The Genesis of the Aortic Standing Wave

By Robert S. Alexander, Ph.D.

On the basis of observations of arterial pulse forms in anesthetized dogs, a hypothesis is developed to account for the genesis of the aortic standing wave which is responsible for the dicrotic characteristics of pulses recorded from the abdominal-femoral system. A sudden lowering of resistance to pulse wave propagation in the region of the diaphragm and an increase in resistance in the peripheral femoral bed produce reflections which, because of the equivalent wave-lengths of the thoracic and abdominal-femoral segments of the aortic system, create a resonant oscillation of the aortic blood column between the root of the aorta and the femoral bed.

The precise mode of origin of the dicrotic undulations in pressure observed in arterial pulses has long remained an enigma. The time relationships of these pressure waves exclude the possibility that they might be simple transmitted manifestations of the rebound of blood against the aortic valves at the end of systole. Frank recognized that the dicrotic waves resembled a low frequency vibration of the entire aortic system. Experimental proof of this concept was presented by Hamilton and Dow who showed that the dicrotic waves were pressure surges that occurred simultaneously throughout the system, oscillating about a node located near the diaphragm. To distinguish them from the transmitted pulse wave, they have identified these oscillations as the aortic "standing wave."

The physical phenomena responsible for these standing waves, however, have not been satisfactorily explained. Frank suggested that the initiating factor was a sudden drop in resistance to the advancing pressure wave at the point at which it reached the iliac bifurcation. If this were so, occlusion of the aorta at this point should be expected to invert the phase of the standing wave system. Experimental tests of this hypothesis have failed to confirm such a supposition. Hamilton and Dow explained the genesis of these oscillations by the sudden increase in resistance as the advancing pressure wave reached the peripheral arterioles. We have used a modified form of this concept in previous studies, although we have become convinced that it fails to offer a satisfactory explanation for the actual genesis of the oscillation. The major argument against this latter concept is that, as apparently recognized by Hamilton, there occurs, simultaneously with the arrival of the pulse wave at the periphery, a deficit in pressure at the root of the aorta. Such a negative pressure variation in the aorta could not be explained by peripheral reflection of the pulse wave.

We have recently proposed a new hypothesis for the genesis of the standing wave which, while taking cognizance of the increased resistance in the femoral bed, also stresses the importance of a decrease in resistance to pulse wave propagation that is encountered in the splanchnic region. The purpose of this communication is to present the experimental evidence in support of this hypothesis.

Methods

These observations were obtained from acute experiments on anesthetized dogs in which lateral pressures were recorded from unoccluded vessels by means of membrane manometers as has been described previously. Central aortic pressures were recorded through a cannula introduced through the left carotid until its tip came to reside at the base of the innominate artery. Abdominal aortic pressure was obtained by performing a left nephrectomy and introducing a cannula via the remaining stump of the renal artery. To perform experiments on the aorta at the level of the diaphragm, a transthoracic approach was used which involved resection of the tenth and eleventh ribs on the left and dissection at the region of the hiatus so as to expose freely the desired vessels.

For optimal identification of the standing wave pressure undulations, particularly in the central
aorta, it is essential to have a moderately slow heart rate and good stroke volume. When necessary, this was achieved in these experiments by applying weak faradic stimulation to the right vagus nerve. In all instances where such a vagal bradycardia was employed, control observations were made without vagal stimulation to verify that the vagal stimulation itself had not qualitatively altered the standing wave relationships.

**RESULTS**

**The Contribution of the Standing Wave to Pulse Contours**

Figure 1 illustrates pressure pulses in an anesthetized dog in which the standing wave components are particularly well shown. It will be noted that, occurring approximately simultaneously with the femoral systolic peak, there is a peaking of the abdominal aortic pulse and a dip in pressure in the pulse recorded at the arch. This dip in pressure during late systole in the arch, the "preincisural slump" (fig. 1-I), is followed by a definite rise in pressure, the "postincisural hump" (fig. 1-II) which occurs at the same time that the dicrotic dip is observed in the abdominal and femoral pulses. Persistence of this undulating feature in the central aortic pressure is observed (fig. 1-III, IV) occurring simultaneously but 180 degrees out of phase with the dicrotic pressure oscillations in the lower aortic system. These pressure variations correspond to the analysis of the standing wave system as presented by Hamilton and Dow.

To make absolutely certain that the standing waves are not related to cardiac phenomena associated with the termination of systole and the closure of the aortic valves, it would be desirable to separate the standing waves from the end of systole. A particularly instructive illustration of this has come to our attention in studies of the cardiac affects of hypothermia being conducted in this laboratory by Dr. Robert M. Berne. Figure 2 presents typical curves obtained by Dr. Berne, to whom we are indebted for this illustration. On the left is shown a control pulse with a pressure of 157/122, recorded while the animal had a normal rectal temperature of 38.5°C, recording on the right obtained after rectal temperature had been lowered to 26.0°C. With the great prolongation of systole produced by cooling, the incisura (INC.) is markedly delayed. The standing wave shows some prolongation which can be explained as due to the lowered pressure level, but this delay is not nearly as great as is the prolongation of systole. In consequence, there is now a dip in pressure near the ankleotic crest (I) and a hump in pressure in midsystole (II). These recordings demonstrate that the normal position of the standing waves, in association with the incisura of the central pulse, is coincidental. The explanation of the standing wave genesis must therefore reside not in any direct cardiac factor but in some phenomenon which occurs as the pulse wave progresses through the aortic system.

**The Time and Site of Origin of the Standing Wave**

To determine the moment of first appearance of these undulations in pressure, experi-
Ments from eight dogs have been selected in which the standing wave components in the central pulses were particularly prominent. Measurements of these recordings revealed that the time interval between the initiation of the pulse wave and the start of the standing wave is approximately the same as the round trip transmission time of the foot of the pulse between the aortic arch and the origin of the renal arteries, as shown in the first two columns of table 1. The third column of table 1 shows a further correspondence of these data with the half-period of the standing wave as estimated on the same recordings in terms of the time interval between the dicrotic dip and the positive dicrotic wave in the femoral pulse, measured in reference to a line constructed to indicate the mean rate of pressure fall. The data of table 1 might therefore suggest that the initiation of the standing wave phenomena is related to the arrival of the pulse wave at the level of the renal artery.

Since the speed of propagation of the foot of the pulse wave is somewhat more rapid than that for the succeeding portions of the wave,\(^8\) however, measurements based upon the transmission rate of the foot of the pulse will exaggerate the actual anatomic distance over which the returning wave traveled. When this correction is introduced, the conclusion is reached that the phenomena giving rise to the initiation of the standing wave occur at the time that the foot of the pulse wave reaches the approximate level of the diaphragm.

A Working Hypothesis for the Genesis of the Standing Wave

The localization of the point of origin of the standing wave and also its node at the level of the diaphragm prompts inquiry into the anatomic peculiarities of this region. As has been emphasized elsewhere,\(^7\) the pulse traverses a considerable length of thoracic aorta without encountering any major branches. On passing through the diaphragm, however, it reaches a series of very large vessels represented by the celiac and superior mesenteric arteries followed shortly by the two renal arteries. In terms of cross sectional areas, the lower thoracic aorta has an area of 1.15 sq. cm., while the celiac and superior mesenteric arteries together with the abdominal aorta just below represent an area of 1.39 sq. cm. Including the renal arteries would raise this to a total cross sectional area of 1.62 sq. cm. Considering these vessels as parallel resistances of unit length, such a widening of the effective lumen should result in a sudden drop in resistance (impedance) to wave propagation, unless one wishes to postulate a sudden change in the modulus of elasticity in the walls of the vessels to counteract this change in cross sectional area.

Basic principles of fluid dynamics dictate that, with an increase in resistance, there should be a momentary pyramiding of pressure energy in the wave front with the resulting retrograde reflection of a positive pressure wave; when the wave front encounters a point of lowered resistance, there should be an attenuation of the pressure rise with the generation of a retrograde wave negative in sign. Thus, when the front of the pulse wave transmitted down the thoracic aorta encounters the lowered resistance at the level of the diaphragm, there should result a surge towards the femoral bed with a corresponding decrement in pressure transmitted back to the arch, thereby accounting for the preincisural slump. As soon as the wave reaches the peripheral femoral bed, however, an increase in resistance is encountered which produces a marked peaking of pressure in the femoral pulse. This would result in a positive reflection and a rebound of the aortic blood column.

The basis of the standing wave oscillation resides in the fact that the retrograde trans-

\(\text{Table 1}\)

<table>
<thead>
<tr>
<th>Dog No.</th>
<th>Foot of Aortic Pulse to Beginning of Standing Wave; milliseconds</th>
<th>Transmission Time to Renal Artery X 2; milliseconds</th>
<th>Standing Wave Half-period*; milliseconds</th>
</tr>
</thead>
<tbody>
<tr>
<td>30</td>
<td>63</td>
<td>60</td>
<td>68</td>
</tr>
<tr>
<td>31</td>
<td>62</td>
<td>65</td>
<td>69</td>
</tr>
<tr>
<td>33</td>
<td>62</td>
<td>64</td>
<td>68</td>
</tr>
<tr>
<td>34</td>
<td>86</td>
<td>90</td>
<td>81</td>
</tr>
<tr>
<td>36</td>
<td>82</td>
<td>82</td>
<td>89</td>
</tr>
<tr>
<td>62</td>
<td>112</td>
<td>106</td>
<td>115</td>
</tr>
<tr>
<td>64</td>
<td>89</td>
<td>84</td>
<td>84</td>
</tr>
<tr>
<td>68</td>
<td>102</td>
<td>96</td>
<td>108</td>
</tr>
</tbody>
</table>

* Estimated from the dicrotic waves in the femoral pulse as described in text.
mission of the initial surge at the diaphragm back to the root of the aorta requires the same time as for the forward pulse wave to reach the resistance of the femoral bed. We therefore have two "closed" segments with

![Diagram of pulse waves](image1)

**Fig. 3.** Effects of progressive aortic constriction at the level of the diaphragm upon central aortic and femoral pulse pressures (above) as compared with the postincisural standing wave component in the central aortic pulse (below).

**Test of the Hypothesis**

The simplest manner of testing the basic validity of this hypothesis would be to eliminate the reduction in resistance which occurs normally at the diaphragm. Closely related experiments have already been reported by Gupta and Wiggers, who noted obliteration of dicrotism in the femoral pulse incidental to studies of experimental coarctation. We have repeated their experiments in modified form by placing an adjustable clamp around the aorta at the level of the diaphragm just above the origin of the celiac axis. To estimate the magnitude of the standing wave component in the central aortic pulse, the height of the postincisural hump was approximated by drawing a straight line from the incisura to the end of diastole and measuring the maximum deviation from this line during the postincisural interval. As shown in figure 3, constriction of the aorta by gradual tightening of the clamp leads to a progressive reduction in femoral pulse pressure, some augmentation of central aortic pressure, and a progressive reduction in the magnitude of the postincisural hump. It is significant that the postincisural hump disappears at the point at which femoral pulse pressure has been reduced to equal the aortic pulse pressure, as indicated in figure 4 by the crossing of the pulse pressure plots. Still

![Diagram of pulse waves](image2)

**Fig. 4.** Central aortic and femoral pulses during progressive aortic constriction showing control (left), sufficient constriction to produce equal pulse pressures (middle), and constriction sufficient to almost obliterate the femoral pulse (right).
further constriction of the aorta leads to a reduction in femoral pulse pressure to values significantly below those in the aortic arch and the appearance in the central aorta of a preincisural hump in pressure followed by a negative variation in pressure after the incisura. The actual change in pulse contours in this type of experiment is illustrated in figure 4 which demonstrates the disappearance of the normal aortic standing wave with moderate constriction (middle tracing) and the creation of a new reflected wave with opposite phase relations by extreme constriction (righthand tracing).

The Importance of the Splanchnic Bed

Further evidence that the low resistance associated with the origin of the splanchnic bed is of major importance in the genesis of the standing wave was obtained by experimentally altering this route of aortic drainage. After transthoracic exposure of the aorta as it passed through the diaphragm and dissection so as to visualize the celiac and superior mesenteric arteries, a control pulse recording was made and then these two arteries were immediately occluded with hemostats. A second pulse recording revealed changes in contour as indicated on the left of figure 5. The preincisural slump and the postincisural hump, which are obvious in the control recording, are absent on the curve with occlusion of these arteries. The femoral pulse still exhibits dicrotic waves, but the form of these waves has changed considerably and measurements reveal a marked alteration in their timing.

The converse experiment was performed by injecting 0.2 mg. of histamine into the celiac axis. As shown on the right of figure 5, dilation of the splanchnic bed leads to an exaggeration of the preincisural slump in pressure and a more pronounced positive wave following the incisura. Because of the large amount of drainage from the central aorta into the splanchnic bed, the standing wave component in the pulses, which is initially exaggerated, becomes rapidly damped, accounting for the reduced dicrotic waves in the femoral pulse.

**DISCUSSION**

A definitive analysis of this problem is made difficult because pulse waves in the arterial system are distorted in transmission by the hysteresis properties of the vessels, and by local reflection phenomena. One is therefore unable to make accurately quantitative measurements of wave length or wave amplitude, as would be demanded for a rigorous solution to the problem. Nevertheless, we feel that the evidence presented above offers some substantial support for a working hypothesis as to the genesis of the standing wave system.

As described here, the standing wave is dependent upon four requirements: (a) a functionally closed system at the root of the aorta, (b) an "open" system (lowered resistance) at the level of the diaphragm, (c) a relatively closed system at the femoral bed, and (d) matched wave lengths in the thoracic and abdominal-femoral segments so as to produce resonance. In accord with these re-
quirements, opening the system at the central end by aortic insufficiency, closing off the drainage into the splanchnic bed (fig. 5) or opening the system at the peripheral end by creating femoral arteriovenous fistulae all act to reduce or abolish dicrotism and the aortic standing wave system.

In referring to the systems as "open" or "closed" or having lower or higher "resistance," it should be understood that these properties should not be confused as being synonymous with the familiar concept of Poiseuille resistance to blood flow. As employed here, "resistance" refers to the degree of impedance to forward propagation of the pulse wave as determined only in part by blood flow; of greater significance in pulse propagation is the cross sectional area and the modulus of elasticity of the walls of the blood vessels which transmit the pressure wave. In basing the argument for resistance changes of this latter type upon changes in cross-sectional area alone, it is assumed that there are not opposite changes of the elasticity properties of the vessels of sufficient magnitude to negate the changes in cross-sectional area. Direct confirmation of this assumption would require an intimate knowledge of both the elastic properties of the vessels and also the Laplacian effects associated with the exact geometry of the wave front entering the regions of bifurcation. This would require data of a considerably higher degree of accuracy than are at present available. By analogy with physical systems, however, it might be noted that distortion of an advancing wave front is the accepted basis for concluding the presence of a change in impedance to wave propagation.

It should not be inferred that the standing wave is the only source of dicrotism. Any type of reflection process, if properly timed, may yield dicrotic features in any specific pulse. In figure 5 the femoral pulse continues to exhibit dicrotic characteristics in spite of the abolition of the normal aortic standing wave. As Hamilton has previously suggested, there may be several segments of the vascular bed which are capable of being thrown into some type of pressure oscillation. Experimental occlusions of the aortic system at various sites can produce standing waves of different frequencies and quite different node locations than that observed normally. The standing wave system described here, therefore, while appearing to represent the normal source of dicrotism in the femoral system of the dog, is perhaps best regarded as a prototype of this form of dynamic distortion of the pulse wave in the arterial system.

SUMMARY

A standing wave oscillation of the aortic blood column, which is the dominant source of the dicrotism observed in the femoral arterial pulses of the normal dog, is described as originating because of the lowered resistance associated with the origin of the major splanchnic vessels at the level of the diaphragm and the increase in resistance in the femoral bed. Negative and positive pressure waves produced when the advancing pulse wave encounters these changes in resistance create a disturbance in the aortic system which, due to the equivalent wave lengths of the thoracic and abdominal-femoral segments, results in a resonant oscillation of the entire aortic blood column. This hypothesis appears adequate to explain the standing wave pressure undulations observed in normal pulses, and has been given experimental support by altering aortic or splanchnic resistance and recording changes in pulse form that are in accord with this concept.

REFERENCES

6 Hamilton, W. F.: The patterns of the arterial
7 Alexander, R. S.: Factors determining the con-
tour of pressure pulses recorded from the aorta.
8 Hamilton, W. F., Remington, J. W., and Dow,
P.: The determination of the propagation ve-
locity of the arterial pulse wave. Am. J. Physiol.
144: 521, 1945.
9 Gupta, T. C., and Wiggers, C. J.: Basic hemo-
dynamic changes produced by aortic coarcta-
10 Remington, J. W., Hamilton, W. F., and Dow,
P.: Some difficulties involved in the predica-
tion of the stroke volume from pulse wave velocity.
11 Bramwell, J. C., and Hill, A. V.: The formation
of "breakers" in the transmission of the pulse
12 Alexander, R. S.: Unpublished observations.
The Genesis of the Aortic Standing Wave
ROBERT S. ALEXANDER

doi: 10.1161/01.RES.1.2.145

_Circulation Research_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1953 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7330. Online ISSN: 1524-4571

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circres.ahajournals.org/content/1/2/145