THE PATTERNS OF THE ARTERIAL PRESSURE PULSE

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During the past ten years there have accumulated in this laboratory a large number of records of the arterial pressure pulses taken with the Hypodermic Manometer (4). These are faithful records of the changes in the pressure of the blood in various arteries of man and of other animals. The form of the pressure pulse curve is quite variable. Some of the types are drawn roughly to scale in the figures presented herewith, but there are many intergrading examples which can not be shown for lack of space.

It is the purpose of this paper to describe the manner in which these typical patterns take form and to present a simple hypothesis that will account physically for the variations seen in the pressure pulses in different arteries and under different conditions.

The simplest pulse form is that seen in very small animals such as the mouse, canary, frog or turtle (1). During early systole the ejection from the heart is rapid, blood is coming into the arterial tree faster than it is leaving through the arterioles and the pressure in the arteries rises abruptly. Later the rate of ejection falls, the arterial pressure rise continues but becomes less abrupt. Toward the end of systole there is a balance between the rate of ejection and the rate of arteriolar drainage. The curve is then horizontal. An actual decline in the curve is sometimes seen during late systole, when ejection falls to a rate that is less than arteriolar drainage.

During diastole the curve is a perfectly smooth fall of pressure with time. It falls faster at first when the pressure is high. Blood drains out of the arterioles more rapidly and each unit of blood leaving the arterioles makes a greater pressure difference when the pressure is high than when the pressure is low.

This smooth pressure curve is the simple consequence of filling and emptying of the arterial compression chamber. However it is seen only in small animals. The tiny arteries of these creatures respond so quickly that they follow faithfully the pressure changes which are produced by the heart.

In ordinary laboratory animals such as the dog and in man the form of the pressure pulse is much more complex. Superimposed upon the first described filling and emptying curve are waves of arterial origin. This is because the mass of the arterial blood column is so large and the arterial walls are so distensible that the action of the heart sets up oscillations of pressure in the artery that are reflected back and forth over its length as they gradually damp out. The natural period of these waves varies directly with the time it takes the pulse wave to be transmitted from one end of the artery to the other and their amplitude depends upon the abruptness of the initial upstroke of the pulse and the sharpness with which reflection takes place at the end of the artery. These waves, therefore,
differ in different arteries and in different parts of the same artery. They differ with the degree of peripheral constriction, with changes in the blood pressure and can be modified by occluding (pinching) the artery beyond the place where the manometer needle is inserted.

The most important of these waves of arterial origin is the great standing wave of the aorta (2). This wave oscillates about a node which is situated in the descending thoracic aorta. When the wave is reaching its trough above this node it is reaching its peak below. It is called a standing wave because the wave reaches its peaks and troughs simultaneously in all parts below the node. Similarly, above the node the peaks and troughs are simultaneous all the way up the ascending, transverse and descending aorta and even in the ventricle during systole. The wave is called a free pressure oscillation because it is the dying down of a highly damped surging which is determined by the elastic characteristics of the arterial tree and the mass of blood within. The damping factor is supplied by the viscosity of the blood and by incomplete reflection.

Since it is difficult to record the pressure pulses from the node and thus make a recording of the fundamental filling and emptying curve of the aortic compression chamber, it is convenient in many experiments to derive this curve from records taken from points above and below the node. In figure 1 this has been done using curves from the root of the brachiocephalic artery of the dog and from the femoral. The brachiocephalic curve was made with a sound down the carotid artery connected with a hypodermic manometer and the femoral curve was made from a needle inserted into the artery so as not to occlude flow and connected to another manometer. The two curves were redrawn to the same pressure and time scales.

It is seen that the two curves interlace very much as would the curves of two tuning forks, one vibrating strongly and one vibrating weakly and 180° out of phase. The base line on which the oscillations are inscribed goes up and down with the arterial filling and emptying. A smooth curve can then be drawn through the points of intersection to approximate the fundamental filling and emptying curve of the arterial compression chamber. Theoretically it should be closer to the brachiocephalic than to the femoral curve because the latter has the stronger oscillations. It should have the same area as both and should look something like the dotted curve in figure 1. The damped oscillation drawn in below is in phase with the difference between the femoral and fundamental filling and emptying curve.

The standing waves of the carotid artery are superimposed not upon the fundamental filling and emptying curve of the arterial tree but rather upon the pressure curve which actually occurs in the root of the aorta. The standing wave of the aorta and that of the carotid are therefore superimposed upon the fundamental filling and emptying curve to make the pulse pattern as it is seen in the carotid artery.

The carotid standing waves themselves are seen in figure 2. They begin to appear 2 or 3 cm. from the arch and are simultaneous, peak and trough, from there up to the head. They are, therefore, standing waves of the same sort as
those in the aorta. The amplitude of the carotid standing waves may be increased by vasoconstriction and also by pinching the artery between the fingers peripheral to the manometer needle. Their frequency can be increased somewhat by raising the blood pressure (which increases the rate of pulse wave transmission) or greatly and progressively by pinching the artery closer and closer to the needle which is in the root of the neck. This shortens the resonating column over which the pulse wave must be transmitted. It will be noticed from figure 2 that the free vibrations are set off not only by systole itself but also by the aortic pressure changes at the incisura.

Fig. 1. Comparison of the femoral (peaked curve) and aortic pressure pulses. An approximation of the fundamental filling curve of the aortic tree is dotted in. Below is a rough estimate of the free pressure oscillations of the aortic tree.

Fig. 2. Comparison of the pressure pulses in the root of the aorta, and of the carotid artery which starts later and has the transient pulses. Below is a rough estimate of the free pressure oscillations of the carotid tree.

Fig. 3A. Pressure pulses from the two femorals a few seconds after one (the lower curve) has been injected with a small dose of acetylcholine.
B. The same ten seconds later.
C. Pressure pulses in carotid and femoral.
D. The same after a small intravenous dose of acetylcholine.

The relation of the pulse pattern in the aorta and the fore leg arteries has not been determined in the dog. In man the arm arteries do not usually give clear evidence of a separate standing wave system unless the artery has been occluded below the needle (3). Very occasionally there is a brachial standing wave which is recognizable. Its frequency is greater than that of the aorta but considerably less than that of the carotid.

In the dog the hind leg arteries follow the standing wave system of the aorta, the peak and dips being simultaneous all the way down from the aortic node to the dorsalis pedis. In man the dorsalis pedis and femoral pulsations are not
simultaneous (4). They seem to differ according to the same principles as do the aortic and carotid curves. The free pressure oscillations of the large leg arteries are of course much slower than those of the carotid.

Occluding the artery below the manometer needle produces a sharp point of reflection and sets up standing waves of different form than the natural waves. The fact that during occlusion the systolic pressure usually reaches greater height than normal has given rise to the fiction of "systolic end pressure." The rise in pressure (when it occurs) central to the point of occlusion is due to three factors. First, there is an increase in the peripheral resistance over the whole body because the occlusion shunts the blood which would ordinarily go out the arterioles of the stopped artery into the remaining arterioles and it takes a few millimeters more of pressure to force the cardiac output through the fewer arterioles. This is an increase of both systolic and diastolic pressure and is not usually maintained very long. It is regulated against reflexly. Secondly, there is the moot question as to whether the velocity head may not appear as pressure when the artery is occluded. If the artery in question is a branch of the aortic tree, occluding the artery makes it a part of the manometer which measures the pressure below the bifurcation and in the larger trunk where a certain part of the energy is in the form of velocity and a certain part in the form of pressure. Conditions are so complex that it seems impossible to say whether the balance between these two energies will be significantly changed by occluding the branch. Certainly the amount of velocity energy which had been at the needle point is irrelevant in explaining the change in the pressure pulsation upon occlusion. The third and principal factor in explaining the increase in systolic pressure upon occlusion of the artery is the result of setting up an entirely new set of standing waves. These are conditioned by the fact that the artery is stopped and have no relation to pressures or energies present in the unstopped artery.

Normally, the waves of the oscillating aortic system are reflected at one end from the heart or from the semi-lunar valves. At the other end they are reflected, not from large bifurcations but from the terminal arterioles. In these small vessels there is classically an increased resistance to flow which is the physical condition for the reflection of pulsatile waves (2). In case of the secondary systems such as the carotid and human leg arteries the site of peripheral reflection is the same as that of the aortic system, i.e., the arterioles. The central site of reflection is probably the opening of the artery into the aorta which acts as the open end of a sounding tube. In the sense that fluctuations of pressure are minimal and fluctuations of movement maximal it is analogous to a node.

To show that it is the peripheral resistance which is responsible for the reflected waves it is necessary to produce a vasodilation in the peripheral bed of the artery under examination. If this can be done without changing the level of blood pressure and without producing any great change in the manner of wave reflection in other arteries the experiment will be more convincing.

The two femoral arteries were exposed and the needles of two hypodermic manometers (4) inserted in each so as not to obstruct flow of blood. The pulse curves were identical and showed a systolic peak that was 30 or 40 mm. higher
than the systolic pressure in a simultaneous carotid pressure pulse (fig. 3 C). Now a very small dose of acetylcholine was injected into the right femoral artery. The systolic peak in this artery disappeared at once and the pressure during systole became 30 or 40 mm. lower in the right femoral artery than in the left (fig. 3 A). During diastole the pressure in the two femoral arteries was equal, showing that the arteriolar run off was not sufficiently great in the dilated bed to make a continued difference in pressure between the two femorals. The difference seemed to be due simply to the absence of the systolic peak in the injected artery. The systolic pressure pulse in the left femoral artery was almost unaffected and the second wave (negative) was reduced somewhat but equally in both arteries. A few seconds later the second wave had disappeared from both arteries (fig. 3 B). The wave reflected from the periphery had been lessened by general vasodilation so that the second wave cannot be seen. The drug had worked no change in the arterial wall which prevented the transmission of reflected waves because these waves reappeared when the leg was grasped tightly at the knee making a pseudo-vasoconstriction. They could also be brought back by intra-arterial injections of epinephrine in small doses which produced constriction of the dilated arterioles in the femoral bed.

When somewhat larger doses of acetylcholine are given intravenously the standing waves are eliminated from all arteries. The femoral pressure pulse curve is very similar in systolic height to that of the aortic arch, being delayed and smoothed out. In the carotid artery the initial standing waves are eliminated by dilatation in its peripheral bed but there appears another wave that is transmitted up the carotid artery appearing later in the upper parts of the vessel. In appearance it is not very different from the carotid standing wave but it is not always a continuation of the upstroke of the carotid pulse nor is it repetitive as are the standing waves (fig. 3 D). It only occurs when the diastolic pressure in the arterial system as a whole is very low and the arterial column becomes more or less stationary between heart beats. When ejection begins against the inertia of this stationary column, there is a sudden rise of pressure in the aorta and its lower branches. At the same time there is a surge of pressure which travels out the carotid and which accounts for the wave in question.

The sudden increase of pressure in the lower arteries gives rise to the pistol shot sound which can be heard over the artery whenever diastolic pressure gets so low that flow is definitely reduced in rate in these vessels. (Aortic regurgitation, A.V. fistula, etc.) The carotid surge seems to be a sort of back fire from the water hammer that is responsible for the pistol shot sound.

The presence or absence of reflected waves can be used in hypotension to determine the cause of the fall in blood pressure. If it is due to reduced circulatory blood volume and occurs in spite of vasoconstriction, the reflected waves can clearly be recognized. If the primary cause of the low blood pressure is vasodilation or if a terminal vasodilatation occurs its indication can be seen from the absence of reflected waves from the pressure pulse curves.

As the blood pressure falls during an experiment the pulse wave transmission time increases and the frequency of the standing waves decreases. The heart
may accelerate and the second wave be partly or wholly obliterated from the femoral pressure pulse. The second wave of the carotid (or preferably upper aortic) pulse then is the only easy guide to the presence of reflected waves because the femoral systolic pressure may be a little higher than that at the root of the aorta. The reciprocal relationship between such curves is shown in figure 4 and is an indication that there is enough peripheral resistance to cause the reflection of the waves.

On the other hand, blood pressure may be low because of generalized relaxation of arterioles and the reflected waves thereby be eliminated. The arteriolar relaxation may be due to direct action of drugs, to stimulation of depressor receptors from certain internal injuries such as exploration in the upper abdominal region. The relaxation may also result from vasoconstrictor “exhaustion” that follows long continued low blood pressure. In these cases the typical relationship of the pressure pulses is shown in figure 5. The femoral and aortic contours are then very similar and there are none of the reciprocal relationships indicating standing waves which are seen when the blood pressure is low in spite of vasoconstriction. The carotid surge and quick upstroke which are responsible for the pistol shot sound (see fig. 3 D) may not be present if the circulation has seriously deteriorated.

Studies of pulse contours offer certain advantages over plethysmography in determining the degree of vasodilatation and hence in helping to decide whether low pressure is due to vascular relaxation or to oligemia accompanied by vasoconstriction. When blood pressure is extremely low the pulsation of and the blood flow into the various peripheral organs is low whether there is peripheral constriction or not. This is the direct consequence of the greatly reduced blood flow (cardiac output) and this in turn results from the low venous pressure. The venous pressure may be low whether the blood volume is reduced or whether the vascular bed is relaxed.

**SUMMARY**

The pressure pulse patterns in various arteries are described in terms of the filling and emptying of the arterial tree and the added reflected waves which are
contributed by the various arteries. It is shown that these waves are reflected from constricted arterioles and that the form of the pulse may be used to evaluate the rôle of vasodilation in producing hypotension.

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REFERENCES