatory adaptation to extraterine life, and this process is presumed to be accentuated in infants who have polycythemia. (44)

Polycythemia and hyperviscosity have been implicated as pathogenic factors in necrotizing enterocolitis (NEC), particularly in term or near-term neonates. (45)(46)(47)(48)(49) Historically, polycythemia has been identified in up to half of all term infants who have NEC. (46)(47)(48) Although altered splanchnic perfusion is widely considered to cause gut mucosal injury in affected infants, recent data indicate that attempts to reduce the hematocrit with partial exchange transfusions (PET) also could contribute to the risk of NEC. (36)(42)(49)

Renal manifestations of polycythemia include decreased glomerular filtration rates, oliguria, hematuria, proteinuria, and renal vein thrombosis. (5)(7) Thrombocytopenia can also be seen in other sites. Thrombocytopenia can be seen in up to one third of all cases, presumably due to platelet consumption in the microvasculature. (50)(51) In neonates who have polycythemia due to increased erythropoiesis, thrombocytopenia may also be related to
“progenitor steal,” the diversion of hematopoietic progenitors toward erythropoiesis at the expense of other lineages. (8)(18) Overt disseminated intravascular coagulation is rare. (51)

**Diagnosis**

The diagnosis of polycythemia requires detection of a venous hematocrit of at least 65% (0.65). (2)(3)(4) This cut-off finds its origins in studies on blood viscosity that show an exponential increase in viscosity above this value. (3)(7) However, clinical evidence for this threshold remains scant; studies have shown only modest concordance between a hematocrit greater than 65% (0.65) and actual demonstration of hyperviscosity. (7) Although blood viscosity could be a useful guide for deciding appropriate management strategies in affected patients, hematocrits continue to be widely used surrogate markers of hyperviscosity due to limited availability of tools for direct measurement of blood viscosity.

Detection of high hematocrits (≥55% [0.55]) in cord blood could help predict the risk of polycythemia at 2 hours postnatal age, but such “screening” has not gained acceptance because of the lack of data showing that treatment of asymptomatic newborns who have elevated hematocrits alters outcomes. (3) The possibility of polycythemia should be considered in any infant exhibiting signs of hyperviscosity. Detection of a high hematocrit in the first few hours after birth should trigger a follow-up measurement in a few hours to identify any further rise with normal postnatal fluid shifts. (3)

Close attention must be paid to the technique of sample collection while interpreting the test results. Capillary blood samples often show hematocrits that are 5% to 15% higher than venous samples, and, therefore, high hematocrit measurements in samples obtained by heelsticks should be confirmed in a free-flowing venous sample. (3)(52) The technique of sample analyses should also be taken into account during serial evaluation of results because microcentrifuge hematocrit may be slightly higher (due to trapped plasma of about 2%) than that calculated from RBC volume and RBC count determined by hematology analyzer. (1)

Neonates who have polycythemia should be evaluated for underlying causes such as intrapartum growth restriction, maternal diabetes, or birth asphyxia. Because clinical manifestations of hyperviscosity can overlap with other conditions, alternative causes for the symptoms should always be carefully excluded. Infants also should be monitored for systemic complications of polycythemia such as thromboses, NEC, hypoglycemia, hypercalcemia, hyperbilirubinemia, and thrombocytopenia.

**Treatment**

The management of polycythemia is controversial because of the lack of evidence showing that aggressive treatment improves long-term outcomes. Asymptomatic infants whose central hematocrits are between 60% (0.60) and 70% (0.70) can be monitored closely and aggressively hydrated with adequate enteral intake or administration of intravenous fluids. The hematocrit should be reassessed in 12 to 24 hours, and plasma glucose and bilirubin and cardiorespiratory status should be monitored. If the hematocrit decreases or remains stable and the patient remains asymptomatic, monitoring can be continued for a further 24 to 48 hours. In asymptomatic infants whose hematocrits are greater than 70% (0.70), the treatment options are controversial. Although traditional treatment has been PET, studies show a lack of difference in outcomes with continued hydration and expectant management versus aggressive management with PET. (36)(42) In the absence of a consensus, the decision to perform PET is usually taken on a case-by-case basis, with a careful analysis of risks and potential benefits.

To perform PET, a precalculated volume of blood (calculated to reduce the central hematocrit to 50% [0.50] to 55% [0.55]) is replaced by an equivalent volume of normal saline, 5% albumin, commercially available solutions of human plasma protein fraction, or fresh frozen plasma. Colloid solutions do not offer any therapeutic advantages over normal saline and, at least in some studies, have been associated with a higher incidence of NEC. (53) Because of its lower cost, ready availability, and absence of risk of transfusion-associated infection, normal saline is generally accepted as the replacement fluid of choice for PET in infants who have polycythemia. (54)(55)

The total blood volume to be exchanged is determined as follows:

\[
\text{[Total blood volume} \times (\text{patient’s hematocrit} - \text{desired hematocrit})]/(\text{patient’s hematocrit})
\]

where total blood volume = the patient’s weight in kilograms multiplied by a presumed blood volume of 90 mL/kg. PET can be performed through a single umbilical venous catheter using a pull-push technique (withdrawal of blood alternated with replacement of fluid through a single catheter) or by withdrawing blood from an umbilical or peripheral arterial catheter and administering replacement fluid simultaneously through an umbilical or peripheral venous catheter. Regardless of the sites used, small aliquots of 5 mL/kg or less should
be used for removal or delivery, with each step carried out over 2 to 3 minutes.

Several randomized studies have evaluated the efficacy of PET in patients who have polycythemia (Table 2). Malan and de V Heese (n=49), (56) Goldberg and colleagues (n=20), (57) Black and coworkers (n=94), (58) and Ratrisawadi and associates (n=42) (59) randomly assigned infants to receive either PET using plasma or supportive care. Bada and colleagues (n=28) (60) compared PET using a commercially available human plasma protein solution with supportive care. Kumar and Ramji (61) randomized 45 infants to peripheral PET using normal saline or to routine medical management. None of these six studies documented a beneficial effect of PET on neurodevelopmental outcome. Dempsey and Barrington (42) systematically reviewed five of these studies to investigate whether PET was associated with improved short- and long-term outcomes in neonates who had polycythemia. They documented no improvement in long-term neurologic outcome (mental developmental index, incidence of developmental delay, and incidence of neurologic diagnoses) after PET in symptomatic or asymptomatic infants. There was also no evidence of improvement in early neurobehavioral assessment scores (Brazelton neonatal behavioral assessment scale). PET could have been associated with an earlier improvement in symptoms, but the data were insufficient to calculate the size of the effect.

Ozek and coworkers (36) reviewed six randomized trials to determine the effect of PET on primary outcomes of mortality and neurodevelopmental outcomes at 2 years and at school age. Secondary outcomes included seizures, cerebral infarction, NEC (Bell stage 2 or greater), hypoglycemia, hyperbilirubinemia, and thrombocytopenia. Only one study reported data on mortality, and no significant increase was noted with PET (RR, 5.23; 95% CI, 0.66, 41.26). Four studies reported neurodevelopmental outcomes at 18 months or older, and no significant delay was reported in the PET group (typical RR, 1.45; 95% CI, 0.83 to 2.54 including all studies and typical RR, 1.35; 95% CI, 0.68 to 2.69 when only randomized, controlled trials were included). However, these results were based on data limited by poor follow-up and did not account for patients who were lost to follow-up. The authors performed a worst case/best case scenario post hoc analysis, which showed a significant skewing toward or away from the association of PET with poor neurodevelopmental outcomes and was considered to reflect the wide distribution of data points rather than actual outcomes. The authors concluded that there is no significant benefit of PET in asymptomatic patients or those who have mild symptoms.

Patients who have polycythemia and show signs or symptoms that may be related to hyperviscosity are frequently treated with PET, even though the practice is not supported by high-quality evidence. The arguments in favor of PET are based on the pathophysiology of hyperviscosity syndrome because most of the symptoms are presumed to be related to altered microcirculatory perfusion and tissue hypoxia. (5)(7) By replacing some of the circulating RBC mass with a crystalloid solution, PET is believed to reduce blood viscosity and improve end-organ perfusion. However, no randomized clinical studies demonstrate a clear benefit of PET in the treatment of symptomatic polycythemia. The groups led by Bada, (60) Ratrisawadi, (59) and Kumar (61) randomized only asymptomatic polycythemic infants, while those led by Black, (58) Goldberg, (57) and Malan (56) made no distinction between symptomatic and asymptomatic newborns. In nonrandomized clinical reports, PET has been shown to lower pulmonary vascular resistance, improve cerebral blood flow velocity, (63)(64) and possibly normalize cerebral hemodynamics and improve the clinical status of infants who have polycythemia. (65) However, the long-term benefits of PET remain unclear.

Concerns remain about potential adverse events following PET. PET was associated with an increased risk of NEC in the systematic reviews performed by both Dempsey (42) (RR, 8.68; 95% CI, 1.06 to 71.1) and Ozek (36) (two studies: typical RR, 11.18; 95% CI, 1.49, 83.64 and typical risk difference, 0.14; 95% CI, 0.05, 0.22). Malan and de V Heese (56) reported that 1 of their 24 patients in the exchanged group developed NEC within the first 24 hours after PET compared with none of the control patients. Black and associates (58) recorded NEC in 8 of their 43 infants in the PET group compared with none of the 50 control infants. PET also did not alter the frequency of hypoglycemia (two studies) or thrombocytopenia (one study). (36) Given the risks of PET for polycythemia in the newborn and the lack of evidence indicating clear benefit, we are generally reluctant to use PET in asymptomatic infants. We do offer PET for treatment of infants who have symptoms that could be ascribed to hyperviscosity, but this decision is taken cautiously, with a careful review of all risk factors. Routine use of PET in neonatal polycythemia is not supported by current evidence, and further study is needed to identify patients who would be more likely to benefit from aggressive correction of polycythemia.
<table>
<thead>
<tr>
<th>Study</th>
<th>Randomization/ Sample Size</th>
<th>Hematocrit (Hct) at Enrollment</th>
<th>Mode of Exchange/ Replacement fluid</th>
<th>Outcome Measurements</th>
</tr>
</thead>
<tbody>
<tr>
<td>Malan and de V Heese (56)</td>
<td>Not clearly stated; n=49</td>
<td>Hct &gt;65% (0.65)</td>
<td>UVC</td>
<td>Brazelton neonatal scale</td>
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<td>FFP</td>
<td>Prechtl neurologic assessment at 10 days</td>
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<td>Neurodevelopmental assessment at 8 months</td>
</tr>
<tr>
<td>Goldberg et al (57)</td>
<td>Not clearly stated; n=20</td>
<td>Hct &gt;64% (0.64) and hyperviscosity</td>
<td>UVC</td>
<td>Brazelton neonatal scale</td>
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<td></td>
<td></td>
<td></td>
<td>FFP</td>
<td>at 8 hours, 24 hours, 72 hours, and 2 weeks</td>
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<td>BSID and neurologic assessment at 8 months</td>
</tr>
<tr>
<td>Black et al (58)</td>
<td>Randomized; n=93</td>
<td>Antecubital venous Hct &gt;65% (0.65) and hyperviscosity</td>
<td>UVC</td>
<td>Neonatal signs</td>
</tr>
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<td></td>
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<td>FFP</td>
<td>BSID and neurologic assessment at 1 and 2 years</td>
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<td></td>
<td>Cognitive testing at 7 years</td>
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<td></td>
<td>Cerebral artery Doppler measurement</td>
</tr>
<tr>
<td>Radrzawadi et al (59)</td>
<td>Randomized; n=28</td>
<td>Radial artery Hct &gt;65% (0.65)</td>
<td>Route not stated; Plasma protein solution</td>
<td>BSID or cognitive testing at 30 months</td>
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<td></td>
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<td>Gasel Development at 1.5 to 2 years</td>
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<tr>
<td>Ratnison et al (62)*</td>
<td>Randomized; n=105</td>
<td>Central venous Hct &gt;65% (0.65)</td>
<td>Route not stated</td>
<td>BSID at 8 months</td>
</tr>
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<td>Not clearly stated; n=24</td>
<td>Hyperviscosity (undefined)</td>
<td>Not clearly stated; FFP</td>
<td>Neurologic deficits at 3, 6, 9, 12, 18 months</td>
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<td>Peripheral venous Hct &gt;70% (0.70)</td>
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*FFP = fresh frozen plasma, BSID = Bayley Scale of Infant Development, UVC = umbilical venous line, * = abstract only.
American Board of Pediatrics Neonatal–Perinatal Medicine Content Specifications

- Know the causes of neonatal polycythemia.
- Know the clinical manifestations, management, and outcomes of neonatal polycythemia.

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logic function is the viscosity and not the polycythemia. Clin Hemorheol Microcirc. 1997;17:67–72
NeoReviews Quiz

6. Polycythemia represents an abnormal elevation of the circulating red blood cell mass, which increases the risk of hyperviscosity, microcirculatory hypoperfusion, and multisystem organ dysfunction. Of the following, the hematocrit threshold that best defines polycythemia is:

A. 55% (0.50).
B. 60% (0.60).
C. 65% (0.65).
D. 70% (0.70).
E. 75% (0.75).

7. Polycythemia in neonates is often associated with metabolic complications. Of the following, the most common metabolic abnormality associated with polycythemia in neonates is:

A. Hyperchloremia.
B. Hyperkalemia.
C. Hypernatremia.
D. Hypocalcemia.
E. Hypoglycemia.

8. The treatment of polycythemia includes partial exchange transfusion in which a precalculated volume of blood is replaced by an equivalent volume of fluid. Of the following, the most accepted choice of fluid for partial exchange transfusion is:

A. Albumin solution.
B. Fresh frozen plasma.
C. Lactated Ringer solution.
D. Normal saline.
E. Plasma protein fraction.
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<thead>
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</tr>
</tbody>
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