Physical basis of the dependence of blood viscosity on tube radius

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FAHRÆUS AND LINDQVIST (1) were the first to study the effect of the radius of the tube used in measurements of the apparent viscosity of blood. They found that the apparent viscosity at high flow rates is reduced in tubes of radius less than about 0.2 mm. A similar effect had been demonstrated previously by Bingham and Green (2) in paint, and it is now realized to be a general property of suspensions whose particles are the order of a micron or more in size. However, with special reference to the phenomenon in blood we shall call it the 'Fahraeus-Lindqvist effect.'

The theories discussed in this paper apply to tube radii about 25 μ and greater; however, in tubes of capillary dimensions the conditions for plug flow are present (since the red cell diameter and tube diameter are approximately the same) and the special problems that arise in this region will not be considered here.

Unfortunately, the basic mechanical problem of the behavior of a large number of colliding particles suspended in a liquid in steady flow through a cylindrical tube has not been solved, and indeed, it may well be insoluble even statistically (3, 4). Thus, the theories that have been proposed to account for the effect of tube radius on the viscosity of suspensions are phenomenological in character, and are based on qualitative models designed to approximate the effect of the particles in suspension. There appear to be only two such theories that can be used in the analysis of the Fahraeus-Lindqvist effect; one is based on the existence, in steady flow, of a marginal zone at the tube wall which is presumed to have a lower viscosity than the rest of the fluid, the other is based on the existence of unheated laminae in the fluid which arise from the presence of particles of finite size. Both of these theories can be used to account for the Fahraeus-Lindqvist effect in blood (5, 6); in each case the equations have essentially the same form and the difference arises primarily in the way a certain parameter is interpreted. Neither theory grossly offends one's physical intuition, and it seems probable that both postulated mechanisms are present, although it is difficult to decide their relative importance. From the experimental standpoint, Taylor (7) has made direct optical studies of a marginal zone that is, on the average, particle-free, and whose existence could be explained on the basis of the axial accumulation of the red cells (8, 9). On the other hand, there is no doubt that the red cells possess sufficient rigidity to resist the local shearing stress and thereby give rise to finite unheated laminae in steady flow.

One of the first marginal zone theories was proposed by Schofield and Scott Blair (10) who postulated the existence of a lubricating layer at the wall whose thickness was independent of tube radius and which had a lower viscosity than the fluid nearer the tube axis. In the analogous theory used in this paper, the marginal zone will be assumed to be a cell-free layer of the suspending fluid (or plasma) whose thickness is likewise independent of the tube radius. The other theory was proposed originally by Dix and Scott Blair (11) and is called the theory of the 'sigma phenomenon' because the existence of the unheated laminae implies that the flow must be calculated by a summation (∑) rather than an integration.

In this paper, both the marginal zone and sigma
phenomenon theories are applied to the experimental data of Haynes and Burton (19) and Kûmin (13). The mathematical derivation given for the sigma phenomenon equation and the technique used to determine the parameters in the theory are simpler than the methods originally proposed by Dix and Scott Blair. It should be noted that the theories in this paper apply only at high flow rates (i.e. at high shear rates and shear stresses), however, some slight evidence will be presented which suggests that the Fahraeus-Lindqvist effect either vanishes or reverses at very low flow rates. Finally, the existence of the Fahraeus-Lindqvist effect makes possible the calculation of the effective blood vessel diameter of a vascular bed, and although only an approximate value can be obtained with the available data, nevertheless, it does indicate that the vascular bed of the dog's hind leg can be replaced theoretically by a single equivalent vessel of arteriolar dimensions.

**PROCEDURE AND RESULTS**

**Sigma phenomenon.** In the derivation of Poiseuille's law, three integrations are performed. In the final integration, the volume rate of flow through the tube is calculated from the parabolic velocity profile. This implies that the velocity is a continuous, well-behaved function of the radial distance from the tube axis (denoted by \( r \)). This, in turn, is based on the assumption that the shearing laminae are infinitesimally thin, so that the methods of integral calculus are applicable to the problem. In the case of suspensions of particles of finite size, (i.e. particles whose size is not infinitesimal compared with the tube radius) this is an unsatisfactory assumption since there probably exist unsheared laminae whose thickness is at least of the order of the linear dimensions of the particles, and which could be greater if the concentration is such that the particles are effectively in continuous contact with one another. On this basis the total flow from the tube should not be calculated by an integration of the contributions from an infinite number of coaxial laminae, but rather by a summation of the contributions from a finite number of laminae of finite thickness. In this model the rate of shear across the laminae is assumed to be zero, while the rate of shear between the laminae is assumed to be infinite; thus the velocity profile becomes a 'staircase' approximation to a parabola rather than a smooth curve. The summation procedure for the sigma phenomenon will be referred to as the 'discontinuous' model, as opposed to the usual 'continuous' model. The transition from the discontinuous to the continuous case occurs in the limit \( b/R \rightarrow 0 \), where \( b \) is the thickness of the finite laminae and \( R \) is the tube radius. Thus, the equations and parameters that occur in the discontinuous model should be such that they reduce to the ordinary continuous results under this transformation.

In the continuous case, the general equilibrium condition in steady flow between the inertial force resulting from the applied stress, and the tangential stress resulting from the viscous drag between the shearing laminae, is given in the usual derivation of Poiseuille's law by,

\[
\frac{P r}{2} = \eta \frac{dr}{d\chi}
\]

where \( P \) is the applied pressure gradient, \( r \) is the radial distance from the tube axis, \( \eta \) is the coefficient of viscosity, and \( dr/d\chi \) is the velocity gradient (or rate of shear). The volume rate of flow through the tube, \( \dot{Q} \), is obtained by integrating across the velocity profile \( u(r) \), and after an integration by parts and substitution for \( dr/d\chi \) from equation 1, the flow can finally be written in the form,

\[
\dot{Q} = \frac{xP}{2\eta_\infty} \int_0^R r^2 \, dr
\]

where \( \eta_\infty \) denotes the apparent asymptotic viscosity (i.e. viscosity at high flow rates) in a tube of large radius. For blood and similar suspensions, the apparent viscosity depends on both the rate of shear and the tube radius except, of course, at high shear rates and in large tubes. Hence, the only coefficient of viscosity that could be considered to be independent of \( r \) and so taken outside the integral is \( \eta_\infty \). It should also be noted that it was not necessary to specify the particular form of the velocity profile and so this equation is valid for both non-Newtonian and Newtonian fluids.

To generalize equation 2 to the discontinuous case, we assume that the fluid flows in \( N \) concentric laminae of thickness \( \delta \), such that \( N\delta = R \) and \( n\delta = r \), where \( n = 1, 2, 3, \ldots N \). Thus, the integral in equation 2 should be written as the summation,

\[
\dot{Q} = \frac{xP}{2\eta_\infty} \sum_{n=1}^N (n\delta)^2 \delta
\]

This can be summed using the formula for the sum of the cubes of the first \( N \) integers, and then substituting \( N = R/\delta \) and simplifying, it becomes,

\[
\dot{Q} = \frac{xPR^2}{2\eta_\infty} \left(1 + b/R\right)^2
\]

Equation 4 is the generalization of Poiseuille's law for the discontinuous model proposed by Dix and Scott Blair. It reduces to Poiseuille's law as \( b/R \rightarrow 0 \), and this derivation clearly shows that the sigma phenomenon reflects the difference between the sum of a series of cubic terms and the integral of a cubic function. Thus, the efflux of a suspension from a tube such that \( b/R \) is not small compared with unity is greater than that predicted by a direct application of Poiseuille's law; and so, \( \eta_\infty(R) \), the apparent asymptotic viscosity of a tube of radius \( R \), is less than \( \eta_\infty \). Therefore, we have finally,

\[
\frac{1}{\eta_\infty(R)} = \frac{1}{\eta_\infty} \left(1 + b/R\right)^2
\]
BLOOD VISCOSITY IN NARROW TUBES

In order to apply this theory to experimental data it is necessary to evaluate the parameters \( \eta_0(\infty) \) and \( \delta \). This can be done most simply by linearizing equation 5; that is, by taking the square root of both sides, it is clear that a plot of \( [\eta_0(R)]^{-1/2} \) versus \( 1/R \) should yield a straight line from whose slope and intercept best-fit values of the parameters can be obtained by the method of least squares.

Marginal zone theory. In this model, one assumes the existence of a marginal zone of thickness \( \epsilon \) which is independent of the tube radius. The viscosity in the marginal zone is that of the suspending liquid, and will be denoted by \( \eta_s \). The viscosity of the suspension in the central region of the tube is assumed to be constant and equal to the apparent viscosity of the suspension as a whole in a tube of infinite radius, i.e. \( \eta_0(\infty) \). Thus, because \( \epsilon \) is independent of \( R \), the presence of the marginal zone has little effect in tubes of large diameter, but it reduces the apparent viscosity in tubes of small diameter. It can be shown by a direct application of Poiseuille’s law to the marginal and central zones that the total flow under such conditions in a tube of radius \( R \) is given by,

\[
Q(R) = \frac{\pi PR^4}{8\eta_s(\infty)} \left[ \frac{\eta_s(\infty)}{\eta_0} + \left( 1 - \frac{\eta_s(\infty)}{\eta_0} \right) \left( 1 - \frac{\epsilon}{R} \right) \right] \tag{6}
\]

and so the apparent viscosity, to the first order in \( 1/R \) is,

\[
\frac{1}{\eta_0(R)} = \frac{1}{\eta_0(\infty)} + \frac{\epsilon}{R} \left( \frac{\eta_s(\infty)}{\eta_0} - 1 \right) \tag{7}
\]

Equations 5 and 7 both have the same form to the first order in \( 1/R \) and so the experimental data can be explained to first order accuracy by either the sigma phenomenon or the marginal zone effect. The relation between \( \epsilon \) and \( \delta \) can be obtained by equating the coefficients of \( 1/R \) from each equation, viz.

\[
\epsilon(H) = \eta_0(H)/\sqrt{\eta_0(\infty) - \eta_s} \tag{8}
\]

The fact that \( \delta \) and \( \epsilon \) depend upon the total hematocrit \( H \) has been indicated in equation 8.

Application of theory to experimental data. It was shown in a previous paper (12) that the pressure-flow curves of human erythrocyte suspensions in standard acid-citrate-dextrose solution can be represented by an equation of the form,

\[
Q = MP - B(1 - e^{-kP}) \tag{9}
\]

where \( M \) is the slope of the linear segment of the pressure-flow curves, and is a function of both the hematocrit \( H \) and the tube radius \( R \); \( B \) is the nodal point on the negative flow axis and depends only on the tube radius; and \( k \) is an empirical parameter which characterizes the nonlinear portion of the curves and depends on both \( H \) and \( R \). The apparent asymptotic viscosity in a tube of radius \( R \) is therefore given by,

\[
\eta_0(R) = \frac{\pi H^*/BM}{(100H/R)} \tag{10}
\]

and is plotted in figure 1 for various hematocrits at 25.5°C. A similar curve calculated from the measurements of Kumin (13) on ox blood of 40% hematocrit at 38°C is shown in figure 2. The viscosity of the acid-citrate-dextrose solution, indicated by the horizontal line marked ACD in figure 1, was 1.093 centipoise; the viscosity of serum used in Kumin’s suspension was 1.30 centipoise.

The smooth curves drawn in figures 1 and 2 were obtained from equation 5, using the values of \( \delta \) and \( \eta_0(\infty) \) that were obtained from a least squares fit of the linearized data. The values of the parameters are given in table 1, together with the correlation coefficient for each hematocrit. The correlation for all hematocrits averages 0.97, except for \( H = 10\% \), which indicates a satisfactory agreement between the experimental data and an equation of the form of 5. The standard error of estimate is shown in figure 1 by the vertical bars on the asymptotes, and in figure 3, \( \eta_0(\infty) \) is plotted semilogarithmically.
TABLE 1. Summary of Calculated Parameters

<table>
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<tr>
<th>Hct</th>
<th>$\tau_m^{(*)}$</th>
<th>$\delta(H)$</th>
<th>$\epsilon(H)$</th>
<th>$R_{0}$</th>
<th>Marginal Flow at $R_{0}$</th>
<th>Corr. Coef</th>
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<td>16.0</td>
<td>-97</td>
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* Calculated from data of K"{u}min (13) for ox blood at 38°C.

As a function of hematocrit. The lack of scatter in this graph results from the exponential interpolation used to obtain particular values of $M$ from the experimental curves (12).

In figure 4 the best-fit values of $\delta(H)$ are plotted, and also the corresponding values of $\epsilon(H)$ as given by equation 6. The general features of these curves can be explained in terms of the theoretical models to which they apply. Thus, one would expect the effective value of $\delta$ to increase with hematocrit, since there would be a greater probability of unshaped cell clusters being formed, and these would tend to have both a larger size and longer 'life-time' as the number of cells per unit volume increased. On the other hand, axial accumulation is necessarily restricted as the cells become more closely packed, and so one would expect a corresponding decline in the marginal zone thickness with increasing hematocrit. Furthermore, it would appear from figure 4 that $\epsilon$ approaches an asymptotic value of 10 $\mu$ at high hematocrits, and this could be interpreted simply as a 'wall effect' or 'effective slippage zone.' The range of variations of $\delta$ and $\epsilon$ over the hematocrit range 10–80% differ considerably: $\delta$ changes by a factor of 10, whereas $\epsilon$ changes only by a factor of 4. The fact that the variation of $\epsilon$ is small over a wide range of hematocrits is undoubtedly a strong point in favor of the marginal zone hypothesis.

It is of interest to know the tube radius at which it might be said that the Fahraeus-Lindqvist effect first manifests itself. This requires the adoption of some arbitrary criterion for the onset of the effect. The radius at which the apparent viscosity is 10% less than the asymptotic value in large tubes seem to be quite suitable for this purpose, since such a viscosity change, while not large, is still not so small as to pass undetected or to be without physiological significance. This radius will be denoted by $R_{0}$, and it can be calculated from equation 5 which gives,

$$R_{0} = 8.48\delta(H)$$ (11)

so that the variation of $R_{0}$ with hematocrit is of the same form as $\delta(H)$. The values of $R_{0}$ are given in table 1, from which it is clear that for any hematocrit the effect can be considered to be virtually nonexistent in tubes whose diameter is 1 mm or more: for hematocrits about 40%, and taking into account the rule-of-thumb value ($R = 175 \mu$) suggested by Fahraeus (14) and the value obtained from K"{u}min's data, it appears that the effect first manifests itself in the range 0.5–0.2 mm diameter.

In view of the spread in the values of $\epsilon$ and $R_{0}$ over the hematocrit range, and between our values at $H = 40\%$ and those of K"{u}min, it is of interest to see if there is any physical quantity that is independent of hematocrit and the conditions of the measurements. One possibility that comes to mind is the fractional flow in the marginal zone. Thus, it seems not unreasonable to expect that the tube radius associated with the onset of the effect is such that the flow in the marginal zone is a constant fraction of the total flow in the tube. It can be shown that the fractional marginal flow is given by

$$Q(\epsilon)/Q(R_0) = [1 - (1 - \epsilon/R_0)^4] \tau_m(R_0) / \tau_m$$

(12)

where $Q(\epsilon)$ is the flow in the marginal zone. The values are given in table 1 and the hematocrit variation is plotted in figure 5, from which it is clear that the marginal flow in a tube of radius $R_0$ remains close to 13% of the total flow over quite a wide range of hematocrits (30–80%); furthermore, the same criterion holds reasonably well for K"{u}min's data despite the difference in the value of $R_{0}$ itself.

Effect of very low flow rates. So far in this paper only the dependence of the apparent viscosity on tube radius at infinite flow rates or shear stresses has been discussed. The question that now arises is whether or not the Fahraeus-Lindqvist effect persists at very low flow rates; in other words, how does the function behave as the shear stress $P/2$ approaches zero? It is difficult to give a confident answer to this question on account of the scatter in the data that is presently available; however, upon extrapolating to zero shear stress ($P/2 \rightarrow 0$), it would appear that there is a reversal of the Fahraeus-Lindqvist effect in the region of very low flow rates.

It can be shown from equation 9 that the apparent viscosity at any shear stress in a tube of radius $R$ is given by

$$1/\eta(R) = 1/\eta_m(R) - 8B(1 - e^{-k_p})/\pi PR^4$$

(13)

As the shear stress increases the second term approaches zero. The apparent viscosity at vanishing shear stress (i.e. $P \rightarrow 0$) is given by

$$1/\eta_m(R) - 1/\eta_m(R) - 8Bk_p/k_p R^4$$

(14)

The values of $\eta_m(R)$ for four tube radii and 40% hematocrit are plotted semilogarithmically in figure 6, together with the corresponding curve of $\eta_m(R)$ taken from figure 1. The plotted points are best-fit extrapolations of the data, that is, in calculating $\eta_m(R)$ from equa
tion 14, the values of $\eta(R)$ were read off the theoretical curve, and similarly the values of $B$ and $k$ were taken from the smooth curves that best fit the experimental data (12). It should also be remembered that in writing down equation 14 it is implicitly assumed that equation 9 is valid in the limit $P \to 0$. However, hearing in mind both the experimental and theoretical uncertainties, it would appear from figure 6 that at vanishingly small flow rates the apparent viscosity of blood rises in small tubes; that is to say, there is a reversal of the Fahraeus-Lindqvist effect.

The effect of hematocrit on the function $\eta(R)$ is analogous to its effect on $\eta_w(R)$: the higher the hematocrit, the greater is the viscosity increase in small tubes. At intermediate shear stresses (the order of 1-10 dynes/cm), the Fahraeus-Lindqvist effect is indeterminate; that is, there is no unique crossover value of $PR/2$ for which the viscosity is a constant independent of $R$, and so in this region the function $\eta(R)$ oscillates.

A more detailed mathematical discussion of this problem can be found elsewhere (15), and so for the purposes of this paper it is sufficient to note that the possibility of a reversal of the Fahraeus-Lindqvist effect can be predicted from the fact that the second term in equation 14 is negative; the extent of the reversal depends upon the way in which the exponential constant $k$ varies with tube radius.

Hematocrit dependence and the effective radius of a vascular bed. The apparent asymptotic viscosity can be plotted as a function of hematocrit for various tube radii; such a plot for infinite radius is shown in figure 3, which indicates that viscosity increases exponentially with hematocrit. Similar plots for the four tubes used in the original measurements, and a detailed discussion of the hematocrit dependence have already been published (12, 15), so that only a brief summary of this aspect of blood rheology will be given here.

The problem of accounting for the shape of the viscosity-hematocrit curves is just a particular case of the more general problem of the viscosity-concentration relations of suspensions, which has been reviewed recently by Frisch and Simha (10). This is a very difficult problem that has been solved only for extremely dilute suspensions. There are three concentration regions that are characterized by the relative importance of interparticle effects in each region. First, there is the extremely dilute suspension in which the apparent viscosity is made up additively from the contribution of each of the suspended particles, between which there is no interaction, so that each behaves as though none of the others were present. The second region is that of the dilute suspension in which the departure from additivity introduced by the hydrodynamic interactions between the particles can no longer be considered negligible. The third is the concentrated suspension in which the mutual hydrodynamic interactions of the suspended particles are of major importance. The first and second regions are separated by the so-called 'critical concentration' and a corresponding 'critical viscosity.' Because the property of additivity is associated with the first region, the relation between the apparent viscosity and concentration is linear; the critical concentration marks the change to a nonlinear relation that continues through the second and third regions. The value of the critical concentration is not uniquely defined, but rather depends on the shape and size of the particles; it also does not signal any marked discontinuity in the viscosity-concentration curve since the effect of the hydrodynamic interactions becomes only gradually apparent as the concentration is increased.

In the case of blood the critical concentration (hematocrit) that separates the first and second regions is about 15%; the third region of high concentration begins near the point of close packing of the cells, which corresponds approximately to a hematocrit of 60%. The viscosity-hematocrit curves of blood can be described qualitatively in much the same way as the general suspension considered above; in the first region below $H = 15\%$, the relation is almost linear, and it is nonlinear in the second and third regions. However, none of the concentration power series that have been discussed in the rheological literature are completely satisfactory for blood, whereas the data does follow an exponential relation quite well except for hematocrits less than 10%. Thus, for all practical purposes one can write for all hematocrits,

$$\eta_w(H) = \eta_0 e^{(\chi k)H}$$  \hspace{1cm} (15)

where the exponential constant $\chi$ depends on the tube radius. Such an exponential relation has been given a phenomenological interpretation by Richardson (17) in which it is assumed that, as the particle concentration increases, the fractional decrease in mean particle sepa-
ration is proportional to the fractional increase in apparent viscosity, from which equation 15 immediately follows. Richardson has also applied this relation to blood (10) using the data of Trevan (19); however, Trevan used a low velocity Ostwald viscosimeter for his measurements and did not obtain true asymptotic conditions.

The degree of agreement between the measurements

![Graph 1](image1)

![Graph 2](image2)

![Graph 3](image3)

**FIG. 4.** Parameters of the sigma phenomenon and marginal zone theories which are required to account for the Fahraeus-Lindqvist effect in erythrocyte suspensions at 25.5°C.

**FIG. 5.** Percentage of total flow that is in the marginal zone at the onset of the Fahraeus-Lindqvist effect.

**FIG. 6.** Reversal of the Fahraeus-Lindqvist as the flow rate or shear stress approaches zero. The lower curve for infinite shear stress is taken directly from fig. 1; upper curve was drawn by eye through the points.

**FIG. 7.** Linearization of $x(R)$ which facilitates the calculation of the effective diameter of vascular beds according to the method proposed in the text.
and an exponential relation can be seen in the semilogarithmic plots of $M(R, H)$ given in a previous paper (12). The dependence of the exponential constant $\chi$ on the tube radius is a direct manifestation of the Fahraeus-Lindqvist effect. It was found that $\chi$ increases hyperbolically with increasing radius, that is,

$$\chi(R) = \psi(1 - \alpha/R)$$

This equation can be linearized by plotting $\chi(R) \cdot R$ versus $R$ as shown in figure 7. Thus, $\psi$ is the slope of this line and $\alpha$ is the intercept on the $R$-axis. The values of $\psi$ and $\alpha$ obtained from figure 7 were $3.26$ and $22 \mu$, respectively.

Equation 16 immediately suggests a simple method for measuring the ‘effective diameter’ of a vascular bed, which could be used in a ‘single equivalent vessel’ theory for in vivo pressure-flow curves. Such an effective diameter can be obtained by using a given vascular bed as a viscosimeter in which the apparent viscosity of blood is measured at high flow rates for several hematocrits. This viscosity data could then be plotted semilogarithmically to get a value of $\chi$, and the effective diameter then obtained from the corresponding value of $R$ given by equation 16. Unfortunately, there is no set of completely consistent data that can be used in this way, but an approximate value can be obtained from the measurements of Whittaker and Winton (20) on the hind leg of the dog. Their data are exponential up to hematocrits of 55%, and over this range the value of $\chi$ was found to be $0.6 \pm 0.2$. This means that the effective diameter is $55 \pm 5 \mu$, which corresponds to that of a medium sized arteriole. This is in line with the view that the arterioles are the most important vessels insofar as the peripheral control of the circulation is concerned. Furthermore, it suggests that in vivo pressure-flow curves, such as those obtained by Girling (21), might be deduced theoretically by replacing the vascular bed by a single equivalent vessel, $55 \mu$ in diameter, under constant vasomotor tone and whose elastic diagram has the same shape as that of a small artery (A. C. Burton, personal communication).

### DISCUSSION

Two theoretical models have been invoked to account for the Fahraeus-Lindqvist effect at high shear rates, and the experimental data can be fitted with equal precision in each case since equations 5 and 7 both have the same form to the first order in $1/R$. The values of $\delta(H)$ and $\epsilon(H)$ plotted in figure 4 were obtained on the assumption that the sigma phenomenon and the marginal zone effect are mutually exclusive, and so one must choose either one explanation or the other. Unfortunately, each theory can be criticized as being unrealistic in some respects, but neither can be rejected outright. For example, it is hard to accept the large variation of $\delta$ with hematocrit; but at the same time, the sharp boundary of the marginal zone and the assumption that the core viscosity is independent of the core radius seem equally untenable. However, these difficulties can be resolved to some extent since the two theories are not in fact mutually exclusive, and it is possible that both effects are present simultaneously. Thus, the theories can be combined by assuming that the marginal zone is somewhat thinner than that shown in figure 4, and that the sigma phenomenon occurs in the core.

For example, if the marginal zone is assumed to be $15\%$ less than $\epsilon(H)$, the corresponding value of $\delta$ at 80% hematocrit is reduced to only $5.3 \mu$ as opposed to the original value of $34.2 \mu$. In this way, the Fahraeus-Lindqvist effect might be explained at all hematocrits by the presence of a cell-free marginal zone $1-5 \mu$ thick, and unsheared laminae in the core the thickness of which is the order of the red cell dimensions.

Although the evidence presented in this paper for the reversal of the Fahraeus-Lindqvist effect at low shear rates should not be considered conclusive, nevertheless, it is not difficult to explain such a reversal on the basis of the establishment of the marginal zone by axial accumulation. At vanishing shear rates, the marginal zone would not be present and red cells would be in intimate contact with the tube wall. This added friction between the cells and wall would contribute relatively more to the apparent viscosity in small tubes than in large tubes. Once the shear rate is great enough so that the marginal zone is well established, the flow of the suspension is “lubricated” by it, and the apparent viscosity then is less in small tubes. About the only other possible explanation for the reversal is based on the electrokinetic effect of flow retardation arising from the streaming potential. However, even if one assumes the most optimistic values for the streaming potential of blood in glass tubes, the effect is still about 1000 times too small to account for a reversal of the Fahraeus-Lindqvist effect.

In conclusion, it would appear that the Fahraeus-Lindqvist effect occurs in tubes less than about 0.4 mm in diameter. The effect can be explained on the basis of the sigma phenomenon or as arising from the presence of a cell-free marginal zone, or as some undetermined combination of these theories. If the marginal zone theory alone is used, then the marginal flow at the onset of the effect must be about $13\%$ of the total flow in the tube. In a combined theory, this marginal flow would be slightly less ($\sim 11\%$). It was possible to use the data to calculate the effective diameter of a mammalian vascular bed, which was found to lie in the range of arteriolar dimensions ($30-60 \mu$).

I am greatly indebted to Professor Alan C. Burton, of the Biophysics Department, University of Western Ontario, for his advice and encouragement throughout the course of this work. I also express my thanks to Dr. M. G. Taylor, now at the University of Sydney, Australia, for several useful discussions, and to Mr. J. E. Baptist, of this laboratory for his help with the calculations.
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