Studies Made by Simulating Systole at Necropsy

VIII. Significance of the Pulse Pressure

By Isaac Starr, M.D.

In a previous publication we defined the relation between cardiac work and blood pressure measurements by means of multiple regression equations. In this communication we seek a simple method of estimating cardiac work from observations any doctor could make in the clinic.

Long before blood pressure could be measured in the clinic, the nature of the relation between the amplitude of the pulse and the cardiac stroke volume was discussed. It is mentioned in a report of studies of the recorded pulse by Roy and Adami in 1890,1 in a similar report by Mackenzie in 1902,2 and surely the same idea must have occurred much earlier to countless imaginative doctors who, when feeling the pulse, speculated on the aspect of cardiac performance that produced the sensation they perceived through their fingers. During this era, exact knowledge of the relationship was not possible, for the amplitude of the recorded pulse varied with the pressure with which the pick-up unit was applied to the artery, and it was therefore difficult to interpret in quantitative terms.

As soon as blood pressure could be measured in the clinic, more exact knowledge was sought.3 Thus, a direct relation between pulse pressure and cardiac stroke volume was proposed by Erlanger and Hooker in 1905.4 Later, many attempts were made to define the relation more exactly, and several methods designed to estimate cardiac stroke volume from pulse pressure were proposed, based on the theoretic conceptions favored by the various authors at that time.5-9 Recently, workers from this laboratory have brought forward methods of estimating stroke volume from pulse pressure, based not on any theory, but on regression equations derived from measurements made after simulating systole in experiments on cadavers.10

Indeed, it seems almost self-evident that the amplitude of the pulse wave, expressed as the pulse pressure, should be somehow related to the size of the cardiac stroke volume, which is its genesis. But a relationship between the magnitude of the pulse pressure and the work performed by the heart at each stroke did not seem self-evident at all, at least to me. So, when results indicating that pulse pressure was related much more closely to stroke work than to stroke volume began to turn up in the data secured in our cadaver experiments, I was taken by surprise. But I soon discovered that, by reasoning from their theoretic conceptions, the Scandinavian school had anticipated this finding.

In 1928, Liljestrand and Zander5 published the results of experiments supporting a very simple view of the relation between stroke volume and the blood pressure, which has been expressed as follows:

\[ \text{Stroke volume} = L \frac{\text{pulse pressure}}{\text{mean pressure}} \]  

(1)

\( L \) being a constant that was expected to vary from subject to subject. However, the validity of such a simple relationship was disputed by the German school6-11 whose theoretic conceptions and experimental results indicated a much more complicated relationship between stroke volume and blood pressure, dependent on many additional factors such as the elasticity and size of the vessels. At that time, the only way
to test the validity of such ideas was to compare the blood pressure data with the results secured by the cardiac output methods then available. Since there was little knowledge of the absolute accuracy of these flow methods, it is not surprising that the results secured by one group were not always confirmed by those obtained by another. In our experiments in which systole was simulated in cadavers at necropsy,10 the stroke volumes employed were accurately known, and the results supported the view of Liljestrand and Zander that their simple formula provided a useful approximation of stroke volume.

Twelve years later, a logical extension of Liljestrand and Zander’s conception was made by Apériɑ11 but, buried in an advanced mathematic treatise on the dynamic theory of the circulation, the simple conception he proposed seems to have been altogether overlooked. Apériɑ produced no evidence for the correctness of his view, but he clearly pointed out that, since the approximate formula for cardiac work was:

\[
\text{Cardiac work} = \text{stroke volume} \times \text{mean pressure} \quad (2)
\]

then, by substituting from the Liljestrand and Zander equation (1), we have:

\[
\text{Cardiac work} = L \text{ pulse pressure}. \quad (3)
\]

By this simple mathematic expression, Apériɑ set forth the view that the pulse pressure provides a quantitative measure of changes occurring in the work of the heart of any patient. But, I know of no clinician using the pulse pressure for this purpose at the present time, although a simple method of estimating the heart’s work would be welcomed by everyone.

A second feature of this conception will also take clinicians by surprise. The height of the blood pressure is an important factor in the estimation of cardiac work, but Apériɑ’s formula indicates that pulse pressure measures the work independently of the height of the blood pressure.

The purpose of this paper is to record a test of Apériɑ’s idea by means of the results secured by simulating systole in our cadaver experiments; for in these, curves of cardiac ejection and of both central and peripheral blood pressure had been recorded graphically, and the “left ventricular” work performed could be estimated by integration with an accuracy not approached in experiments performed on living men or mammals. Our results support the simple conceptions of the Scandinavian school in emphatic fashion. Indeed, the pulse pressure proves much more closely related to the work performed by the heart than to its output.

**Material and Methods**

The technic of the experiments in which systole was simulated in cadavers at necropsy has already been described in detail.12 In brief, after raising the pressure to the diastolic level by perfusing fluid into a femoral artery, systole was simulated by injecting fluid into the root of the aorta and pulmonary artery from syringes, the injection being powered by a blow of known energy. The amount injected at every instant was recorded by an optical system. Central and peripheral blood pressures were secured by 2 optical manometers connected with small tubes, the tip of 1 lying in the root of the aorta, that of the other in the femoral artery. The ballistocardiogram was recorded also, but this paper is not concerned with that measurement.

In the series of 144 “systoles” conducted on 16 cadavers, the stroke volume, blood pressure, and the contour of the ejection curve were varied widely as we aimed to explore the ranges found clinically.13 In some subjects, the vessels were altogether normal in appearance, in others, arteriosclerosis was advanced. The experiments themselves must be divided into 2 groups; those in which blood was used to fill the vessels and simulate systole, and those in which water was used for these purposes. The results secured in each group will be analyzed separately.

The experiments in which blood was used were performed at the end of our long series after our technic had been brought to its highest point. The clinical and necropsy findings on the 6 cadavers of these series have been recorded. The curves of cardiac injection at each instant of systole have been published, and the values of stroke volume, central and peripheral systolic and diastolic pressure, pulse wave velocity, etc. are also on record.14 From the curves of cardiac injection and of central blood pressure, “left ventricular” work was calculated for me by Dr. Walter Feder,15 by multiplying the values instant by instant and integrating the resulting curves, thus securing values for work, as work was defined by Sir Isaac Newton, which are certainly far more accurate than any that have been secured in living men or mammals. These accurate values of work, which will be designated as “true work,” can be compared with the peripheral pulse pressures secured in the same “systoles.” Statistical analysis has been performed
on the results secured in 51 such “systoles” in 6 cadavers; that is, on all the results obtained except 3 in which the duration of ejection was far longer than any systole to be expected during life, and 1 in which, through an oversight, work was not measured by integration.

The experiments in which water was used both to fill the vessels and simulate systole, performed earlier, were not so technically perfect as the experiments in which blood was used, and the various reasons for this have been discussed. Technical deficiencies made us discard all the results secured in several experiments. Most of these were lost because of the difficulty inherent in photographic recording; errors of technic that ruined the records and were not recognized until the film was developed at the end of the experiment. All the results secured in 2 other cadavers were disregarded for reasons of a different kind. In 1, the brain, of great interest to the pathologist, was removed before our experiments began, in the fear that our injections of water might distort the pathologic picture by creating edema; so the common carotid and innominate arteries were tied before reasonable diastolic pressures could be secured, greatly reducing the vascular bed. In another experiment, a mediastinal tumor so distorted the anatomy that the rigid glass cannulas used at the time had to be forced into their usual position in the aorta and pulmonary artery; distortions were produced thereby that might well have kinked the vessels. While it seemed wise to omit all data secured from these last 2 subjects, they could have been included without damage to any of our conclusions.

After thus omitting all data from 5 subjects, we still had satisfactory data from 93 “systoles” secured in 10 other cadavers perfused by water, and this seemed a sample of ample size. Except for the omissions noted above, this sample includes all the results secured after the technic had been improved by powering the injections mechanically by a swinging mallet, in place of pushing in the syringe plungers by hand.

Because the different physical properties of water and blood made the results of the “water” experiments less clearly applicable to conditions existing during life, the labor of estimating “cardiac” work by integration was not undertaken in this group. We contented ourselves by calculating work without regard to time by formula (2), which we have shown to give a reasonably good estimate of the true work.14

The results of such estimates will be called “approximate work.”

The clinical diagnosis and autopsy findings of most of the cases perfused with water have already been published.13,15 However, in this communication additional data will be used from 4 other subjects not previously described. The essential clinical data and necropsy findings of these were as follows:

Subject 24. V.S., a man of 49, 75 Kg. weight and 174 cm. tall, died of acute lymphatic leukemia. At necropsy, the heart showed subacute bacterial endocarditis of mitral and aortic valves in addition to widespread findings characteristic of leukemia. The aorta and arteries were normal.

Subject 25. L.E., a man of 54, 78 Kg. weight and 170 cm. tall, had formerly been hugely obese, once weighing 135 Kg. He had been in congestive failure repeatedly without any obvious clinical explanation. Necropsy showed old myocardial infarction and coronary atherosclerosis. The aorta showed severe atherosclerosis with numerous yellow plaques, many of which were calcified and ulcerated.

Subject 26. F.C., a woman of 64, 52 Kg. weight and 165 cm. tall, died of carcinoma of the ovary with widespread metastasis. There was moderate atherosclerosis of the aorta without calcification.

Subject 27. R. P., a woman of 49, 60 Kg. weight and 166 cm. tall, after suffering from diabetes mellitus and hypertension for many years, died of renal involvement and terminal uremia. Two toes had been amputated for peripheral vascular disease. Necropsy showed renal nephrosclerosis. The aorta showed only moderate atherosclerosis, little more than would be expected for her age. There was some calcification below the bifurcation, but none above.

Results and Discussion

The relations between pulse pressure and stroke volume, and pulse pressure and true “left ventricular” work, secured in subjects perfused with blood are shown in figures 1–3, and the regression equations have been placed in tables 1 and 3. Similar relations for the subjects perfused with water are shown in figures 4–6 and in tables 2 and 4. In these tables, the regression equations have been numbered serially with those of our preceding communications derived from data secured in our cadaver experiments. The multiple regression equations recorded in tables 3–5 were calculated for me by Dr. Albert Schild.

In the statistical analysis of our data, the results secured on subjects perfused with blood have not been combined with those secured on subjects perfused with water, because the great differences in viscosity between water and blood made it unlikely that similar quantitative relations would be found for the 2 series. This was borne out by the results, for certain significant differences were found, and they will be mentioned before the much greater similarities will be discussed. Thus, the slopes of the comparable regressions in figures 1 and 4, equations...
TABLE 1.—Regression Equations from Data Secured in Fifty-one Simulated Systoles Performed in Six Cadavers Perfused by Blood Relating Stroke Volume and True "Left Ventricular" Work to Peripheral Pulse Pressure; and Such Work to Mean Peripheral Blood Pressure

<table>
<thead>
<tr>
<th>Equation Number</th>
<th>Equation</th>
<th>( \sigma )</th>
<th>Correlation coefficient</th>
</tr>
</thead>
<tbody>
<tr>
<td>94</td>
<td>Stroke Volume ( ml. = 35.9 + 0.21 ) pulse pressure ( mm. Hg )</td>
<td>13.45 ( ml. )</td>
<td>0.40</td>
</tr>
<tr>
<td>95</td>
<td>True Work ( Gm. M. = 1.1 + 1.13 ) pulse pressure ( mm. Hg )</td>
<td>18.3 ( Gm. M. )</td>
<td>0.86</td>
</tr>
<tr>
<td>96</td>
<td>True Work ( Gm. M. = 0.54 + 68.4 ) pulse pressure ( mm. Hg ) ( \frac{\text{age years}}{\text{age}} )</td>
<td>16.6 ( Gm. M. )</td>
<td>0.89</td>
</tr>
<tr>
<td>97</td>
<td>True Work ( Gm. M. = -8.0 + 0.78 ) mean blood pressure ( mm. Hg )</td>
<td>26.2 ( Gm. M. )</td>
<td>0.69</td>
</tr>
</tbody>
</table>

TABLE 2.—Regression Equations from Data Secured in Ninety-three Simulated Systoles Performed in Cadavers Perfused with Water Relating Stroke Volume and Approximate "Left Ventricular" Work to Peripheral Pulse Pressure

<table>
<thead>
<tr>
<th>Equation Number</th>
<th>Equation</th>
<th>( \sigma )</th>
<th>Correlation coefficient</th>
</tr>
</thead>
<tbody>
<tr>
<td>98</td>
<td>Stroke Volume ( ml. = 32.1 + 0.21 ) pulse pressure ( mm. Hg )</td>
<td>12.9 ( ml. )</td>
<td>0.47</td>
</tr>
<tr>
<td>99</td>
<td>Approx. Work ( Gm. M. = 25.2 + 0.50 ) pulse pressure ( mm. Hg )</td>
<td>17.1 ( Gm. M. )</td>
<td>0.74</td>
</tr>
<tr>
<td>100</td>
<td>Approx. Work ( Gm. M. = 18.6 + 44.8 ) pulse pressure ( mm. Hg ) ( \frac{\text{age years}}{\text{age}} )</td>
<td>15.3 ( Gm. M. )</td>
<td>0.80</td>
</tr>
</tbody>
</table>

95 and 99, are significantly different \( (t = 4.67) \). In the experiments in which blood was employed, a given increase in pulse pressure represents a greater increase in work. This difference would be expected from the more viscous nature of blood, which causes greater loss of energy in friction during its passage to the periphery. Also, in the data from "blood" experiments, the regressions of pulse pressure with both work, and work divided by age, pass very close to the origin indeed; in the data secured in the corresponding water experiments they pass somewhat farther away. Despite these differences, the results of the 2 sets of experiments confirm each other in surprising fashion as far as their main features are concerned, as will now be set forth.

Pulse Pressure and Stroke Volume

A glance at figures 1 and 5 shows that this relationship is a poor one; the regressions pass far from the origin and the scatter is large. If one divides figure 1 by a line from the origin to the opposite corner, all the points fall to the right of and below this line and they are scattered quite evenly throughout this triangular area. The points in figure 5 show a similar distribution.

From this distribution of the data, several deductions of general interest to clinicians can be pointed out. If peripheral pulse pressure is unduly large, it is safe to believe that stroke volume is abnormally increased. But if this pulse pressure is small, no deduction about the
size of the stroke volume can be safely made, for it may be either small or large. When the tension in the large arteries is low, a stroke volume of considerable size may not produce a pressure wave that reaches the periphery in the identifiable form of a pulse wave. Therefore, when the patient is pulseless, or if blood pressure cannot be secured, it does not necessarily mean that stroke volume is 0. This may well account for experiences in which patients, pulseless for considerable periods of time and believed to be dead, have revived unexpectedly. Our findings are also consistent with the observations that in conditions of syncope, when blood pressure and the pulse often cannot be secured, the circulation through an arm may still be large.

**Pulse Pressure and Stroke Work**

Comparisons between figures 1 and 2 and between figures 4 and 5 show clearly that the relation between peripheral pulse pressure and **work** is the greatest improvement over that between pulse pressure and stroke volume. The relationship between pulse pressure and work is clearly linear and so good that it is no exaggeration to say that the pulse pressure measures the work of the left ventricle.

When the relations between pulse pressure and cardiac work are examined in individual subjects, figures 3 and 6, one sees that most of the points are surprisingly close to their regressions. The scatter around the best line of the groups taken as a whole is largely due to differences between individuals. The slopes of the regressions differ from one subject to another, but if the slopes are arranged according to the age of the subjects, it becomes evident that age is an important factor in this difference, although it may not be the whole story. Thus, in figure 3, the regressions of R.R. and J.W., the 2 oldest subjects, are the most nearly vertical, while the regressions of M.M. and M.L., the youngest, are much more nearly horizontal. Similarly in figure 6, the most upright regressions, those of E.S. and A.McG., pertain to the 2 oldest subjects, while the most horizontal, V.S. and P.L., are those of the 2 youngest subjects. The effect is that, as one grows older, a given heart work results in a larger pulse pressure. The constant "L" of Apéria's formula (3) is thus found to vary with age.
Fig. 4. Relation between peripheral pulse pressure and stroke volume in 10 cadavers perfused with water. The data secured on the different subjects are represented as follows: A.McG., dots; P.L., triangles with point up; E.B., circles; E. Sco., squares; J.I., crosses; E. Sch., x's; R.P., T's; F.C., diamonds; L.E., triangles with point down; V.S. inverted T's. The solid line is the calculated best line for the group, corresponding to regression equation 98 of table 2. \( r = 0.47 \).

Fig. 5. Relation between peripheral pulse pressure and approximate stroke work. Data from 10 cadavers perfused with water; symbols as in figure 4. The solid line is the calculated best line of the group corresponding to regression equation 99 of table 2. \( r = 0.74 \).

Fig. 6. Relation between peripheral pulse pressure and approximate stroke work in 8 of the 10 subjects perfused with water. The solid lines are the regressions for each subject proper for estimating work from pulse pressure. \( r = \) : for L.E., 0.96; J.I., 0.86; A.McG., 0.53; E.B., 0.85; V.S., 0.88; P.L., 0.95; E. Sco., 0.91; R.P., 0.87. In the 2 remaining subjects the data were so concentrated that the true slope of the regressions could not be defined with confidence.

Therefore, if one estimates left ventricular work from blood pressure, a correction for the age of the subjects will improve the results. The magnitude of the necessary correction has been investigated in 2 ways. In the first, work was compared with pulse pressure divided by age; the results are shown in figures 7 and 8, and the corresponding regression equations are in tables 1 and 2. Considerable improvement has resulted by this means, the standard deviations about the regressions diminishing by 9 and 11 per cent.

To secure maximum improvement from a consideration of the subject's age, and to see if consideration of the subject's size and the size and elasticity of his aorta would also improve estimates of heart work from pulse pressure,
multiple regression equations were computed by Dr. Albert Schild (tables 3–5). In the subjects perfused with blood, the improvement in the estimate of work from pulse pressure and age, gained by using the multiple regression equation 101, is very little better than that secured by simply dividing pulse pressure by age and using the simple regression equation 96; in subjects perfused with water, there is no material gain in using the corresponding multiple regression equation 104 over the ratio equation 100. Also, the additional use of body surface area to provide a factor related to the size of the subject (equation 102), accomplished very little to improve the relationship in either series. The use of pulse wave velocity (equation 107), and the measurements of cross section area of the ascending aorta made at necropsy (equation 108), also provide little improvement, while their combination (equation 109) has a standard deviation indicating a little more scatter than when age (equation 101) is employed alone. These are interesting findings because one would have expected that advancing age affected the results by producing changes in aortic size and elasticity, and that direct measurements of size and elasticity would improve the results far more than age itself in the regression equations.

In looking over the regression equations in tables 1–5, readers may well wonder why, in our search for means to improve the simple estimates of work, we failed to include the most obvious factor, namely the height of the blood pressure itself. This factor does not appear because this aspect of the situation was studied first and the results have already been reported in a previous paper, where multiple regression equations including both pulse pressure and mean blood pressure are to be found. It should be emphasized that the best of the simple regression equations given in this present paper does not provide so good an estimate of cardiac work as can be obtained from the best of the multiple regression equations published in this previous communication. Thus, equation 85, an estimate of cardiac work from pulse pressure, mean pressure, diastolic pressure, and age, has a standard deviation about the regression of 14.9 Gm.M. The corresponding standard deviations for equation 95, an estimate of work from pulse pressure alone, and equation 96, an
TABLE 3.—Multiple Regression Equations, Computed by Dr. Albert Schild from Data Secured in Cadavers Perfused with Blood, Concerned with the Estimation of “Left Ventricular” Work from Clinical Data

<table>
<thead>
<tr>
<th>Equation Number</th>
<th>Equation</th>
<th>Gm. M.</th>
</tr>
</thead>
<tbody>
<tr>
<td>101</td>
<td>True Work Gm. M. = 44.9 + 1.21 pulse pressure mm. Hg - 0.79 age years</td>
<td>15.3</td>
</tr>
<tr>
<td>102</td>
<td>True Work Gm. M. = -51.5 + 1.13 pulse pressure mm. Hg + 31.48 surface area M.²</td>
<td>16.6</td>
</tr>
<tr>
<td>103</td>
<td>True Work Gm. M. = 15.5 + 1.20 P.P. mm. Hg - 0.65 age years + 12.9 surface area M.²</td>
<td>15.1</td>
</tr>
</tbody>
</table>

TABLE 4.—Multiple Regression Equations, Computed by Dr. Albert Schild from Data Secured in Cadavers Perfused with Water Concerned with the Estimation of “Left Ventricular” Work from Clinical Data

<table>
<thead>
<tr>
<th>Equation Number</th>
<th>Equation</th>
<th>Gm. M.</th>
</tr>
</thead>
<tbody>
<tr>
<td>104</td>
<td>Approx. Work Gm. M. = 62.57 + 0.72 pulse pressure mm. Hg - 0.73 age years</td>
<td>15.4</td>
</tr>
<tr>
<td>105</td>
<td>Approx. Work Gm. M. = 15.54 + 0.59 pulse pressure mm. Hg + 3.98 surface area M.²</td>
<td>17.2</td>
</tr>
<tr>
<td>106</td>
<td>Approx. Work Gm. M. = 133.85 + 0.67 P.P. mm. Hg - 0.95 age years - 33.07 surface area M.²</td>
<td>14.9</td>
</tr>
</tbody>
</table>

TABLE 5.—Further Multiple Regression Equations, Computed by Dr. Schild from Data Secured in Cadavers Perfused with Blood, Concerned with Attempts to Improve the Estimation of “Left Ventricular” Work from Pulse Pressure by the Use of Data Not Readily Secured in the Clinic

<table>
<thead>
<tr>
<th>Equation Number</th>
<th>Equation</th>
<th>Gm. M.</th>
</tr>
</thead>
<tbody>
<tr>
<td>107</td>
<td>True Work Gm. M. = 13.60 + 1.30 P.P. mm. Hg - 3.12 pulse wave velocity M. per sec.</td>
<td>17.2</td>
</tr>
<tr>
<td>108</td>
<td>True Work Gm. M. = 15.16 + 1.13 P.P. mm. Hg - 2.22 aortic section cm.³</td>
<td>16.7</td>
</tr>
<tr>
<td>109</td>
<td>True Work Gm. M. = 29.57 + 1.31 P.P. mm. Hg - 3.38 P.W.V. M. per sec. - 2.35 aortic section cm.³</td>
<td>15.4</td>
</tr>
</tbody>
</table>

estimate from pulse pressure and age, are 18.3 and 16.6 Gm.M, respectively. Nevertheless, great interest lies in the fact that the difference in scatter is so small.

Our data indicate that one can make a reasonably good estimate of the work of the left ventricle from the magnitude of the peripheral pulse pressure alone. Indeed, it is a striking but altogether fortuitous fact, clearly shown in figure 2 and equation 95, that the magnitude of the pulse pressure in mm. Hg bears a remarkably close relation to the left ventricular work expressed as Gm.M.

Our data also indicate that in clinical estimations of work from pulse pressure one should expect to detect differences in the left ventricular work of a single patient better than differences between one patient and another. Doubtless, large differences of cardiac work could be detected by palpation of the pulse alone. By considering the patient’s age, estimates of work from pulse pressure could be improved, as the same pulse pressure indicates less work as age advances. On the other hand, consideration of the size of the subject or the size of his aorta is not necessary, an estimate of pulse wave velocity is not necessary, and one does surprisingly well without considering the height of the blood pressure.

It is of great interest to compare the accuracy of such an estimate of cardiac work, made from the pulse pressure alone (equation 95), with the accuracy secured by estimating cardiac work from the height of the blood pressure.
alone, for certainly most doctors now consider the latter to be the best simple clinical method of estimating the work of the heart. Accordingly, figure 9 shows the relation between mean peripheral mean blood pressure and true work. Data from 6 cadavers perfused with blood. Mean blood pressure = \( \frac{1}{2} \) (systolic + diastolic pressure). The calculated best line corresponds to equation 97 of table 1. \( r = 0.69 \).

![Fig. 9. Relation between peripheral mean blood pressure and true work. Data from 6 cadavers perfused with blood. Mean blood pressure = \( \frac{1}{2} \) (systolic + diastolic pressure). The calculated best line corresponds to equation 97 of table 1. \( r = 0.69 \).](image)

I repeat that this was a surprising finding, but, on reflection, an aspect of the situation became apparent to me that went far to explain it and helped me to understand how it was possible that the pulse pressure could provide such a good estimate of the heart’s work without considering the height of the blood pressure at all. Figure 10 may give more persons a better grasp of my explanation for this unexpected finding than would a detailed exposition in the text. The explanation lies in an important difference between the magnitude of the pressure changes occurring in a tense system and those occurring in a flaccid system, when fluid is injected into each.

Another analogy has helped some people to understand this finding: in a tense system, a small volume injected causes a high rise of pressure; in a flaccid system, a large volume must be injected to cause a same rise of pressure.

![Fig. 10. A schematic diagram to illustrate the reason why the relation between heart work and pulse pressure is so largely independent of the height of the blood pressure. In the situation pictured at the top, at high pressure, WORK = Small stroke volume \times \text{large mean pressure. In the situation pictured at the bottom, at low pressure, WORK = Large stroke volume \times \text{small mean pressure. Therefore, when pulse pressures are similar WORK is quite similar, despite differences in height of blood pressure.}](image)
UNDERSTAND better what I have in mind. Suppose one patient has a blood pressure of 120/80 mm. Hg, another a pressure of 220/180; could their hearts be doing the same amount of work? It takes the injection of only a little fluid to produce 40 mm. pulse pressure at the higher level of blood pressure, and the work performed is roughly analogous to carrying 1 bucket of water up 2 flights of stairs; at the lower pressure level the injection of much more fluid is required to raise the pressure 40 mm., and the work performed may be likened to carrying 2 buckets of water up 1 flight of stairs.

I must emphasize that I have not said that the heart does not perform increased work in hypertension. Certainly, in a great majority of these cases, not only is the blood pressure elevated, but the pulse pressure is increased also. In such cases there is abundant evidence that heart work has increased and this is a reasonable explanation for the cardiac hypertrophy so often found. Nevertheless, cases of prolonged hypertension without cardiac enlargement are fairly common. I am hopeful that we now have a rational explanation for what appeared to be an inconsistency.

I must close as I began with some reflections on the countless doctors who, in the past, have felt the pulse of their patients to determine how the heart was "working," without attempting to employ that term in its exact sense. Although the art today is largely neglected, as a young man I recall elderly practitioners whose fingers automatically sought the wrist of any patient before them. Doubtless their spokesman was Mackenzie, who wrote,2 "It is the strength of the left ventricle that we are gauging when we seek to determine the strength of the pulse." This view is strongly upheld by the results of this investigation.

**Summary**

The relationship between peripheral pulse pressure and left ventricular work, predicted by Apéria from theoretical considerations, has been explored by a statistical investigation of results secured in experiments in which systole was simulated in cadavers.

From results secured in 51 simulated systoles in 6 cadavers in which blood was used as perfusing fluid, "left ventricular" work had been estimated by integration of central blood pressure and cardiac ejection curves by Dr. Walter Feder. The correlation coefficient between this "true" work and the peripheral pulse pressure was 0.86. In the same data, the correlation coefficient between stroke volume and peripheral pulse pressure was only 0.40.

In results secured in 93 simulated systoles in 10 cadavers perfused with water, "left ventricular" work was estimated approximately as the product of stroke volume and mean central blood pressure. The correlation between this work and the corresponding peripheral pulse pressure was 0.74. In the same data, the correlation between stroke volume and peripheral pulse pressure was only 0.47.

Therefore, our results demonstrate clearly that peripheral pulse pressure is much more closely related to the work performed by the left ventricle than to its stroke volume.

The relation between pulse pressure and stroke work in individuals is closer than that in groups, for the slope of the regression differs from subject to subject, being related to the age. Therefore, consideration of the age of the subject improves one's ability to estimate left ventricular work from peripheral pulse pressure. Consideration of the size of the subject, the pulse wave velocity, and the size of the aorta, leads to little additional improvement in the estimation. The use of the height of the mean peripheral blood pressure benefits the estimation much less than was expected.

The magnitude of the pulse pressure (used alone) provides a much better estimate of the left ventricular work performed than does the height of the mean blood pressure (used alone).

While the simple estimates of cardiac work from pulse pressure here described are not as accurate as are the estimates of such work made by the more elaborate equations published previously, they seem accurate enough for many clinical purposes, especially for the detection of changes in cardiac work in single patients.

The data indicate that by careful consideration of the pulse pressure, or indeed perhaps simply by skillful palpation of the pulse, any clinician could detect the larger deviations in the work being performed by the left ventricle
without making any numerical computation whatsoever.

**SUMMARIO IN INTERLINGUA**

Le relation inter peripheric pression pulsatile e labor sinistro-ventricular, predicite per Apériu ab considerationes theoretic, esseva explorate per medio del investigation statistic de resultatos obtenite in experimentos in que systole esseva simulae in cadaveres.

Super le base de resultatos obtenite in 51 simulate systoles in 6 cadaveres (in que sanguine esseva usate como fluido de perfusion), le labor “sinistro-ventricular” esseva estimate per Dr. Walter Feder per medio del collation de curvas del pression de sanguine central con curvas del ejection cardiac. Le correlation de iste “ver” labor con le peripheric pression pulsatile habeva un coefficiente de 0,86. Le mesme datos mostrava un correlation inter volumine per pulso e peripheric pression pulsatile con un coefficiente de solmente 0,40.

Super le base de resultatos obtenite in 93 simulate systoles in 10 cadaveres (in que aqua esseva usate como fluido de perfusion), le labor “sinistro-ventricular” esseva estimate approximativamente como producto de volumine per pulso e pression de sanguine central medie. Le coefficiente de correlation inter iste labor e le correspondente peripheric pression pulsatile esseva 0,74. Le mesme datos mostrava un coefficiente de correlation de solmente 0,47 inter volumine per pulso e peripheric pression pulsatile.

Assi nostre resultatos demonstra claramente que peripheric pression pulsatile es molto plus nettemente correlationate con le labor del pulso cardiac que con le volumine per pulso cardiac.

Le relation inter pression pulsatile e labor de pulso in individuos es plus stricte que in gruppos, proque le inclino del regression differre ab un subjecto al altere in relation al etate. Per consequente, le consideration del etate de un date subjecto meliora le possibilitate de estimar le labor sinistro-ventricular super le base del peripheric pression pulsatile. Le consideration del largor del subjecto, del velocitate del unda pulsatile, e del dimension del aorta adde paucu al precision del estimationes. Le uso del valor medie del peripheric pression de sanguine beneficià le estimation multo minus que lo que nos haberea expectate.

Le magnitude del pression pulsatile (usate sol) provide un mulito melior estimation del labor cardiac que le valor medie del pression sanguine (usate sol).

Ben que le simple estimationes del labor cardiac super le base del pression pulsatile (secundo le supra-descritite metodo) es minus accurate que le estimationes del labor cardiac per medio del complexe computationes prevemente publicate, illos pare esser satis accurate pro multe objectivos clinic, specialmente pro le detection de alterationes del labor cardiac in patientes individual.

Le datos indica que le caute consideration del pression pulsatile—o forsan mesmo solmente le habile palpation del pulso—permite a omne clinico deterger le plus marcate deviationes in le labor del ventriculo sinistre, sin recurso a ulle computation numeric del toto.

**REFERENCES**


The authors studied a group of 29 patients in whom arterial reconstruction has been performed for the treatment of arteriosclerosis obliterans involving the iliofemoral arterial trunk. Twenty-one operations were done. The follow-up period was from 2 to 29 months. Seven of the patients had associated diabetes. Only individuals with no palpable pulsations below the iliac or femoral areas but with patent popliteal arteries, as demonstrated by arteriography, were considered to be suitable candidates for the operation. Homologous arterial and autogenous venous grafts were used.

All but 3 patients left the hospital with a patent graft. Of the 16 arterial grafts, 9 closed within 7 months. Of the 13 autogenous vein grafts, 8 reclosed, 6 within 6 months and the other 2 at 8 and 9 months.

In the 19 cases of closure, 4 limbs were improved, 9 showed no change and 6 were made worse. All of the latter came to amputation.

Twelve grafts continued to function. Seven of these were arterial and 5 were venous. Seven of the patients were followed over 6 months.

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