Critical Arterial Stenosis:

A Theoretical and Experimental Solution

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The mechanics of critical stenosis of a blood vessel are studied by means of a comprehensive theoretical model in terms of energy changes and dissipation. These theoretical assumptions correlate well with experimental data obtained in vivo. Previous work in this field is analyzed. This new treatment of the phenomenon of blood vessel stenosis allows explanation of apparent contradictions in previous studies. When the velocity of flow in the unstenosed portion and the geometry of the stenosis are known, the drop in pressure at flow can be predicted.

THE FUNCTIONAL SIGNIFICANCE of the narrowing in an arterial segment is a central problem in cardiovascular pathology. For many years, since Mann's original work,[7] it has been known that a substantial decrease in the lumen of a vessel must occur before a drop in pressure or flow can be measured distal to the narrowed point. It is also well known that beyond a certain degree of constriction, small decrements in the area result in abrupt changes of pressure and flow distal to the constriction. The cross-sectional area value beyond which this phenomenon occurs has been termed the "critical" area of a stenosis (Fig. 1).

This term, however, has been defined in different ways; some arbitrary, some contradictory. Most workers[3,4,12] describe this "critical" area as that beyond which small decreases in vessel lumen areas will result in significant or "marked" effects in pressure drop or flow-rate decrease beyond the stenosis. Others, such as Brice,[1] have adopted an arbitrary definition; ". . . that constriction which would produce a 5% fall in distal mean pressure or a 10% decrease in distal mean flow." Under certain conditions, such as high flow rates, the above definitions may represent substantially different values than the one that would be obtained by Weale's standard ". . . that cross-sectional area at which 80% of the pressure gradient obtainable on total occlusion occurs."

The problem of explaining what happens in a critical stenosis has been approached following basically three lines, or any combination of them: mathematical models, physical models of the circulation and experimentation in animal or human subjects (biological models). The pitfalls inherent in physical and biological simulation have led some workers to make conclusions that are often incomplete and sometimes incorrect. Thus, for some workers, the critical stenosis value would be defined by the percentage reduction of the area at the stenosis site.[7] For others,[1,4] the factors determining the critical value would be the absolute cross-section of the stenosis and the value of the "peripheral resistance," while they claim that the area of the un-stenosed portion of the vessel bears no relation to the value of the critical area. Lastly, others[8] insist that the most important factors in determining the value of the critical area are the velocity of the flow in the unobstructed segment and the area ratio between the former segment and the stenosis. Clearly, there is a fair amount of confusion around the question of which are the parameters that define the "critical" value of narrowing that will produce

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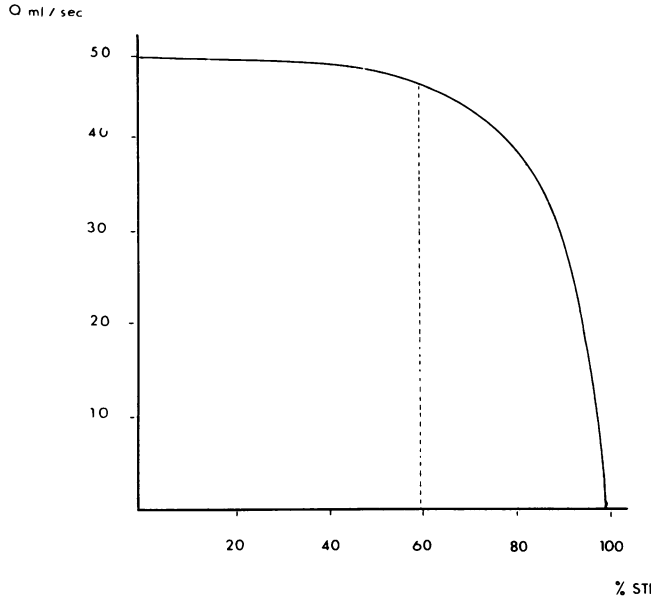

FIG. 1. The phenomenon of critical arterial stenosis. Flow rates plotted versus percentage stenosis of vessel lumen.

a significant pressure gradient or a drop in the flow rate through a vessel.

The problem of the critical stenosis, the prediction of its occurrence and the physical grasp of its mechanics are not therefore theoretical or abstract problems, but matters that have a direct importance on everyday surgical decisions and planning. Narrowing of an artery is the most common problem in our circulatory system beyond middle age and, a very common reason for which vascular surgery operations are performed. Having established its importance, its elucidation must be pursued for, being a problem of fluid mechanics, however particular, it must have a theoretical solution.

Theoretical Solution

Kinetic Energy and the Area Ratio of the Constriction

Our approach has been to describe the circulatory phenomenon in terms of its cause: energy gradients causing blood mass displacements from a high to a low energy point (the traditional proposition that blood flows from a high to a low pressure point is only a specific case of this general proposition).

We shall recall briefly that the total energy (Et) in a unit weight of blood can be expressed as the sum of three components $Et = h + V^2/2g + P/\gamma$ where the term h is the gravitational potential energy, $V^2/2g$ is the kinetic energy (Ek) and P/γ is the lateral pressure energy (Ep). In as much as we shall be considering vessels in a horizontal position, the gravitational component (h) can be neglected.

To calculate the total energy across an arterial segment, all we need to know is the geometry involved, and the values of the systemic pressure (P) and its bulk velocity (V). The pressure (P) can be measured directly and the velocity (V) can be obtained from the volume flow rate (Q), since V = Q/A. These data (P, A) can be measured in both the pre (P_1V_1) and post (P_2V_2) stenotic segments, from where the values of P and V at the stenosis site ($P_{s1}V_s$) can be computed once the geometry of the arterial segment is known.

Let us consider (Fig. 2) a continuous arterial segment of known geometry which is divided into three sections, 1 (pre-stenotic), S (stenotic) and 2 (post stenotic). The velocity at the stenosis (V_s) can be computed as follows:

$$\left.\begin{aligned} Q_1 &= A_1V_1 \\ Q_s &= A_sV_s \end{aligned}\right\} \qquad [1]$$

$$A_1 - A_s = \pi(r_1^2 - r_s^2) = \Delta A \qquad [2]$$

from [2] and [1]:

$$\frac{Q_s}{Q_1} = \left(\frac{A_s}{A_1}\right)\left(\frac{V_s}{V_1}\right) = \left(1 - \frac{\Delta A}{A_1}\right)\frac{V_s}{V_1};$$

solving for V_s:

$$V_s = V_1\frac{1}{\left(1 - \frac{\Delta A}{A_1}\right)} = V_1\frac{1}{(r_s/r_1)^2};$$

substituting for $\omega = r_s/r_s$,

$$V_s = V_1 \cdot \omega^{-2} \qquad [3]$$

From the value of V_s, the corresponding value of kinetic energy at the stenosis (Ek_s) can be derived since: $Ek_s = \frac{1}{2}m \cdot V_s^2$

$$\left.\begin{aligned} Ek_s &= \frac{\rho}{2}A_sV_s^3 \\ Ek_1 &= \frac{\rho}{2}A_1V_1^3 \end{aligned}\right\}, \qquad [4]$$

where P is the density of the fluid

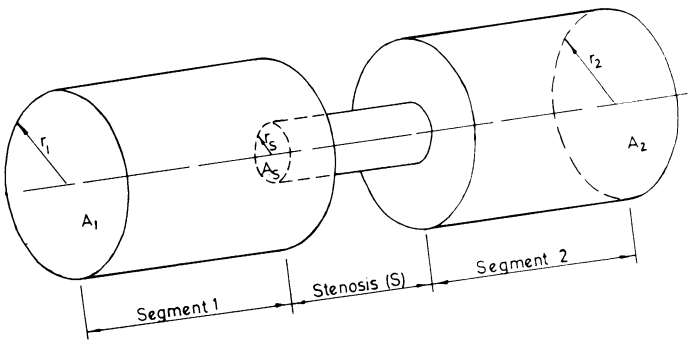

FIG. 2. Schematic representation of the stenosis model with the three segments under consideration (1, S and 2). Flow direction $1 \rightarrow 2$.

$$\frac{Ek_s}{Ek_1} = \frac{A_sV_s^3}{A_1V_1^3};$$

and substituting by [3]:

$$\frac{Ek_s}{Ek_1} = \omega^{-4} \qquad [5]$$

Figure 3 is a graphical representation of this function which states that the ratio of kinetic energy in the stenosed segment (Ek_s) to kinetic energy in the prestenotic segment (Ek_1) is a fourth power function of the decrease in the radius of the vessel.*

Let us assume a stenosis of minimal length whose physical approximation could be the constriction caused by a tight suture around a vessel (see experimental device in Fig. 6). If we now plot the ratio Ek_s/Ek_1 for this constriction, versus the values of ω (that is the ratio of radii) we obtain the curve displayed in Fig. 3. Due to the effect of the fourth power function, a sharply increasing amount of kinetic energy is required to "carry" the flow through the stenosis (Ek_s). Now, the total energy in the stenosis segment ($Ek_s + Ep_s$) cannot be greater than the energy in the prestenotic segment; thus, if Ek_s is increased, then Ep_s must decrease. Hence a drop in lateral pressure energy is inevitable as the kinetic energy through the stenosis increases. In fact, as ω approaches zero, a great deal of pressure energy (Ep) must be converted at an accelerated rate into kinetic energy in order to maintain flow through the stenosis.

Evidently, as the demand for kinetic energy in the stenosis increases with progressive reduction of the stenotic area, a point will be reached when the remaining pressure energy can no longer meet the demand. At this point the flow through the stenosis will have to "slow down." This also implies a decrease in velocity at the prestenotic segment, ΔV_1, and hence a decrease in the flow rate ΔQ_2.

Normally, the fraction of energy being used as kinetic energy in a peripheral medium-sized artery is rather small and less than 1% of the total energy† during peak systole. As the stenosis progresses, the graphic representation of the function $Ek_s/Ek_1 = \omega^{-4}$ (Fig. 3) shows that Ek_s has to rise considerably before its magnitude becomes appreciable (Ek_1 being originally so small). From this point on, as Ek_s increases drastically at an exponential rate, a correspondingly speedy fall in pressure and flow will occur.

According to the Bernoulli principle, an "ideal" fluid

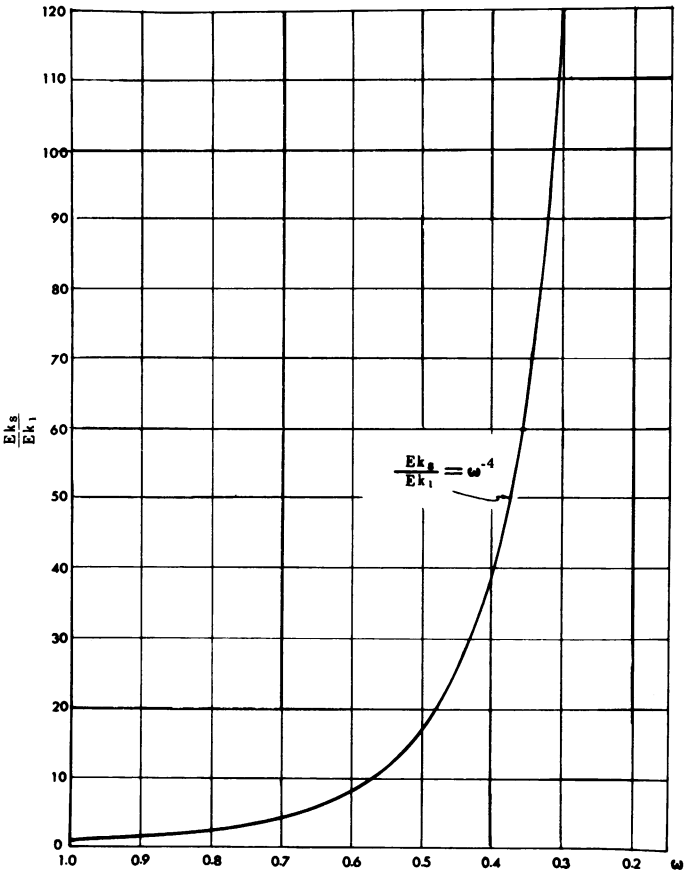

FIG. 3. Graphic representation of the ratio Ek_s/Ek_1 plotted versus the ratio of radii (ω).

will not generate a pressure gradient across a stenosis but a transfer of energy from one form to another ($Ep_1 \rightarrow Ek_s \rightarrow Ep_2$). However, the simple fact that, in a viscous fluid flowing through a tight stenosis, an otherwise laminated flow gives rise to a turbulent jet, (which is later dissipated in the distal stream as heat) indicates that energy must be lost in the process. That blood flowing through a tight stenosis results in a turbulent jet which quickly dies out in the distal segment is beyond doubt: this phenomenon can be observed with the naked eye and, more precisely, with high speed cinematography.

Application of Conventional Hydraulic Principles to the Problem of Stenosis

Normally, in a continuous conduit having a contracted segment, the transfer of lateral pressure energy to kinetic energy in the contraction ($Ep_1 \rightarrow Ek_s$) is made with relatively little energy loss. Conversely, the transfer of kinetic energy to lateral pressure energy ($Ek_s \rightarrow Ep_2$) at the expansion of a conduit is a very expensive one (in terms of energy). Energy is lost in the expanded area by the formation and maintenance of vortices and in providing the viscous drag which

* The theoretical errors that may be incurred in the computation of kinetic energy from mean velocities as opposed to doing so by differentiation of elements are discussed in the Appendix.

† See appendix.

slows down the jet. These energy losses are usually accounted by as contraction loss (h_c) and expansion loss (h_e) as the fluid enters and leaves the stenosis respectively.

Expansion Loss. Energy loss due to sudden expansion has a common place in hydraulics where—for the particular case of sudden expansion in a pipe—the following formula is used:[6]

$$h_e = \frac{(V_s - V_2)^2}{2g} \quad [6]$$

This equation is used in hydraulic engineering where flow in pipes is generally considered to be under turbulent conditions. In our case where a high speed jet discharges into a relatively slow moving fluid mass (poststenotic segment) the use of this formula seems to be justified. Figure 4 illustrates the flow profile of a stenosis in a photograph obtained from a flow model (based on the Hele-Shaw theory). If we now insert this term h_e (Eq. 6) into the continuity equation derived from Bernoulli's principle:

$$P_s/\gamma + V_s^2/2g = P_2/\gamma + V_2^2/2g + h_e,$$

we have:

$$P_s/\gamma + V_s^2/2g = P_2/\gamma + V_2^2/2g + V_s^2/2g - V_2^2/2g - 2V_sV_2/2g;$$

$$P_s/\gamma = P_2/\gamma + V_2^2/g - V_sV_2/g$$

Neither in the work presented in this paper nor from the data from Fry is there any evidence to suggest that the loss in lateral pressure energy that occurs at stenosis is regained in any substantial amount in the post stenotic segment. In other words:

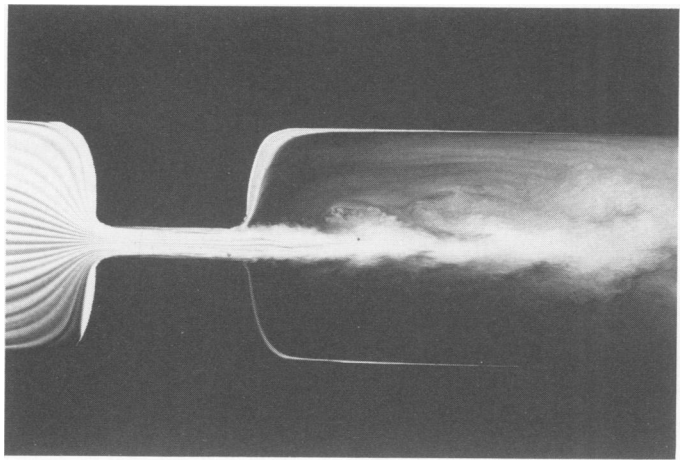

FIG. 4. Photograph of flow through a stenosis taken using the author's flow model (based on the Hele-Shaw theory). The laminar flow in the prestenotic segment remains laminated through the stenosis. At the outlet, a disturbed flow pattern is noted immediately distal to the stenosis. In the post-stenotic segment the energy loss (i.e. expansion loss) through eddies is obvious.

$$P_s/\gamma \simeq P_2/\gamma \quad [7]$$

and therefore the term $V_2^2 - V_sV_2/g$ must be very small. For practical cases, this means that the lateral pressure energy at the stenosis is approximately the same as the lateral pressure energy in the post-stenotic segment ($Ep_s \simeq Ep_2$). Thus, the kinetic energy involved in carrying the flow through the stenosis is lost at the sudden expansion in the formation and maintenance of local turbulence (it is possible, however, that in an elastic system, such as an artery, a small amount of pressure recovery could occur). The conventional representation of the increase in lateral pressure energy at the post-stenotic segment does not really apply to turbulent viscous flow, which is the case of a critical arterial stenosis. The loss in lateral pressure energy that occurs at the stenosis is mostly non-recoverable in the post-stenotic segment.

Contraction Loss. The energy loss due to a sudden contraction in a pipe is usually much smaller than the corresponding expansion loss. There is also a common hydraulic formula for this loss[6] $h_c = 0.5\ V_s^2/2g$. This empirical formula however is not applicable to our problem because: 1) the flow condition proximal to a stenosis in an artery is quasi laminar, while the hydraulic formula is the result of experiments performed in turbulent pipe flow. At the inlet of the stenosis most of the contraction loss occurs in the boundary layer, the characteristics of which will vary greatly between quasi laminar and turbulent flow conditions;[11] 2) this hydraulic formula is commonly used to calculate the contraction loss in water pipes. The geometrical reduction in area of water piper that normally takes place is, in practical terms, of much lesser magnitude than the one under consideration here: a critical stenosis.

Fraction Loss through the Stenosis. The pressure drop or flow decrease in a stenosis is also known to be a function of its length. It is primarily the result of fraction forces that develop between the moving fluid and the wall boundary. For a steady flow system—laminar or turbulent—this loss of energy through length is expressed by the Darcy-Weisbach formula:[9]

$$h_L = f\frac{L}{D}\frac{V^2}{2g} \quad [8]$$

(f) being a coefficient (Darcy-Weisbach resistance coefficient) that varies with the Reynolds number, (L) length of the segment (in our case, the length of the stenotic segment), (D) diameter of the conduit and ($V^2/2g$) the kinetic energy per unit weight of the fluid flowing through the segment under consideration.

It is obvious that in a stenosis this friction loss will be directly proportional to the length and to the kinetic energy of the blood flowing through it, and inversely proportional to the diameter of the vessel. The graph

of the energy loss as a function of the length of the stenosis, can therefore be anticipated to be a curve whose slope will become steeper as the internal radius of the stenotic area under consideration decreases.

Experimental Method

Experiments were carried out in greyhound dogs. Anaesthesia was obtained with intravenous Nembutal (1 mg/kg weight) and the animals were systemically heparinized with 5,000 units of heparin. The iliofemoral trunk was dissected and collateral vessels were ligated with the exception of the internal iliac and the saphenous arteries; these were used as side branches and connected by 13 cm of plastic tubing to pressure transducers (Bell & Howell L 221) (Fig. 5). The pressure transducers were calibrated against a mercury column at the beginning and end of each experimental run. The external diameter of the exposed arterial segment was measured in three points using both a vernier caliper and a fine suture to determine the circumference of the vessel. Two of these points of known external diameter were chosen for the placement of suitable electromagnetic flow probes; the third (middle) point was the site elected for placement of the constricting device. The electromagnetic flowmeter used was the Nycotron 372 with cuff probes, type PS. Occlusion zero was obtained before every flow determination. Time-averaged flow rates ($\bar{Q}$) through the artery were read out on a digital display. Both pulsatile flow and pressure tracings were recorded on an ultraviolet 6-channel recorder (S. E. Laboratories). At the end of each experimental run and occlusion zero sufficient time was allowed for hyperhaemic compensation. The stability of the preparations was excellent throughout the experiments, as evaluated by heart rate, systemic pressure and periodic unrestricted flow rate measurements.

Our constricting device consists of a 2-0 nylon suture looped through a very small ring, and held at each end by two blocks of plastic. The two plastic blocks are mounted on a pair of metal rods; one of the blocks is moveable, thereby altering the loop diameter. The distance between these blocks can be measured accurately with a vernier caliper, the relation between this distance and the corresponding decrease in radius of the loop being linear.

Arterial wall thickness was estimated at the end of the experiments on frozen and fixed tissue sections using a travelling microscope. A certain amount of error must have been incorporated by not taking into account the changes in wall thickness due to distending pressure (although the artery is distended against a constricting and unyielding loop) and due to the unavoidable tissue volume changes secondary to processing. Corrections for wall mass displacement which occur in the stenosed segment in the range of severe constrictions, were made on the curve representing internal radii by extrapolating the values of zero flow and maximal pressure gradient of those of zero internal area.

In order to study the effect of length of the stenosis on the energy loss of the blood flowing through it, we used split and bored plastic blocks of known length and contricting diameters. Accepting the Shippley and Gregg[10] demonstration, we assumed not significant wall mass displacement in the experiments carried out using plastic blocks as external constrictors.

Pressure data were obtained by averaging the reading of 15 cardiac cycles. Kinetic energy computations were made by differentiating the values of the velocity at very small time intervals and computing the kinetic energy for each one of these elements.* Plots of kinetic and lateral pressure energy were made in terms of a height of a water column (cm).

* See appendix.

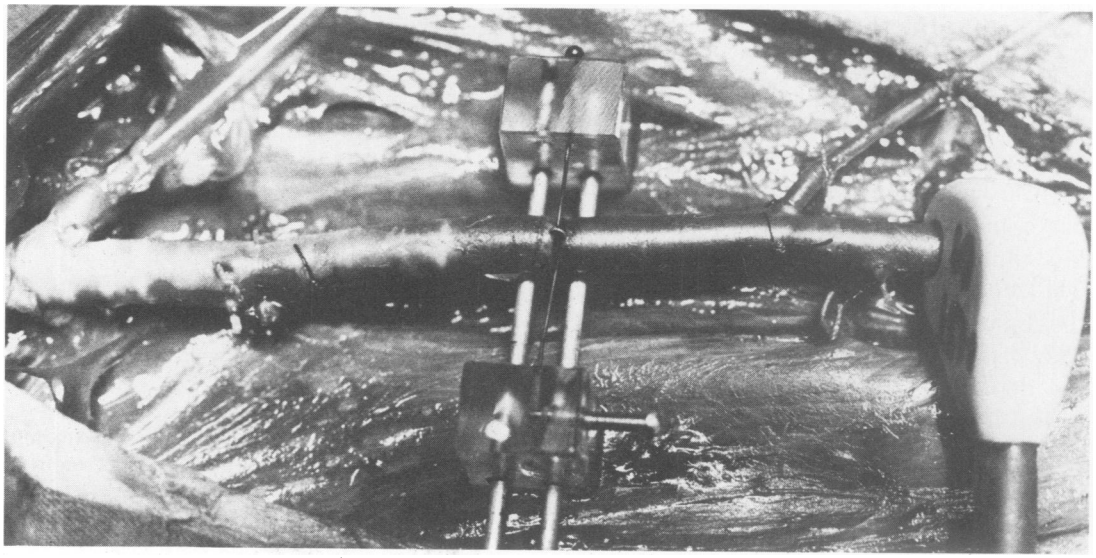

FIG. 5. Photograph of the experimental preparation showing the two side branches cannulated for pressure measurement, and the electromagnetic flow transducer *in situ*. The constricting loop is seen in the mid portion of the femoral artery.

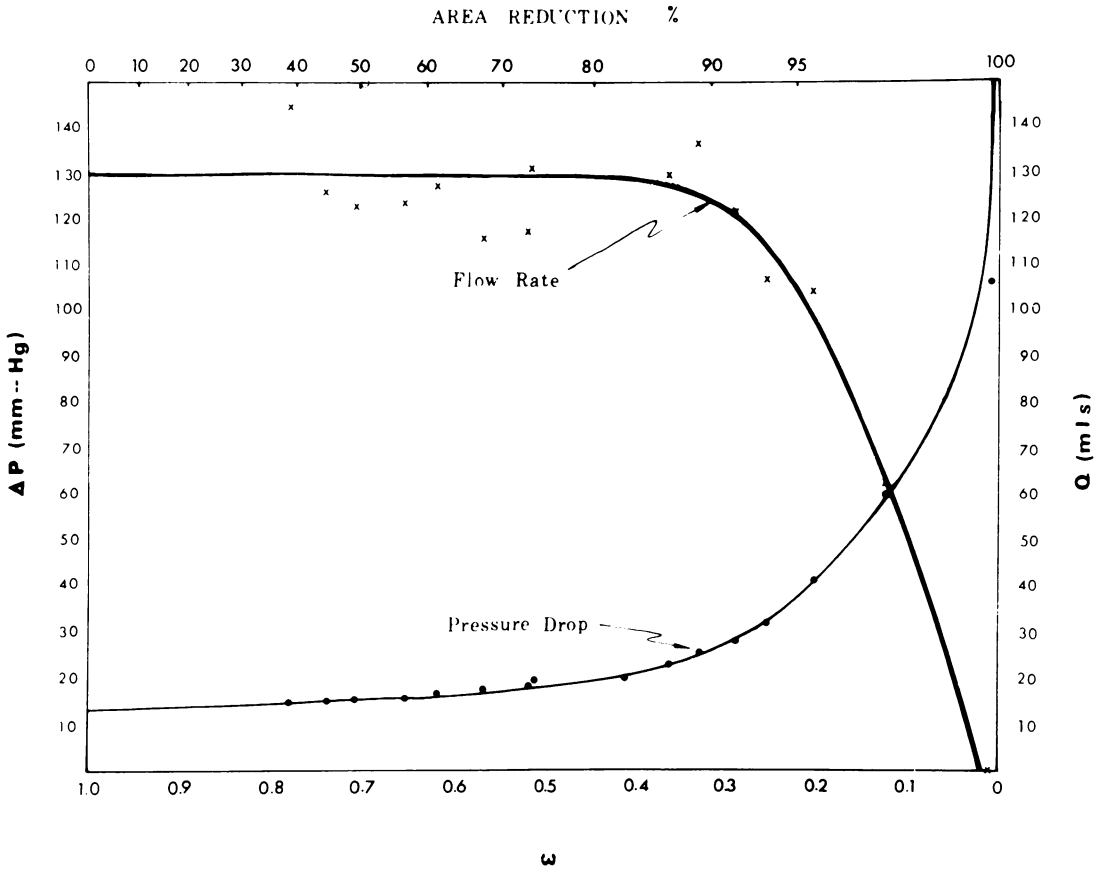

FIG. 6. Flow and pressure changes with increasing stenosis, plotted against radii ratio (ω). The scale showing percentage of stenosis is added to the top of the graph for comparative reference.

Results

A typical tracing of the flow and pressure changes with increasing stenosis (loop constrictor) is shown in Fig. 6. The scale showing percentage of occlusion is added to the ω scale for reference.

We had predicted that before reaching the critical range, the experimental values of Ek_s/Ek_1 would follow the theoretical curve. This close correlation can be seen in Fig. 7 which displays data measured in two experiments in the same animal against the theoretical curve. Within the critical range, the experimental values should be smaller and fall from the theoretical curve because, having incurred a drop in flow volume, hence a drop in V_1 and Ek_1, the value of Ek_s will be smaller since it is expressed as "n" times that of a smaller Ek_1.

In order to show this departure from the theoretical curve more clearly, we have plotted the latter against the experimental data in logarithmic scales (Fig. 8). The solid straight line with the 4 to 1 slope represents the theoretical fourth powered function (equation 5). The beginning of the critical area is clearly shown by the departure of the experimental data from the theoretical line. Beyond this point of departure, the velocity, and, hence Ek_s, continues to increase although it does so at a slower rate than before the critical point was reached. This increase can only continue for a very short distance (as indicated by AB in Fig. 8) until a point is reached where the velocity through the stenosis reaches a maximal value (Max) and then decreases. After this maximal point, the fall to zero flow is nearly instantaneous.

The stenotic values between th critical (departure)

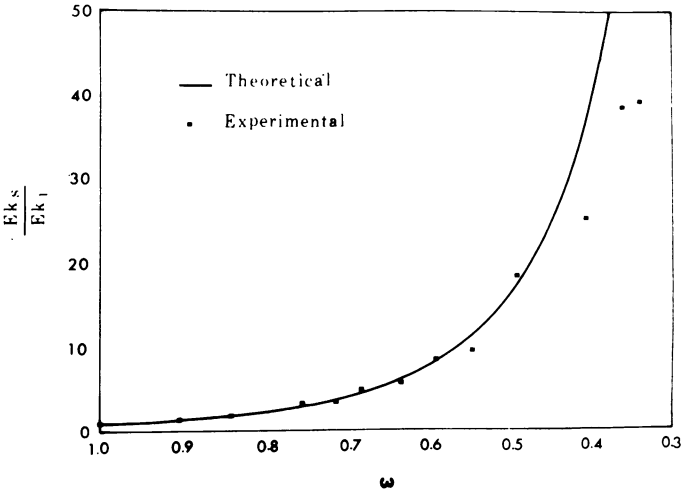

FIG. 7. Theoretical and experimental values of kinetic energy plotted against the ratio of radii (ω). Note the close correlation between theoretical and experimental values up to a value of $\omega = 0.45$.

point and the Max point could be termed the critical range. The departure point is that point beyond which pressure and flow decrements will occur with even the slightest additional constriction and is, therefore, the "critical" point.

Figure 9 shows the effect of increasing the length of two different stenotic segments on the flow rate in the femoral artery. Two series of drilled blocks (4 mm and 3 mm) were used which provided vessel lumen of 0.063 mm^2 and 0.0165 mm^2 respectively in this particular animal. The steeper curve for the 3 mm series correlates well with the theoretical prediction (Equation 8).

From the above derivations and data, it is clear that the "critical" value of a stenosis depends mainly on the velocity of the flow and on the area ratio (or radii ratio = ω) between the stenosed and unstenosed segments of the vessel. Changes in length and viscosity (within physiological limits) are less important in determining the critical value.

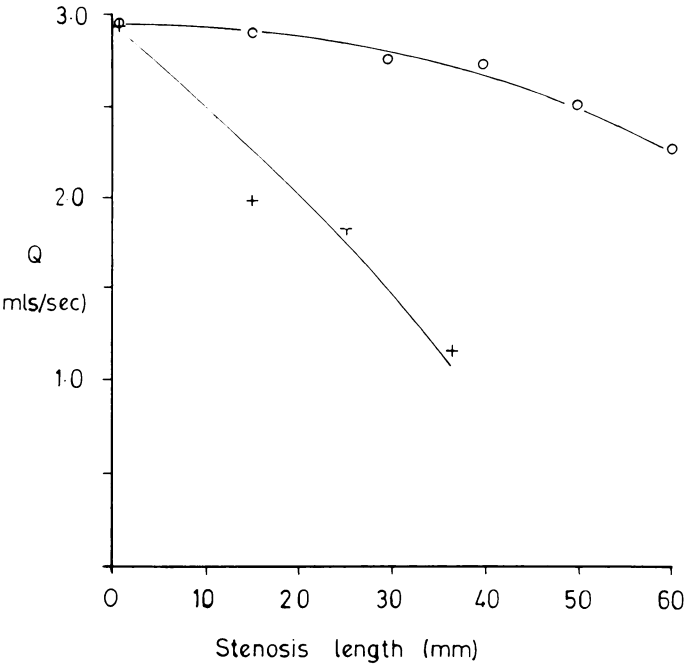

FIG. 9. Graph showing the effect of increasing the length of the stenosis on the flow rate. (o): Constricting block giving a vessel outside diameter of 4.0 mm; (+) idem. of 3.0 mm.

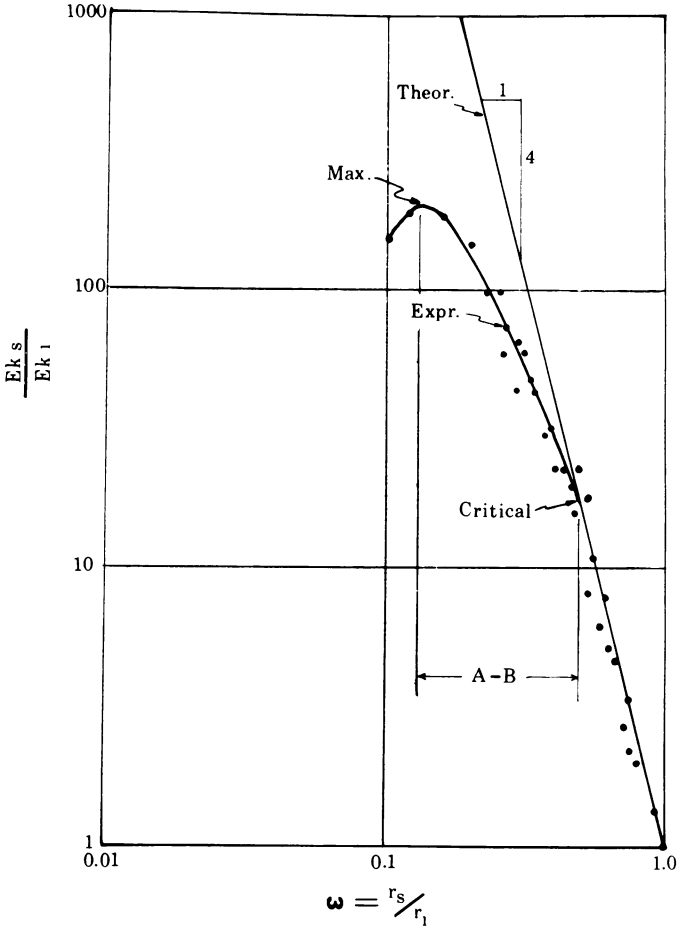

FIG. 8. Experimental data (Expr.) plotted with the theoretical function (Theor.) against the ratio of radii (ω) on log-log scales. The critical (departure) point and the point of maximal kinetic energy (max) are indicated. The critical range is designated (A-B).

Discussion

Review of the Previous Experimental Models on Arterial Stenosis

The experimental work on the problem of arterial stenosis has been performed using mainly two types of models: physical and biological systems. Mathematical models have been infrequently used, probably because of the complexity involved in describing arterial blood flow in precise fluid dynamic terms. Although the Poiseuille equation has been extensively quoted, its inadequacy for predicting changes in flow or pressure secondary to stenosis has been the subject of some studies, such as those of Byar[2] and of Delin.[3] This fact should come as no surprise for the conditions implicit in the derivation of Poiseuille's equation make it non-applicable to the problem of localised stenosis.

Empirically derived formulas—such as the one presented by Byar[2]—throw little light on the subject in terms of providing either a tool or an understanding of the phenomenon under consideration.

May[8] introduced a mathematical model of a stenosis that, because of its apparent simplicity, is worthwhile discussing. Their model equation has three terms representing the pressure losses contributed by the stenosis, the contraction and the expansion of flow, respectively. The first term $(8\ \mu L/R_1^2)V_1(A_1/A_s)^2$ is derived from

Poiseuille's equation through a continuity proposition. It must, therefore, apply only where Poiseuille's equation does apply: pulsating flow through a localised constriction is not such a case. Their second term $(4.8\ \mu/R_1)\ V_1(A_1/A_s)^{1\ 1/2}$ is an empirical one and is obviously based in Poiseuille's equation as well. The third term of their model $\rho V_1^2(A_1/A_s)^2$ is somewhat puzzling. It is presented as being derived from the calculation of head loss in a pipe due to sudden expansion. Such loss in hydraulics has been quoted above (equation 6) as $(V_s - V_1)^2/2g$. By applying Bernoulli's principle through a mass-continuity proposition, this term can be easily transformed into the form of $(\rho/2)V_1^2(A_1/A_s - 1)^2$ which although different does have a certain similarity with that of the third term used by May.[8] However, even if this term were the same as the one that they used, it must be borne in mind that this hydraulic formula has been experimentally derived and applies only to turbulent flow. It is hardly acceptable to introduce into a 3-term equation two terms applicable to laminar flow (Poiseuille) and a third one which is only applicable to turbulent flow and whose derivation is not clear. We agree however, with their conclusions regarding the important factors that determine the value of the critical area of a vessel.

Among the physical models, the most often used are either a constant head of pressure (gravity flow) or a rotary pulsatile system. Mann's[7] original experiments involved both types of systems. His results are clear evidence that the two systems are not comparable, and this should be borne in mind when making deductions to be applied to animal or human arteries.

The animal models have generally been complementary to physical models. They reproduce in a more realistic way what happens in the human arteries, but they have the usual drawbacks of lacking the steadiness of a physical system and of incorporating variables that change the parameters under investigation (autoregulation, etc). In spite of this, they are the ultimate test of any arterial haemodynamic problem before inferences are made which are applicable to human arteries. Some of the drawbacks in the use of physical and animal models are discussed in the next two headings.

The Calculation of the Area of Stenosis Caused by a Constriction

Much of the work on critical stenosis has been done using external constrictions imposed on an artery. From the known values of the external constriction, the value of the internal lumen is derived. When this is done in arteries, whether interposed in a physical model or "in situ," the calculation of this inside lumen implies some assumptions.

Mann,[7] assumed that the percentage of external constriction of a vessel would be directly proportional to the percentage of internal constriction. Assuming the wall thickness to be constant is a source of serious error, as was pointed out by Shipley and Gregg.[10] They showed that with Mann's assumption, the total occlusion of the vessel would occur earlier than would be expected from calculations. Shippley and Gregg convincingly argued that with the constricting blocks they used (10 mm) there was no mass extrusion and, therefore, the internal lumen can be predicted from the values of external diameter and the assumption of constant wall mass. These conclusions apply to constricting blocks of over a certain length but do not apply to tight, minimal-length stenoses, such as those produced by a fine thread around an artery.

In those of our experiments that were performed using constricting blocks (minimum length 10 mm) the assumption of a constant wall volume was made. In the experiments using the nylon loop the curve that defined the internal radius for progressive constrictions was extrapolated to the point where flow rate is zero and the pressure drop reaches its maximum value to correct for the wall mass shift in the constricted vessel.

Some workers[3] have used radiographic measurements of the stenotic areas. The precision of this technique at high values of stenosis does not seem beyond question, even when 2 plane projections are used.

Resistance to Flow

The establishment of a "fixed" peripheral resistance in flow models has been the source of some interpretative errors. Most of the physical models used in previous work[2,4,7,10] have achieved a "fixed peripheral resistance" by placing a small bore tube or a screw-clamp at the distal end of the circuit. It must be said beforehand that one cannot "fix" the peripheral resistance by simply placing an obstruction at the end of a tube while varying either Q or P. For if Q is kept constant, varying P by ΔP would result in a variation of the resistance, ΔR

$$\frac{P + \Delta P}{Q} = R + \Delta R$$

Similarly, if P is kept constant, varying Q by ΔQ would also result in a change in resistance, $\Delta R'$,

$$\frac{P}{Q + \Delta Q} = R + \Delta R'$$

To place a "high peripheral resisistance" at the distal end of the tubing is basically creating a second critical stenosis downstream. The result of this second critical stenosis is to decrease Q_1 and hence V_1. This decrease in velocity in the pre-stenotic segment under study will

result in a much narrower stenosis being now needed to achieve the "critical" constriction.

This is the flaw in Fiddian's work which led him to conclude that the area ratio between unstenosed and stenosed areas would not influence the value of the critical area. By placing a "high peripheral resistance" in the three different diameters of tubing which he used to simulate the sizes of human aorta, dog's aorta and dog's femoral artery in his experiments, he concluded that the critical stenosis area was about 2.3 mm^2 for all three systems. However, if we use his data to calculate the respective mean velocities, we shall find them to be approximately 1.8 cm per second for the human aorta model, 8.4 cm per second for the dog aorta, and 57 cm per second for the dog femoral model. The actual mean velocity in the human aorta is about 20 times higher. His model, therefore, did not represent the conditions that he was trying to simulate. In fact, under a mean pressure of 100 mm Hg his flow data shows that for both the aortic models, and specially for the human, the peripheral resistance in his model acts as a critical stenosis as shown by the very slow velocity in the tubes.

The Different Families of Flow and Pressure Curves in Progressive Stenosis

Previous research in this field of arterial stenosis[4,8,10,12,13] has shown that when plotting stenotic areas versus flow rate (or pressure values distal to the stenosis) the resulting curves have different shapes and give different values for critical areas for the same vessel, depending on whether it is a high or a low flow rate situation. Thus, with progressive constrictions in a high flow rate system, the critical stenosis appears sooner and the curve representing the fall in flow rate (or in post-stenotic pressure) has a smaller slope than in a low flow rate system. In the latter, the critical point appears later and the fall in flow rate (or in pressure) is more precipitous beyond the critical point. This is illustrated in Fig. 10.

We have seen that the gain in kinetic energy (Ek_s) through the stenosis is at the expense of the lateral pressure energy (Ep_s), and that this gained kinetic energy is mostly lost in the post-stenotic segment. Thus, the curve representing the gain in kinetic energy through the stenosis is nearly the same as the curve representing the drop in lateral pressure energy (Ep_2) in the post-stenotic segment. There is a small difference between both curves accounted for by the energy lost in contracting the flow at the inlet and the friction loss at the stenosis. We will now show how the function $Ek_s/Ek_1 = \omega^{-4}$ can explain these different families of curves for different flow velocity systems.

In an arterial segment, providing we are dealing with stable experimental conditions, the total energy per unit

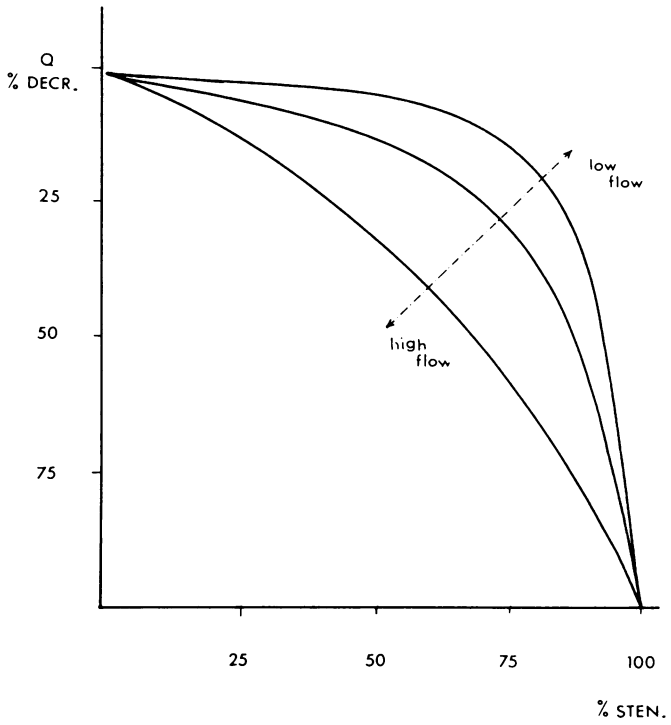

FIG. 10. Graph of percentage decrease in flow obtained for progressive stenosis under high, medium and low flow conditions.

weight (Et) contained in the blood which flows through a cross-section is constant. The kinetic (Ek) and the lateral pressure (Ep) components, can be expressed as percent fractions of the total energy (Fig. 11). In a high flow condition (h) such as exercise, arteriovenous shunting, etc., the amount of energy allocated to the kinetic component—$Ek_1(h)$—will be a substantial proportion of the total energy available. Let us give a high

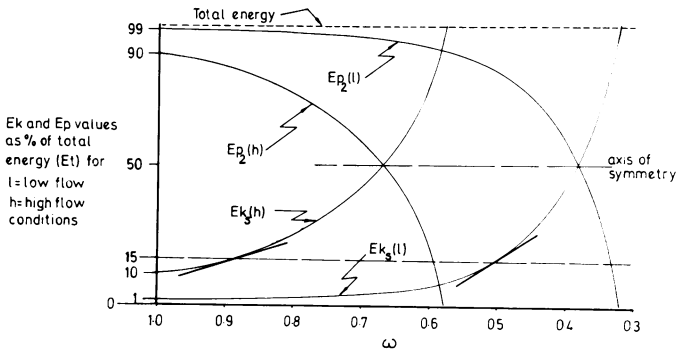

FIG. 11. Graph showing changes in the lateral pressure (Ep) and kinetic (Ek) components of the total energy in a vessel for different degrees of stenosis, considering kinetic energy at the stenosis (Ek_s) and lateral pressure energy in the post-stenotic segment (although for practical purposes $Ep_s = Ep_2$). Two cases are considered: a high flow condition (h) with the kinetic component being 10% of the total energy, prior to any stenosis ($\omega = 1$), and a low flow condition (l) with the kinetic component representing 1% of the total energy at $\omega = 1$. The 15% level of total energy has been marked and is considered to be within the critical range.

value, say 10% of the total energy, to $Ek_1(h)$ in the curve representing the function $Ek_s(h)/Ek_1(h)$, for a progressive decrease of the lumen area ($\omega \rightarrow 0$), the function $Ek_s(h)/Ek_1(h)$ will rapidly approach the level of 15% of the total energy. This rise will be reflected by a similar (and detectable) drop in lateral pressure energy as shown in Fig. 11 by $Ep_2(h)$. One may notice the similarity between the graph of this drop and that depicted as typical of high flow conditions (Fig. 10 high flow curve).

Conversely, the percentage of total energy allocated to the kinetic component in a low flow situation—$Ek_1(1)$—is a small fraction, generally less than 1%. Thus, it requires a much higher degree of stenosis for the function $Ek_s(1)/Ek_1(1)$ to approach a value of 15% of the total energy, and hence to show a measurable drop in lateral pressure energy—$Ep_2(1)$—than is required in the high flow condition to obtain the same result. When this value (15%) is approached, that is when the stenosis is within the critical area, we are dealing with that portion of the function ω^{-4} that has a very steep slope (Fig. 11). If we consider that, beyond the stenosis, the changes in kinetic energy are approximately inversely proportional to the changes in lateral pressure energy, we can see that, in a low flow condition, the changes observed within the critical area occur at a faster rate than in a high flow condition.

In an experimental situation, a 15% drop in the lateral pressure energy is definitely a measurable change. When this occurs across a stenosis, we can assume that the critical range has been entered. The drop in lateral pressure energy (post-stenotic) being proportional to the gain in kinetic energy through the stenosis, we shall arbitrarily set this value of 15% as one which is definitely within the critical range, in order to discuss the behaviour of the function that describes energy changes through a stenosis. This is not to say that 15% is what we define as a critical stenosis but is a deliberately chosen value which we know must be within the critical range.

Conclusions

Although the mechanics of blood flow through a stenosis is known to be a dynamic problem, its interpretation as a static, purely geometrical problem has been used in both research and clinical work. Thus some authors have defined the "critical" narrowing of a vessel in terms of the area ratio between the stenosed and unstenosed portions.

On the other hand, experimental results have led others to conclude that the "critical" area of a stenosis depends on the peripheral resistance and on the actual area of the stenosis while the area of the unstenosed segment of the vessel would bear no relation to the value of the "critical" area of stenosis.

By considering the mechanics of the blood flow through an arterial stenosis from the standpoint of energy transfers, we have attempted to provide a physical grasp of the fluid dynamics involved, which could be used as a tool in evaluating arterial lesions in man. Our experimental results correlate well with the theoretical solution. In the calculation of the values, the assumption of a flat velocity profile was made. The possible magnitude of error involved with this assumption is discussed in the appendix. The changes induced in the system by the elastic properties of the arterial wall are not incorporated in the computation as their relative magnitude is not thought to be of importance. For the size of artery of our interest, the role of viscosity has been studied by other workers[2] and follows the simple Newtonian law. The importance of the length factor as seen in the experimental data, correlates well with the theoretical expectations. The most important factors in determining the "critical" value for the stenosis of a vessel are the flow rate in the unstenosed vessel and the ratio of areas between the stenosed and unstenosed segments.

The development of a pressure gradient or the decrease in volume flow secondary to a critical stenosis is a reflection of energy loss through it. Most of this loss occurs through the conversion of lateral pressure energy into kinetic energy, the subsequent loss of the latter being mostly irretrievable as it is spent in the generation of jet turbulence in the post-stenotic segment. The critical area is defined, for each particular flow rate, as the area ratio where the values of the kinetic energy through the stenosis deviate from the theoretical curve. The critical range of a stenosis is defined as the range of values between the critical area value and that where the velocity reaches its maximum value.

The influence of the peripheral resistance on the values for critical stenosis is affected through the changes that it produces in flow velocity (V_1).

The geometrical ratio between stenosed and pre-stenotic segments and the velocity in the pre-stenotic segment provide sufficient information to anticipate the approximate value of a critical stenosis if the total energy of the system is known. In most large and medium sized arteries, the value of the lateral pressure energy at rest (in the horizontal position) approximates to the total energy value in the vessel, kinetic energy being a small fraction of the latter. Under conditions of high flow rates (exercise, drug-induced vasodilation, arterio-venous communication) the kinetic energy component becomes important and must be taken into account. Measurement of pressure gradients across stenosis under operative conditions (low cardiac output, increased peripheral resistance; hence, low velocity, etc) does not provide an answer to the significance of a lesion. Vasodilating drugs may be used to induce high volume flows, although the magni-

tude of this increase may not correspond to that obtained by exercise.

Appendix

In most fluid systems, velocity distributions over a cross-section are quite pronounced. This variation in velocity, plus the fact that kinetic energy is a power function of velocity, can introduce errors in the computation of kinetic energy when using the space-average value of velocity, that is, assuming the velocity profile to be flat.

A similar type of consideration has to be made with respect to the time-average velocity, when dealing with any type of time dependent flow, such as pulsatile arterial flow. In the same manner, the use of time-averaged values of velocity can introduce considerable errors in the computation of kinetic energy values.

Errors Introduced by the Assumption of a Flat Velocity Profile

Considering steady flow, the weight of the fluid that passes through a cross section of the vessel in unit time equals γQ; Q: discharge, γ (specific weight) $= \rho g$.

The true average kinetic energy over the cross-section of the vessel, expressed in K.E. per unit weight would be:

$$\frac{1}{2gQ}\int_A v_{(s)}{}^3\,dA \qquad [9]$$

Comparing equation 9 with the conventional way of computing kinetic energy:

$$V^2/2g \qquad [10]$$

we have:

$$\frac{\frac{1}{2gQ}\int_A v_{(s)}{}^3\,dA}{V^2/2g} = \frac{\frac{1}{2gQ}\int_A v_{(s)}{}^3\,dA}{V^3A/2gQ} = \frac{\int_A v_{(s)}{}^3\,dA}{V^3A} = \alpha \qquad [11]$$

This ratio (α) between the true value of the kinetic energy and the value of the kinetic energy computed by using the average velocity $v = Q/A$ is called the "coefficient of kinetic energy." This coefficient will be larger than 1.0 unless the velocity is uniform across the entire cross-section. In steady laminar flow (parabolic distribution) the value of $\alpha = 2.00$, while for turbulent flow in a smooth pipe $\alpha = 1.06$. Hence, the error involved in using the space-average value of the velocity in the term $v^2/2g$ for computation of kinetic energy amounts to 6% in fully developed turbulent flow and to 200% in laminar flow.

Error Introduced by Using the Time-averaged Value of Velocity ($\bar{V}$) for the Computation of Kinetic Energy

Assuming now a flat flow profile, the true time-average of velocity in unsteady flow is:

$$\bar{V} = \frac{1}{T}\int_T v_{(t)}\,dt$$

T being the time period over which the average is taken. We have then,

$$Ek = \frac{\bar{V}^2}{2g} = \frac{\left[\frac{1}{T}\int_T v_{(t)}\,dt\right]^2}{2g} \qquad [12]$$

The true average kinetic energy can be calculated from the velocity recordings (as functions of time) by considering small increments of time (dt) when the instantaneous velocity is $v_{(t)}$. The volume of fluid that passes through the vessel cross-section in this period (dt) is $Q = Av_t\,dt$ and its weight: $\gamma Av_{(t)}\,dt$

Kinetic energy per unit weight in this time period will be $v_t^2/2g$ and the kinetic energy of the total fluid weight passing through the cross-section will be:

$$\frac{v_{(t)}{}^2}{2g}(\gamma Av_{(t)}\,dt) = \frac{\rho}{2}Av_{(t)}{}^3\,dt$$

which over a long period of time (T) will equal:

$$\frac{\rho}{2}\int_T Av_{(t)}{}^3\,dt \qquad [13]$$

The time averaging Ek per unit weight will be:

$$\frac{\frac{\rho}{2}\int_T Av_{(t)}{}^3\,dt}{\gamma\int_T Av_{(t)}\,dt} = \frac{1}{2g}\frac{\int_T v_{(t)}{}^3\,dt}{\int_T v_{(t)}\,dt} \qquad [14]$$

comparing the values from equations [14] and [12]:

$$\frac{-\frac{1}{2g}\frac{\int_T v_{(t)}{}^3\,dt}{\int_T v_{(t)}\,dt}}{\frac{1}{2gT^2}\left[\int_T v_{(t)}\,dt\right]^2} = \frac{\int_T v_{(t)}{}^3\,dt}{\frac{1}{T^2}\left[\int_T v_{(t)}\,dt\right]^3} = \alpha_{(t)} \qquad [15]$$

We shall call $\alpha_{(t)}$ the "time coefficient of kinetic energy." Its value depends entirely on the waveform.

Considering arterial blood flow, the errors involved in computing kinetic energy values by using its time-averaged velocities ($\bar{V}$) are greater than those involved in using the space-averaged velocities ($\bar{V}$). In space-averaged velocities, the largest possible error in computing kinetic energy values would be in the case of a parbolic flow profile (laminar) when the true kinetic energy value would be 200% higher than the estimated assuming the same average velocity for the entire cross-section. It

is doubtful that a true parabolic profile ever exists in arterial blood flow and, if it does, it could only occur in certain locations and for a short period in each cardiac cycle.

The errors involved in using time-averaged velocities for the computation of kinetic energy values are of greater magnitude. In certain physiological waveforms we have found the true values of kinetic energy to be over 1,000% higher than those obtained by computing kinetic energy from recorded time-averaged velocities.

In this study we have computed the kinetic energy values from differential time elements in the waveform according to equation (14). We have assumed a flat velocity profile. The reason for this lies in the difficulty of plotting the flow profile through the stenosis and in the fact that, as the stenotic area decreases, we are dealing with a turbulent jet in which the coefficient of kinetic energy must approach the 1.06 value.*

In fact, in the range of very small areas of stenosis, the errors incurred in the computation of kinetic energy depend more on the difficulty of estimating with accuracy the cross-sectional area of the vessel, than in time or space velocity distributions, for the flow becomes a turbulent jet (flat profile), and the velocity wave is damped and approaches the configuration of the time-averaged velocity tracing.

In detailed haemodynamic studies, it may be necessary to consider both the space and time distributions of velocities simultaneously. The basic mechanics are provided in equations (1 and 15), and the computations provided in equations (11 and 15), and the computations space (A) and time (T).

* The errors incurred by computing kinetic energy values from time-averaged velocity values also become smaller as the stenotic area decreases because of the damping effect of the velocity wave amplitude. These are still of sufficient magnitude to be unacceptable for computation of kinetic energy values.

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