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Blood Viscosity and Optimal Hematocrit in Preterm and Full-Term Neonates in 50- to 500-μm Tubes

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ABSTRACT. Blood viscosity is an important determinant of blood flow resistance. Because a substantial part of flow resistance arises in small arteries and arterioles with diameters of 100 μm and less, rheologic properties of blood from preterm infants (24 to 36 wk of gestation), full-term neonates, and adults were measured in glass tubes with diameters of 50, 100, and 500 μm for a wide range of adjusted feed hematocrits (0.15–0.70). At each of the feed hematocrits, blood viscosity decreased when going from a 500-μm tube to a 50-μm tube. The viscosity reduction increased with increasing hematocrit. Moreover, the viscosity reduction was more pronounced in the neonates than in the adults. At a hematocrit of 0.70, the viscosity reduction averaged 56% in preterm infants, 50% in full-term neonates, and 39% in adults ($p < 0.005$). However, the viscosity reductions at a hematocrit of 0.30 were only 35, 29, and 19%, respectively ($p < 0.05$). In all four groups, blood viscosity increased exponentially with increasing hematocrit. The steepness of the hematocrit-viscosity curves decreased with decreasing tube diameter and with decreasing maturity of the infants. Erythrocyte transport efficiency (hematocrit/blood viscosity) was calculated to estimate the optimal hematocrit (*i.e.* hematocrit with maximum erythrocyte transport). In 500-μm tubes, the optimal hematocrit was about 0.40 in all of the groups. In 100-μm tubes, the optimal hematocrit was 0.44 ± 0.05 in the adults and 0.52 ± 0.04 in the neonates ($p < 0.05$). In 50-μm tubes, the optimal hematocrit was 0.51 ± 0.04 in adults and 0.60 ± 0.05 in the neonates. There was no significant difference in the optimal hematocrit among preterm and full-term infants. Our results suggest that the strong viscosity reduction at high hematocrits may help to maintain oxygen transport in polycythemic neonates. (*Pediatr Res* 32: 97–102, 1992)

Abbreviations

RBC, red blood cell

Polycythemia, defined as a venous hematocrit greater than or equal to 0.65, occurs in 2 to 20% of all newborn infants (1–3). Clinical manifestations have been observed in approximately 50% of newborn infants with hematocrit values of 0.65 or greater (2, 4). The rise in blood viscosity accounts for clinical manifestations of polycythemia (5). Blood viscosity is an important determinant of the resistance to blood flow, and hyperviscosity can impede blood flow to various organs and compromise their oxygen supply (5, 6). In neonates, hyperviscosity increases the risk of pulmonary hypertension, renal failure, necrotizing enterocolitis, cerebral ischemia, intracranial hemorrhage, and developmental retardation (2, 4, 7).

Blood viscosity is mainly determined by the hematocrit. Moreover, increased plasma viscosity, strong RBC aggregation, and decreased RBC deformability can also contribute to an increase in blood viscosity (6.8). In artificial tubes with diameters of less than 500 μm, both the hematocrit (Fahraeus effect) and the blood viscosity (Fahraeus-Lindqvist effect) decrease with decreasing diameter (9, 10). This has been attributed to the migration of RBC to the axis, thereby creating a cell-poor plasma layer on the wall and a cell-rich central core (11–14). Because the flow velocity increases from the vessel wall to the axis, the central cell core leaves the tube more rapidly than the slow-flowing plasma layer at the wall. This decreases the hematocrit and blood viscosity in narrow tubes. The Fahraeus-Lindqvist effect is enhanced at high hematocrit levels (10–12) and may therefore aid in the maintenance of sufficient oxygen transport in polycythemia (5, 6). Because during normal circulation a substantial part of the flow resistance arises in small arteries and arterioles with diameters of less than 500 μm (6, 14), the viscosity reduction may be an important prerequisite for adequate circulation, particularly in polycythemia.

If tubes are perfused with a constant driving pressure, RBC transport increases with increasing hematocrit until an "optimal" hematocrit is reached. Further increases in hematocrit cause a decrease in RBC transport (8, 15). A study on the optimal hematocrit of human adult blood has shown that the optimal hematocrit increases from 0.38 in 500-μm tubes to 0.51 in 50-μm tubes (16).

Determination of the optimal hematocrit requires the measurement of blood viscosities over a wide range of hematocrits. In newborn infants, the optimal hematocrit has been determined only for full-term neonates using tubes with a diameter of 100 μm (17). We have constructed a tube viscometer for viscosity measurements of small blood samples of 50 μL or less (16). With this device, the viscosities of blood with hematocrits adjusted to values of 0.15 to 0.70 (in 0.05 increments) in 50-, 100-, and 500-μm tubes were measured. Based on viscometric data obtained in preterm and full-term neonates and adults, the optimal hematocrit in these different tubes was estimated.

MATERIALS AND METHODS

Blood samples. Placental blood samples from 30 newborn infants were studied with the approval of the Department of Pediatrics Human Subjects Research Committee. Ten of the newborn infants were healthy full-term neonates with gestational age of 38 to 41 wk and birth weight of 3340 to 3690 g, 10 were preterm infants with gestational age of 24 to 29 wk and birth weight of 960 to 1410 g, and 10 had gestational age of 30 to 36

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wk and birth weight of 1400 to 2620 g. The gestational age of each infant was derived from the maternal history and confirmed by clinical assessment of maturity. All infants had birth weight appropriate for gestational age (10th to 90th percentile according to Munich growth charts). Infants with malformations, erythroblastosis, diabetic mothers, or intrauterine asphyxia were excluded, as were twins and infants with high risk of infection. The umbilical artery cord pH was above 7.25 in all cases. Apgar scores at 1 to 5 min of birth were 7 or more in the preterm infants and 9 to 10 in the full-term neonates. Rectal temperature was above 36°C in all cases. Respiratory distress syndrome developed in four of the preterm infants. Infants who developed intracranial hemorrhage and infants who died were retrospectively excluded. Thus, the preterm infants were highly selected to avoid a strong influence of complications on the results.

For the 30 neonates, 10 mL of blood were collected from the placenta into EDTA (1 mg/mL) immediately after cord clamping before delivery of the placenta. Umbilical cords were clamped within 10 s of birth. Adult blood samples were collected from 10 healthy adults via venipuncture into EDTA. The results obtained in the adults have already been reported (16).

RBC were isolated by centrifugation at 2000 × *g* for 10 min and, by gentle aspiration, the plasma was removed and the buffy coat discarded. The RBC were resuspended in the autologous plasma at hematocrits of 0.15 ± 0.01 to 0.70 ± 0.01 in 0.05 increments for measurements with the tube viscometer. Thus, 12 RBC suspensions were studied in each donor. In addition, viscosity of the plasma from each sample was measured. All measurements were made within 4 h after collection. Previous studies have shown that keeping blood for this duration does not alter its rheologic properties (17).

Miscellaneous techniques. Feed and discharge hematocrits were measured by the microhematocrit method. RBC count, mean corpuscular volume, and Hb concentration were determined with a Coulter Counter (Coulter Electronics, Herts, UK). Total plasma protein concentration was measured by the biuret reaction.

Viscosity measurement. Details of the tube viscometer have been described elsewhere (16). Blood suspensions flow from a wide, calibrated feed tube (diameter of 0.5 cm) through the capillary tube to a calibrated receiver tube for 5 to 50 μL of volume. The feeding tube is connected to compressed air and a water manometer and maintained at 37°C. Glass tubes with internal diameters (D) of 50 and 100 μm were 1 cm in length (L), and tubes with internal diameters of 500 μm were 10 cm in length. Experiments were performed by using pressure drops (P) of 5 kPa or 50 cm H_2O (50- and 500-μm tubes) and 2.5 kPa (100-μm tubes). The wall shear stress (=0.25 P·D/L) was 6.25 Pa (62.5 dyne/cm^2) independent of the tube diameter. Because the arterial blood pressure in newborn infants is markedly lower than in adults, the pressure-dependent shear stress may also be lower in neonatal vessels. To study the effect of shear stress on blood viscosity, the hematocrit of three full-term neonatal and three adult blood samples was adjusted at 20, 40, and 60%. Apparent viscosities of these blood samples were measured in 50-, 100-, and 500-μm tubes at six different pressures resulting in wall shear stresses between 3 and 19 Pa. Variation of the shear stress did not affect blood viscosity measurements significantly (16). The chosen wall shear stress of 6.25 Pa is within the range of *in vivo* values (6).

For each of the RBC suspensions, the passage times of equal volumes of buffer solution, plasma, and RBC suspension through the same capillary tube were measured by stop watch. The passage times were between 30 s and 5 min. Sedimentation of RBC was prevented by a magnetic stirrer in the feeding reservoir and short RBC residence times (<1.5 s) in the tubes (16). First, the tube was perfused with buffer solution, then with autologous plasma, and finally with the RBC suspension. For each of the three samples, two receiver tubes were subsequently filled and the second passage time was used to minimize dilution by the previous sample. After each blood viscosity measurement, the tube was cleaned with buffer solution until no blood was visible in the system. The discharge hematocrits of all RBC suspensions were measured by using a microcentrifuge. For all three tube diameters, the feeding and the discharge hematocrits agreed within 2%.

Because of potential errors of tube viscometry (15, 16), the uncertainty in the viscosity measurements was studied in blood from five adults and five full-term neonates. Hematocrits of the 10 blood samples were adjusted at 0.20, 0.40, and 0.60. Apparent viscosities of buffer, plasma, and RBC suspensions were measured twice in 50-, 100-, and 500-μm tubes at a wall shear stress of 6.25 Pa. The difference between two paired measurements was always below 5%.

Calculations. Viscosity (η) of Newtonian fluids in circular tubes is determined by the Hagen-Poiseuille equation (15);

$$\eta = (\Delta P/Q)\cdot(r^4/L)\cdot(\pi/8) = t\cdot(\Delta P/V)\cdot(r^4/L)\cdot(\pi/8) \quad (1)$$

where V is the volume and t the passage time of the fluid (Q = V/t). Because only t varies for a given tube and all other parameters are constant, tube viscosity of Newtonian fluids can be calculated from:

$$\eta = t\cdot C \quad (2)$$

Blood viscosities (η_{blood}) were calculated from:

$$\eta_{blood} = (t_{blood}/t_{H_2O})\cdot(0.6915\ mPa\cdot s) \quad (3)$$

where t_{blood} and t_{H_2O} are the passage times of the blood sample and water and 0.6915 mPa·s (1 mPa·s = 1 centipoise) is the viscosity of water at 37°C. Relative viscosity (η_{rel}) is calculated as ratio of blood to plasma viscosity (η_{plasma}):

$$\eta_{rel} = \eta_{blood}/\eta_{plasma} \quad (4)$$

The tube RBC flow (Q_{RBC}) can be calculated as product of tube blood flow (Q_{blood}) and discharge hematocrit (Hct):

$$Q_{RBC} = Hct\cdot Q_{blood} \quad (5)$$

$$Q_{blood} = V\cdot C/\eta_{blood} \quad (6)$$

$$Q_{RBC} = (Hct/\eta_{blood})\cdot V\cdot C \quad (7)$$

V and C are constant for a given tube. RBC flow in a given tube thus depends on the hematocrit-to-blood viscosity ratio (*i.e.* RBC transport efficiency, T) (15):

$$T = Hct/\eta_{blood} \quad (8)$$

The "optimal hematocrit" (*i.e.* peak RBC flow) was derived from individual RBC transport efficiency-hematocrit relationships.

Statistics. Statistical analyses were performed to test for differences in measurements among the three tube diameters using analysis of variance for paired comparisons. Analysis of variance for unpaired observations was used to test for differences in measurements among the preterm infants, full-term neonates, and adults. Regression analyses were used to determine correlations between plasma viscosity and total plasma protein concentration (linear function), between blood viscosity and hematocrit (exponential functions), and between RBC transport efficiency and hematocrit (5th order polynomial functions). The functions were selected because they gave the best fits and the highest correlation coefficients.

RESULTS

Total plasma protein and plasma viscosity increased with increasing gestational age and reached the highest values in the adults (Table 1). There was a significant ($p < 0.001$) linear correlation between total plasma protein and plasma viscosity (Fig. 1). Plasma viscosity did not differ significantly among the three tube diameters.

Table 1. *Hematologic and viscosity data for blood from preterm and full-term infants and adults**

	Hematocrit	Tube diameter (μm)	Neonates (wk of gestation) A 24–29	B 30–36	C 38–41	Adults D	Significant differences among four groups ($p < 0.05$)
General hematologic data							
MCV (fL)			120 ± 9	116 ± 10	107 ± 6	91 ± 5	A > B > C > D
MCH (pg)			39.1 ± 3.5	38.4 ± 3.3	34.9 ± 2.7	30.6 ± 2.3	A > B > C > D
MCHC (g/dL)			329 ± 20	330 ± 22	327 ± 15	331 ± 12	A = B = C = D
Total plasma protein (g/L)			43 ± 4	50 ± 4	56 ± 7	71 ± 7	A < B < C < D
Viscosity data							
Plasma viscosity (mPa·s)		100	0.83 ± 0.07	0.93 ± 0.11	1.04 ± 0.10	1.26 ± 0.13	A < B < C < D
Blood viscosity (mPa·s)	0.40	50	1.2 ± 0.1	1.5 ± 0.2	1.8 ± 0.2	2.4 ± 0.3	A < B < C < D
(relative viscosity)			(1.5 ± 0.1)	(1.6 ± 0.2)	(1.7 ± 0.2)	(1.9 ± 0.2)	A = B < C < D
	0.40	100	1.5 ± 0.2	1.8 ± 0.2	2.1 ± 0.3	2.7 ± 0.3	A < B < C < D
			(1.8 ± 0.2)	(1.9 ± 0.2)	(2.0 ± 0.2)	(2.2 ± 0.3)	A = B = C < D
	0.40	500	2.1 ± 0.3	2.3 ± 0.3	2.6 ± 0.4	3.2 ± 0.4	A = B < C < D
			(2.6 ± 0.4)	(2.4 ± 0.3)	(2.5 ± 0.4)	(2.6 ± 0.3)	A = B = C = D
	0.70	50	2.1 ± 0.2	2.4 ± 0.2	3.0 ± 0.4	4.5 ± 0.5	A < B < C < D
			(2.4 ± 0.2)	(2.6 ± 0.3)	(2.9 ± 0.4)	(3.6 ± 0.4)	A = B < C < D
	0.70	100	2.7 ± 0.4	3.2 ± 0.5	4.2 ± 0.7	5.7 ± 0.7	A < B < C < D
			(3.2 ± 0.5)	(3.5 ± 0.4)	(4.0 ± 0.5)	(4.5 ± 0.6)	A = B < C < D
	0.70	500	4.7 ± 0.8	5.4 ± 0.9	6.0 ± 1.1	7.3 ± 1.0	A < B < C < D
			(5.5 ± 1.4)	(5.7 ± 1.0)	(5.7 ± 0.9)	(5.9 ± 1.2)	A = B = C = D
Optimal hematocrit		50	0.59 ± 0.06	0.62 ± 0.05	0.60 ± 0.05	0.51 ± 0.04	A = B = C > D
		100	0.52 ± 0.05	0.53 ± 0.04	0.50 ± 0.03	0.44 ± 0.05	A = B = C > D
		500	0.42 ± 0.05	0.39 ± 0.03	0.41 ± 0.04	0.38 ± 0.04	A = B = C = D

* $p < 0.05$ compared with the next larger tube (analysis of variance). Values are mean ± 1 SD for 10 adults (D), 10 term neonates (C), and 20 preterm neonates (10 in group A and 10 in group B). MCV, mean cell volume; MCH, mean cell Hb; and MCHC, mean cell Hb concentration.

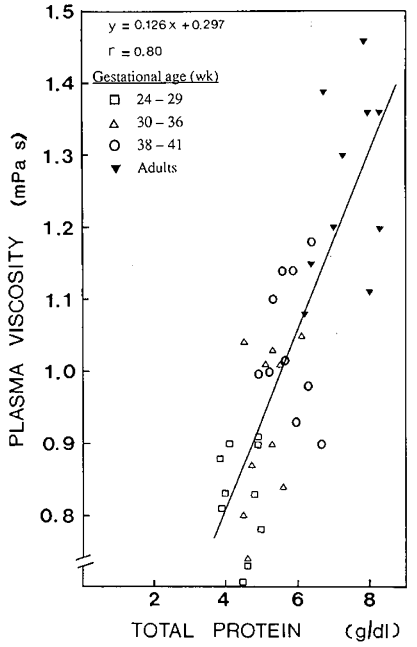

Fig. 1. Relationship of plasma viscosity to total plasma protein concentration. 1 mPa·s = 1 centipoise.

Blood viscosity decreased significantly ($p < 0.05$), at every adjusted hematocrit, when going from a 500-μm tube to a 50-μm tube (Table 1). In each of the four groups, the viscosity reduction increased with increasing hematocrit. The strongest viscosity reductions were found in the preterm infants, the smallest in the adults. Figure 2 shows the hematocrit-viscosity relationships for the four groups and the three tube diameters. The steepness of the curves decreased with decreasing tube diameter. The flattest curves were observed for blood from preterm infants in 50-μm tubes. In the smallest preterm infants, the regression coefficient for the 50-μm tube was 44% less than that for the 500-μm tube, whereas in the adults the difference was only 24% (Table 2).

At given hematocrits, whole blood viscosities were lower in the preterm infants than in the full-term infants, who in turn showed lower whole blood viscosities than the adults. Relative viscosity (*i.e.* whole blood viscosity divided by plasma viscosity) in 500-μm tubes was similar for the four groups, thereby indicating that the differences in these large tubes was merely the result of different plasma viscosities. Relative viscosities in the 50- and 100-μm tubes were, however, lower in the neonates than in the adults (Table 1).

The RBC transport efficiency (*i.e.* hematocrit divided by blood viscosity) was higher in the narrow tubes than in the wider tubes at each of the adjusted hematocrits (Fig. 3). Moreover, RBC transport efficiency was higher in the neonates than in the adults. The hematocrits with the greatest RBC transport efficiency (*i.e.* the optimal hematocrits) were obtained for each of the 40 blood samples for each tube size. These data were used to calculate the mean ± 1 SD of the optimal hematocrit for each tube size and each group (Table 1). Figure 3 shows the mean RBC transport efficiencies calculated for the 10 samples at each of the adjusted hematocrits. The curves in Figure 3 were computed from these mean values. This explains why the optimal hematocrits of the curves slightly deviate from the optimal hematocrits in Table 1. The optimal hematocrit increased with decreasing tube diameter. The 50-μm tubes had a plateau region where the transport efficiency was about constant over a hematocrit range of 0.40 to 0.60 in the adults and 0.50 to 0.70 in the neonates. The optimal hematocrits determined for the 500-μm tubes were about 0.40 in all four groups. The optimal hematocrits in the smaller tubes were significantly higher in the neonates than in the adults.

DISCUSSION

From the present data, we conclude that blood viscosity (at given hematocrit) measured in narrow tubes is lower in preterm infants than in full-term neonates, who in turn show lower blood viscosity than adults. The lower blood viscosity was due to low plasma viscosity and to a more pronounced viscosity reduction

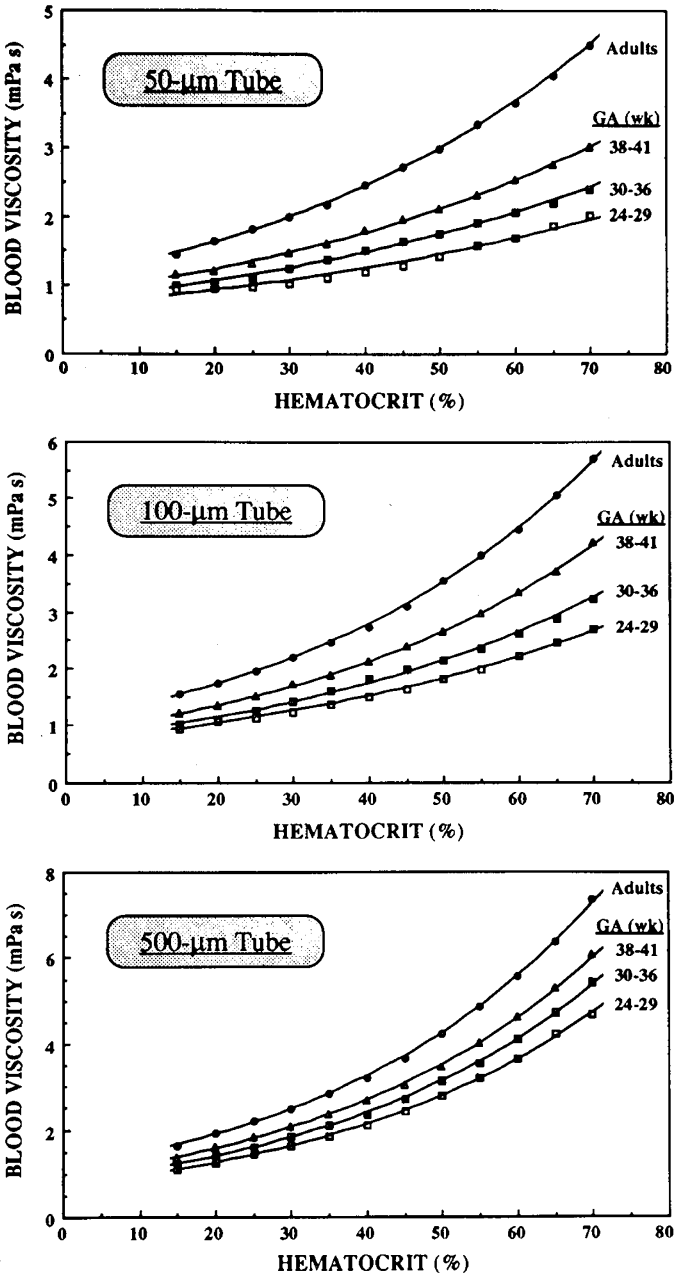

Fig. 2. Blood viscosity in tubes with diameters of 50, 100, and 500 µm plotted against the feed hematocrits. Exponential functions (Table 2) were computed for fitting the data. Note that the viscosity of adult blood in 50- and 100-µm tubes increased more with increasing hematocrit than that of neonatal blood. 1 mPa·s = 1 centipoise; *GA*, gestational age in wk.

in narrow tubes (Fahraeus-Lindqvist effect). Plasma viscosity depends on the total plasma protein concentration (Fig. 1). However, the influence of high molecular weight proteins (*e.g.* fibrinogen) is greater than that of smaller proteins (*e.g.* albumin) (8). Total plasma protein, fibrinogen, and plasma viscosity are very low in small, preterm infants, but increase with increasing gestational age and reach the highest values in adults (18). Plasma viscosities measured with a tube viscometer are lower than those determined with a rotational viscometer (17). This may be due to flow instability in rotational viscometers (19). For this reason, capillary viscometers are considered more reliable for plasma viscosity measurements than rotational viscometers (19).

In the neonates, blood samples were taken from the placenta. It could be argued that our results are representative of the time before birth rather than of the neonatal period. However, plasma viscosity, RBC aggregation, and RBC deformability are not different when cord blood and venous blood taken at 2 and 24 h after birth are compared (20). The hematocrit may change markedly during the first day (1, 3), particularly after late cord clamping (20), but we adjusted the hematocrit at fixed values.

In agreement with previous reports, we found that the viscosity reduction in narrow tubes increases with increasing hematocrit (10–12). Barbee and Cokelet (21) demonstrated that down to a tube diameter of 29 µm the viscosity reduction can be predicted completely by the hematocrit reduction. At high hematocrits, a small reduction in the hematocrit causes a marked viscosity reduction. This explains why the Fahraeus-Lindqvist effect (*i.e.* viscosity reduction) tends to increase with increasing hematocrit although the Fahraeus effect (*i.e.* hematocrit reduction) tends to decrease (14). The more pronounced viscosity reduction of neonatal RBC can also be explained by a stronger hematocrit reduction when compared with adult RBC (22). The large volume and increased membrane elasticity (23) of neonatal RBC may facilitate their packing in the rapidly flowing tube center (13, 24), particularly at high hematocrit.

The principal thrust of studies on blood flow in glass tubes is to provide quantitative data for physical variables that control blood flow dynamics *in vivo*. In stiff glass tubes, the tube diameter and length, perfusion pressure, and flow rate (and thus shear rate and shear stress) can be controlled; the composition of blood and physical properties of the blood components can be altered. On the other hand, the vessel diameter *in vivo* decreases steadily from the aorta to the capillaries, and vessel diameters may quickly change as a result of passive and active processes. Perfusion pressure, flow rate, and diameter of elastic vessels change during each heart cycle as a result of periodic vasomotion (14) and in response to the needs of the body and the tissues. Nevertheless, the biophysical properties of blood flow behavior studied under well-controlled conditions *in vitro* are principally also valid *in vivo* (6, 14, 15).

The decrease in hematocrit and blood viscosity with decreasing tube diameter occurs both *in vitro* and *in vivo*. Direct microscopic observation of vessels in adult animals revealed that the hematocrit decreases by about 75% as the blood flows from wide arteries to narrow capillaries with diameters approaching the resting RBC diameter (25, 26). The hematocrit reduction in narrow vessels explains why the total body hematocrit is 10% lower than the large vessel hematocrit (25). The viscosity reduction in narrow vessels explains why the viscosity measured *in vivo* is lower than that determined by means of a wide tube or rotational viscometer (27, 28). *In vivo* studies of the Fahraeus and Fahraeus-Lindqvist effect do not appear to exist for neonates.

RBC flow through a tube varies with the hematocrit-to-viscosity ratio (8, 15). RBC flow rises with increasing hematocrit as long as the increase in hematocrit is greater than the increase in

Table 2. *Exponential functions between blood viscosity (η in mPa·s) and hematocrit (Hct)*

Gestational age (wk)	Vessel diameter		
	50 µm	100 µm	500 µm
24–29	$\lg \eta = -0.1640 + 0.65$ Hct	$\lg \eta = -0.1516 + 0.82$ Hct	$\lg \eta = -0.1368 + 1.16$ Hct
30–36	$\lg \eta = -0.1225 + 0.72$ Hct	$\lg \eta = -0.1293 + 0.92$ Hct	$\lg \eta = -0.0915 + 1.17$ Hct
38–41	$\lg \eta = -0.0697 + 0.78$ Hct	$\lg \eta = -0.0735 + 0.99$ Hct	$\lg \eta = -0.0391 + 1.17$ Hct
Adults	$\lg \eta = 0.0311 + 0.89$ Hct	$\lg \eta = 0.0289 + 1.06$ Hct	$\lg \eta = 0.0421 + 1.17$ Hct

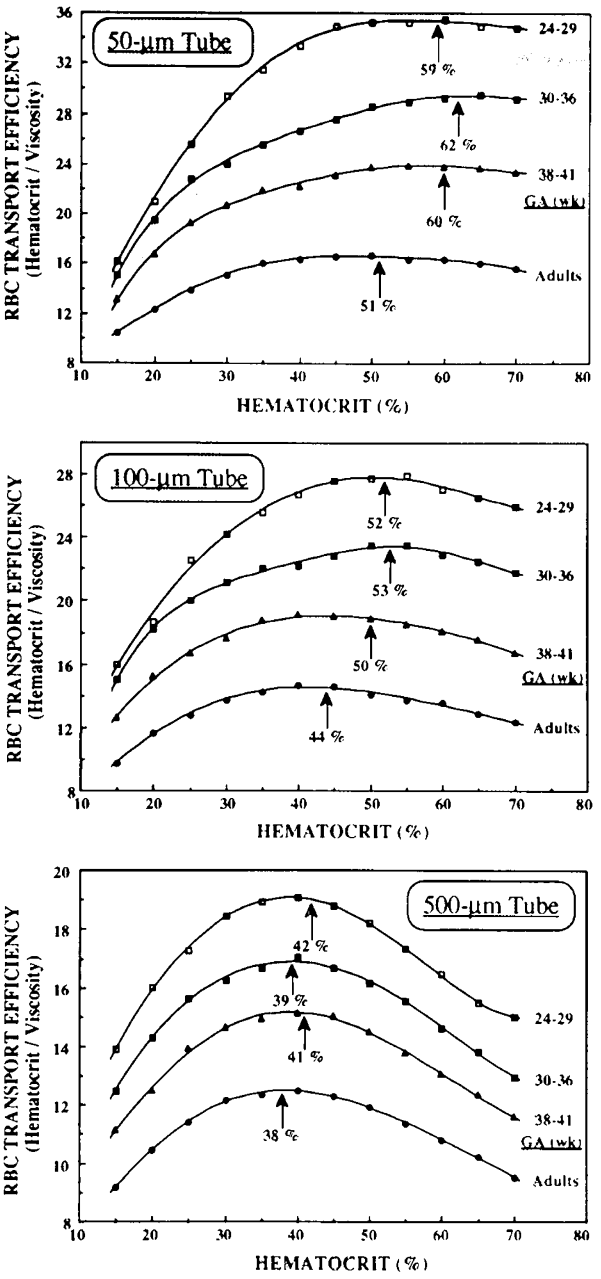

Fig. 3. Effect of tube diameter and feed hematocrit on RBC transport efficiency. Fifth order polynomial functions were computed for fitting the data. The *arrows* indicate the mean maximum RBC transport efficiencies presented in Table 1. *GA*, gestational age in wk.

blood viscosity. Above the optimal hematocrit (*i.e.* hematocrit with maximum RBC transport efficiency), the steep increase in blood viscosity results in a decreased RBC flow (Fig. 3). RBC transport efficiency was higher in narrow tubes than in wide tubes and in neonates than in adults over the whole range of hematocrits due to lower blood viscosities (Fig. 2). Particularly in neonates, the optimal hematocrit increased with decreasing tube diameter (Table 1).

The results of our *in vitro* study may have implications for the physiology and pathophysiology of *in vivo* circulation in the fetus and neonate. The circulation in the fetus and neonate is characterized by low blood pressures and high flow conditions (29–32). Systemic flow resistance calculated as the ratio of the mean arterial blood pressure (33) and cardiac output (31) increases from ~10 to ~15 mm Hg/(L/min/m^2) when the gestational age increases from 26 to 40 wk and to ~30 mm Hg/(L/min/m^2) in healthy adults. According to the Hagen-Poiseuille equation (see equation 1) the flow resistance decreases with decreasing blood viscosity, decreasing vessel length, and increasing vessel radius. Comparative studies on the architecture and geometry of microvessels in neonates and adults do not appear to exist in the literature. However, the rise in blood viscosity in 50-μm tubes from the preterm infants to the adults (Table 1) is strikingly similar to the increase in the systemic resistance. Thus, because a substantial part of the flow resistance arises in small arteries and arterioles with diameters of 100 μm and less (6, 14), the pronounced viscosity reduction of neonatal blood may be necessary for adequate circulation.

In 50-μm tubes, we observed an optimal hematocrit of about 0.60 for neonatal blood and 0.51 for adult blood (Table 1, Fig. 3). This suggests that in neonates the hematocrit may increase to higher values than in adults before the oxygen transport is compromised and clinical manifestations develop. Optimal hematocrits for overall systemic and organ RBC flow have been determined in adult animals (5, 34) and in human adults (6). RBC transport to the brain of human adults appears to be maximal at a hematocrit of about 0.50 (6). In human neonates, systemic RBC flow and cerebral RBC flow velocity [= blood flow (velocity) × hematocrit] remain about constant over a hematocrit range of 0.50 to 0.65 (30, 32). The critical hematocrit at which the risk of complications rises markedly is approximately 0.55 in adults (6) and 0.65 in neonates (2, 4). Because the optimal hematocrit is strongly dependent on the shear forces acting on the blood (8), a low driving pressure (*e.g.* after an artery stenosis or in shock) (6, 8) causes a steep rise in the viscosity of polycythemic blood. This increases the risk of blood stasis and ischemia. It is, therefore, extremely important to maintain blood pressure in polycythemic infants.

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