Role of Abdominal Aortic Branches in Pulse Wave Contour Genesis

By Joseph M. Ryan, M.D., Ralph W. Stacy, Ph.D. and Robert N. Watman, M.D.

Central and peripheral pulse wave contours were recorded in dogs before and after occlusion of the major visceral arteries. Comparison of the pulse wave contours indicates that occlusion of the visceral branches produces very little effect upon the wave form of the central and peripheral pulse. The major changes noted were slight shifts in the timing of reflected components, and it would appear that the timing changes can be explained on the basis of the increase in pulse wave velocity attendant with a rise in the arterial blood pressure. Both the importance of these findings in the understanding of pulse wave contour genesis and the basic assumptions of the standing wave hypothesis are discussed.

It is generally considered that the pulse wave contour (central and peripheral) is the result of summation of a single fundamental resulting from the ejection of blood by the heart into the aorta and a series of reflected energies originating at points of change of the resistance to pulse wave transmission in the system. These points of resistance change have been considered by Alexander and others to be a point in the abdominal aorta immediately below the diaphragm and a point in the femoral system corresponding to the functional branching of the large arteries. In the case of the abdominal reflection point, the change of pulse wave transmission resistance is assumed to be from a higher to a lower resistance, while that of the femoral bed is assumed to be from a lower to a higher resistance. Reflected components from these points would, therefore, be negative pressure reflections and positive pressure reflections respectively.

Alexander's measurements of the pulse wave contours before and after clamping the major abdominal vessels would appear to demonstrate conclusively that clamping off of these vessels produces major alterations in the reflected components. In his experiments, however, the clamping was applied only to the celiac and superior mesenteric arteries, while one of the renal arteries was permanently closed and the other was apparently left open. Furthermore, Alexander's analysis did not include consideration of the effects of changes of mean pressure; there also remained a possibility that the mass of the occlusive hemostats themselves altered the mechanical system sufficiently to provide changes in the pulse wave contour.

In view of these points of question, it was considered advisable to repeat the experiments with special attention on the techniques for occluding the major abdominal arteries.

Methods

Measurements were made on a total of 8 adult mongrel dogs, anesthetized with intravenous pentobarbital sodium. Long metal cannulae were inserted into the right carotid artery and the right femoral artery and pushed inward until the opening lay at the root of the aorta and the bifurcation of the iliacs respectively. These cannulae were made of long no. 10 spinal needles.

Each cannula was attached to a Statham type P23D physiological pressure transducer. The Statham gages were powered with a standard DC control box and the output was fed into a chopper type amplifier. This amplifier operated at a carrier frequency of 50,000 c.p.s so that its frequency response was flat to well above the maximum frequency response of the pressure pickups. The output of the amplifier was recorded by means of a Hathaway oscillograph using galvanometers with a frequency response flat to 2,000 c.p.s.

The control boxes and amplifiers were adjusted so that the 0 set and excursions on the 2 channels represented exactly the same positions and sensitivities (5 cm. final excursion per 100 mm. Hg) causing superimposition of the central and femoral pulse.

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In the course of an experiment, recordings of central and peripheral pulses were made before opening the abdomen; after opening the abdomen; after placing ties on the superior mesenteric, celiac, right and left renal and inferior mesenteric arteries; and finally, after tying off these arteries. The ties were made with fine suture material so that little or no mass was introduced into the system.

Comparison of the pulse wave contours before and after ligation was made by transferring equivalent central and peripheral pulse curves to tracing cloth, one on top of the other. In most cases this necessitated an adjustment of the 0 pressure level. Note was taken of such adjustment.

**RESULTS**

An example of the curves obtained in this study is shown in figure 1. A typical comparison picture of a central pulse is shown in figure 2A and a typical picture of a peripheral pulse is shown in figure 2B. The shaded areas in these curves represent the changes which have occurred on tying off the major abdominal arteries. It will be noted that one of these major changes is a lessening of the slope of the preincisural slump. The postincisural hump appears earlier and is very often greater in magnitude. In a few cases (where the pulse rate was slow enough) there appeared a secondary wave near the end of diastole. This wave was always small in magnitude.

In the peripheral pulse, the major change seemed to be a shift of the dicrotic wave. The wave appeared earlier and was narrower. This made the dicrotic notch appear shallower in many cases. In both the central and peripheral pulses the pulse pressure was usually greater after ligating the major abdominal arteries.

In 7 of the 8 experiments performed there was a distinct increase in both systolic and diastolic pressures after the vessels were ligated. The mean pressure elevations corresponding with these changes ranged from 20 to 45 mm. Hg. In the 1 case where the pressures did not change appreciably, the alterations noted in the pulse wave contours were minimal.

Actual quantitation of the relation between pressures and the velocity of propagation of transmitted and reflected components is difficult to achieve. Table 1 represents our efforts in this direction giving average data obtained from our experiments.

**Fig. 1.** Original tracings of central and peripheral pulse waves. Note (a) the increase in mean pressure attendant on ligation of abdominal aortic branches, (b) the shift of postincisural and postdicrotic notch waves, and (c) that we have deliberately lowered the central pressure (ordinate) an amount equivalent to 10 mm. Hg to prevent obscuring some changes. Time marks = 0.1 sec.

**Fig. 2.** Effect of ligation of major abdominal aortic branches on pulse wave contours.

It may be seen that these values reveal an increased pulse wave velocity and a decreased reflection time, and that these changes are concomitant with increases in the systolic and diastolic pressure levels.

It is worthy of note that in 3 of the 8 experiments pulsus alternans developed a few minutes after tying the major abdominal vessels. The pulsus alternans persisted for more than one-half hour after the ties were made, and was
Table 1.—Some Values Indicating Transmission and Reflection Conditions in the Arterial System Before and After Ligation of the Major Abdominal Aortic Branches

<table>
<thead>
<tr>
<th>Condition</th>
<th>Pulse wave velocity (central to peripheral)</th>
<th>Pressures</th>
<th>Reflection time*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Aortic</td>
<td>Femoral</td>
</tr>
<tr>
<td>Ties in place…..</td>
<td>6.0 M./sec</td>
<td>128/103</td>
<td>142/100</td>
</tr>
<tr>
<td>Branches ligated</td>
<td>7.5 M./sec</td>
<td>154/122</td>
<td>190/120</td>
</tr>
</tbody>
</table>

* The "reflection time" is arbitrarily selected as the time between the foot of the initial pressure wave and the peak of the reflected wave, i.e., the postincisural hump of the central pulse or the dicrotic wave of the peripheral pulse.

still present at the termination of the experiment.

DISCUSSION

Careful examination of the pulse wave contours indicates that the change in the preincisural slump may not be an alteration in the magnitude of the slump itself, but rather an interference with the slump due to moving of the postincisural hump forward on the pulse curve. It is unfortunate that the incisura obscures some of the details of these changes.

The alterations noted in the peripheral pulse are clear and straightforward. The magnitude of the wave which follows the dicrotic notch appears to be increased somewhat and the wave definitely appears earlier in the sequence of events. The total energy of this wave does not appear to be altered appreciably (i.e., the area under the curve remains about as it was).

In the 1 instance where the pressures did not change appreciably, the peripheral pulse contour changes were also minimal.

The fact that all of these changes are in timing and magnitude of the reflected components of the pulse wave contour, and that in every case where such changes appeared there was a concomitant major increase in all arterial pressures, indicates the existence of a significant relationship between the pressure levels and the occurrence of these reflected waves. Since the pulse wave velocity increases with increased pressure, the relationship between these factors is fairly apparent.

It is possible (as Alexander suggests) that the change of the preincisural pressure level represents merely the return of this pressure level to the magnitude which the pressure would have attained in the course of normal drainage had the preincisural slump not occurred. It is, of course, equally possible that the slope level represented by the preincisural slump is actually a level which would be normally produced by simple drainage of the root of the aorta and that the actual reflected components would be a positive wave represented by the foot of the postincisural hump. If the slump is the reflected component, timing would indicate that the source of the slump is indeed 20 to 25 cm. below the root of the aorta. If, however, the postincisural hump is the real reflected component, the distance traveled would be of the order of magnitude of 1 M. in a complete circuit. Thus, the source of the reflection would be about \( \frac{1}{5} \) M. from the root of the aorta or approximately at the bifurcation of the aorta.

The possibility remains, of course, that both types of reflections might occur. The fact that the level of the preincisural slump is altered but very little by the tying off of the major abdominal vessels would indicate, however, that this represents at best a minor component of the pulse wave contour.

The occurrence of the reflected wave in the peripheral pulse is at such a time that the round trip distance would appear to be fairly great. Thus, this reflection must occur at the mouths of very small vessels far down in the femoral bed. The shift of this reflected wave forward on the pulse contour after tying off the abdominal arteries may merely reflect the increase in the pressure in the vessels through which the wave is being propagated. This wave does not appear in the central pulse unless the pulse rate is slowed down to the point where sufficient time is allowed for transmission up the aorta in a backward direction.

Peterson has observed that artificially induced pressure waves are attenuated severely in their travel in a peripheral-central direction. Such a concept is completely in accord with the
results noted here, for all of the "reflected" wave components of the central pulse are small in magnitude, while those of the peripheral pulse are comparatively large.

Lastly, it would appear that the alterations noted by Alexander in his experiments were reasonably accurate in spite of the existing feeling of doubt. A few of the interpretations he has put upon his data, however, are open to some question. It is believed that the exact nature of changes occurring in the region of the incisura are masked by the occurrence of the incisural waves and, therefore, one cannot be sure of the precise nature of the physical system producing these waves.

**Summary**

The work of Alexander on the role of the major abdominal vessels in genesis of the pulse wave contour has been repeated with attention to the method of occlusion and to the inclusion of the renal arteries in the ligation. The alterations in pulse wave contours noted were essentially the same as those noted by Alexander, with the exception that the changes in the pre-incisural slump were relatively minor.

Examination reveals that all of the changes noted can be explained on the basis of increased pulse wave velocity associated with the increase in arterial pressures attendant with occlusion of the major visceral arteries.

It is believed that under the conditions of measurement it is impossible to arrive at definite conclusions concerning the genesis of the reflected waves of the pulse. This is largely due to the occurrence of the incisura at a time when the change from negative to positive reflected pressure components is occurring.

**Summario in Interlingua**

Le investigationes de Alexander in re le role del major vasos abdominal in le genese del configuration del undas pulsative ha essite repetite, con attention prestate al methodo de occlusion e al inclusion del arterias renal in le ligation. Le alterationes in le configurationes del undas pulsative esseva essentialmente identic con illos observate per Alexander, con le exception que le alterationes in le cardita pre-incisural esseva relativamente leve.

Le analyse del datos revela que omne le alterationes observate es explicabile super le base del augmentate velocitate del unda pulsative que es associate con le augmento del pressiones arterial effectuate per le occlusion del major arterias visceral.

Nos opina que sub le conditiones de mesuramento empleate il es impossibile arrivar a definite conclusiones in re le genese del reflectite undas del pulso. Isto es debite in grande mesura al occurrentia del incisura a un tempore quando le alteration occurre ab le negative al positive componente del pression reflectite.

**REFERENCES**

1. ALEXANDER, R. S.: The genesis of the aortic standing wave. Circulation Research 1: 145, 1953. *Note: The references given by Alexander are adequate and reprinting is deemed unnecessary.*

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