On the Physical Equilibrium of Small Blood Vessels

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Physiologists have paid a great deal of attention to the forces that govern the passage of water, electrolytes and protein across the wall of the capillaries and other small blood vessels; much less to those which hold in equilibrium the wall of the vessel itself, and which determine what diameter it will hold under given conditions of blood pressure and vascular 'tone.' It is true that it has been recognized in hemodynamics that the distensibility of the small vessels may play an important role in determining how much the resistance to flow will passively change with the pressure, and elaborate formulas have been suggested to introduce this factor (1). Such investigations start by assuming a value for the distensibility often based on data obtained only on vessels of very much larger size, e.g. aorta or vena cava, without having made any fundamental analysis of the equilibrium of small vessels, and of what type the equilibrium must necessarily be. Even the theoretical mathematical analyses of Raschevsky in hemodynamics (2) lack emphasis on this fundamental approach. Yet the basic physical laws concerned are classical. The only application of them to a vascular problem we have found is that of Woods (3) in 1892, who applied the law of Laplace to the heart.

Forces Concerned

The blood vessels are to be considered as cylinders, open at both ends and filled with a fluid whose hydrostatic pressure is greater than that existing outside the wall of the vessels (tissue pressure). Normally we consider the latter pressure to be zero or nearly so, while the pressure inside the cylinder is the blood pressure pertaining to that type of blood vessel, i.e. the arterial pressure diminished by the gradient due to the flow down the resistance to flow before the blood stream reaches that point. Figure 1 shows the two forces that are in equilibrium in the wall of the blood vessel. The hydrostatic pressure acts everywhere at right angles to the wall, tending further to distend the vessel and increase its diameter. This is opposed by the tension in the wall of the vessel, tangential at every point, tending to diminish the diameter of the vessel. This tension may be reckoned in dynes per cm. length of vessel. If a longitudinal slit were made through the vessel wall, the two edges of this slit would be pulled apart with this force for each cm. length of slit.

The classical law of Laplace, introduced in general form in his famous theory of capillarity, states that for these two forces to be in equilibrium in the case of a cylinder, the relation must be:

\[ P = \frac{T}{R} \]  

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where \( P \) is the excess of hydrostatic pressure, inside the vessel over outside, in dynes per cm.\(^2\), \( T \) is the tension in the wall in dynes per cm. length, and \( R \) is the radius of the cylinder in cm. This law must govern the equilibrium of the wall of all cylindrical blood vessels.

**Total Tension in the Wall and Its Components**

The total tension in the wall obviously may consist of several components. For convenience we may classify these as three types: 

**a) Elastic tension**, due to stretch of the vessel wall and the elastic fibers and other tissues capable of resisting stretch by tension. The elastic tension will be a function (a linear one if Hooke’s law applies) of the ‘elongation,’ i.e. of the circumference of the vessel minus the ‘unstretched’ circumference. It will primarily be due to elastic fibers but all other tissues possess elasticity, i.e. resist stretch by developing tension, to varying degrees.

**b) Active tension**, due to contraction of smooth muscle fibers in the wall. This part of the tension will be a function of the vasomotor tone, due to nervous impulses or depending on the presence of pressor and dilator substances in the blood stream which have diffused to the smooth muscle. In that the force of contraction of muscle is affected by its initial length, there is also a dependence, as with the elastic tension, on the elongation, but for convenience we may group this part of the tension exerted by smooth muscle under ‘elastic tension’ rather than as ‘active tension,’ and consider that ‘active tension’ is independent of stretch.

**c) Interfacial tension**, due to a possible ‘surface tension’ between the fluid in the vessel and the wall, which will be present unless the wall is completely ‘wettable’ by the fluid. This type of tension (surface tension) is what maintains a flowing jet of water from a tap in the cylindrical shape although the pressure within it is greater than outside. Since there is much evidence that suggests that normal blood vessel possess a degree of unwettability by the blood plasma (4) we cannot neglect to include this as a possible component contributing to the total tension.

**Total Tension in Various Mammalian Vessels**

If the diameter of a blood vessel and the hydrostatic pressure within it are known, the total tension in the wall is at once given by Laplace’s equation, as \( T = P \times R \). As we pass down the vascular tree from the aorta to the smallest vessels (the capillaries) the radius changes by a factor of 10,000 times. The mean pressure changes by a factor of only about four times. The size factor, \( R \) in the equation, is therefore greatly predominant in determining the magnitude of the total maintenance tension in the wall.

Table 1 shows that in spite of the decrease in pressure as we pass from capillaries
to the veins, the tension required in the wall rises from a minimum to values for the
great veins comparable to that in large arteries, because their radius increases so
greatly. As the final columns show, the maintenance tension is well correlated with
the presence and amount of elastic fibers. The usual explanation for the reappearance
of elastic fibers in the venules and veins was that they were needed to withstand
pressures from outside structures. This was unconvincing, and the application of
Laplace's law gives a much more satisfactory reason, if we consider that the provision
of elastic fibers and elastic tension is the most efficient way of providing a 'main-
tenance tension' to hold the wall of the vessels against the prevailing hydrostatic
pressure of the blood without any continuous expenditure of energy. A second im-
portant function of elastic tissue will emerge in a later section of this paper.

**Table 1. Application of the pressure-curvature-tension relation to the
blood vessels (T = P × R)**

<table>
<thead>
<tr>
<th>TYPE OF VESSEL</th>
<th>MEAN PRESSURE WITHIN</th>
<th>RADIUS R</th>
<th>TENSION T IN WALL</th>
<th>AMOUNT OF ELASTIC TISSUE</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>mm. Hg</td>
<td>dynes/cm.</td>
<td>dynes/cm.</td>
<td></td>
</tr>
<tr>
<td>Aorta and large</td>
<td>100</td>
<td>1.3 × 10^6</td>
<td>1.3 cm. down</td>
<td>170,000</td>
</tr>
<tr>
<td>arteries</td>
<td></td>
<td></td>
<td></td>
<td>Very elastic, two coats</td>
</tr>
<tr>
<td>Small distributary</td>
<td>90</td>
<td>1.2 × 10^6</td>
<td>0.5 cm.</td>
<td>60,000</td>
</tr>
<tr>
<td>arteries</td>
<td></td>
<td></td>
<td></td>
<td>Much elastic tissue</td>
</tr>
<tr>
<td>Arterioles</td>
<td>60</td>
<td>8 × 10^4</td>
<td>0.15 mm. – 62 µ</td>
<td>1,200–500</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Thin elastica intima only</td>
</tr>
<tr>
<td>Capillaries</td>
<td>30</td>
<td>4 × 10^4</td>
<td>4 µ</td>
<td>16</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>None</td>
</tr>
<tr>
<td>Venules</td>
<td>20</td>
<td>2.6 × 10^4</td>
<td>10 µ</td>
<td>26</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>None except in largest</td>
</tr>
<tr>
<td>Veins</td>
<td>15</td>
<td>2 × 10^4</td>
<td>200 µ up</td>
<td>400</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Elastic fibers reappear</td>
</tr>
<tr>
<td>Vena cava</td>
<td>10</td>
<td>1.3 × 10^4</td>
<td>1.6 cm.</td>
<td>21,000</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Very elastic increasing</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>with size.</td>
</tr>
</tbody>
</table>

Table 1 also shows how very small a total tension is required in the wall of
capillaries to withstand the prevailing blood pressure there. The breaking strength of
a strip of lens paper or 'kleenex' tissue, one cm. wide, will be found to be about 50
gm. weight, or 50,000 dynes per cm., which is over 3000 times as great as the main-
tenance tension required in a capillary wall. It has been astonishing to some that
a structure apparently so fragile as the capillary wall could withstand pressures up
to the arterial pressure (as occur in any occlusion experiment on the human arm)
without rupture. We see that, entirely because of the small size of the capillary, no
great strength is actually needed. The engineer uses this principle in high-pressure
lines; if they be made of tubing of small enough diameter they will not burst even
if the metal is comparatively thin.

**Elastic Diagram for a Blood Vessel**

From data on the volume contained in blood vessels at different pressures, the
tension in the wall at different pressure can readily be calculated by Laplace's law
\( T = P \times R \). A plot of the tension as ordinate against the radius (or the circumference) of the vessel gives an ‘elastic diagram’ of classical form. There is much data on pressure-volume relations for the large vessels of animals and some for the aorta in man taken at autopsy \((5, 6)\). It has been shown that the difference between results on vessels \textit{in vivo} and the same vessels after death is not very great, and there is general agreement with distensibilities deduced in man from data on pulse-wave velocity \((7, 8)\). Figure 2a reproduces the well known results for the aorta and for the vena cava as usually presented. In figure 2b the same data are transformed to give the elastic diagrams of these vessels. These show that the elasticity of the wall does not follow Hooke’s Law, which would give a straight line, except for small amounts of stretch. For greater stretch the curve becomes steeper (steadily in the case of aorta but quite abruptly for the vena cava) indicating that the elastic constant has increased. The shape of the curve may be considered to be due to the composite character of the wall; the elastic fibers giving the initial linear part of the curve where Hooke’s law approximately applies, and restricting ‘wall’ or ‘jacket’ of fibrous tissue limiting the distensibility when the distension becomes great.

The use of the elastic diagrams also greatly illuminates the well known results of Halloch and Benson \((6)\) on the change in the elasticity of the aorta with age (fig. 3a). The curves for different ages cross each other in a way very difficult to
explain in terms of the changes seen by the pathologist, namely a progressive destruction of elastic fibers and simultaneous increase in fibrosis. The elastic diagrams deduced from the same data (fig. 3b) however are easily interpretable on this basis. With increasing age the initial slope, associated with the elastic fibers, decreases as it would if these fibers were weakened, while the relative rigid 'wall' due to fibrous tissue becomes dominant at smaller degrees of stretch. Unfortunately comparable data for the smaller vessels is completely lacking and we can only assume that the elastic diagram for these would be of similar shape.

**Equilibrium Under Elastic Tension Alone**

The reverse process to that by which the elastic diagram was deduced may be followed. From the elastic diagram the change in diameter of the vessel with change of pressure (when elastic tension only is present) may be deduced very easily. To do this the total tension required by the law of Laplace is plotted on the same diagram. It is a straight line through the origin \( T = P \times R \), the slope of which is determined by the pressure \( P \) (fig. 4). The intersection of this straight line with the curve for the elastic tension gives the point of equilibrium for the vessel under the given pressure. If the pressure be lowered the new straight line is less steep and the intersection occurs at a smaller value for the radius \( D \), fig. 4).

A point of equilibrium may not always be stable, even though the equations of equilibrium be satisfied. It is important to see whether or not the equilibrium of a blood vessel under elastic tension alone is stable. If the radius increased from \( R \), that of the equilibrium point \( A \), to \( R + \Delta R \), figure 4 shows that the elastic tension will increase more than the increase in total tension required for equilibrium at the new point \( B \). The tension will therefore dominate and the vessel will return to equilibrium at \( A \). Similarly the equilibrium is stable for a casual decrease in radius of the blood vessel, to \( R - \Delta R \) (point \( C \)).

Raschevsky (2) has pointed out that if Hooke's law were obeyed, or, as with simple elastic substances, the slope of the elastic line decreased as the stretch increased, no equilibrium would be possible if the pressure exceeded a critical value, and 'blow-out' would occur. This is shown on the diagram (fig. 5a) when there is no longer any intersection of the two lines. The Laplace lines for pressures 1 and 2

![Equilibrium diagram for a blood vessel.](http://ajplegacy.physiology.org/Downloaded_from/http://ajplegacy.physiology.org/2017/06/26/332)
give intersections, but there is no equilibrium for the pressure. Since however we have seen that the curve for a blood vessel is always likely to turn upwards because of the fibrous jacket, this is probably without application. However in an aorta where the elastic tissue is weakened by disease without accompanying increase in the fibrous 'jacket' the curve may well be as in figure 5b. Here a slight increase in the pressure above a critical value will cause a sudden very great increase in diameter, (from $R_2 - R_3$). This may be the correct view of the mechanism of aortic aneurism.

**Equilibrium Under Active Tension Alone**

Under active tension alone the equilibrium would be completely unstable, because of what might be called 'the elastic paradox' for a cylinder. Suppose that the active tension has the value correct to be in equilibrium with the pressure at the moment (i.e. $T = P \times R$). Now suppose there were a casual increase in the pressure to a value slightly greater than this equilibrium value. A tendency for the radius of the vessel to increase would result. At the new increased radius, Laplace's law demands an increased tension to withstand even the original pressure, and the tension will be still less able to hold the pressure in check. The vessel would expand progressively in an 'explosive' manner. Similarly with a casual decrease in pressure, the vessel would continuously become smaller. The same complete instability is shown to casual fluctuations in the active tension, under a constant pressure. Unless there be some mechanism by which the tension automatically changes with diameter of the vessel to an extent greater than is required by the law of Laplace, only a fictitious, precarious, equilibrium is possible. A blood vessel without elasticity, provided by elastic fibers or by the elastic properties of other tissues, could only be completely closed or completely open when under vasomotor tone. No grading of constriction would be possible.

Mathematically the condition that the equilibrium be stable is that at the point of equilibrium

$$\frac{dT}{dR} = \frac{dT_1}{dR}$$

where $T$ is the tension in the wall, and $T_1$ is the equilibrium tension demanded by the equation of Laplace.

Since $T_1 = P \times R$

$$\frac{dT_1}{dR} = P$$
in terms of elastic constant, $E$ of the wall

$$T = E \frac{(R - R_o)}{R_o}$$

where $R_o$ is the unstretched radius.

$$\frac{dT}{dR} = \frac{E}{R_o}$$

and the condition for stability then becomes

$$\frac{E}{R_o} = P$$

(3)

or

$$E = P \times R_o$$

The application of the law of Laplace then reveals an important role of elastic tissue in the wall of blood vessels that has been hitherto unrecognized. In addition to its function in maintenance of a steady tension against the prevailing pressure that has been already discussed, elastic tissue is necessary to make possible a graded constriction or dilation under vasomotor tone. Where it is absent, or in very small amount, as it may be in some sphincters, vessels could only be either open or closed; equilibrium at intermediate, graded, diameters would be impossible. The true elastic constant $E$ for smooth muscle, that for very slow stretch, is very low indeed (9), and probably elastic fibers are necessary in most vessels. Equation 3 shows that the need for a high elastic constant $E$ is less the smaller the vessel and the lower the pressure. This may explain why capillaries and precapillary sphincters may be able to function without apparent elastic fibers (though their ability to show any considerable gradation of contraction may be in doubt).

Equilibrium under interfacial tension alone would be similarly unstable, for it is the nature of such tensions that they are completely independent of stretch (i.e. $E$ is zero). (The soap bubble cannot remain in equilibrium if its interior is connected to the outside pressure.)

**Equilibrium Under Elastic Plus Active Tension**

When there is elastic tension as well as active tension stable equilibrium is possible over a range of diameters of the vessel. The same type of elastic diagram shows how this is possible. In figure 6 the blood vessel would be in equilibrium under the pressure, for which the straight line of Laplace's law is drawn, at the radius $R_1$ corresponding to the point of equilibrium $A$. Suppose that under active tension the vessel contracts to radius $R_2$. The new total tension required for equilibrium is given by the ordinate $R_2B$. This total tension is less than the tension required before ($R_1A$), but the elastic tension has decreased even more, to that represented by $R_2C$. The difference in tension $BC$ therefore represents the active tension that will cause constriction to the radius $R_2$. The vertical intercepts between the straight line and the elastic tension curve thus give us the relation between the amount of active tension and the constriction that will result. It is obvious that a relatively great increase in active tension is required to produce the first constriction, but slight further increase
in active tension will greatly increase the degree of constriction. The intercepts reach a maximum length at some point close to the point where the elastic tension has reached zero, (DE in fig. 6). This means that an active tension greater than this maximum value seen in the diagram will cause complete closure of the vessel. The point of maximum intercept between the curves represents the limit of stability of the equilibrium. Beyond this point the vessel is as unstable as with active tension alone. An active tension greater than that represented by the maximum intercept (DE in fig. 6) will be sufficient to close the vessel completely.

The lower the pressure, the less will be the maximum active tension before the point of instability and complete closure is reached. Thus with a given active tension, if the pressure in the vessel be lowered, the vessel will eventually reach the point of instability, and have to close completely. For a given active tension there is then what may be called a ‘critical closing pressure.’ If the pressure falls below this critical value the vessel will close completely. The greater the active tension, the higher will be the critical closing pressure.

There is an alternative graphical method of demonstrating this. We may plot the sum of the elastic tension plus a given active tension (fig. 7), assumed to be independent of the stretch. The family of lines through the origin for Laplace’s law at different pressures then, as before, defines the equilibrium radius under these pressures. It will be seen that there is no intersection possible if the slope of the straight line is less than a critical value, i.e. the pressure is less than a critical value. The critical closing pressure will be approximately given by

\[ P_c = \frac{T_e}{R_u} \]

where \( T_e \) is the active tension in dynes per cm. and \( R_u \) is the ‘unstretched’ radius of the vessel. Measurement of the critical closing pressure should therefore give us a way of estimating the active tension in the wall in absolute value, provided we know the radius of the vessels that are closing.
CRITICAL CLOSING PRESSURE AS AN INDEX OF ‘TONE’

If the critical closing pressure be measurable for any vascular bed, under vasomotor tone, its value should be a valuable index of the magnitude of that tone. At present the most-used index is the resistance to flow, defined as the ratio of flow of blood through the vessels to the pressure gradient (arterial pressure minus venous pressure) which drives this flow. Two difficulties arise in using the peripheral resistance as an index of vasomotor tone. The first is that the relation between flow and pressure is not a linear one, due to the factors that the vessels are not rigid and change their resistance with change of pressure within them, and that the viscosity of blood is not a constant but depends on the velocity of flow, diameter of vessel etc. The second difficulty is that the resistance depends on two variables, the geometry of the vessels which is directly related to the vasomotor tone, and the viscosity of the blood, which may vary physiologically. The ‘critical closing pressure’ as an index of tone would suffer from either of these defects. To evaluate it the pressure must be lowered in a vascular bed to the point at which the flow abruptly becomes zero (the pressure would then be the same throughout the system). At this point, since there is no flow, viscosity cannot be involved at all. The critical closing pressure should be the same whatever the viscosity of the perfusing fluid, provided that the muscular tone is the same. The tone that is measured by such a critical closing pressure would be that of the ‘critical’ vessels in the vascular system. Due to their small size and the degree of tension that can be developed by their muscular walls, we would expect these to be the arterioles.

DISCUSSION

Two difficulties may be raised to the application of this simple theory to actual blood vessels, both concerned with the fact that these are not in general thin walled cylinders but may have a thickness of wall greater than the diameter of their lumena. The first is the question whether or not the forces in the different co-axial layers of such a wall can at all be represented by a single total tension \( T \) to which Laplace’s law applies. The difficulty is easily removed by the use of the methods of the differential calculus. The wall may be considered as a series of contiguous co-axial shells, each having its radius \( r \) and specific tension \( t, \) i.e. the tension in a shell of thickness \( dr \) will be \( t,dr \). By the law of Laplace, the difference of pressure from the inside to the outside of any such shell is equal to \( \frac{t,dr}{r} \) Adding together all the differences of pressure across successive shells we obtain the total difference of pressure \( B \) from the lumen of the blood vessel to the outside tissues where the tension is zero. By the integration we see that this must equal \( \int \frac{t,dr}{r} \) There is no doubt that, though we may not be able to integrate this as any specific case because of our ignorance of the specific tensions in the layers, yet we can equate the integral to a single total tension \( T, \) or if we prefer, \( I \times d, \) where \( I, \) is an average specific tension and \( d, \) the total thickness of the wall. The use of Laplace’s law is therefore justified in a thick wall or blood vessel.

The second difficulty is to visualize how a thick walled vessel can close completely.
so that the lumen is obliterated. Even though elastic forces may disappear when the wall is unstretched, the rigidity of the tissues might prevent complete closure. The facts of direct observation are that very thick walled arteries do close their lumena when, for example, the wall is traumatized. Some form of crenation of the endothelial wall with plastic deformation of the cells must make this possible. The conclusions from the theory should be qualified to state that 'unless the rigidity of the tissues prevent,' the vessels must close completely at the critical pressure.

The clinical evidence as to how kidney function is affected in hypotensive shock certainly would suggest that a high critical closing pressure exists, in this condition, for the glomerular vessels. The picture is certainly not that of a kidney function and circulation which declines in proportion to the level of mean blood-pressure. Rather it is that the function (blood flow) decreases out of proportion, and ceases altogether when the blood pressure falls below a critical level (possibly as high as 60 or 70 mm. Hg). Experimental studies of the perfused animal kidney have shown that flow becomes zero when the perfusion pressure reaches 20 mm. Hg (10). Again the remarkable benefits of intra-arterial transfusions under high pressure in shock compared to the usual low pressure intravenous method of transfusion, have been difficult to explain physiologically. If a critical closure has resulted from the low blood pressure, so that circulation has ceased in certain vascular beds, the difference would be expected. Intravenous transfusion would not immediately restore circulation to these 'closed' areas, while the intra-arterial 'pressure' transfusion, which raises the arterial pressure everywhere, would open the closed vessels. When these were restored to normality, they would remain open. There is therefore suggestive evidence that the 'critical closing pressure' may be of more than academic interest.

**SUMMARY AND CONCLUSIONS**

By the application of the law of Laplace, which relates the total tension in the wall to the radius of a cylinder and the excess hydrostatic pressure within it, the total tension in the wall of the various blood vessels in normal conditions can easily be calculated. The calculation reveals that in the mammalian vascular bed the total tension varies from 200,000 dynes per cm. for the aorta to a minimum of only 16 dynes per cm. for the capillaries, rising for the veins to about 20,000 dynes per cm. for the vena cava. The size of the vessels, rather than the difference in pressure prevailing in the various categories of vessel is the dominating factor. The very small radius of the capillaries explains how so delicate a structure can withstand such relatively high internal pressures. The amount of elastic tissue in the wall of the various vessels is well correlated with the total tension in the wall that is required to hold the pressure within them in equilibrium. This suggests that one function of elastic fibers is to provide automatically this 'maintenance tension' in the wall, without the expenditure of the energy required to do this by the contraction of smooth muscle.

Under elastic tension alone, blood vessels can maintain a stable equilibrium under a varying hydrostatic pressure within them. From the data on volume at different pressures and the law of Laplace the elastic diagram for the wall may be deduced, as for the data on the human aorta. Such diagrams deduced for the data of different age groups show clearly the progressive decrease in 'elastic constant'
with age, with a simultaneous increase in the rigidity of a 'jacket' of fibrous tissue. Considerations of the equilibrium diagram offer an explanation as to how aortic aneurism can occur when the elastic fibers are weakened by disease. Under 'active tension' due to the contraction of smooth muscle (or to an interfacial tension between blood and the endothelial wall), no stable equilibrium is possible unless there be an automatic adjustment of tension with stretch (elasticity). Any slight departure from the point of equilibrium given by Laplace's law would result in either complete closure or 'bursting' of the vessel. A second important role of elastic tissue in the blood vessel wall is therefore to make possible a constriction that can be graded by vasomotor tone. With elastic tension as well as 'active tension,' grading of constriction and equilibrium under different pressures and active tension is possible, but still only over a limited range. If the pressure falls below a certain critical value, determined by the 'unstretched' radius of the vessel and the vasomotor tone, complete closure and cessation of flow must result. The same is true if the pressure be constant and the tension in the wall increases above a critical value. If the 'critical closing pressure' as defined above be measurable in any vascular bed, its magnitude should provide a valuable index of vasomotor tone, independent of the viscosity of the blood or perfusion fluid used in its determination. The measurement of critical closing pressure requires the finding of the pressure at which the flow ceases as the driving pressure in a perfusion is progressively reduced.

An account of the experimental verification of these predictions from simple physical laws will be given in succeeding papers. It may be mentioned here that the fundamental limitation of the range of stability of the small vessels has been completely confirmed, and critical closing pressures have been measured which under vasomotor tone may rise above the available mean blood pressure (100 mm. Hg).

REFERENCES