The Aortic Flow Pulse as Related to Differential Pressure

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The flow pulsations in the dog's descending aorta were recorded with a noncannulating square-wave electromagnetic flowmeter and found to be at considerable variance with previously published curves. The validity of the square-wave electromagnetic flows was established by tests of the instrument's adequacy and by comparison with the simultaneously recorded differential pressures. In the genesis of the aortic flow pulse, inertia is dominant and resistance secondary, so that flow is related to the differential pressure pulse mainly through acceleration.

Little is known concerning the pulsatile movement of the blood along the aorta, chiefly because of inadequacies and difficulties in applying existing pulsatile flowmeters. Most of our knowledge has been deduced from pressure pulses recorded at various points along the aorta or by use of differential pressure flowmeters. Aortic flow pulses obtained by various types of differential pressure flowmeters have been published by Frank, Broemser, and Green.

Machella published a curve of the time-velocity sequence of the aortic blood flow derived from application of the hot wire principle, wherein a heated wire was passed across the lumen of the ascending aorta. This instrument suffers from an inadequate frequency response and failure to differentiate between forward and backward flow.

X-ray cinematography was used to study the movement of the aortic blood by Timni who followed the moment-to-moment position of radiopaque oil droplets along this structure. McDonald, using reflected light, photographed the passage of air bubbles along the rabbit's aorta to derive a velocity curve.

The electromagnetic induction principle has been used on the ascending aorta by Wetterer. Each of the flow contours found in these previous studies will be critically compared in this paper to those obtained with the square-wave electromagnetic flowmeter.

No investigator has compared aortic flow pulses with the simultaneous differential pressures existing across the segment of vessel under study; yet, the close relationship between flow and differential pressure has long been recognized. The pressure transmission studies made by Hamilton and Dow demonstrated a system of standing pressure waves in the aorta which they predicted would act on the flowing stream to produce alternate acceleration and retardation. Their concept established the aorta physically as an elastic and relatively frictionless tube. Frank recognized this in his theoretical treatment of the stream of velocity in vessels and stated in his paper "the pressure difference, which represents the moving force, can, in a frictionless current, only be balanced by forces of inertia." The exact phase relationship of differential pressure to acceleration in the large arteries was, however, apparently not clear to him.

This communication will present what we believe to be the true blood flow curves of the aorta and establish their validity by their relationship to the differential pressures existing along that structure. At the same time we will point out some artifacts that may be introduced into flow curves when recorded under differential pressure.

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certain conditions which considerably alter the normal physical characteristics of the arterial system.

METHODS

A modification of the newly developed square-wave electromagnetic flowmeter has been utilized in these experiments to obtain high frequency recordings of the volume flow with minimal disturbance of the aorta. Electrocardiographic potentials, which limited this electromagnetic flowmeter, and earlier ones when used near the heart, were suppressed by increasing the square-wave frequency from 30 cycles/sec. to 240 cycles/sec. By so doing, electrical interference voltages of low frequency, such as that of electrocardiogram, can be filtered out without materially affecting the voltage generated by the flowing blood.

In order to establish that the square-wave flowmeter, as used here, is capable of recording flow frequencies above 100 cps, a simple test was applied. The magnetic probe was mounted on one end of a steel bar and immersed in saline, while the other end was held firm in a vise. By striking the bar with a rubber hammer, the probe was caused to vibrate so that saline flowed to and fro between the magnetized poles. The flowmeter system used in these experiments recorded to-and-fro flow frequencies well over 100 cps. Response to higher flow frequencies was limited by the ink-writing pen-motor.*

The magnetic probes were so constructed that the dog's aorta was compressed between the magnet poles by 20 or 30 per cent but allowed to expand and pulsate freely in the interelectrode diameter. Changes in the interelectrode dimension of the artery do not affect the flow calibration of the meter because such changes alter the length of the moving cross section of blood and thus directly change the number of magnetic lines of force being cut. The net rate at which magnetic lines are cut produces a voltage directly proportional to the net flow, thus preserving the linearity of calibration.

The compression in one diameter of a vessel by 20 per cent reduces the cross sectional area by only 6 per cent if the other diameter is allowed to expand freely. In return for this small sacrifice, one gains greater sensitivity and stability. On occasion, smaller probes which caused greater compression were applied to study the effect of flowmeter resistance on the velocity contours.

Pressure pulses were recorded from the summit of the aortic arch by means of a cannula introduced through a carotid artery. The cannula tip was placed just within the orifice of the innominate artery at its origin from the aorta and connected at its other end to a Statham P23D pressure transducer.

Aortic differential pressures across the point of application of the flowmeter probe were recorded simultaneously with the arch pressure pulse and the flow pulse. The differential pressure pulses, hereafter symbolized by \( \Delta P \), were obtained by means of two no. 18 spinal needles puncturing the aortic wall at right angles, as diagrammed in figure 1. The bevel of each needle was always oriented so that its opening faced the aortic wall rather than upstream or downstream.

Each needle was attached to one of a pair of Statham P23D transducers, also shown in figure 1. The electrical terminals of the transducers were connected in parallel so that their combined output was proportional to the difference in pressure between them. This differential pressure system was carefully checked for adequate natural frequency and linearity. The gages were adjusted to equal sensitivity by appropriate attenuation of the most sensitive one. Since the position of the paired gages affected this sensitivity adjustment, they were rechecked after being fixed to the dog board in their operating position.

The plan of the experiment was to explore the flow pulse contour along the thoracic and abdominal portions of the descending aorta while the aortic

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* The frequency response of the Brush Recorder used here is sharply limited above 100 cps. This places no limitation on the validity of flow pulses which require no greater fidelity than that necessary for accurate pressure pulse recording.
APs were simultaneously recorded from dual needle punctures immediately above and below the point of flow measurement. The flow and AP curves of 30 dogs were examined. The effects on flow through a section whose resistance and elasticity were altered were studied. In addition, the effects of increased and decreased peripheral resistance were examined.

RESULTS

The Normal Aortic Flow Pulse

The flow pulses recorded by this method represent the instantaneous net forward or reverse volume flow of the streams as they pass the magnetic field. Because of the confinement of the vessel to the space between the magnetic poles, each magnet calibrates in ml./min. The exact vessel size is not critical so long as it touches the walls formed by the poles.

The radial components of blood movement concurrent with pulsations in the interelectrode dimension are not picked up because the electrodes are not oriented to pick up the voltage generated by this movement. Only linear flow parallel to the vessel axis is recorded.

Flow Pulses and Pressure Pulses in the Thoracic Aorta. A normal flow curve and pressure pulse are depicted in figure 2. The following generalities may be stated concerning the relationship of the flow pulse in the thoracic descending aorta to the pressure pulse in the arch. The flow pulse consists of a series of smooth, damped oscillations beginning soon after the upstroke of the arch pressure pulse and gradually diminishing in amplitude throughout the cardiac cycle. The first peak tends to be flattened, is reached in midsystole near the peak of the pressure pulse, and is at-

Fig. 2. Normal flow and pressure pulses in thoracic aorta. Upper record, pressure pulse at summit of aortic arch. (I, incisura.) Middle record, pulsatile pressure difference between two points 3.5 cm. apart. (UL, incisura at the upstream needle; DL, incisura at the downstream needle.) The direction of the pressure gradient constantly alternates with the standing wave. Lower record, pulsatile aortic flow between two points of differential pressure measurement. Fine waves on this record represent 60 cycle hum. Average velocity of flow streams at peak systolic flow, 101 cm./sec. (calculated from the flow and systolic internal diameter of the aorta).
tended by an initial period of acceleration, \(A_1\), and a period of deceleration, \(D_1\). The first trough immediately follows the incisura and is attended by \(D_1\) and the second period of acceleration, \(A_2\). The first trough is invariably the time of lowest velocity and may (not always) dip below zero as backflow. The period and amplitude of these waves of alternate acceleration and deceleration continue to diminish to zero or until begun again by the next cardiac cycle. The character of \(D_1\) is particularly influenced by the incisura and is discussed in a subsequent paragraph.

When our flow pulse curves are compared with previous curves obtained by others using the differential pressure flowmeters\(^1\) and bubble cinematography,\(^\text{a}\) a great phase discrepancy is immediately apparent. The previous curves record the first flow peak in early systole and the first trough (always backflow) as synchronous with the moment the incisura passes the flowmeter. In order to clarify this discrepancy, \(\Delta P\)s were measured across the segment of aorta on which the electromagnetic flowmeter was applied. Simultaneous \(\Delta P\) and flow curves could then be compared directly with the expectation that the two would be related through acceleration.\(^*\)

**Differential Pressures.** Figure 2 depicts (in addition to pressure pulses and flow pulses) the \(\Delta P\) between two points 3.5 cm. separated, obtained by needle punctures of the thoracic descending aorta. The first positive wave of this pulse begins after the upstroke of the arch pulse, being delayed by the time required for transmission of the pulse between the arch and the upstream needle. It rises sharply just as a normal pressure pulse, but is interrupted when the pressure pulse reaches the downstream needle. During mid-systole it tends to plateau at a pressure of +5 to +8 mm. of Hg. The peak volume flows of 6.7 L./min. seen in figure 2 and 5.3 L./min. in figure 3A are within the normal range. The systolic aortic circumferences in these two instances were 11.5 mm. and 12.5 mm. The mean velocity calculated from an assumed wall thickness of 1 mm. was 101 and 112 cm./sec., respectively.

When the \(\Delta P\) recording indicates the pressure is greater in the upstream needle, the gradient is said to be positive; and when greater in the downstream needle, it is said to be negative. The first negative wave (frequently the only one) begins in late systole and is shorter in duration and lower in amplitude than the first positive wave. The lowest point of the negative wave corresponds to the time the incisura reaches the upstream needle. A small, sharp peak rising immediately out of the negative wave represents the incisura passing the downstream needle.

The \(\Delta P\) pulse in the aorta is a direct representation of the standing wave. Its frequency diminishes throughout the cardiac cycle as the wall tension under systolic pressure diminishes to diastolic pressure. These standing waves always correspond in number with the flow waves.

**Relationship Between Flow Pulse and \(\Delta P\) Pulse.** When the electromagnetic flow pulses are compared to the \(\Delta P\) pulses, a great phase difference is apparent. The beginning initial upstrokes of both curves coincide closely; but when the first positive wave of the \(\Delta P\) reaches its peak, the electromagnetic meter indicates flow is still accelerating and at the greatest rate seen during the cardiac cycle. When the flow recording reaches its mid-systolic peak, the \(\Delta P\) pulse is almost back to zero. This phase lag continues throughout the cycle so that when the first negative wave of the \(\Delta P\) reaches the bottom of its trough, the flow is decelerating and at a rate unequalled during any other time in the cardiac cycle. In terms of a standing wave cycle, this lag represents almost a 90° phase shift. It is always apparent when the aorta is unrestricted by the flowmeter probe. This relationship may be briefly summarized as follows: Flow peaks or troughs occur each time the pressure gradient is reversed in direction and the acceleration of blood (slope of the flow curve) at any time is nearly proportional.
THE AORTIC FLOW PULSE

Fig. 3. Transformation of flow pulse along descending aorta. Single magnetic probe applied at three different levels, (A, B, and C) and the records superimposed by retracing. Carotid pressure pulse measured simultaneously at times 1, 2, and 3. The three levels were A, just above the 3rd intercostal arteries; B, between the 8th and 9th intercostal arteries; and C, 1.5 cm. below the left renal artery. At each level the systolic circumference and peak systolic velocity (calculated from flow and circumference, assuming 1 mm. wall thickness) were as follows: A, 37.5 mm., 112 cm./sec; B, 30.5 mm., 128 cm./sec; C, 23 mm., 141 cm./sec.

to the difference in pressure across the segment under study.

The contribution of the incisura of the thoracic pressure pulse to the APs and flow curves is significant. The first negative wave reaches its peak value (trough) as the incisura passes through the flowmeter, therefore, the flow curve displays the greatest deceleration slope at this time. This finding is another point of phase variance with the curves of the differential flowmeter and bubble cinematography which recorded a considerable degree of backflow occurring at the time of the incisura.

Propagation of the Flow Pulse Along the Aorta. As one applies the probe to various levels along the aorta, one finds the entire course of the flow wave is progressively delayed in the cardiac cycle (fig. 3). This delay or transmission time coincides with that seen in the pressure pulses. Little transformation occurs in the flow contour while it traverses the descending aorta. Mainly, one notices a lessening of the A1 and D1 gradients, coincident with the disappearance of the incisura of the pressure pulse. Backflow during the diastolic trough is more prominent in the lower aorta, particularly below the renal arteries. Peak systolic velocity becomes increased in the smaller lower aorta.

Factors Altering the Normal Flow Pulse

Loss of Elasticity. When a large, rigid cannulating probe is substituted for the aortic wall by division of vessel and cannulation, the recorded flow contour through the rigid segment is altered little from that found using noncannulating probes. This is illustrated by comparing the noncannulating flow curves of figure 2 with the cannula curve 1 of figure 4. The same phase shift, rounded contours, and incisural effects are seen in both curves. Also, the ΔP pulse is affected little when measured from needles introduced immediately above and below the cannulae.

Aortic Constriction. A segment of the aorta was progressively constricted by application of smaller and smaller flowmeters and by constriction with a clamp placed around the aorta and just beside the magnetic probe. Beyond reduction in the mean flow and reduction in peak forward flow, the most noticeable change occurs around the time of the incisura (late systole and early diastole). The contours of both flow and ΔP curves rise, backflow disappears, and the negative ΔP waves become positive. Both curves move toward a closer approximation of the central pressure pulse. Changes in the flow pulse and ΔP pulse become apparent when the lumen is restricted beyond 15 per cent. The flow contour under these conditions is termed "viscous" or "resistant" flow because it is proportional to the ΔP without phase lag.

Increased Peripheral Resistance. When the aorta is gradually constricted downstream to the flowmeter, (fig. 4) mean flow and peak forward flow are diminished. In addition, backflow becomes more prominent and a considerable change in contour takes place. Upon complete occlusion, the contour is that of a series of forward and backward waves oscillating around zero flow and at the frequency of the standing wave; the amplitude falls off to
Fig. 4. Effects of progressive constriction of aorta 9 cm. below flowmeter. Records obtained by "can-
nulating type" flow study in the midthoracic de-
sending aorta and superimposed by retracing. 1,
normal control; 2, partial constriction; 3, complete
occlusion. The net forward movement of flow in
record 3 is the amount that flows out of intercostal
branches between the meter and clamp. The phase
shift between flow and differential pressure is ap-
parent and attains 90° at all times in record 3. The
standing wave frequency increases, because of short-
ening of the Windkessel.

zero in diastole unless interrupted by the next
systole. During the period of severe occlusion,
nean perfect 90° phase shift exists between the
flow and ΔP.

The ΔP curves display both an increase in
frequency of the aortic standing wave which cor-
cresponds to the increased frequency of the
main flow pulsations, and a loss of the mid-
systolic plateau. The intermediate curves of
partial aortic occlusion (fig. 4) include flow
contours which are remarkably similar to
those published by Frank,1 Broemser,2 Green,3
and McDonald.4

Diminished Peripheral Resistance. The effects
of diminished peripheral resistance were ob-
served during reactive hyperemia which oc-
curred following release of aortic occlusion.
Upon release of the clamp during diastole, a
large flow pulse passes along the aorta to fill
the lower end. This initial surge is not unlike a
normal flow pulse and is accompanied by an
appropriate ΔP pulse. The first cardiac cycle
after release causes a supernormal ΔP and a
flow pulse which is increased in volume. The
duration of systole is increased, and flow
reaches a greater than normal peak value.
Backflow does not occur even if normally
present. The increased systolic peak flow is
reflected in the ΔP pulse by a higher (more
positive) mid-systolic plateau. The succeeding
pressure and flow pulses approach the normal
state.

DISCUSSION

Since little net fall in pressure occurs along
the length of the aorta, it must be accepted as
a relatively frictionless structure. In addition,
the carotid-femoral differential pulse waves
clearly affirm its elastic nature. The flow pulsa-
tions and ΔP pulses measured in short segmen-
t of the aorta by this study are in agreement with
the premise that blood is conducted along this
elastic tube primarily as a wave phenomenon
superimposed on a net forward flow. Normally
resistance does play a small role and is mani-
fest in two ways: 1. A complete 90° phase shift
between ΔP and flow pulse is not quite attained.
2. During the peak systolic flow, when the
flatness of the flow curve indicates little change
in flow rate, a small, relatively constant differ-
ence in pressure is apparent along the aorta in
the direction of the flow.

The discrepancy between our flow contours
and those previously published led us to ex-
amine some of the factors which might affect
the flow curves of all meters. When the factors
of segmental rigidity, segmental resistance, and
peripheral resistance were carefully studied,
the normal ΔP-flow relationship in the aorta
became clearer. In addition, we could see the
pathologic ΔP-flow relationships to be expected
when a disease process introduces the same
factors we accomplished experimentally.

The normal movement of blood through the
aorta is a resultant of two basic types of flow.
First and foremost is that of wave motion flow or "inertia" flow, and second that of resistant flow or "viscous" flow. The part played by each in any given flow pulse can be seen in the relationship of flow to the ΔP across the point of flow measurement. Inertia flow dictates that the ΔP will be related to the flow through acceleration and completely dominates when the flow rate is minimal. Viscous flow dictates that the velocity through a segment under study will be directly related at each instant to the magnitude of the ΔP across the same segment. It is most apparent in mid-systole when flow is maximal.

Pure inertia flow can be approached by reducing the velocity, by increasing the peripheral resistance, or by reducing the cardiac output. The introduction of a rigid but wide cannula into a short segment of the aorta does not greatly alter the inertia flow seen normally in the same segment. Pure viscous flow occurs when the segment under study is constricted, when the velocity is very high, or when wave motion is abolished by damping.

We feel that the flow pulses recorded under control conditions of this study represent the normal aortic contours. Their validity is based on three general principles. 1. The instrument is adequate to record forward or backflow with linearity and at any frequency to be expected in the aorta. 2. The flow sensing unit is applied directly to the unopened vessel with negligible interference with the dynamics of the flowing stream. 3. The observed velocity sequence through a given aortic segment is that expected from the simultaneously measured ΔPs across the same segment when the aorta is considered as an elastic and relatively frictionless tube.

Womersley, in a solution of the equations of viscous fluid motion, predicted the phase lag to be expected between flow and ΔP when the net forward flow is zero. McDonald found this phase lag in the dog's femoral artery by first measuring the pressure gradient indicated by two manometers recorded from small plastic tubes in side branches of the femoral and then measuring the flow through the same artery by means of bubble cinematography.

We cannot say with assurance why others have not obtained the same aortic flow curves as we have because the exact experimental conditions under which they worked are unknown. We have shown, however, that a great increase in peripheral resistance can produce curves similar to those obtained by Frank, Bröemser, Green, and McDonald. It seems likely that the injected air bubbles, necessary to McDonald's method, can produce an increased peripheral resistance by air embolism.

With the ΔP flowmeters another possible explanation arises from the similarity of such "flow" curves with the ΔPs. The ΔP flowmeter in theory measures the difference in pressure created by velocity of blood. If, however, there is an elastic element between the two points of pressure measurement, considerable standing wave ΔP, which generates the flow, will be recorded in addition. The velocity-generated ΔP will be in phase with the flow, but the standing wave ΔP will be 90° out of phase as it generates velocity. Green's ΔP meter seeks to avoid this difficulty by cannulating the vessel with a rigid tube in which the pressure ports are mounted. The rubber membrane of the differential manometer works against the attainment of this perfection.

The flow curves of Wetterer were recorded in the ascending aorta and show a very brief period of backflow at the incisura. The degree of compression applied by the magnet pole pieces is not given, but from the pressure pulses measured downstream it does not appear to be excessive. Backflow near the heart should be related to closure of aortic valves and coronary flow. A special study will be required to relate the ΔP to flow pulse here.

**SUMMARY AND CONCLUSIONS**

The phasic flows at various points along the dog's descending aorta were recorded by means of the square-wave electromagnetic flow meter and related to the differential pressure (ΔP) simultaneously existing across the point of flow measurement. The following conclusions were reached:

An adequate pulsatile flow meter requires a frequency response as great as that necessary for arterial pulse pressure recording. Its application should not restrict the lumen of the vessel by more than 15 per cent of the cross sectional...
area. The elasticity of a short segment of the aorta may be entirely abolished with only slight effect on the flow contour.

The noncannulating square-wave meter is an adequate pulsatile meter for quantitative measurement of arterial flow pulsations.

The aorta is an elastic tube through which the blood is conducted primarily as a wave motion "inertia flow" superimposed on a net forward movement. The main flow waves correspond in number but not in phase with the standing waves represented by ΔPs. Except at very high flow rates, the phase delay between ΔPs and flow pulses closely approaches 90°.

Resistant or "viscous flow" is apparent at the high flow rates of the mid systolic flow plateau. The pressure drop along the aorta during this phase of the cardiac cycle accounts for most of the mean pressure drop along the aorta.

Except during the phase of viscous flow, the slope of the flow pulse (i.e., the acceleration or deceleration of blood at any instant) is determined by the magnitude and direction of the ΔP across the point of measurement.

Backflow frequently occurs during the normal cardiac cycle at all levels of the descending aorta. Reversal of flow begins at a time early in diastole when the deceleration produced by the first negative component of the standing wave is sufficient to exceed the kinetic energy of the initial caudal (systolic) movement of blood. Backflow is more prominent in the lower abdominal aorta.

Aortic flow curves derived from previous methods differ markedly from these and may be affected by increased resistance at the meter, in the peripheral vascular bed and in the case of AP flow meters by the AP accelerating the blood.

When peripheral resistance is increased by compression of the aorta distal to the point of flow measurement, mean flow is reduced, the standing wave frequency is increased, and backflow is increased as the velocity sequence oscillates about zero flow.

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SUMMARIO IN INTERLINGUA

Le fluxos phasic a varie punctos del aorta descendent del can esseva registrate per medio del fluxometro electromagnetique a unda quadrate e relationate al pression differential (ΔP) que existe simultaneamente a transverso le puncto de mesuration. Le sequente conclusiones esseva establite:

Un adequate fluxometro pulsatil require un responsa de frequentia tanto grande como illo requisite in le registration de pulsos de pression arterial. Su application non deberea restringer le passage aperte del vaso per plus que 10 pro cento del area transversal. Le elasticitate de un curte segmento del aorta pote esser abolite completemente con non plus que leve effectos super le contorno del fluxo.

Le non-cannulante registrator a unda quadrate es un adequate registrator pulsatil pro le mesuration quantitative del pulsationes de fluxo arterial.

Le aorta es un tubo elastic in que le sanguine progrede primarimente in le forma de "fluxo inertial" a motion undulatori, superimponite a un nette movimento in avante. Le major undas de fluxo corresponde in numero sed non in phase al undas stationari representate per ΔPs. Excepte sub conditiones de rapidissime fluxos, le retardo phasic inter ΔPs e pulsos de fluxo es multo proxime a 90°.

Fluxo resistente (o viscose) es apparente al alte rapiditate occurrente al plateau de fluxo medie-systolic. Le decrescente pression a punctos successive del aorta que occurre durante iste phase del cyclo cardiac suffice a explicar un parte major del decrescentia in pressiones median a punctos successive del aorta.

Excepte durante le phase del fluxo viscose, le inclino del pulso de fluxo (i.e., le acceleration o le deceleration del sanguine a un momento specific) es determinate per le magnitude e le direction de ΔP a transverso le punto de mesuration.

Refluxo occurre frequentemente durante le normal cyclo cardiac a omne nivellos del aorta descendente. Le reversion del fluxo comencia
tosto in le diastole, quando le deceleration producute per le prime componente negative del unda stationari suffice a eccedere le energia kinetic del initial movimento caudal (systolic) del sanguine. Refluxo es plus prominente in le aorta infero-abdominal.

Curvas de fluxo aortic establite per previe methodos differe marcatemente ab le nostres e es possibilemente afficite per augmentos de resistencia al registratore e in le rete periphero-vascular e, in le casos de fluxometros de ΔP, per le ΔP que acceler a sanguine.

Quando le resistencia peripheric es augmentate per le compression del aorta a un puncto distal al measurement del fluxo, le fluxo median es reduce, le frequentia del unda stationari es augmentate, e le refluxo es augmentate durante que le sequentia de velocitate oscilla in le vicinitate de fluxos a magnitude zero.

REFERENCES

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