Optimal Hematocrit for Oxygenation of Canine Intestine

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SUMMARY. To determine the direct effects of hematocrit on intestinal oxygen consumption and to determine whether an optimal hematocrit exists for intestinal oxygenation, we perfused isolated canine gut loops at a constant pressure (120 mm Hg) and varied hematocrit from 80% to 10%. As hematocrit fell, blood flow rose while arterial oxygen content fell. The regression of blood flow on hematocrit was linear, whereas the relationship between oxygen uptake and hematocrit was parabolic, showing a maximal oxygen uptake at an hematocrit of 48.7%. To determine whether the optimal hematocrit for intestinal oxygenation could be altered by changes in vasomotor tone, we performed two other series of experiments. Raising perfusion pressure to 180 mm Hg did not significantly alter the optimal hematocrit for oxygen uptake. However, when we increased the oxygen demands of the gut by placing transportable solutes within the intestinal lumen, the optimal hematocrit for oxygen uptake increased markedly. We conclude that the optimal hematocrit for intestinal oxygenation is slightly higher than the normal range, a finding that could possibly be explained by the plasma skimming known to occur in the intestinal mucosa. Our experimental design and method of data analysis could be used to determine the optimal hematocrit in other organs. (Circ Res 51: 233–240, 1982)
the experiment, the packed red blood cells and 10,000 units of heparin were mixed with plasma and 5% dextran in saline to yield a whole blood with the desired hematocrit. The reservoir contained a sufficient quantity of blood to study the effect of changing hematocrit at a given perfusion pressure (see Experimental Protocols). The blood was equilibrated with air and 5% CO₂ until the hemoglobin was 95-100% saturated with oxygen. The dextran employed had approximately the same viscosity as blood plasma (Dawidson et al., 1980).

The air pressure in the arterial reservoir was controlled with a tank of compressed air containing 5% CO₂ and a conventional low-range air pressure regulator (model 10BLR, Bellofram). Thus, a perfusion pressure could be selected and maintained by adjusting the pressure regulator. Blood flow was measured with an extracorporeal probe (Zepeda Instruments) immediately distal to the pressure reservoir. The signal from the flow probe was processed by a Zepeda SWF-3RD electromagnetic flowmeter. Since the venous blood draining from the perfused gut segment simply collected in a beaker, the calibration of the flowmeter was easily established by collecting the venous effluent during each experiment. This recalibration was necessary because the sensitivity of the flowmeter was affected by the hematocrit alterations. Details of the surgical preparations and perfusion apparatus have appeared in previous reports (Shepherd et al., 1973; Shepherd, 1979, 1980).

To study the effect of hematocrit on intestinal oxygen consumption, it was desirable to determine when oxygen uptake reached a steady state following each change in hematocrit. This is most easily accomplished by registering the arteriovenous oxygen (A-V O₂) difference continuously as the arterial and venous blood flows through the cuvettes of a spectrophotometric A-V O₂ difference analyzer (Axon Systems, Inc.). Shepherd and Burgar (1977) have shown that the A-V O₂ analyzer is not affected by hematocrit changes. Thus, when our tracings indicated that both blood flow and A-V O₂ were in a steady state, we drew samples of arterial and venous blood anaerobically, placed them on ice in sealed syringes, and determined their oxygen content after having an hematocrit of 80, 60, 40, 20, and 10%. In the first control experiment (perfusion pressure = 120 mm Hg) have control experiment (perfusion pressure = 120 mm Hg). Thus, a perfusion pressure of 180 mm Hg should simply produce passive vascular distension without significantly altering the metabolic demand for oxygen.

To determine whether the optimal hematocrit for intestinal oxygenation was affected by metabolic vasodilation, we perfused a third group of six gut loops at 120 mm Hg and determined their basal oxygen consumption rate when the animal's own blood perfused the intestinal loop. After the basal oxygen uptake had been measured, we instilled a solution of the commercially available diet substitute, Vivenox, into the intestinal lumen. Earlier studies (Shepherd, 1979, 1980) have shown that Vivenox stimulates the intestinal absorption of glucose and water, and increases oxygen consumption. When the expected increase in oxygen uptake occurred and had reached a steady state, we began the hematocrit protocol previously described.

Experimental Protocols

To determine the effect of hematocrit on oxygen transport in the intestine, we perfused each gut loop with blood having an hematocrit of 80, 60, 40, 20, and 10%. In the first series of experiments, each gut loop was perfused at an arterial pressure of 120 mm Hg. Initially the hematocrit was 80%; this hematocrit was maintained until blood flow and arteriovenous oxygen difference reached a steady state. This usually required 2-4 minutes. Upon observing an unequivocal steady state, we pumped dextran into the arterial reservoir to reduce the hematocrit to the next desired level and waited until both blood flow and A-V O₂ reached a new steady state. Thus, the hematocrit-flow relationship was determined by lowering the hematocrit in the previously mentioned steps from 80% to 10%. The hematocrits were determined in arterial blood by centrifuging samples in glass capillary tubes for 3 minutes at 8000 rpm. No correction was made for trapped plasma. After the experiment, each gut loop was weighed so that the blood flow and oxygen consumption data could be expressed per 100 g of tissue. Normalizing the data in this manner facilitated comparing blood flow and oxygen consumption data obtained from gut loops of various sizes, but it did not influence the analysis of the optimal hematocrit described below.

The oxygen content of arterial and venous blood was determined as described previously and the oxygen content values from the Lex-O₂-Con technique were used to calculate (1) oxygen delivery as the product of blood flow and arterial oxygen content, (2) oxygen uptake as the product of blood flow and the arteriovenous oxygen difference, and (3) the oxygen extraction ratio as the arteriovenous oxygen difference divided by the arterial oxygen content.

In a second series of experiments, we studied the effect of increased perfusion pressure on the optimal hematocrit (described in Results). To do this, we perfused six preparations with arterial pressures of 180 mm Hg. Shepherd (1980) has shown that this perfusion pressure is slightly above the autoregulatory range and thus should lead to passive vascular distension because of the increased transmural pressures. Shepherd (1980) has also shown that the oxygen uptake vs. blood flow relationship is essentially flat between the blood flow values produced by perfusion pressures of 120 to 180 mm Hg. Thus, a perfusion pressure of 180 mm Hg should simply produce passive vascular distension without significantly altering the metabolic demand for oxygen.

Analysis of Data

In all experiments we calculated—for each hematocrit level—the oxygen transport parameters defined in Table 1. To determine the relationship between any one of these oxygen transport parameters and hematocrit, e.g., between oxygen delivery and hematocrit, we used a Hewlett-Packard model 9825A or an Apple II+ computer to determine, by the method of least squares, the equation that best fit the data from each experiment. The data were fit to eight different equations, including linear, exponential, logarithmic, and quadratic models (Warner, 1980). Statistical analyses, as noted in Results, included conventional Student's t-tests, analysis of variance, and Dunnett's test (Zar, 1974).

Figure 1 illustrates the methods we used to analyze the data from each experiment. In Figure 1, blood flow, arterial oxygen content, and oxygen delivery data from a single control experiment (perfusion pressure = 120 mm Hg) have been plotted as functions of hematocrit. In the control experiments, arterial oxygen content was linearly related to hematocrit. Several considerations lead to this conclusion. First, visual inspection of the graphs indicated that the oxygen content vs. hematocrit relationship was linear. Second, the values of the correlation coefficients were greater for a linear least-square fit than for any of the seven other equations to which the data were fitted. Third, in the case...
of arterial oxygen content (CaO₂), one would expect the relationship to be linear provided oxyhemoglobin saturation and mean corpuscular hemoglobin concentration are constant in the reservoir blood. Note that

\[ \text{CaO}_2 = b_1 H + a_1 \]  

(1)

where \( b_1 \) is a constant denoting the oxygen carrying capacity per unit hematocrit (H).

One would not necessarily expect the blood flow vs. hematocrit relationship to be linear. In fact, the relationship between hematocrit and flow has been shown to be exponential in rigid tubes (Haynes and Burton, 1959; Haynes, 1960). However, many physiological reasons prevent our knowing a priori the relationship between hematocrit and flow in a perfused organ. Such unpredictable physiological factors include a possible vasodilation or constriction secondary to oxygenation changes induced by the hematocrit alterations, the well-known nonlinear relationship between hematocrit and viscosity (Whittaker and Winton, 1933), and the Fahraeus-Lindqvist (1931) effect (Klitzman and Duling, 1979). Therefore, like earlier investigators (Smith and Crowell, 1963; Crowell and Smith, 1967) we fitted our blood flow data to straight lines and, as Figure 1 shows, found good correlation with a linear model. Thus, we described blood flow empirically as a function of hematocrit.

\[ F = -b_2 H + a_2. \]  

(2)

Note that \( b_2 \) is the slope of the flow vs. hematocrit regression while \( a_2 \) is the plasma flow at zero hematocrit.

If we define oxygen delivery (F-CaO₂) as the convective flux of oxygen, multiplying Equation 1 by Equation 2 and combining constants yields a quadratic equation in the form:

\[ F \cdot \text{CaO}_2 = a_3 H^2 + b_3 H + c. \]  

(3)

This equation describes a parabola as shown in Figure 1 and we note that maximal oxygen delivery occurs at the vertex of the parabola. Because the vertex has the abscissa \((-b_3/2a_3)\), the "optimal hematocrit for oxygen delivery" (Hopt) can be computed as

\[ H_{\text{opt}} = -b_3/2a_3. \]  

(4)

Thus, in each experiment, the blood flow and arterial oxygen content data were fit to straight lines while the oxygen delivery vs. hematocrit data were fit to a quadratic equation from which we computed the hematocrit at which maximal oxygen delivery occurred. In a similar manner, we also fitted the venous oxygen efflux and oxygen consumption data to quadratic equations from which we calculated the hematocrits at which maximal oxygen consumption occurred or at which the venous efflux of oxygen was maximal. This treatment of the data seemed justified since the regressions of venous oxygen content and arteriovenous oxygen difference on hematocrit appeared linear on visual inspection and because the venous efflux and oxygen uptake data were well correlated with hematocrit when fit to a quadratic equation.

Finally, we also analyzed the data using the Smith-Crowell equation. Smith and Crowell (1967) showed that if the regression of flow on hematocrit is known, the hematocrit at which the maximal red blood cell flux occurs can be computed as

\[ \text{Hopt} = -a_2/2b_2. \]  

(5)
where $b_2$ and $a_2$ are the slope and intercept of the linear flow vs. hematocrit regression (Eq. 2). The reader will note the similarity of Equations 4 and 5. The utility of the Smith-Crowell equation (Eq. 5) is that, although empirical, it predicts the hematocrit at which the RBC flux and therefore oxygen delivery should be maximal. Equation 5 requires only that the coefficients of the flow vs. hematocrit regression be known. By contrast, Equation 4 can be used when the actual oxygen uptake has been measured. Thus, with Equation 4, one can interpolate to the hematocrit at which maximal uptake occurred.

**Results**

To recapitulate the data analysis, in each experiment the oxygen content data (Fig. 2) were fitted to linear equations while the oxygen flux data (Figs. 1 and 3) were fitted to quadratic equations. Next, we calculated the hematocrits at which the venous oxygen efflux, oxygen delivery, and oxygen uptake were maximal. Finally, we used the Smith-Crowell equation and the linear flow vs. hematocrit regressions to compute the hematocrit at which the RBC flux was maximal. The optimal hematocrits calculated these four different ways are shown in Table 2. As Table 2 indicates, under control conditions, the optimal hematocrits computed from the oxygen flux data were not significantly different from the optimal hematocrit for RBC flux as calculated from the Smith-Crowell equation. Thus, the experimentally measured oxygen flux data confirm the utility of the Smith-Crowell equation for calculating the optimal hematocrit when only the flow vs. hematocrit regression is known. This conclusion, as discussed later, may be valid only under control conditions, since the apparently fortuitous agreement among these four methods for determining the optimal hematocrit was lost in both experimental conditions.

To present representative relationships graphically, we determined the mean regression equations for a particular group of experiments by averaging the coefficients from individual regression equations. Table 3 contains both the regression coefficients from individual experiments and the mean regression coefficients from which the figures were generated. Figure 2 shows curves computed from the mean regression equations for the control group (perfusion pressure = 120 mm Hg). As Figure 2 shows, the oxygen content vs. hematocrit lines intercept the oxygen content axis very near zero. This, we feel, also supports the validity of the analysis, since an intercept near zero would be expected if blood plasma (Hct=0) contained a negligible amount of dissolved oxygen.

Figure 3 shows the analysis of oxygen consumption measured in the control experiments. The mean regression equations were used to generate the curves for blood flow, arteriovenous oxygen difference, and their product, oxygen uptake. As Figure 3 indicates, the optimal hematocrit for oxygen uptake in the control group was approximately 48%.

Figure 4 illustrates the effects of hematocrit on oxygen delivery, on the venous efflux of oxygen, and on their difference, the oxygen uptake. In these control experiments, as mentioned earlier, the maxima in the three oxygen fluxes occurred at approximately the same hematocrit, e.g., 46–48%.

Figure 5 shows the effect of reducing the hematocrit on the oxygen extraction ratio. The oxygen extraction ratio remained relatively constant until the hematocrit reached 25%. Below an hematocrit of 25%, oxygen extraction fell more precipitously. A partial explanation for the near constancy of the oxygen extraction ratio is that, with both the numerator and the denominator falling, the extraction ratio shows only a slight decline over a wide hematocrit range, but because of the diminishing arterial oxygen content (or oxygen delivery), the amount of blood that had to be "cleared of oxygen" to maintain even a falling oxygen uptake increased sharply as hematocrit fell. Although the oxygen extraction ratio is generally a useful index for expressing oxygen transport, Figure 5 shows that the extraction ratio can be misleading in situations in which arterial oxygen content varies markedly.

In the second series of experiments, we studied the effects of vascular distension on the optimal hematocrit. Paired experiments have shown that increasing perfusion pressure to 180 mm Hg lowers vascular resistance by 13.1 ± 1.8% compared with resistance at 120 mm Hg (Shepherd, 1980). Therefore, in this series of experiments, perfusion pressure was held at 180 mm Hg. Table 4 (center column) shows that the optimal hematocrit based on the maximal RBC flux shifted to a higher value with the increased perfusion pressure, i.e., 52%. However, neither the optimal hematocrit computed from oxygen uptake data nor that based on oxygen delivery was significantly different from control.

The purpose of the third series of experiments was

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**Table 2**

Comparison of Optimal Hematocrit Determinations: Control Data

<table>
<thead>
<tr>
<th>O2 transport parameter</th>
<th>Maximal RBC flux</th>
<th>Maximal O2 delivery</th>
<th>Maximal O2 uptake</th>
<th>Maximal venous O2 efflux</th>
</tr>
</thead>
<tbody>
<tr>
<td>Method of computation</td>
<td>Smith-Crowell Eq.</td>
<td>Parabolic fit</td>
<td>Parabolic fit</td>
<td>Parabolic fit</td>
</tr>
<tr>
<td>Mean hematocrit ± ss</td>
<td>46.2 ± 1.9</td>
<td>46.4 ± 2.6</td>
<td>48.7 ± 1.5</td>
<td>46.0 ± 3.2</td>
</tr>
</tbody>
</table>

Control data from gut loops perfused at 120 mm Hg. None of the determinations of the optimal hematocrit are significantly different from that computed for the maximal red blood cell flux; N = 6.
to determine whether increasing the metabolic demand for oxygen alters the optimal hematocrit for oxygen transport. When Vivonex was instilled into the intestinal lumen, the oxygen consumption rate increased 26.6 ± 4.8% above the control level. Table 4 (right column) and Figure 6 show that the optimal hematocrit calculated using the oxygen uptake method increased significantly from 48.7 to 57.1% when the oxygen demands of the gut were stimulated with transportable intraluminal solutes. This shift in the optimal hematocrit was also reflected in the computation of an optimal hematocrit for oxygen delivery but not by the hematocrit for maximal RBC flux.

Discussion

Although the optimal hematocrit concept was proposed nearly 20 years ago, few investigators have accepted the challenge of determining the optimal hematocrit for oxygen transport, and still fewer have attempted to extend the concept to the organ level. Nevertheless, one investigator (Staub, 1981) claims "the concept of optimal hematocrit has great clinical relevance" and Fan et al. (1980) have shown that specific organ blood flows are affected in different ways as hematocrit changes. In that report, coronary and cerebral blood flows appeared to be more affected than flow to the abdominal organs when the hematocrit was changed. Therefore, better data regarding the effects of hematocrit on tissue oxygenation could not only provide greater insight into the mechanisms of oxygen transport, but also could have significant consequences in medical practice. For example, "pseudopolycythemia" patients with hematocrits in the 50–60% range suffer a 6-fold increase in mortality (Burge et al., 1975, as cited by Dintenfass, 1981), while repeated isovolumic hemodilution to an hematocrit of 31% is reported to cause partial or complete healing of intractable leg and arm ulcers in patients with severe arterial occlusive disease (Rieger et al., 1981, as cited by Staub, 1981). Explanations for these clinical observations are not available.

Several recent studies of the effects of hematocrit on oxygen transport at the organ level have failed to show an unmistakable optimal hematocrit at which either oxygen delivery or actual oxygen utilization was maximal. Most of these previous studies were

![Figure 2](http://circres.ahajournals.org/)

**Figure 2.** Effects of hematocrit on oxygen content. Curves generated from mean regression equations show linear relationships for arterial content, venous content, and arteriovenous oxygen difference as functions of hematocrit. Data from control group, arterial pressure = 120 mm Hg, n = 6. (CaO₂ = bH + a, b = 0.43 ± 0.01, a = -0.594 ± 0.021; CVO₂ = bH + a, b = 0.327 ± 0.023, a = -0.319 ± 0.050; CaO₂ - CVO₂ = bH + a, b = 0.102 ± 0.019, a = -0.208 ± 0.021).

![Figure 3](http://circres.ahajournals.org/)

**Figure 3.** Effect of hematocrit on intestinal oxygen uptake. Mean regression equations from control group were used to generate curves shown. Maximal oxygen uptake occurred at an hematocrit of 46–48%. Note that VO₂, as product of two linear equations, shows parabolic relationship with hematocrit. (F = bH + a, b = 1.019 ± 0.115, a = 93.7 ± 10.2; CaO₂ - CVO₂ = bH + a, b = 0.102 ± 0.019, a = 0.288 ± 0.017; VO₂ = cH² + bH + a, c = -0.0007 ± 0.0002, b = 0.0704 ± 0.0172, a = 0.151 ± 0.191).
FIGURE 5. Effect of hematocrit on intestinal $O_2$ extraction. Extraction ratio shows slight decline with hemodilution from 80 to 25% hematocrit. Below hematocrit of 25%, oxygen extraction ratio fell sharply. Compare with Figure 2; note both numerator ($CaO_2 - CvO_2$) and denominator ($CaO_2$) of extraction ratio declined during hemodilution.

FIGURE 4. Effect of hematocrit on oxygen delivery, venous $O_2$ efflux, and oxygen uptake. In the control group, the optimal hematocrit for $O_2$ delivery, venous efflux, and $O_2$ uptake was 46–48%. ($F$: $CaO_2 = ch^2 + bh + a$, $c = -0.0037 \pm 0.0009$, $b = 0.3532 \pm 0.0948$, $a = 0.3567 \pm 1.1287$; $F$: $CvO_2 = ch^2 + bh + a$, $c = -0.0029 \pm 0.0008$, $b = 0.2820 \pm 0.0799$, $a = -0.0553 \pm 0.0942$, $VO_2 = ch^2 + bh + a$, $c = -0.0007 \pm 0.0002$, $b = 0.0704 \pm 0.0172$, $a = 0.1509 \pm 0.1913$).

performed by producing whole-body hemodilution or hemoconcentration (Lautt, 1977; Jan and Chien, 1977; Fan et al., 1980). In our opinion, studies of whole-body hematocrit changes are of limited value in providing information about the direct effects of hematocrit on the oxygenation of individual organs. One reason is that whole-body anemia evokes chemoreceptor reflexes (Hatcher et al., 1978), causes vasodilation in skeletal muscle (Chapter et al., 1981), and may even increase the oxygen demands of skeletal muscle (Chapler et al., 1979). On the other hand, whole-body polycythemia evokes myocardial and cerebral vasodilation and splanchic vasoconstriction (Fan et al., 1980). In addition, polycythemia increases viscosity and increases cardiac work. The latter effect would probably interfere with the determination of an optimal hematocrit because the present study has shown that stimulating the metabolic rate can shift the optimal hematocrit, at least in the gut. Nevertheless, because of the paucity of data directly compa-

rable to ours (Gahtgens et al., 1979) and because of the apparent conflict between previous reports and the present study, we shall compare the present results from an isolated organ with the previous reports on whole-body hematocrit changes.

If the previous data are compared with ours, there are several possible explanations for the apparent absence of optimal hematocrits in the earlier studies. The first explanation is a difference in experimental designs. In each of our experiments, the arterial hematocrit was varied over a wide enough range to ensure that $O_2$ delivery and $O_2$ uptake data were obtained at hematocrits both above and below the optimum (assuming an optimum existed within the 10–80% range). In the study by Jan and Chien (1977), the whole-body hematocrit of dogs was increased in some animals and reduced in others. For practical reasons, hematocrits markedly above and below an optimal (if it existed) were not obtained in each experimental animal. Although the data of Jan and Chien show clearly that whole-body oxygen delivery fell sharply if hematocrit was above or below the "optimal range" of approximately 43–59%, their data give the impression that no optimal hematocrit existed for myocardial oxygen delivery. Myocardial oxygen delivery fell only when hematocrit was reduced below 20% or increased above 60%. Similarly, the reports of Fan et al. (1980) and Kuramoto et al. (1980) suggest that oxygen delivery to the myocardium and to the intestine was changed little by hematocrit manipulations unless the hematocrit fell below 30% or exceeded 60%. Because the experimental designs in these earlier studies did not provide for a sufficiently wide range of hematocrits in each experimental animal to yield a complete oxygen delivery vs. hematocrit relationship, pooled data had to be used to construct such a relationship. However, we feel that pooling the data from individual experiments obscures the optimal hematocrit.

Therefore, a second possible explanation for the absence of an unequivocal optimal hematocrit for oxygen delivery in the previously cited reports may lie in the method of data analysis. Indeed, when our control data are averaged, as shown in Figure 7, intestinal oxygen consumption appears to be relatively unaffected by hematocrit unless the hematocrit is reduced to below 20% or raised above 60%. However this is an apparent distortion of the data caused by the usual experiment-to-experiment variability in not only the absolute values of oxygen consumption (y-axis) but also variability in the optimal hematocrits (x-axis) for, as Figures 3 and 4 show, oxygen uptake fell sharply in individual experiments when hematocrit was changed appreciably from the optimal value. However, our method of analyzing the data can be applied only if, as in the present study, sufficient data are obtained both above and below the optimal hematocrit. Our oxygen delivery and $O_2$ utilization data, as explained in Methods, were analyzed by fitting the data from each experiment to a quadratic equation and by computing the hematocrit at which oxygen...
TABLE 4

Effect of Perfusion Pressure and Metabolic Rate on Optimal Hematocrits

<table>
<thead>
<tr>
<th>Experimental conditions</th>
<th>Optimal hematocrit based on RBC flux</th>
<th>Oxygen uptake</th>
<th>Oxygen delivery</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control (Pa = 120 mm Hg)</td>
<td>46.2 ± 1.9</td>
<td>48.7 ± 2.5</td>
<td>46.4 ± 2.6</td>
</tr>
<tr>
<td>Passive distension (Pa = 180 mm Hg)</td>
<td>52.3 ± 3.3*</td>
<td>49.4 ± 3.5 NS</td>
<td>54.2 ± 1.7 NS</td>
</tr>
<tr>
<td>Increased O₂ demand (Pa = 120 mm Hg)</td>
<td>47.8 ± 1.7 NS</td>
<td>57.1 ± 5.2*</td>
<td>68.4 ± 9.9* NS</td>
</tr>
</tbody>
</table>

Mean optimal hematocrits ± SE for three experimental conditions: perfusion pressure (Pa) = 120 mm Hg (n = 6), Pa = 180 mm Hg (n = 6), and increased metabolic rate at Pa = 120 mm Hg (n = 5). The two experimental groups were compared with the control group by analysis of variance and Dunnett’s test.

* Significance (P < 0.05).

delivery or oxygen consumption was maximal. This “parabolic interpolation” method clearly would not be applicable to individual experiments in which only data below the optimal hematocrit had been obtained. Nevertheless, in addition to identifying the optimal hematocrit for intestinal oxygenation, the present study also demonstrates an experimental design and a method for analyzing data that could be applied to other organs, such as the brain and myocardium.

Besides quantitating the optimal hematocrit for intestinal oxygenation, the present study also shows that raising perfusion pressure to 180 mm Hg failed to alter the hematocrit at which maximal oxygen uptake occurred. The data at Pa = 180 mm Hg also indicate that the optimal hematocrit computed from the Smith-Crowell equation (see maximal RBC flux in Table 4) does not necessarily predict the hematocrit at which the measured oxygen consumption is maximal, and we are therefore reminded that, until all factors that determine the relationship between hematocrit and blood flow are better understood, our empirical equations are likely to disagree with experimental results. Similarly, the present study also showed that the optimal hematocrit for intestinal oxygenation shifted to a higher range during a metabolic stimulation induced by placing transportable solutes within the intestinal lumen. Again, the optimal hematocrits calculated for the maximal RBC flux (47.8%) did not agree, under these conditions, with the optimal hematocrit at which oxygen delivery and the measured intestinal oxygen consumption were maximal (68.4 and 57.1%, respectively).

Several aspects of the present findings deserve further comment and, if we had them, explanations. One unexpected finding was that the optimal hematocrit for intestinal oxygen consumption was somewhat above the normal range, e.g., 48% vs. 40–45%. Much of the oxygen utilized by the small bowel is consumed in the mucosa (Granger et al., 1978). Therefore, two peculiarities of the mucosal circulation that affect oxygen transport could possibly account for the optimal hematocrit being slightly above the normal range. One is that a diffusive oxygen shunt from arteriole to venule is thought to exist because the countercurrent blood flow in the villus (Lundgren, 1967). Although the countercurrent

![Figure 6](http://circres.ahajournals.org/)

**Figure 6.** Effect of increased oxygen demand on optimal hematocrit. Transportable solutes in intestinal lumen increased oxygen uptake and shifted optimal hematocrit to higher value. In control group, n = 6; in group with increased metabolic rate, labeled Vivonex, n = 5. The hematocrits at which maximal oxygen delivery occurred were significantly different (P < 0.05) when compared by analysis of variance and Dunnett’s test.

![Figure 7](http://circres.ahajournals.org/)

**Figure 7.** Optimal hematocrit is obscured by using averaged data. Compare with sharp fall in oxygen delivery seen in single experiment shown in Figure 1. Data shown here are means ± se from control group.
oxygen shunt would reduce the convective oxygen delivery to the villus, the mucosal circulation is also known to suffer a plasma skimming (Jodal and Lundgren, 1970) that apparently results from the essentially perpendicular branching of submucosal vessels that supply the mucosa. Although speculative, these peculiarities of mucosal circulation could account for the optimal hematocrit in the gut being slightly higher than the normal range.

In summary, the present study demonstrates an experimental design and a method for analyzing data that could be used to determine the optimal hematocrit for the oxygenation of other organs. The optimal hematocrit for intestinal oxygenation is shown to be slightly higher than the normal range. Although the optimal hematocrit for intestinal oxygen consumption was unaffected by increased perfusion pressure, an upward shift in the optimal hematocrit occurred during metabolic stimulation. However, the mechanism responsible for this shift remains unknown.

This study was supported by Grant HL-23435 from the National Heart, Lung, and Blood Institute.

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Received November 30, 1981; accepted for publication May 5, 1982.

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INDEX TERMS: Blood flow · Mesenteric circulation · Metabolism · Oxygen transport · Viscosity
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Circ Res. 1982;51:233-240
doi: 10.1161/01.RES.51.2.233

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