Digital Vascular Resistance in Normal, Polycythemic and Hypertensive States

By Milton Mendlowitz, M.D.

The relationship between blood pressure and flow in the digital circulation has been found to be rectilinear. This makes it possible from a single pressure-flow determination to measure (1) digital vascular configuration volume index, (2) initial digital arterial caliber, (3) resistance in dyne second/cm.², (4) resistance in dynes and (5) resistance in dynes/cm.². The normal variations in these values and the deviations from the normal found in hypertension and polycythemia are presented.

Peripheral resistance has in the past been measured by dividing pressure by flow and multiplying this value by a constant. This determination includes the so-called viscosity factor and assumes that with unchanged resistance the relationship between pressure and flow with varying pressures is rectilinear. It has, however, been demonstrated by many workers that especially in small blood vessels this relationship may not be a straight line. It has also been found that apparent viscosity may not be a constant but may change with changes in velocity, in caliber of the perfused vessels and in the nature of the perfusate. It was our purpose to study the pressure-flow relationship in the digital circulation and to attempt separation of the various factors in the peripheral resistance in order to compare the abnormal with the normal circulation.

If the vascular bed of a dog's leg is perfused with Ringer's solution or plasma, the relationship between pressure and flow is rectilinear. If venous pressure is subtracted from arterial pressure in the calculations, a measure neglected by earlier workers, the straight line intercepts the pressure axis at or near zero.

Poiseuille's law is then applicable, the formula being

\[ Q = \frac{P \pi r^4}{8\eta} \] (1)

in which \( Q \) is the volume flow in cm.³/second, \( P \) the mean arterial pressure minus the venous pressure converted into dynes/cm.², \( r \) the radius of the tube in cm., \( l \) the length of the tube in cm. and \( \eta \) the viscosity in poises (dyne second/cm.²). If \( r, l, \) and \( \eta \) are fixed, the only variables are \( P \) and \( Q \) and the formula becomes the familiar \( Q = Pk \) in which \( k \) is a constant. This is clearly a rectilinear equation.

When blood is used as the perfusing fluid the relationship between \( P \) and \( Q \) has been interpreted by different workers to be rectilinear with a shifting intercept on the pressure axis, hyperbolic, approaching an asymptote, or logarithmic. There are several possible factors involved in this deviation from the poiseuillean straight line. Increasing pressure may distend blood vessels progressively or open new capillaries especially at low velocity rates. With maximum vasodilatation, however, the effect of pressure on the vessels becomes minimal. What is more, curves similar to those seen in vivo may be obtained by perfusing glass tubes of appropriate diameter. Changes in the physical elasticity or compressibility of the cells and plasma are too small to account for the effects observed at low pressures. The lower portion of the curve may, however, be affected by changes in alignment and distribution of cells and possibly by changes in their shapes at lower pressures as compared with higher pres-
sures. This is sometimes referred to as plasticity. The slope of the upper portion of the curve is modified at vascular resistances prevailing in the peripheral circulation by the Fähraeus phenomenon. This represents a decrease in apparent viscosity with decreasing tube caliber below a critical radius. This is caused by proportionate increase in the peripheral zone of plasma, which is less viscous than blood, the cells being crowded into the center of the stream. It is this effect which creates considerable discrepancy between viscosity determined in vivo as against in vitro observations. It has also been shown, however, that increasing vasocstriction in the hind limb of the dog increases apparent viscosity. Because of the simultaneously increased intercept of the major pressure-flow slope on the pressure axis, this increase in viscosity is greatest at very low rates of flow. At such low rates of flow, moreover, ischemia can cause capillary dilatation with variable changes in diffusion from capillaries to tissues which may modify pressure-flow curves by changing the red cell and plasma protein concentration of the blood. Such changes in diffusion are theoretically possible at higher flow rates but are probably unimportant in the terminal digit where most of the flow is through arteriovenous anastomoses.

Logarithmic formulas for the pressure-flow relationship have been fitted to empiric data and have been found to be $Q = aP^k$ or $Q = (P/m)^n$ in which $a$ and $k$ or $m$ and $n$ are constants. In such a formula the logarithmic function $k$ or $n$ is the plasticity factor and $a$ or $m$ the variables of Poiseuille's law, $r$, $l$ and $\eta$ together with the Fähraeus phenomenon factor. The exact relationship of these factors has not yet been worked out physically although Lamport has recently presented a logarithmic formula incorporating the factor of caliber. The constant, $k$, has been found to vary from 1.4 to 1.8 in animal perfusion experiments. It must be remembered, however, that these formulas are derived from data obtained by the perfusion of vascular beds in which the preponderance of flow is through capillaries. In the human digital circulation in which sympathetic tone is released the preponderance of flow is through arteriovenous anastomoses. It therefore becomes necessary to determine the character of the pressure-flow curve in the human digital circulation to see if any difference can be detected between such curves and those obtained in animals and also to see if these pressure-flow relationships can be used to analyze digital vascular resistance.

To this end, studies were made after stabilization of the circulation by indirect heating, on a normal subject, on a patient with severe polycythemia vera, and on several patients with hypertension. Pressure-flow relationships at various digital arterial pressures were ob-

Fig. 1. Arterial compression clamp
very little if at all with brachial artery compression. What is more such compression produced no change in the erythrocyte con-

centration and hence viscosity of the perfusing arterial blood.

One possible source of error was the subtraction of 6 mm. Hg from the mean arterial pressure as average venous pressure. With de-

creasing digital arterial pressure the digital venous pressure might well fall. What is more

![Graphs showing logarithmic pressure-flow relationships for different conditions: Normal, Polycythemia, Mild Essential Hypertension, Severe Essential Hypertension.](http://circ.ahajournals.org/doi/abs/10.1161/01.CIR.43.5.696)
sectional area than the arterial pressure which would create an error in estimating resistance in dynes (see below). These factors, however, appeared to be small enough to be negligible.

The validity of the technic for measuring blood flow was exhaustively established elsewhere. Repeated pressure-flow determinations at heart level after maximal vasodilatation by indirect heating were made on different days in the same subject. It had been demonstrated that in any given individual this relationship was nearly identical from day to day. Only those experiments were accepted in which the brachial pressure from day to day was stable and in which the digital pressures before and after the flow observations remained unchanged. That release of sympathetic nerve tone could be satisfactorily effected in the upper extremity by indirect heating was demonstrated by Arnott and Macfie.

When our results were plotted by the method of the least squares on logarithmic paper, using the formula, \( Q = aP^k \), \( k \) was found to vary from 0.8 to 1.2 and not from 1.4 to 1.8 as had been found in animal experiments. Typical graphs are presented in figure 2. It is clear that when \( k \) is 1 or nearly 1 we are dealing again with a linear rather than a logarithmic relationship. The reason for the discrepancy between our studies and animal perfusion experiments probably lies in the fact that in the fingertip flow through arteriovenous anastomoses predominates over that through capillaries.

The results of our observations in normal, polycythemic and hypertensive subjects were hence plotted on cartesian coördinates (fig. 3). Since the rectilinear fit was as good as the logarithmic, the least squares method was employed to determine straight line regressions. The intercepts of these lines hovered around zero. The deviations of the points from the pressure-flow lines were due to slight fluctuations in sympathetic nerve tone, to such errors as are inherent in the method, as well as to the fact that arithmetic and true mean pressures do not always coincide. The return of the pressure-flow ratio to normal in the bled polycythemic patient was nicely demonstrated. Consideration of probable error, however, indicated that the exact intercepts on the pressure axis could not be accurately determined from these lines, especially in hypertension, and could be considered to be 0 ± 10 mm. Hg.

If these observations are valid and the error involved in disregarding the intercepts on the pressure axis is sufficiently small, it becomes possible to apply Poiseuille’s law to the digital circulation. In formula (1), \( \eta \) represents the viscosity of the blood. Whittaker and Winton showed in animal perfusion experiments that normal intravascular blood viscosity is approximately 2.2 times that of Ringer’s solution at a temperature of 37 C. The changes in viscosity, moreover, with varying erythrocyte concentration demonstrated by these workers in animals were reproduced in the human digital circulation. The viscosity of water or Ringer’s solution at 37 C. is 0.0069; that of blood is 0.015 poises. There is an insignificant error involved here because the average temperature of the blood in the fingertip is 33 to 34 rather than 37 C. The increase in apparent viscosity with vasoconstriction described by Pappenheimer and Maes in animal perfusion experiments was due chiefly to the increasing intercept of the pressure-flow slope on the pressure axis. Since such a shift is minimal in the digital circulation, if it occurs at all, the apparent viscosity can fairly safely be assumed to be 2.2 times that of water or Ringer’s solution. It still remains necessary to demonstrate this relationship by perfusion experiments on amputated human extremities.

The only remaining unknowns in the equation are length and caliber. These two factors together represent the physical configuration of the digital blood vessels. Thus, transposing in equation (1)

\[
\frac{\pi r^4}{8l} = \frac{Qe}{P}
\]  

The vascular configuration volume index is represented by \( \frac{\pi r^4}{8l} \). Thus, in a typical calculation, if the arithmetic mean digital arterial pressure minus venous pressure is 52 mm. Hg and the flow 0.0047 cc./second/cm.² of skin, 5.2 cm. multiplied by 13.6 (specific gravity of mercury) and by 980 cm./second² (acceleration
FIG. 3. Cartesian pressure-flow relationships. A. Normal (M.M.): \( Q = 0.006846 + 0.003545P \). B. Polycythemia (M.K.): \( Q = -0.013384 + 0.003705P \). C. Mild essential hypertension (M.G.): \( Q = 0.00974 + 0.00307P \). D. Severe essential hypertension (A.B.): \( Q = 0.011805 + 0.001706P \).
of gravity) equals 69306 dynes/cm.\(^2\). \(\eta\) for normal blood is 0.015 dyne second/cm.\(^2\). Hence, the vascular configuration volume index is

\[
\frac{0.0047 \text{ cm.}^3 \cdot 0.015 \text{ dyne sec.}}{69306 \text{ dynes sec.}} \cdot \frac{\text{cm.}^2}{\text{cm.}^2} = 0.00000001010 \text{ cm.}^3
\]

or 1010 cubic microns. Calculations of this index based on 34 normal subjects, 25 cases of polycythemia. The data on some of these cases have already been published.\(^{15, 16}\)

Separation of the factor of length from that of caliber is impossible in an irregularly branching system. Such separation can, however, be effected theoretically as follows: If it is assumed that Poiseuille's law applies to the systemic circulation as a whole, the number of dynes of force at the sinuses of Valsalva would

### Table 1.—Dimensions and Resistance of the Digital Vascular Bed

<table>
<thead>
<tr>
<th>Determination</th>
<th>Normal Range</th>
<th>Essential Hypertensive Range</th>
<th>Malignant Hypertensive Range</th>
<th>Polycythemia (M.K.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Digital Vascular Volume Index/cm.(^3) of skin in cubic microns</td>
<td>1010-653</td>
<td>653–308</td>
<td>308–177</td>
<td>1177</td>
</tr>
<tr>
<td>Estimated length factor for one digital artery in cm. assuming average radius of 0.03 cm. and adjusting value to average size fingertip</td>
<td>55.6</td>
<td>55.6</td>
<td>55.6</td>
<td>55.6</td>
</tr>
<tr>
<td>Initial caliber of a single digital artery (in cm. of radius) adjusted to average size fingertip</td>
<td>0.031-0.028</td>
<td>0.028-0.023</td>
<td>0.023-0.020</td>
<td>0.033</td>
</tr>
<tr>
<td>Resistance in dyne sec./cm.(^4) of a vascular bed perfused by a single digital artery (adjusting value to average size fingertip)</td>
<td>2,287,000-3,420,800</td>
<td>3,420,800-7,219,300</td>
<td>7,219,300-12,568,700</td>
<td>3,275,200</td>
</tr>
<tr>
<td>Resistance in dynes in a vascular bed perfused by a single digital artery (adjusting value to average size fingertip)</td>
<td>235-301</td>
<td>301-406</td>
<td>286-357</td>
<td>320</td>
</tr>
<tr>
<td>Resistance in dynes/cm.(^2) of cross-sectional area in a vascular bed perfused by a single digital artery (adjusting value to average size fingertip)</td>
<td>69,306-111,955</td>
<td>111,955-211,915</td>
<td>203,918-226,576</td>
<td>95,962</td>
</tr>
</tbody>
</table>

### Essential Hypertension

- Normal Range: 1010-653
- Malignant Hypertensive Range: 308-177
- Hematocrit 72%
- Postphlebotomy Hematocrit 44%

### Malignant Hypertensive

- Normal Range: 1010-653
- Malignant Hypertensive Range: 308-177
- Hematocrit 72%
- Postphlebotomy Hematocrit 44%

### Polycythemia (M.K.)

- Normal Range: 1010-653
- Malignant Hypertensive Range: 308-177
- Hematocrit 72%
- Postphlebotomy Hematocrit 44%

**Note:** The plasticity factor modifies Poiseuille's law in the systemic circulation and the figures are inaccurate to that extent.\(^{3}\)
cm.\(^2\). If the cross sectional area as estimated roentgenographically is approximately 13 cm.\(^2\), at a flow of 80 cm.\(^3\)/second and a mean pressure of 100 mm. Hg, a theoretic length factor can be calculated from equation (1) to be about 7 kilometers. This means that if the aorta were a uniform bore tube 7 kilometers long it would offer the same resistance as is offered by the entire systemic arterial tree, provided the viscosity of the blood were kept unchanged. It is also very probable that this length factor does not change much for any given vascular bed. Hence, if a length factor is established for a vascular bed in this way, the initial cross sectional area can be calculated back from the formula and its normal range of variation estimated.

The same process may be applied to the two digital arteries where Poiseuille's law does apply. Roentgenograms of these arteries in preliminary observations reveal an approximate initial diameter after vasodilatation of 0.06 cm. each as they enter the terminal segment of the finger, and the length factor in each entire digital circulation can be calculated to be approximately 55.6 cm. This means that if each digital artery were a uniform horizontal tube 55.6 cm. long it would offer a resistance comparable to that offered by the entire digital vascular bed supplied by that artery, provided the apparent viscosity remained unchanged. Both digital arteries are assumed to be equal in caliber and since caliber varies with the surface area perfused, a size factor, average surface/actual surface, has been incorporated in the calculation. The variations in caliber presented in table 1 may be modified when more extensive radiographic data on digital arteries in the vasodilated state become available.

To calculate resistance, formula (1) may be rearranged as follows:

\[
\frac{P}{Q} = \frac{8\eta \ell}{\pi r^4} \quad (3)
\]

Since \( P \) is expressed in dynes/cm.\(^2\) and 1 \( Q \) in cm.\(^3\)/second, \( P/Q \) is expressed in dyne second/cm.\(^3\). This method was introduced by Böger and Weizler\(^16\) and is commonly used to measure peripheral resistance. When such resistance is calculated for the human systemic circulation as a whole\(^18\) it is 539 to 1130 dyne second/cm.\(^2\). For the rabbit it varies from 11,670 to 12,590\(^18\) and for the vascular bed of a single digital artery in the normal fingertip from 2,287,000 to 3,420,800 dyne second/cm.\(^3\). The normal and abnormal variations in digital vascular resistance calculated in this way are presented in table 1. Superficially it seems incredible that the resistance of systemic vessels in a rabbit, or in a fingertip, should be so much greater than the resistance of the entire human systemic circulation unless one remembers that the expression dyne second/cm.\(^3\) measures resistance in dynes per unit of blood flow and per unit of cross sectional area. It is therefore relative to the actual blood flow and actual cross sectional area.

The resistance in dynes can only be measured by multiplying the resistance in dyne second/cm.\(^3\) by the actual blood flow and cross sectional area. Thus if equation (3) is converted into physical units,

\[
\frac{\text{dynes/cm.}^2}{\text{cm.}^3/\text{second}} = \frac{\text{cm.}^3 \cdot \text{dyne-second/cm.}^2}{\text{cm.}^3}
\]

or

\[
\frac{\text{dyne-second/cm.}^3}{\text{cm.}^3} = \frac{\text{dyne-second/cm.}^3}{\text{cm.}^3}
\]

Multiplying both sides of the equation by the actual \( Q \) in cm.\(^3\)/second and the actual cross sectional area in cm.\(^2\), we have

\[
\frac{\text{dyne-second/cm.}^3}{\text{cm.}^3} \cdot \frac{\text{cm.}^3}{\text{second}} \cdot \frac{\text{cm.}^3}{\text{second}} = \frac{\text{dyne-second/cm.}^3}{\text{cm.}^3} \cdot \frac{\text{cm.}^3}{\text{second}} \cdot \frac{\text{cm.}^3}{\text{cm.}^3}
\]

or

\[\text{dynes} = \text{dynes}\]

Thus the force in dynes exerted by the pressure acting over the initial cross sectional area is equal to the resistance in dynes provided the flow is constant. In a system in dynamic equilibrium force and opposing resistance in dynes must be exactly equal. Such resistance is the resultant of the frictional forces in the blood stream acting in a direction opposite to that of pressure. To calculate force and hence resistance in dynes, it can be seen from equation (4) that the pressure in dynes/cm.\(^2\) may simply be multiplied by the calculated initial digital arterial cross sectional area in cm.\(^2\), the flow factor being cancelled.
It is to be noted that resistance in dynes varies directly with pressure and directly with cross sectional area. Since the cross sectional area determines flow if the other factors remain constant, resistance in dynes varies directly rather than inversely with flow. The digital vascular resistance in dynes (see table 1) now becomes an intelligible proportion of the total systemic resistance in dynes, which can be calculated from the total systemic force at the sinuses of Valsalva to be approximately 1,732,640 dynes.* Such resistance is proportionately much higher per gram of fingertip than per gram of average tissue because of the greatly increased flow in the fingertip made possible by the arteriovenous anastomoses.

To complete the circle, resistance and force in dynes can each be divided by the actual cross sectional area to give

\[
\frac{\text{dynes}}{\text{cm}^2} = \frac{\text{dynes}}{\text{cm}^2}
\]

It will be recognized that in this sense pressure per unit of cross sectional area is exactly equal to resistance per unit of cross sectional area.

It can be seen from table 1 that vascular configuration volume index and caliber are decreased and resistance is increased in established hypertension. Also, a critical point in vascular configuration volume index, caliber and resistance in dyne second/cm.5 can be found, beyond which the cases of malignant hypertension fall. Resistance in dynes is more variable in malignant hypertension because of the variable decrease in flow and caliber. Resistance in dynes/cm.2 of cross sectional area is also more variable in malignant hypertension. In polycythemia on the other hand, if the viscosity factor is corrected in accordance with erythrocyte concentration16 the vascular configuration volume index and caliber are approximately normal or slightly above normal despite moderately increased resistance. After phlebotomy all these values become normal.

**SUMMARY AND CONCLUSIONS**

1. The pressure-flow relationship in the dilated digital circulation at different levels of digital arterial pressure was found to be rectilinear in contrast to the logarithmic curvilinear relationship found by investigators perfusing the dog's leg. This difference is believed to be attributable to the fact that in the digit, after sympathetic nerve tone is released, the preponderance of flow is through arteriovenous anastomoses rather than capillaries.

2. By the application of Poiseuille’s law to these data it is possible to calculate (a) digital vascular volume index in cubic microns, (b) initial digital vascular caliber in cm. of radius, (c) digital vascular resistance in dyne second/cm.5, (d) in dynes and (e) in dynes/cm.2.

3. The normal digital vascular volume index was found to vary from 1010 to 653 cubic microns, the digital vascular caliber from 0.031 to 0.028 cm. of radius, the digital vascular resistance in dyne second/cm.5 from 2,287,000 to 3,420,800, the digital vascular resistance in dynes from 235 to 301, and in dynes/cm.2 from 69,306 to 111,955.

4. In polycythemia the digital vascular volume index and caliber were normal or slightly above normal and remained normal after phlebotomy. The digital vascular resistance was either above or within normal limits but decreased to normal after phlebotomy.

5. In established essential hypertension the digital vascular volume index varied from 653 to 308 cubic microns. The digital vascular caliber varied from 0.028 to 0.023 cm. of radius, the digital vascular resistance in dyne second/cm.5 from 3,420,800 to 7,219,300, the digital vascular resistance in dynes from 301 to 406 and in dynes/cm.2 from 111,955 to 211,915. In the malignant phase of hypertension the digital vascular volume index was always below 308 cubic microns, the digital vascular caliber always below 0.023 cm. of radius and the digital vascular resistance in dyne second/cm.5 always above 7,219,300. The resistance in dynes varied from 286 to 357 and in dynes/cm.2 from 203,918 to 226,576.

**Acknowledgments**

The author is indebted to S. Feitelberg, M.D., and W. M. Nelson, Ph.D. (Engineering), for advice and to Miss J. Shapiro, Miss M. Spencer, Miss S. Lichtenberg, and Mr. R. E. Schwartz for technical assistance.
REFERENCES


Digital Vascular Resistance in Normal, Polycythemic and Hypertensive States

MILTON MENDLOWITZ

Circulation. 1951;3:694-702
doi: 10.1161/01.CIR.3.5.694

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1951 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/3/5/694

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/