Influence of Deformability of Human Red Cells upon Blood Viscosity

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ABSTRACT

The viscosity of blood at high rates of shear is unusually low compared to other suspensions of similar concentration. The underlying mechanisms were studied by rotational viscometry, red cell filtration, viscometry of packed cells and direct microscopic observation of red cells under flow in a transparent cone plate viscometer. Deformability of red cells was altered osmotically or abolished by aldehyde fixation. The normal red cells under isosmotic conditions passed easily through filter pores (5 to 14 μm diameter). After osmotic crenation, deformability of cells in pore flow was reduced. Normal cells were deformed into a variety of shapes at high rates of shear, while crenated cells tumbled undeformed. Suspensions of these normal cells showed more pronounced shear thinning (reduction of viscosity with increasing shear rate) than suspensions of crenated cells. Suspensions of rigid cells showed greatly increased viscosity and a shear thickening as a function of shear rate and shear time. The physiological deformability is of critical importance to blood flow at high rates of shear. This is possible through a fluid transition of the erythrocyte caused by a rotation of the membrane with and around the cell contents. This phenomenon is the prime cause of the progressive reduction in viscosity with increasing shear.

ADDITIONAL KEY WORDS

erythrocyte membrane rheology
fluid transition of red cells
suspension rheology
capillary blood flow
shear thinning at high shear rate
non-Newtonian viscosity of blood

The almost exponential rise in apparent blood viscosity near zero shear rate has been shown to be due to a reversible aggregation of red cells into a three-dimensional structure of rouleaux (1, 2). It has also been demonstrated that aggregation is not the sole cause of the non-Newtonian viscosity of blood, as nonaggregating suspensions, such as red cells in saline or crenated cells in plasma, also exhibit shear dependent viscosity (2, 3). Shear thinning flow behavior is lost when the deformability of the red cells is abolished. Chien et al. (4) found the viscosity of rigid cells in saline considerably higher at high rates of shear than that of normal red cells in saline. It can be implied from their studies that the physiological decrease of viscosity under high shear must in some fashion be a consequence of red cell deformability. The concept that the viscosity of the red cells affects blood viscosity has long been advocated by Dintenfass (5, 6). However, his assumption that this operates through a shear dependent liquefaction of the red cell membrane is in conflict with the known physical properties of the erythrocyte membrane. The present report is concerned with experiments in which the relationship between cell deformability and the flow be-
behavior of blood was studied under a variety of experimental conditions, including those that permitted the direct microscopic observation of the deformation process under flow. The relative contributions of both membrane and intracellular hemoglobin to this process were quantified.

Methods

Blood samples were obtained from healthy donors by routine blood bank procedures (450 ml of blood anticoagulated in 67.5 ml of citrate phosphate dextrose solutions) and used within 72 hours of withdrawal. The cells were separated from plasma, and theuffy coat was removed. Size, shape, and deformability of red cells were altered osmotically, and the osmolarity of the plasma was changed without altering the concentration of the plasma proteins. NaCl crystals were added to achieve the desired osmolarity between 300 and 900 milliosmols/liter. The osmolarity was measured by freezing point depression of the plasma.1 The hypertonic plasma was mixed with the red cells and then separated again by centrifugation after an equilibration period of 10 minutes. The shape of the red cells was observed in fresh, wet preparations at phase-contrast microscopy. The following arbitrary subdivisions of cell shape were made: (1) normal biconcave disks, (2) crenated disks, and (3) crenated spheres.

In other experiments maximum hardening of cells was accomplished by prolonged exposure to gluteraldehyde. The cells were washed four times in buffered saline and then mixed with 10 times their volume of 1.24% isosmolar solutions of gluteraldehyde at 4°C for 24 hours. Thereafter, the cells were again washed five times in large volumes of saline to remove all aldehyde and then were resuspended in saline or plasma. These suspensions were carefully mixed and passed through 0.3-mm pores of a stainless steel sieve to remove clumps that resulted from packing by the centrifuge.

Two methods were employed to measure deformability of red cells: (1) viscometry of concentrated cell packs (volume concentration 95%) at a wide variety of shear rates as described in detail elsewhere (7), and (2) by filtration of dilute suspensions (hematocrit 2% and 12%) through standardized Millipore filters.2 All filters used were cut from a single sheet by the manufacturer. Cellulose ester filters with entrance pore diameters of 5.0 μ (type SM) and 8.0 μ (type SC) and nylon filters with entrance pore diameters of 14 μ (type NC) were used in a 25-mm filter holder (type XX30-023-00). At 37°C and a driving pressure of 15 ±1 cm H2O, a fixed volume was passed through the filter. After the filter holder had filled and steady flow through the filter itself began, a volume of 2 ml was passed, and the time necessary for this was measured. The flow rate (mm3/sec) was then computed. All measurements were done in triplicate. The flow of cell-free plasma was also measured for comparison. The hematocrit as well as the percentage of normal and crenated red cells were determined both before and after filtration. Viscosity of hardened cells was measured in a GDM viscometer (8) at shear rates between 0.1 and 20 sec−1 with continuous recording of shear stresses. A guard ring was built into the instrument to obviate surface effects, and in some experiments, a thin layer of paraffin oil (viscosity 3.8 centipoises) was placed on the surface of the suspension for the same purpose. As evidenced by comparative studies, this procedure did not influence the results. Due to incomplete packing of hardened cells, the volume fraction (hematocrit) had to be determined indirectly by measuring the volume of the continuous phase. This was achieved by measuring the concentration of albumin in 125I (9). The hematocrit of suspensions of normal and crenated cells was measured in duplicate with the microhematocrit centrifuge at 11,500g (10). The viscosity of suspensions of normal and crenated red cells was also measured in a cone plate viscometer (Wells-Brookfield LVT)3 covering a range of shear rates between 11.5 and 230 sec−1 (11).

This latter viscometer was equipped with transparent cone and plate and mounted on an inverted microscope (12). In this "rheoscope," direct observation of blood cells under uniform shear was possible at magnifications between 40 and 400×. Photomicrographs were made of these using single-flash stroboscopic illumination. The flow behavior of individual red cells was studied by mixing 10% intact red cells with 40% plasma and 50% packed red cell membranes (ghosts). The ghosts were produced by freeze-thaw lysis of packed cells. They were separated from the hemoglobin and washed in isotonic buffered saline by repeated centrifugation at 40,000g as described previously (7).

The viscosity of suspensions of packed cells (95% hematocrit) was also measured in a GDM as well as in a cone plate viscometer. To allow

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1Advanced Instruments, Newton Highlands, Mass.  
3Brookfield Engineering Laboratory, Stoughton, Mass.

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viscometry of packed cells at high rates of shear a Wells-Brookfield 10 X LVT viscometer capable of higher shear stresses (up to 230 dynes/cm²) was used (2). The shear rate and shear stress values were computed for fluidity (rhe), the reciprocal value of viscosity.

Results

PARTIALLY RIGIDIFIED RED CELLS
(OSMOTIC CRENATION)

Suspensions of 2% red cells in isosmolar plasma could be driven through filters with entrance pore diameters of 5 μ at a flow rate of 91 ± 6 mm³/sec (Fig. 1) as compared to 440 ± 40 mm³/sec for clear plasma. An increase of the osmolarity above 500 milliosmols/liter strongly reduced the flow rate although the cells were osmotically shrunken. The passage of 2% cell suspensions through filters with 8.3-μ pores showed similar changes with osmolarity; these, however, were more graded and could be studied in greater detail. The flow rate under isosmolar conditions was 280 to 320 mm³/sec and that of clear plasma was 400 ± 60 mm³/sec. With progressive increase in osmolarity, above the normal range, an increasing percentage of the cells

![Graphs showing fluidity, flow rate, percentage trapping, and red cell morphology](image)

**FIGURE 1**

Effect of plasma osmolarity upon blood rheology. A: Fluidity (rhe) of packed red cells at 95% hematocrit and a shear rate of 115 sec⁻¹ (37°C), decreasing with rising osmolarity. B: Flow rate of red cell suspensions through filters with entrance pore diameters of 5, 8, and 14μ, progressive reduction with increasing osmolarity. C: Percentage of red cell trapping in 8-μ filter pores, fewer cells passing pores at high osmolarity. D: Red cell morphology before and after filtration (8-μ pores). (Blank columns = biconcave disks, hatched = crenated disks, dotted = crenated spheres.) The percentage of normal biconcave cells decreases with increasing osmolarity but is consistently higher in the filtrate.
were crenated. Upon filtration, the flow rate was again reduced, although the cells were shrunken to a diameter considerably less than 8 μ (Fig. 1). At osmolarities above 500 milliosmols/liter, the flow either stopped immediately or came to a stop before the volume of 2 ml had passed the filter. At extremely high osmolarities, only clear plasma was filtered (above 700 milliosmols/liter).

The cell suspensions before and after the filtration were compared, and differences between the two cell populations became evident. At normal osmolarity, the hematocrit before and after the filtration was identical, 100% normal cells appearing in either case. With increasing osmolarity, the hematocrit after filtration was markedly reduced. The morphology of the red cell population before filtration showed an increased proportion of crenated disks and crenated spheres with increasing osmolarity. Even at 450 milliosmols/liter, from 2 to 10% of the cells were seen to be biconcave. After passage through the filter, the distribution of cells was altered: a higher percentage of normal or less crenated cells was consistently found in the filtrate (Fig. 1).

The filtration experiments with 12% cell suspensions through filters with 14-μ pores again showed the same pattern upon changes in osmolarity; maximum flow rates were seen under isosmolar conditions with decreases after cell crenation. A representative experiment showing the influence of osmolarity upon filtration through 5.0-, 8.3-, and 14-μ filters, fluidity (the reciprocal value of viscosity) of packed cells, and distribution of altered cells before and after filtration is shown in Figure 1, while Figure 2 summarizes 50 filtration experiments from 14 blood samples at 2% hematocrit 8.3-μ pores.

**Bulk Viscosity**

The changes in bulk viscosity after osmotic crenation are very complex. At high rates of shear (20 to 230 sec⁻¹) the apparent viscosity for each given hematocrit rises with osmolarity. While at

![Graph](image)

**FIGURE 2**

Flow rate of dilute red cell (2% hematocrit) suspensions through 8.3-μ filters as a function of osmolarity. Each point represents average of three measurements (50 experiments, 14 blood samples).
25% hematocrit and 230 sec\(^{-1}\) the difference between isomolar and hyperosmolar blood amounts to only 60%, at 45% the viscosity is increased by 120%. At low rates of shear (between 0.1 and 2 sec\(^{-1}\)), osmolarity affects viscosity in exactly the opposite manner: between 300 and 450 milliosmols/liter, the apparent viscosity near stasis falls progressively. Upon further increase of the osmolarity, the viscosity near stasis starts to rise again, reaching almost the values of isosmotic controls (Figs. 3 and 4).

Microscopic Flow Adaptation

The microscopic observation of the individual cells under flow in the transparent cone plate viscometer became possible after mixing 10% red cells with 40% plasma and 50% packed red cell ghosts. Observation near the axis of the rotating transparent cone is facilitated by the low velocity and thinness of the blood layer. Normal red cells at low rates of shear (4.6 sec\(^{-1}\)) were seen to move mostly individually and only very seldom in groups of four to eight cells forming a short rouleau. Upon each increase of the shear rate the individual cells were seen with occasional tumbling and orbiting in flow. After further increase in shear rate the cells became orientated and less and less orbiting was observed. At shear rates above 100 sec\(^{-1}\) the individual cells lost their biconcave shape and were constantly transformed into a variety of shapes, many of them resembling prolate ellipsoids. The major axes of these prolate cells were oriented parallel to the direction of flow, and the tumbling was eliminated. Instead, a
Viscosity profiles of normal red cells (osmolarity 300 milliosmols/liter; circles), crenated red cells (osmolarity 580 milliosmols/liter; stars), and rigidified red cells in plasma (diamonds) at four different hematocrits. Note strong influence of cell rigidification at 45% hematocrit.

Twisting, flexing, and bending movement of the cell membrane was seen, resulting in irregular cell indentations. Occasionally, a rotational motion of the membrane around the cell was observed. In the lower surface of such cells, which faced the stationary plate of the viscometer (and the objective of the microscope), the patterns of membrane indentations proceeded in a wave-like fashion along the cell surface. These wave patterns started from the leading end of the moving cell and went on to the trailing end, while the whole cell moved forward. Immediately after stoppage of the flow, the cells resumed their biconcave shape (Fig. 5). All cells exhibited similar flow adaptation, they were constantly aligned parallel to the direction of flow and showed a conspicuous lack of collision.

When crenated red cells were suspended in hypertonic plasma and ghosts, no deformation of the cells was observed. Instead, the cells at all rates of shear were seen in an irregular tumbling motion, rotating as units and colliding frequently.

**TOTALLY RIGIDIFIED CELLS**

**Flow Behavior of Individual Cells**

Red cells treated with gluteraldehyde showed the appearance of biconcave disks; suspended in plasma they were monodispersed without evidence of rouleau formation or clumping. In plasma-ghost suspension they were seen to tumble, orbit, and collide in an irregular fashion under shear. In 2% suspensions these cells could not be made to pass through 8.3-μ filter pores, neither could they be passed through 14-μ filters at 12% concentration, the filtration yielding but a few drops of clear plasma.

**Bulk Viscosity**

The viscometry of a 39% suspension of rigidified cells in plasma at any given shear rate never gave a constant shear stress value. In spite of a constant velocity gradient there
was a continuous increase in the shear stress and consequently in the viscosity (Fig. 6A). At a shear rate of 0.1 sec⁻¹, the apparent viscosity showed a more than 100-fold increase over a period of 30 minutes (which corresponds to three rotations of the inner cylinder of the viscometer) (Fig. 6B). The rise of shear stress was frequently not constant, but interrupted by erratic falls. Over short uninterrupted periods, the increase was observed to be an exponential one. When the rotation of the viscometer was stopped, the suspension mixed gently, and the measurement resumed at the same velocity gradient, the shear stress value was approximately equal to the value at the beginning of the original shear (Fig. 6A). If, however, the suspension was sheared for a period sufficient to allow a doubling of shear stress, then stopped without mixing, the shear stress at the resumption of shearing was identical to the value at the end of the previous shearing. In other words, the viscosity continued to rise from the value reached by the previous shearing experience when it was left to stand without mixing. The same behavior was seen if the rotational direction of shearing was reversed without stirring the suspension.

After gentle mixing, the shear stress values at time zero (t = 0) were fairly reproducible for any given shear rate and hematocrit. Upon each increase of the velocity gradient, the viscosity at the initiation of shearing was equal to or greater than the value obtained at the preceding lower value. Again, the viscosity values rose with continuing applied shear in the manner described above. At the intermediate range of shear rates, these time effects often made it difficult to determine a viscosity value at t = 0, and the values had to be taken arbitrarily when there appeared a plateau in the rising shear stress curve. The flow behavior of a representative sample of rigid cells in plasma is shown in Figure 7. The sample showed an increase in viscosity (shear thickening) both as a function of shear rate and shear time. Shear thickening was slight or absent in suspensions of 24% hematocrit, but was strongly exaggerated as soon as the concentrations were
A: Shear stress recording of rigid red cells in plasma (hematocrit 39 ± 1%) in a GDM viscometer at 37°C. Shear rate 0.1 sec⁻¹. Short stopping of viscometer leaves shear stress unaltered after resumption of rotation. Stopping and mixing of suspension reduces shear stress after resumption of rotation with subsequent increase in shear stress. B: Shear stress and viscosity diagram of four different samples at shear rate 0.1 sec⁻¹, hematocrit at 39 ± 1%, at 37°C.

Discussion

Red Cell Deformation and Viscous Resistance.—It has been established that the viscosity of blood is a function of temperature, plasma viscosity, hematocrit, and the degree of aggregation and dispersion of cells. The latter factors were believed to be the principal reason for the shear dependent viscosity of blood (1). The role of cell deformability has only recently been recognized (2, 4) and is emphasized by the present experiments that define the relationship between blood viscosity and cell deformability. In the presence of highly deformable erythrocytes, the viscosity of blood shows very pronounced shear thinning resulting in a comparatively low viscosity at high rates of shear. At shear rates in excess of 50 sec⁻¹ where the cells are disaggregated, the individual cells are seen to be deformed and a tank tread-like motion of the cell
Deformability of Erythrocytes.—The general laws governing erythrocyte deformability have recently been elaborated by Fung (14). He has shown that the red cell can be considered to be a flexible shell (the membrane) filled incompletely with a viscous, incompressible fluid (mostly hemoglobin solution). The incomplete filling, as an important consequence of the biconcave shape, allows deformation of the cell into an infinite variety of shapes without changes in either volume or surface area of the cell (isochoric deformation). The deformability can thus be limited either by the viscosity of the cell content or by increases of membrane tension (7). The bullet-shape deformation of the red cell in the narrow capillaries of the mammalian circulation (15) is the result of shear forces acting on the cell in different directions (16). The relative ease with which the red cells pass through filter pores smaller than their largest diameter must be related to a similar response (17).

The ability to be deformed in narrow pores is reduced under hyperosmolar conditions. Under the conditions of cell desiccation the viscosity of the hemoglobin rapidly increases from its low value at 32 g/100 ml (7). An intracellular crystallization of the hemoglobin in osmotically crenated red cells

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**Figure 7**

Influence of shear time and shear rate on apparent viscosity of rigid red cells 39% suspended in plasma. Note shear thickening as a function of shear rate and shear time.
TABLE 1

Apparent Viscosity of Rigid Red Cells in Plasma and Saline at Different Hematocrits

<table>
<thead>
<tr>
<th>Hematocrit</th>
<th>Plasma (cp)</th>
<th>Saline (cp)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.1</td>
<td>4.0 6.2</td>
<td>4.2 2.4</td>
</tr>
<tr>
<td>0.2</td>
<td>4.2 4.6</td>
<td>4.4 2.3</td>
</tr>
<tr>
<td>0.5</td>
<td>4.4 4.8</td>
<td>4.4 2.3</td>
</tr>
<tr>
<td>1.0</td>
<td>4.4 4.8</td>
<td>4.4 2.3</td>
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<tr>
<td>2.0</td>
<td>4.4 4.8</td>
<td>4.4 2.3</td>
</tr>
<tr>
<td>4.0</td>
<td>5.0 5.7</td>
<td>5.0 2.7</td>
</tr>
<tr>
<td>10.0</td>
<td>5.7 5.7</td>
<td>5.7 2.7</td>
</tr>
<tr>
<td>20.0</td>
<td>4.8 5.3</td>
<td>4.8 2.7</td>
</tr>
</tbody>
</table>

Hematocrit 24%

Hematocrit 30%

Hematocrit 40%

Hematocrit 48%

Viscosity values after mixing (t = 0) and after 5 minutes of shear (t = +5). cp = centipoise.

* Below range of instrument sensitivity.
† Above range of instrument sensitivity.

Flow rate fell, and cells were trapped with selective passage of less affected cells. The fluidity of packed cells, presumably reflecting individual cell fluidity, showed a similar reduction with rising osmolarity as also shown by Murphy (20). This conclusion seems to stand in contrast to that of Rand and Burton (19) drawn from studies of the membrane deformability; however, a greater membrane looseness of the osmotically deflated red cell may not necessarily reflect overall cell deformability.

Deformation of Red Cells in Laminar Shear.—In either rotational viscometers or in large tubes, the red cell is subjected to shear forces that act largely in one direction (parallel to flow direction). In such a uniform shear field, tensile and compressive forces act upon particles in alternate quadrants (13); the deformability of the particles determines its response to those forces. Rigid particles respond by orbiting, the frequency and velocity of which for any given shear stress is determined by shape, size, and interaction with other particles. Deformable particles as well as fluid drops (21, 22) show quite a different response: a transmission of shear stress across the particle interface is possible, subjecting the interior of the particle to a system of laminar shear.

In earlier experiments (7) in which individual red cells were suspended in concentrated suspensions of red cell ghosts to imitate the crowding conditions of packed cells, a tank-tread-like motion accompanied by continuous deformation of the red cells could be observed easily at low rates of shear. In the present studies, in which ghost and intact red cells were suspended in plasma to imitate the crowding conditions of normal blood, the deformation was seen only at high rates of shear. The accompanying elongation and orientation, as well as the absence of cell orbiting, have also been described by Goldsmith and Mason (23). Under similar experimental conditions they observed cell deformation in tube flow. The present microphotographs strikingly resemble his line drawings. The tank-tread motion of the membrane could have been postulated by Teitel-Bernard (18). This might further reduce viscous deformability of the cell notwithstanding greater membrane "looseness" as shown by Rand and Burton (19). In the present filtration studies, reduction of cell deformability with rising osmolarity was seen without exception; the
best be observed in flowing erythrocytes suspended in highly viscous suspending media in which carbon particles (filtered india ink) were used as tracers and were seen to rotate together with the membrane around the cell content (Schmid-Schönbein and Wells, unpublished observations). Under high rates of shear this response capability of the cell permits it to assume the dynamic properties of a fluid drop. The rheology of fluid drops, as elaborated by Taylor (24) as well as Rumscheidt and Mason (21, 22), has been shown to apply to red cells suspended in highly viscous media (25, 26).

Tank-tread deformation of the red cells greatly reduces the viscosity of blood, conceivably by several mechanisms. The most important effect is probably the participation of the cell content in flow and consequently a reduction of frictional energy dissipation between the laminar planes of shear. For any given velocity gradient, there is less shear in the continuous medium than in the case of non-deformable particles. Furthermore, the elongation and deformation of the cells brings their major axis parallel to the direction of flow. As all cells undergo a similar transition, the incidence of cell collisions is greatly reduced; in the case of collisions, the impact is less than in the case of solid particles.

All these responses of the normal cells are reduced after osmotic crenation. The cells suspended in concentrated ghost suspensions (7) or in 50% ghosts and 40% plasma showed orbiting and tumbling much like totally rigidified cells. With increasing osmolarity, an increasing number of erythrocytes lose their ability to undergo tank tread deformation; consequently, the suspension behaves more like one of rigid particles and viscosity rises. These results therefore explain the rise in high shear viscosity and capillary viscosity described unequivocally by many authors (27, 28, 29) to occur after osmotic crenation.

**Effects of Red Cell Rigidification.** —The hemorheological importance of erythrocyte deformability becomes even more conspicuous when comparing the viscous behavior of normal and totally rigidified red cells suspended in plasma. With increasing volume fraction of rigid particles shear takes place in lesser volumes of continuous phase. As a higher degree of frictional energy loss results for any given velocity gradient, the viscosity of the suspension rises. In the high concentrations studied here, particle interaction, collisions and motion of particles across the planes of shear are likely to further increase the viscous resistance.

The shear thickening seen in the rigid cell suspensions has not previously been recorded in the hemorheological literature. Shear thickening as a function of time (rheopexy) or shear rate (dilatancy) is not an unusual feature of concentrated suspensions of rigid particles, in which the particles' interaction becomes more and more prominent. According to Wilkinson (30) rheopexy is the consequence of a build-up of structure by shear. The erratic changes in shear stress observed with the present suspensions would suggest a continuous make and break of such structures, which are broken down by gentle mixing. This assumption would explain both the observation of a fairly reproducible shear stress value at the onset of viscometry and the increase in apparent viscosity with time. The rigidity of the red cells thus not only greatly increases apparent viscosity (4) but the dilatant behavior would preclude the flow of such suspensions through tapering tubes due to shear blockade (31). This illustrates that the deformation under flow is a critical requirement not only for the flow in capillaries but also for flow in the tapering tubes of arterial and arteriolar vessels at high rates of shear and normal hematocrit.

**Factors Responsible for the Anomalous Viscosity of Blood.** —The present experiments are primarily relevant to blood rheology at high rates of shear. Here, the viscous effect of cell deformability is most conspicuous and highly hematocrit dependent. As can be seen from Figure 4, the viscous effect of cell deformability is comparatively small at 25% and 30% hematocrit, and at shear rates around 1 sec⁻¹, it cannot be detected by bulk viscometry. At the physiological hematocrit
range of 45%, the viscosity is markedly affected by deformability at all rates of shear. The rheological variables governing blood flow at low rates of shear are cell aggregation and dispersion (1, 3, 12). The dynamics of cell aggregation and the effect of osmotic crenation upon it have been discussed in detail elsewhere (2). Those results on low shear viscosity and the present ones on high shear viscosity have shown that the non-Newtonian viscosity of blood is based on two distinct and separate mechanisms: the comparatively low viscosity at high velocity gradients is a consequence of red cell deformation, membrane rotation, and cell fluidity. With decreasing shear rate, less and less deformation takes place, and at shear rates below 50 sec⁻¹, the red cells group into the familiar rouleaux. With further reduction of the shear rate, these rouleaux form a secondary structure that greatly increases blood viscosity near stasis and is also responsible for the existence of a yield shear stress of blood.

In concluding, (1) The mechanisms responsible for the unusually low viscosity of blood at high rates of shear have been examined by a variety of methods. (2) These studies have revealed that normally deformable cells lower blood viscosity as the consequence of a tank tread-like rotation of the red cell membrane around the cell content which renders to the red cell features comparable to those of a fluid drop. (3) When this fluidity of the red cell is reduced or abolished, the viscosity of blood at high rates of shear and normal hematocrit is markedly elevated. (4) It thus becomes evident that the anomalous viscosity of blood is based on two separate mechanisms: (a) red cell deformation lowering viscous resistance at high rates of shear and (b) reversible red cell aggregation increasing blood viscosity with flow retardation and causing the yield shear stress of blood.

References


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