Propagation of Blood Flow Pulse in the Normal Human Pulmonary Arterial System

ANALYSIS OF THE PULSATILE CAPILLARY FLOW

By Nicholas B. Karatzas, M.D., D.Phil., and Grant de J. Lee, M.D., M.A.

ABSTRACT

We studied a number of dynamic events associated with the pulsatile flow of blood in the pulmonary arterial system of six healthy men in the supine position. We used the nitrous oxide-body plethysmograph method to record the pulmonary capillary blood flow pulse and the phonocardiogram to determine the time of opening and closing of the pulmonary valve. The pattern of right ventricular ejection was modified by administration of atropine and isoproterenol and by exercise.

The time of conduction of the flow pulse from the pulmonary valve to the lung capillaries averaged 120 msec. Acceleration of capillary blood during systole averaged 8.2 ml/sec/msec. The fraction of stroke volume which distended the pulmonary arterial system during systole averaged 67.2%. The peak flow rate averaged 186 ml/sec. Tachycardia resulted in a decrease in the fraction of the stroke volume stored in the arterial tree during systole. Isoproterenol and exercise resulted in an increase in average capillary blood acceleration. Conduction time is accounted for by pulmonary arterial distensibility, which also allows storage of the ejected blood during systole, which in turn helps to maintain continuity of blood flow in the capillaries. Despite these modifying effects of the pulmonary arterial system upon the flow wave, changes in the pattern of inflow pulse were detectable in the capillary flow pulse.

ADDITIONAL KEY WORDS

pulmonary capillary flow pulse
nitrous oxide-body plethysmograph
cardiogenic oscillations
atropine
isoproterenol
exercise
pulse conduction time
capillary blood acceleration
pulmonary arterial systolic storage

The transport of blood through the blood vessels is a complex phenomenon and has not yet been fully analyzed. Traditional ways of describing the circulation of blood overlook a number of aspects, including the intermittent ejection of blood by the heart.

From the Department of the Regius Professor of Medicine, University of Oxford, and the Cardiac Department, Radcliffe Infirmary, Oxford, England.

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Dr. Karatzas' present address is the Cardiovascular Research Institute, University of California San Francisco Medical Center, San Francisco, California 94122.

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Recent development of methods for recording instantaneous rate of blood flow has stimulated interest in the pulsatile nature of blood flow, but quantitative data related to the propagation of the flow wave in arteries are generally lacking.

In this study we obtained data on dynamic events associated with the pulsatile movement of blood in the human pulmonary arterial system, namely, the duration of conduction of the flow wave from the pulmonary valve to capillaries, the systolic storage in the pulmonary arteries of blood ejected from the right ventricle, and the average acceleration of capillary blood. These measurements were made from the pulmonary capillary flow pulse obtained by the nitrous oxide (N₂O)-body plethysmograph method (1). We used the
phonocardiogram to relate the capillary flow pulse to the time of right ventricular ejection.

**Methods and Subjects**

We recorded capillary blood flow during slow expiration according to the method reported by Bosman and his colleagues (2). The subject lay supine. The body plethysmograph and the flowmeter we used to record the rate of change in the body plethysmograph gas volume have been described in detail elsewhere (3). The plethysmograph was a modified hyperbaric treatment chamber incorporating a flowmeter with pneumatic feedback which was controlled electronically by means of an optical device sensing the pressure in the plethysmograph. The frequency response of this system was flat to 10 cps with a 25% loss in amplitude at 20 cps. The signal was integrated electronically. The concentration of N$_2$O in the expired gas was recorded continuously by an infrared N$_2$O analyzer in closed circuit with the body plethysmograph.

The validity of the N$_2$O-body plethysmograph method for obtaining instantaneous rate of blood flow in the lung capillary has been critically examined in previous publications (1-7).

Lead II of the electrocardiogram and the high-frequency (8) phonocardiogram from the pulmonary area were also recorded. High-quality phonocardiograms obtained inside the soundproof body plethysmograph enabled us to distinguish the various components of the heart sounds. The second component of the second heart sound is known to correspond in time with the closure of the pulmonary valve in healthy subjects. The time of the pulmonary valve opening was taken to correspond with the third component of the first heart sound. This was based on the known sequence of events associated with ventricular systole (9-11) and the evidence indicating that the two initial components of the first heart sound correspond in time with the periods of left and right ventricular isovolumic contraction and that the last (fourth) component coincides with the onset of left ventricular ejection (12-16).

The time for transmission of the heart sounds from the region of the pulmonary valve to the microphone must be of the order of 20 μsec (17) if heart sounds travel from their point of origin to the point of emergence on the chest wall with the speed of sound in water. Indeed, simultaneous intracardiac and extracardiac phonocardiograms obtained by us and other investigators (18) show no measurable time difference for the components of the second heart sound at a paper speed of 100 mm/sec.

All recordings were made by a 12-channel, electron-beam, photographic recorder, at a paper speed of 100 mm/sec. An example of the records thus obtained is illustrated in Figure 1.

The subjects were six young males (Table 1), all of whom had participated as subjects in respiratory tests many times before. They were trained, with the aid of pneumogram tracings displayed on a large persistent-screen oscilloscope, to perform the respiratory maneuver repeatedly in an identical manner and to maintain constant air flow during slow expiration. The pneumogram was obtained by strain gauges of the mercury-in-rubber type placed around the chest and abdomen. Three studies were performed on every subject on different days, and each study involved exercise or administration of either atropine or isoproterenol. The subject rested on the couch of the body plethysmograph for at least 10 minutes before the plethysmograph was closed for control pulmonary capillary blood flow records to be taken. For the drug studies, 2 mg atropine sulfate was given intravenously, and pulmonary capillary blood flow records were taken 20 minutes later, or 40 mg isoproterenol sulfate was taken sublingually and records were taken 10 minutes later. For the exercise study, the subject pushed steadily with his feet placed in stirrups that were connected to springs fixed at the head end of the plethysmograph. No provision was made for estimating the load of work. Records were taken 5 to 10 minutes after exercise had begun. The phonocardiogram was recorded continuously from the time of administration of the drugs or the beginning of exercise until test recordings were obtained, so that changes in the heart sounds resulting from these procedures could be noted. The intensity of some of the components of the heart sounds gradually changed, but their sequence did not.

### Table 1

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</table>

1 Integrator Unit, Devices Sales Ltd., Welwyn Garden City, England.
2 Infrared Development Ltd., Welwyn Garden City, England.
The volume of pulmonary capillary blood flow per pulse (Qc in ml) was calculated by the usual formula (2).

The pattern of pulmonary capillary blood flow rate was obtained by algebraically subtracting the air control record from that of N₂O at 10-msec intervals. To calibrate the resultant curve in flow rate units (ml/sec), its area mean was determined by planimetry and was assigned the calculated mean flow rate value.

The accuracy of the process of subtraction of the air control record from the N₂O uptake record depends on the reproducibility of the cardiogenic oscillations from one cardiac cycle to another and during successive slow expirations. The factors which determine this reproducibility have been investigated (19). These were the rate of expiration, lung volume, volume relationship between thoracic cage and abdomen, movements of vertebral column or shoulders with respiration, depth of the preceding inspiration, heart rate, state of inotropic stimulation of the heart, and stroke volume. These factors must remain constant to have reproducible cardiogenic oscillations. To achieve this, the subjects had to be selected and trained to perform identical maneuvers and maintain constant air flow during expiration. This requirement presents a real limitation of the method when used to obtain flow waveforms, but we have no reason to believe that serious errors due to mismatching of the air control records were present in the studies included in this report.

From the pulmonary capillary blood flow pulse thus computed, after correcting for the 10-msec instrument delay, the following variables were obtained (Fig. 2): (a) the conduction time of the flow pulse from the pulmonary valve to pulmonary capillaries, measured as the interval between the third major vibration of the first heart sound of the high-frequency phonocardiogram from the pulmonary area, assumed to coincide with the onset of right ventricular ejection, and the foot of the capillary flow pulse; (b) the average acceleration of blood in the capillaries (rate of upstroke), calculated from the difference between the flow rates at the foot and peak of the pulse divided by the time interval between these two points (upstroke time); (c) the percentage of stroke volume stored in the pulmonary arterial tree during systole (systolic storage)—this was measured from the volume flow between the second component of the second heart sound and the third vibration of the first heart sound (diastolic volume), divided by the volume flow during the whole cardiac cycle, and expressed as a percentage; and (d) the peak flow rate (Qc,max). The length of the right ventricular ejection period was also measured from the interval between the third vibration of the first
An illustration of the procedure for analyzing the records. Parts of control and $N_2O$ uptake records with optimally matching heart rates have been superimposed. ECG, PCC, $S_1$, and $S_2$ = as in Figure 1. On the right, the result of the subtraction of one cycle is drawn in real scale ($Q_c$). The shaded area on the left corresponds to the volume of $N_2O$ uptake in one cycle and is given by the integrator signal (SV); $DV =$ the volume uptake during diastole; $C_t =$ conduction time; $U_t =$ upstroke time.

Results

Tables 2, 3, and 4 list the measurements obtained before and after atropine and isoproterenol and during exercise. Examples of the flow pulses obtained under these conditions are shown in Figure 3.

The changes in cardiac output and heart rate were similar to those observed in previous investigations (20, 21). The time of conduction between pulmonary valve and capillaries averaged 120 (range 110 to 140) msec; the average systolic acceleration (rate of upstroke) averaged 8.2 (range 5.8 to 11.5) ml/sec/msec; peak flow averaged 186.1 (range 135 to 240) ml/sec. Peak flow occurred approximately at the time of closure of the pulmonary valve. Systolic storage averaged 67.2% (range 59% to 78%) of the stroke volume.

At the higher heart rates (76.4% increase) associated with administration of atropine, the conduction time was prolonged (17.2%), the systolic storage was decreased (18%), and the peak flow rate was not significantly altered.

Following isoproterenol, there was a large increase in the average rate of upstroke (112%), an increase in peak flow rate (26.6%) and a small shortening of conduction time (17%).

During exercise, similar but less marked changes in upstroke rate (71.8%) and conduction time (7.4%) and an increase (85.2%) in the peak flow rate occurred.

Discussion

Before we discuss the implication of these results, we should consider the factors that determine the waveform of the pulmonary capillary flow pulse and its time relations to the cardiac cycle. These are the pattern of ejection of blood from the right ventricle into the pulmonary artery, the pattern of impedance to flow met at the pulmonary capillaries, the architecture, dimensions, and distensibility properties of the pulmonary arteries, and the physical properties of blood as a fluid. None of these factors has been adequately investigated in man, but reasonable assumptions can be made, based on data obtained in the dog and...
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<th>Systolic storage (% stroke vol.)</th>
<th>Cycle length (msec)</th>
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**MEAN**

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<th>Systolic storage (% stroke vol.)</th>
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**% CHANGE**

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**t* | P**

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C = control, A = atropine. * Student's paired t-test.

**FIGURE 3**

Computed carotid flow pulses from one subject obtained at rest, after atropine, after isoproterenol and during static exercise.
### TABLE 3

Measurements before and after Isoproterenol

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C = control, I = isoproterenol. * Student's paired t-test.

### TABLE 4

Measurements before and during Exercise

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C = control, E = exercise. * Student's paired t-test.
on the human pulmonary capillary blood flow pulse itself. The capillary flow pulse recorded by the N₂O method, however, represents an instantaneous summation of individual flow pulses of various capillary groups because the method determines blood flow through the entire gas-exchange area of the lungs. So, when this flow pulse is taken to correspond to the pattern of outflow of blood from the pulmonary arterial system, the assumption is implied that the capillary flow elements of all regions of the lungs are in phase, or expressed in another way, it is assumed that length and conduction properties of the various pathways from the pulmonary valve to the capillaries are so matched that the conduction of the flow wave is uniform through all of them.

Bearing in mind this assumption, we shall now consider the above factors individually. The pattern of ejection of blood by the right ventricle cannot be readily recorded in man, and the electromagnetic and ultrasonic flowmeter methods, which allow its registration in animals, may interfere with the pattern of capillary blood flow because the transducer prevents systolic distension of the part of the pulmonary artery it surrounds. However, the volume of blood ejected from the right ventricle can be estimated by a physiological calculation. The flow pulse in the pulmonary arterial system is recorded with the aid of the N₂O method.

**FIGURE 4**

Illustration explaining the use of the phonocardiogram for measurements of the pulmonary arterial systolic storage (shaded areas in A). The phonocardiogram, the capillary flow pulse (Qₖ) and the hypothetical pulmonary artery flow pulse (Q_PA) are shown. In A, Q_PA pattern assumed during our measurements. In B, a more realistic pattern of Q_PA; the shaded, triangular area corresponds to the error of our measurements. In C, the pattern of the rate of change of arterial storage, ΔS_PA, is shown, computed from the curves of B.
ventricle in systole must equal the volume
flow per cycle in the pulmonary capillaries
during a steady state.

It is also reasonable to presume that blood
accelerates rapidly out of the right ventricle
after the opening of the pulmonary valve, flow
rate reaches its peak value early in systole, and
flow decelerates rapidly before the closure of
the pulmonary valve (Fig. 4).

Variation in capillary impedance during the
heart cycle could exist as a result of transmis-
sion of pressure waves from the left atrium. If
such variations were important, they should
affect the contour of the capillary flow pulse
(7). However, we have not seen any clear
evidence to suggest that this is the case in
healthy subjects.

Our knowledge of the architecture and
dimension of the pulmonary arteries of
animals or man is also inadequate. Their total
volume capacity for a male adult of average
size is probably of the order of 115 ml,
evaculating from measurements made for
the dog by the method of ether evolution
(22). The volume distensibility of the pulmo-
nary arterial system or the way it is appor-
tioned in the various elements of the system is
also unknown, although there is evidence that
the smaller arteries of the isolated rabbit lung
are less distensible than the larger ones
(23).

The rheological properties of the blood are
customarily neglected as factors influencing
pulsatile flow in arteries, but they may indeed
be important in the microcirculation, such as
in the lung capillary circulation. For the
purpose of this discussion, however, we are
regarding pulmonary capillary flow pulse
obtained by the N₂O method as the outflow
pulse from the pulmonary arteries rather than
the flow pulse after passing through the
capillary bed.

All the above limitations determine the
validity of the following description of the
dynamic events associated with the flow of
blood in the pulmonary arteries of a male
adult resting in the supine position.

During approximately the first tenth of a
second after the opening of the pulmonary
valve, blood enters the pulmonary arteries
rapidly from the right ventricle, but no
increase in outflow from the arteries takes
place. The arteries therefore must distend to
accommodate the incoming blood. This dis-
tension could be accomplished by an increase
in both the diameter and length of the vessels.
The radial expansion may be thought of as a
circular swelling of the artery which travels
the entire length of the arterial tree from the
pulmonary valve to the capillaries within this
time period. This swelling spreads and its
amplitude decreases along its course because
part of its volume is used to expand the
arteries, which remain somewhat distended
behind the moving front. This is because
throughout this period blood entering the
pulmonary arteries exceeds the outflow; hence
the volume of blood stored increases. The flow
wave arriving in the smaller arteries, such as
arterioles, if indeed these were relatively
indistensible, would not cause a circular
expansion of their lumen but only local
acceleration of blood.

When the front of the flow wave reaches
the capillaries, blood begins to accelerate out
of the arterial system but not as rapidly as it
did when entering the system because a
considerable part of the flow wave has already
been used in storage.

Throughout the rest of systole the rate of
blood flow out of the arterial system is
increasing and reaches a peak at approximat-
tely the end of systole. At this time the rate of
outflow of blood should exceed the rate of
inflow and so the volume of blood stored in
the arteries should reach a maximum. Thus
systolic storage should approximately equal
peak storage. A considerable proportion of the
stroke volume (average 67%) is stored during
systole. This corresponds to an average of 57
ml, which may represent 50% of the mean
pulmonary arterial blood volume. Thus the
total volume of the pulmonary arteries may
expand during systole an average of 1.6 times
the volume at the end of diastole. We can not
say, however, how this volume expansion is
distributed between the larger and smaller
arteries.
The distensibility of the pulmonary arteries accounts for the delay of conduction of the flow wave from the one end of the system to the other and for the storage of blood during systole, and hence for the spread of an almost square wave of input to a more continuous output, just as the distensibility of the systemic arteries helps to provide steady flow in the systemic capillaries. Thus if the distensibility of the pulmonary arteries increased without alteration in their static dimensions, then the same inflow pulse would take a longer time to be conducted to the capillaries and would be dispersed within the cardiac period to a greater extent because the systolic storage would be greater. The inverse would result from a decrease in the distensibility of the pulmonary arteries, and in the extreme case of completely rigid pulmonary arteries, the rate of blood flow at all points along their length would always be in phase because blood is a practically incompressible fluid.

During diastole, forward flow is maintained by the volume of blood stored in the pulmonary arteries in systole and then discharged to the capillaries. The pattern of this discharge, and hence the rate of diastolic capillary blood flow, should be an exponential function with time if the impedance to capillary flow is uniform and the pulmonary arterial distensibility linear. However, tidal movements of blood within the pulmonary arterial system during diastole may also distort the exponential pattern of diastolic flow.

The modifications of the pulmonary capillary flow pulse following administration of atropine and isoproterenol and during exercise can be explained by the passive effects of the altered pattern of right ventricular ejection on the dynamics of flow in the pulmonary arterial system. Thus after atropine the frequency of ejection was increased, the stroke volume was somewhat decreased, the ejection period occupied a greater part of the cycle, and hence the inflow in the pulmonary arteries was more continuous than at slow frequencies. The systolic storage represented a lesser proportion of the stroke volume, and the expansion of the pulmonary arterial blood volume during the heart cycle should have been smaller. The small increase in conduction time which we observed under these conditions, however, is more difficult to explain. It suggests an increase in distensibility or in the blood volume in the pulmonary arteries at the end of diastole. The latter might have been true as the result of pooling of blood in systemic veins produced by atropine.

Following isoproterenol administration, both heart rate and stroke volume showed small increases, but the major effect was the higher acceleration of blood into the pulmonary artery from the right ventricle, as reflected in the increase in average systolic acceleration of capillary blood. Under these conditions, we could visualize the radial expansion of the pulmonary arteries formed by the flow wave to be of greater amplitude with a more square front and traveling faster along their length. Thus during systole the proportions of stroke volume carried by the flow wave front to the capillaries and stored in the arteries were the same as during the control period. This suggests that the total volume distensibility of the pulmonary artery is curvilinear, decreasing at greater volume expansion, or that isoproterenol caused stiffening of the pulmonary arteries.

Similar conclusions may be drawn from the changes seen during exercise. The most striking change of the capillary flow pulse in exercise, however, was the large increase in peak flow rate, obviously the result of a high peak inflow velocity of blood to the pulmonary arteries.

The above considerations suggest that although the pulmonary arterial elastic reservoir does not convert pulsatile flow to steady flow, it does impose a considerable delay in the transmission of the flow wave and allows an appreciable amount of diastolic flow to take place in the capillaries. This effect becomes more important at slow heart rates, when the inflow into the pulmonary arteries is more discontinuous, and should play a part in improving flow continuity during irregularities of heart rate such as extrasystoles or arrhyth-
mias. Whether or not it plays a part in the function of the total pulmonary vascular system as a volume reservoir for the left ventricle (24) we can not say on the basis of these results.

The volume distensibility of the pulmonary arteries appears to be so adjusted to their dimensions that changes in the inflow pattern are detectable at the site of outflow. Furthermore, the pulmonary capillary network appears to allow flow pulses of large amplitude and rapid rate of upstroke, like those during exercise, to be manifested. This suggests that physical characteristics of the pulmonary capillary network do not appreciably limit rapid changes in flow rate. We believe this supports the theory that pulsatile flow is accommodated in the pulmonary capillaries by the variation throughout the cardiac cycle of the total area of the capillary network in which flow takes place.

It is for this reason that we find attractive the description of pulmonary capillary blood flow as “sheet” flow between two membranes as has been elaborated by Fung and his associates (25).

References


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NICHOLAS B. KARATZAS and GRANT DE J. LEE

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