Pressure Transients Occurring in Diastole in the Central Aorta

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With the development of modern pressure recording systems, and more especially with the advent of currently available catheter tip transducers,* intravascular pressure may now be recorded with an exceedingly high degree of accuracy. Small oscillations in pressure traces that could not previously be clearly distinguished from "catheter-whip" artifact, or that were frequently not seen because of damping in low frequency response systems, take on new meaning and may be studied profitably.

Previous authors, using low frequency response systems, have noticed the presence of definite low amplitude positive waves occurring in arterial tracings during diastole. In some instances, e.g., complete heart block, these waves have been shown clearly to be a result of atrial activity. Early workers have, with painstaking technique, recorded small waves during isometric ventricular contraction. With the use of more modern systems, the recording of central aortic pressure in man, or in the dog, invariably shows a small positive pressure wave immediately preceding the anacrotic limb of the arterial pressure pulse. Although a great many published tracings display such a wave, no adequate explanation for its presence has been offered. Indeed, textbook illustrations of comparative timing of events in the cardiac cycle have recently shown the presence of this wave during ventricular isometric contraction; yet, no mention is made of its origin or significance.

The present communication re-examines the nature of the small positive pressure waves recorded in the central aorta of the dog during diastole under varying circulatory conditions. The instrumentation used permits instantaneous recording of pressure from within any desired cardiac chamber and thereby permits the precise timing of these aortic pressure oscillations with other events of the cardiac cycle.

Methods

Numerous recordings of pressure have been made from all cardiac chambers of healthy anesthetized dogs, of dogs following recovery from myocardial infarction, and of dogs given infusions of pressor amines. All pressures were recorded with the Dallons-Telco variable inductance microtransducer, a catheter tip manometer with a frequency response that has been tested in this laboratory and found to be flat to over 500 cycles/sec. Intracardiac sound was obtained by filtering frequencies below 40 cycles/sec from the pressure output and amplifying the result. This latter function is an integral part of the Dallons-Telco amplifier.

Where indicated, differentiation of left ventricular pressure was obtained by means of the Philbrick P-2 differential operational amplifier* with a time constant such that amplitude was within 5% of correct through 150 cycles/sec. This provided excellent fidelity for the differentiation of all physiologically relevant frequencies contained within the pressure pulse.

The electrocardiogram was taken from a modified lead I with the electrodes placed in the axillae. Care was taken to reduce the resistance between the electrodes to below 500 ohms.

All recordings were made on an Electronics-for-Medicine electron beam photographic recorder.†

*Such as the Dallons-Telco variable inductance transducer, Dallons Laboratories, Inc.; 5066 Santa Monica Blvd., Los Angeles 29, California, and the Statham SP-1 transducer, Statham Transducers, Inc., Hato Rey, Puerto Rico.

†Electronics for Medicine, Inc., 30 Virginia Road, White Plains, New York.
Typical pulse tracing recorded with the Dallons-Telco transducer in the central aorta. Intracardiac sound was recorded simultaneously from the same transducer. Recording speed was 200 mm/sec. Time lines represent .04 sec intervals. From the inscription of the dicrotic notch, the pressure slopes gently to a point .04 sec before systolic upstroke where a small positive wave is written. It is seen to be coincident with the first major component of the first heart sound beginning .12 sec after the onset of the P wave and .04 sec after the onset of the QRS complex of the electrocardiogram. The duration of the wave in this instance is .03 sec.

With this instrumentation, all recordings are virtually instantaneous from the site of origin in the animal preparation. The direct comparison of one to another with respect to timing is valid.

Results

A typical aortic pressure pulse recorded with the Dallons-Telco transducer is shown in figure 1. From the inscription of the dicrotic notch, the pressure slopes gradually to a point, .03 to .04 sec before the anacrotic limb of the systolic pressure pulse where a small positive wave is written. Under normal circumstances, this wave is invariably present, usually positive, but occasionally biphasic. The fundamental frequency of this wave is on the order of 20 to 35 cycles/sec, reflecting a period of .03 to .04 sec. It follows the onset of the P wave of the electrocardiogram by .12 to .16 sec and is coincident with the first component of the first heart sound.

The relationship of the above wave to other events of the cardiac cycle is shown in figure 2, where the aortic pressure trace is compared with left atrial, left ventricular, pulmonary arterial, and right atrial pressure tracings. The aortic wave is seen to coincide in time precisely with the "c wave" of the left atrium and with early isometric ventricular contraction. It may be noted further that the "c wave" of the left atrium is not consistently positive, as classically taught, but may be biphasic. Figure 3 represents three recordings in three dogs demonstrating the variations in appearance of the small presystolic aortic wave, and the variations in appearance of the left atrial "c wave." The relationship of the aortic presystolic wave to the left atrial "a wave" is seen to be variable and a function of the P-R interval, but the relationship to the "c wave" of the left atrium is consistent.

Although the rise of ventricular pressure during isometric contraction appears to be a smooth function, differentiation of the left ventricular pressure pulse reveals a subtle change in slope in early isometric contraction that coincides in time exactly with the aortic presystolic wave and the atrial "c wave" (fig. 4). Thus, there appears to be a pressure oscillation superimposed upon isometric ventricular pressure generation. This, too, is a consistent finding.

In the right heart as well, pressure oscillations are seen to coincide in time with the above-described aortic presystolic wave (fig. 2). Since the rise of pulmonary artery pressure precedes the rise of aortic pressure, the oscillation is frequently seen as a notch on the anacrotic limb of the pulmonary arterial pressure trace, as shown in figure 2C. The simultaneously occurring wave in the right atrium is one of many similar low amplitude waves that may be recorded from that chamber.

In fifteen of the dogs studied, hypertension was induced by norepinephrine and methox...
Relationship of aortic isometric contraction wave to other events of the cardiac cycle.
Details of recording as in figure 1.
A. Isometric contraction wave in the aorta ($A_o$) coincides identically in time with sharp biphasic "c wave" of the left atrium.
B. Isometric contraction wave in $A_o$ is seen to occur during early isometric ventricular contraction. Atrial "a wave" reflection in the left ventricle is seen in end diastole, clearly preceding the isometric contraction wave.
C. Simultaneous recording of aortic pressure with pulmonary arterial pressure reveals that left ventricular isometric contraction is reflected in a notching on the upstroke of the earlier occurring pulmonary arterial anacrotic limb. Small wave prior to systole in pulmonary artery may reflect atrial activity or, more probably, right ventricular isometric contraction.
D. Central aortic pressure compared with right atrial pressure. The very sharp deflection seen following the right atrial "a wave" probably reflects right ventricular isometric contraction, while the succeeding wave represents left ventricular isometric contraction. Their separation is .02 sec. The finding of numerous small pressure waves interposed between the "a wave" and "v wave" in the right atrium is characteristic of this chamber. (Note also the clear reflection of the aortic dicrotic notch in left atrium, pulmonary artery, and right atrium.)

amine. Both drugs induced bradycardia and a pronounced change in form of the central aortic pressure pulse. On occasion, isometric ventricular contraction became more prolonged, and the above-described presystolic pressure wave moved away from the anacrotic limb of the systolic pressure pulse by .01 to .02 sec. Almost simultaneously, a second wave usually appeared in diastole. This wave was longer in duration, more rounded,
Variability in appearance of left atrial "c wave." Details of recording as in figure 1. A. The "c wave" is small and superimposed upon the "x-descent" of the "a wave." B. The "c wave" is wider and biphasic. C. The "c wave" is a complex wave-form. Isometric contraction in this instance was quite brief. In each case, the central aortic presystolic wave follows the "a wave" of the left atrium by at least .06 sec but is in identical time relation with the left atrial "c wave."

and consistently positive. It followed the onset of the P wave of the electrocardiogram by .03 to .05 sec, and occurred simultaneously with the "a wave" of the left atrium and a fourth heart sound that could be recorded from the central aorta (fig. 5). The duration of this wave was .04 to .06 sec, giving it a fundamental frequency of 15 to 25 cycles/sec.

Left ventricular failure and elevation of left ventricular end diastolic pressure was induced in one of several ways. Six dogs previously given myocardial infarction by ligation of the anterior descending coronary artery were either in failure at the time of study or could be put into failure by infusion of moderate doses of pressor amines. In dogs with an intact myocardium, failure was induced by sustained pharmacological hypertension, with or without simultaneous infusion of several hundred milliliters of dextran. The onset of left ventricular failure was usually accompanied by the appearance of a third aortic diastolic wave appearing at the end of ventricular isometric relaxation, a time corresponding to the rapid filling phase of the left ventricle. This was associated further with the appearance of a discernible third heart sound, recorded easily from the central aorta and left ventricle (fig. 6). This wave, when present, was invariably positive, lasted .06 to .10 sec, and only subtly emerged as the normally concave; early diastolic phase of the aortic pressure became convex.

Discussion

Small diastolic arterial pressure waves were first noted by Chaveau and Marey in 1863. Draper in 1910 recorded the superficial pulsations of a giant aneurysm of the ascending aorta and noted two distinct positive waves occurring in diastole. The first was low and broad, appearing two-thirds of the way through diastole; while the second was sharp.
and narrow, immediately preceding systolic upstroke. It is noteworthy that, since the aneurysm presented superficially, it was not necessary to conduct pressure through a long column of saline to the transducer. Thus, the frequency response of the system permitted far more accuracy of detail than had been obtained previously. Webster in 1901 noted that small, broad, positive, aortic pressure waves were seen to occur following each atrial systole in complete heart block. Older textbooks of cardiology gave the phenomenon due notice and attributed the presence of all such diastolic waves, whether in normal sinus rhythm or in complete heart block, to atrial contraction. Disagreement arose as to whether these waves were caused by the bulging of the closed aortic valve as atrial systole was transmitted through the left ventricle or whether they were caused by an extra-arterial impact of the left atrium on the aortic blood column. Garten and Wiggers described a positive pressure wave in the central aorta during isometric contraction "under favorable circumstances." This wave was considered by these authors to be separable from the wave reflections of atrial activity, which they were also able to record. In recent years, two workers have studied diastolic waves in peripheral arteries. Howarth recorded them in normal sinus rhythm, in complete heart block, and in atrial flutter. Heyman made an exhaustive study of the phenomenon in complete heart block, noting that the waves are frequently double humped. Both of these latter authors thought that all such waves were somehow initiated by atrial contraction.

The studies reported here demonstrate the presence of three different kinds of diastolic waves that may appear in central arterial tracings under varying physiological (and pathological) conditions. Their characteristics, as well as their timing with respect to other events of the cardiac cycle, are different.

The first of these waves is consistently recorded in both normal and pathological situations. It coincides with ventricular isometric contraction, the atrial "c wave," and the first major component of the first heart sound. It will be referred to as the isometric contraction wave. That the isometric contraction wave is not seen on routine brachial arterial pulse tracings is partially due to its being damped in transmission from central aorta to brachial artery. Furthermore, since its fundamental frequency is greater than 20 cycles/sec, it would not be recorded by many catheter-transducer systems used for the purpose of recording arterial pressures. The unusual method by which Draper obtained a pulse tracing from a patient with a large aneurysm did, however, permit the recording of this higher frequency event. In retrospect, the second of his two diastolic waves is clearly an isometric contraction wave. In some recent studies where retrograde aortic catheterization has been done, and in which exceeding care has been taken to insure adequate fre-
The isometric contraction wave may be clearly distinguished from other diastolic waves by its relation to the P wave of the electrocardiogram. The isometric contraction wave follows the P wave by more than .10 sec from wherever it is recorded.

The second of these diastolic waves appears in the presence of systemic hypertension and coincides with the atrial "a wave" and the appearance of a fourth heart sound. This wave will be referred to as the atrial contraction wave. It follows the P wave of the electrocardiogram by .03 to .05 sec. Its fundamental frequency is lower than that of the isometric contraction wave, falling within the range of most reasonable catheter-manometer recording systems. It is certainly this wave that was recorded by Francois-Franck, by Webster, by Howarth, and by Heyman. As in the case of the isometric contraction wave, simultaneous waves may be recorded from all chambers of the heart. When this wave occurs in peripheral arterial tracings, it frequently appears as a double humped impulse. This is undoubtedly due to asynchronicity of the two atria.

The third of these diastolic waves appears in the presence of left ventricular failure, coincides with the period of rapid ventricular filling, and is associated with the appearance of a third heart sound. This will be referred to as the ventricular filling wave.

As long as all aortic diastolic waves were presumed to be of atrial origin, little difficulty was encountered in their explanation even though disagreement existed as to whether these waves appeared in the aorta.
Effect of left ventricular failure upon the aortic pressure trace. Three distinct positive waves (a, b, and c) are seen during diastole in the central aorta timing, respectively, with rapid ventricular filling, atrial contraction, and isometric ventricular contraction. Corresponding heart sounds are numbered. Failure was induced by sustained infusion of norepinephrine following administration of chloralose in a large volume of fluid.

Because of bulging of the aortic valve or because of extra-arterial impact of the atrium on the aorta. The presence of three distinct diastolic waves in the aorta presents more difficulty. Any explanation must account for their simultaneous occurrence in all chambers.

The heart, anatomically arranged in the thorax as it is, represents a physical system of a mass of fluid-filled muscle rather loosely suspended from the great vessels within the pericardial cavity. Thus, the elements of inertia, elastance, and resistance are present, and the system is therefore capable of demonstrating the phenomenon of resonance. Should parts of this system be underdamped, oscillations will occur in the presence of an applied force. The frequency and amplitude of these oscillations will be a function of the mass at the instant of displacement, the "spring" of suspension, and the resistance offered by the myocardium, pericardium, and surrounding mediastinal structures. This is not a simple, lumped second order system because the mass is constantly changing, as are elastance and resistance, as blood flows in and out of the chambers. Nevertheless, the analogy holds. This oscillatory mechanism would be expected to give rise to induced pressure waves within the system. These may be recorded by a sufficiently sensitive apparatus for the measurement of pressure. Furthermore, they would be expected to be complex wave-forms with a fairly wide frequency spectrum. The lower frequencies would be seen on conventional pressure tracings as low amplitude events superimposed
upon otherwise smooth intracardiac pressure functions. The higher frequencies would be reflected as intracardiac "sound."

Sudden acceleration or deceleration of blood flow provides a sufficient energy source to displace the system. This follows from the relationship of force as the product of a mass and its acceleration. One could then predict that at instants of large forces, resulting from impedance to the momentum of blood flow, pressure oscillations would be recordable from all chambers of the heart as well as the great vessels. The implications of this system for the production of cardiac sound will be explored in another communication from this laboratory. The present discussion will be limited to the implications of the system for the low frequency events recorded in the aorta in diastole and simultaneous events in other chambers.

Wiggers, in noting the isometric contraction wave, attributed it to an impact on the semilunar valves by the steep rise of intraventricular pressure. Numerous explanations have been given for the simultaneously occurring, and often recorded, atrial "c wave." The upstroke of the "c wave" has been attributed to the bulging of the A-V valve into the atrium while the downstroke is said to be due to the depression of the valve ring toward the apex of the heart during ventricular ejection. Such an explanation does not account for the frequently biphasic, and often more complex, form of the "c wave" when accurately recorded. That some contribution may occur as a result of displacement of the valve curtain will not be disputed, but this cannot be the entire explanation. An alternative explanation has been that the wave is formed as a result of ejection of blood from the thorax. This is untenable since ventricular ejection does not take place until after the "c wave" is written. In any event, neither explanation can account for the presence of a distinct wave in the adjacent aorta.

It is proposed that at the onset of ventricular isometric contraction blood flows toward the low impedance A-V valves, and is suddenly decelerated when these valves are stretched to their elastic limit. This results in a transfer of energy to an oscillation of the entire heart and great vessels that is seen on accurately rendered pressure tracings from all chambers as a distinct complex pressure transient. Isometric contraction of both right and left ventricles contribute to the complex wave-form seen. Their individual contribution to the system response is not discernible, however, since the period of oscillation is longer than any possible asynchronicity of the two ventricles. Certainly, the more powerful left ventricular isometric impact must dominate.

The presence of the atrial isometric contraction wave in all chambers may be similarly argued. For this wave to appear in the aorta, either as a result of the ventricular "a wave" bulging the valve or as a result of atrial impact against the aorta, requires an acceptance of aortic valvular or wall displacement against a very large pressure difference. The atrium and ventricle during atrial contraction act as a single chamber. Under the influence of a very slight pressure gradient, blood flows from the atrium into an already distended ventricle. As the elastic limit of the ventricular musculature is reached, this flow will be impeded, the deceleration again resulting in an oscillation of all chambers, and a coincident pressure transient. This effect would be augmented in the presence of higher-than-normal left ventricular end diastolic pressure or in high flow states. This mechanism is consistent with the mechanism proposed by Kincade-Smith and Barlow for the initiation of the fourth heart sound.

It follows from the above discussion that a similar mechanism accounts for the production of the ventricular filling wave. The opening of the A-V valves initiates the period of rapid ventricular filling. Should ventricular relaxation be an active process, literally "sucking" blood into the ventricles, it may be that deceleration of inflow may at no time be sufficiently abrupt to result in a transfer of energy to a discernible pressure transient.
The alteration of the volume-elastic characteristics of the ventricle by dilatation, or disease restricting filling, might, however, set the stage for the occurrence of these pressure transients when momentum of inflow is halted.

In conclusion, a unitarian argument is invoked to explain the presence of pressure transients appearing in the aortic pressure pulse and, in fact, in all chambers in cardiac diastole. Rushmer has stated: "Since the chambers of the heart are filled with blood, none of these structures can vibrate independently without producing movements of the blood. Similarly, vibrations in the blood must be transmitted to the surrounding structures. . . . It is futile to consider vibrations of the heart walls, valves, arterial walls, and blood individually when, in fact, they constitute an interdependent system and all vibrate at the same time." We assume the heart to be analogous to a mass and spring. Sudden changes of impedance to local flow results in abrupt deceleration of the blood. Energy is transferred to a force that sets the system in motion, producing the observed pressure transients.

Proof for this concept would rest upon the measurement of instantaneous flow in local areas within the ventricular chambers and the demonstration that acceleration of this flow may be quantitatively related to the magnitude of the observed pressure transients. The observed facts, however, fit the concept as predicted from consideration of the heart and great vessels as a physical system.

Summary
From the observations noted and from physical considerations, the following may be concluded:

Cardiac events of sufficient energy content to cause sudden displacement of the heart and great vessels give rise to pressure transients that may be seen in all chambers if carefully looked for. These are, in reality, "acceleration" waves superimposed upon otherwise smooth pressure functions.

Such an oscillation occurs normally at the time of isometric ventricular contraction. The energy content of isometric ventricular contraction results in an induced pressure wave in all chambers, but is most prominently seen in the low pressure atria as the "c wave," and in the aorta and pulmonary artery as small, relatively high frequency, presystolic impulses. The event is obscured in the ventricles by the rapidly rising intraventricular pressure but may be identified when the ventricular pressure is differentiated.

Under pathological circumstances, atrial contraction and early ventricular filling may result in peripherally recordable pressure deflections.

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