Pulsatile Flow and Pressure in Human Systemic Arteries
Studies in Man and in a Multibranched Model of the Human Systemic Arterial Tree

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SUMMARY This study seeks to explain mechanisms responsible for the contour of pressure and flow waves and the pattern of vascular impedance in human systemic arteries. Pulsatile pressure and flow were recorded from the ascending aorta of seven patients undergoing open heart surgery and from the ascending aorta and other arteries of 17 patients at diagnostic catheterization. Ascending aortic pressure/flow relationships in the seven surgical patients were expressed as input impedance to the systemic circulation. Pressure and flow wave contour and impedance results were interpreted with the aid of a multibranched model of the systemic arterial tree, whose parameters could be manipulated to simulate different physiological and pathological conditions. Our data and data previously published on pressure and flow waves and their relationship in human subjects could be explained on the basis of two reflecting sites in the systemic circulation—one representing the resultant of all arterial terminations in the upper part of the body, and the other, some 1.5 times further away, the resultant of all arterial terminations in the lower body. The concept of the arterial system as an asymmetric T tube with two discrete ends has been advanced previously to explain the main features of pressure and flow waves and their relationship in different experimental animals. This concept appears equally applicable to human subjects. Circ Res 46: 363-372, 1980

Pulse contour in any artery is determined by the time course of ventricular ejection and by vascular properties. Separation of cardiac from vascular effects has been aided by determination of vascular impedance (McDonald and Taylor, 1959; McDonald, 1974), which permits characterization of vascular properties downstream from the point of measurement. Impedance determinations in various arteries of different experimental animals have shown great consistency and have been interpreted to show evidence of strong wave reflection in peripheral vascular beds (McDonald, 1974; O’Rourke and Taylor, 1966, 1967; Avolio et al., 1976). Such studies have suggested that the main features of pressure and flow waves in major arteries are explicable on the basis of wave reflection between two functionally discrete reflecting sites in the upper and lower parts of the body (O’Rourke, 1967; McDonald, 1974; Avolio et al., 1976).

Studies of vascular impedance and of pulse wave contour in man generally have been interpreted in terms of peripheral wave reflection (Gabe et al., 1964; Mills et al., 1970; Nichols et al., 1977; Pepine et al., 1978), but there is no agreement as to the intensity of wave reflection or the site or sites of reflection. Recent studies (Murgo et al., 1978a, 1978b; Westerhof et al., 1979) have been interpreted in terms of one apparent reflecting site in the vicinity of the distal aorta, with variable reflection, ranging from strong to minimal, in different patients and under different conditions.

We undertook this investigation to seek a comprehensive explanation for the main features of pressure and flow waves in major human systemic arteries. We wished to explain vascular impedance and pulse contour in humans on the same basis as previously proposed for experimental animals.

In an attempt to overcome the difficulties of obtaining data for humans under different conditions, we developed a digital computer model of the human systemic arterial tree; parameters of this model could be manipulated to simulate different physiological and pathological conditions. Thus, the present study comprises collection and analysis of experimental data for humans, interpretation of data for humans already published by others, and simulation in the human arterial model.

Methods

Patients

Pulsatile arterial pressure and flow were measured in the ascending aorta of seven patients at...
open heart surgery, immediately before starting cardiopulmonary bypass, and in systemic arteries of 17 patients during diagnostic catheterization. The experimental procedures were considered and endorsed by the Cardiovascular Unit at St. Vincent's Hospital, and informed consent was obtained from all participating patients. The subjects' ages were 23-60 (average 47) years. None had clinical evidence of aortic or peripheral arterial disease. In the group of seven subjects in whom ascending aortic impedance was determined, five had coronary artery disease, one had mitral stenosis, and one an atrial septal defect; ages were 40-52 (average 47), and mean blood pressure was 62-118 (average 100) mm Hg.

For the seven subjects studied at surgery, flow was measured with a cuff-type electromagnetic transducer applied to the exposed ascending aorta. Pressure was measured immediately distal to the transducer through a 21-gauge needle inserted directly through the aortic wall and with its bevel facing the direction of flow, as in previous experiments on animals (O'Rourke and Taylor, 1967). The needle was connected to a Statham pressure transducer by a short rigid catheter. The flow transducer was connected to an E.M.I. (E.M.I. Co.) flowmeter instrument. Data were recorded on magnetic tape.

For the 17 subjects studied at diagnostic catheterization, data were obtained with an S.E. (S.E. Laboratories) catheter tip electromagnetic flow transducer incorporating a lumen for measuring arterial pressure. The catheter was inserted in the brachial artery and advanced, when possible, to the ascending aorta. Flow was measured by an E.M.I. floor flowmeter. Pressure was measured with a Statham strain gauge manometer. Data were recorded on magnetic tape.

We calibrated flow transducers in vitro by passing physiological saline at known velocity over (for the catheter) or through (for the cuff) the transducer. Frequency response of the E.M.I. flowmeter showed amplitude flat to 30 Hz and a phase delay of 3.0°/Hz (Goodman, 1966). The S.E. instrument had a flat amplitude response to 30 Hz and a phase delay of 2.0°/Hz (manufacturer's specifications). The manometer was calibrated against a column of mercury and frequency response determined by the pressure-step or "pop" test; natural frequency of the system used in the operative studies was in the range of 50-70 Hz, and the damping coefficient averaged 0.18. It was difficult to maintain an adequate frequency response for the manometer system through the S.E. flow catheter lumen; because of this and apparent motion artifact on ascending aortic flow tracings, impedance was not determined from records obtained at diagnostic catheterization. Sampling rate of waves recorded at surgery was 100/sec.

The relationship between pulsatile pressure and flow in the ascending aorta was expressed as vascular impedance by relating corresponding harmonics of simultaneously recorded pressure and flow waves, as described elsewhere (O'Rourke and Taylor, 1966, 1967). Vascular impedance is analogous to electrical impedance and takes the form of a graph of modulus (amplitude of pressure + amplitude of flow) and phase (delay between pressure and flow) plotted against frequency. Allowance was made for frequency characteristics of flowmeter and manometer systems in calculation of vascular impedance. Impedance was plotted only for the first eight harmonics corresponding to a frequency range of 0-12 Hz. Impedance values were rejected if obtained from either pressure or flow harmonics which were considered to be within the noise level of recording instruments (<0.5 mm Hg for pressure, <1 cm/sec for flow).

The Arterial Model

The multibranched model of the human arterial system was developed on a digital computer (Avolio, 1976; Avolio and O'Rourke, 1976) (Fig. 1). The configurations and dimensions of major arteries for this model were obtained from anatomic texts. This model bore a close relationship to the analog computer model of Noordergraaf et al. (1963) but was more realistic in its representation of the vascular bed, particularly that in the upper part of the body. Values of wall thickness, diameter, and elasticity were identical to those used by Noordergraaf et al. (1963) and by Westerhof et al. (1969). Properties of the model with respect to pressure wave transmission and pressure/flow relationship were determined through application of Womersley's theory of fluid flow in elastic tubes (Womersley, 1957) and uniform electrical transmission line theory. Computational techniques were virtually identical to those used by Taylor (1966), the main difference being that this model used anatomically realistic values of arterial length, whereas Taylor's model generated arterial segments of random length within the computer.

When the general algorithm of this digital computer model was used with the same arterial dimensions and properties as used by Noordergraaf (1963), identical results were obtained as with Noordergraaf's analog technique. The general algorithm also has been used with vessel dimensions obtained from angiography to simulate the arterial system of various experimental animals. There has been very good agreement between these studies and the experimentally recorded pressure and flow waves and derived impedance patterns (Avolio et al., 1976; Avolio 1976).

Results

Human Subjects

Contour of pressure and flow waves recorded in the ascending aorta, descending thoracic aorta, and brachiocephalic artery were similar to those re-
mental animals also show the same distinctive difference in flow patterns as in man between vessels supplying the upper and lower parts of the body (O'Rourke, 1967; Avolio et al., 1976).

Input impedance to the systemic circulation was determined from data obtained at cardiac surgery (Fig. 3). Modulus and phase of impedance in these subjects were similar to those found by others in man (Gabe et al., 1964; Patel et al., 1965; Mills et al., 1970; Nichols et al., 1977; Pepine et al., 1978; Murgo et al., 1978a; Westerhof et al., 1979). Characteristic impedance (the value of impedance modulus about which fluctuations occur) was approximately 600 dyne sec cm$^{-3}$ or with aortic diameter reported by others for man. There was little difference in pressure pulse contour between the ascending aorta and the proximal brachiocephalic artery and proximal descending aorta. In contrast, the brachiocephalic flow contour was quite different from that recorded in the ascending and descending aorta (Fig. 2). In the brachiocephalic artery, the peak of flow occurs early in systole, and this peak usually is followed by a plateau or notch on the systolic downstroke of the wave some 50–150 msec before aortic valve closure. Similar patterns are seen in the subclavian (Mills et al., 1970) and brachial arteries (Gault et al., 1966). Different flow patterns in arteries supplying the upper and lower parts of the human body have been noted previously and stressed by Mills et al. (1970).

Patterns of pressure and flow waves in the proximal aorta and brachiocephalic artery of human subjects were similar to those seen in experimental animals, although in general diastolic pressure and flow fluctuations were less marked in man. Experimental animals also show the same distinctive difference in flow patterns as in man between vessels supplying the upper and lower parts of the body (O'Rourke, 1967; Avolio et al., 1976).

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The distance to peripheral reflecting sites in the upper and lower parts of the body was calculated from brachiocephalic and descending thoracic impedance, respectively, assuming the minimal value of modulus to be reached at a frequency corresponding to one-quarter wave length from a functionally discrete reflecting site (McDonald and Taylor, 1959; McDonald, 1974). Wave velocity [calculated from the Moens Korteweg formula (McDonald, 1974)] was 514 and 478 cm/sec, respec-

**FIGURE 3** Input impedance of the systemic circulation in seven subjects and of the multibranched model. Values of peripheral resistance are given at upper left; values of mean arterial pressure (from above down) were (◼)99, (△)117, (□)114, (●)88, (▼)115, (○)103 mm Hg. Subjects, (△), (□), (●), (▼), (○), had coronary artery disease and underwent coronary artery bypass.

28 mm, approximately 97 dyne sec cm\(^{-2}\). This is in the range of 53-202 dyne sec cm\(^{-2}\) found by other workers. Humans show the same general patterns of ascending aortic impedance as experimental animals. However, characteristic impedance determined from linear velocity and expressed as dyne sec cm\(^{-2}\) appears to be greater in man and to show greater variability from subject to subject; in addition, fluctuations of impedance modulus appear to be greater than in dogs and other animals.

**The Arterial Model**

There was good agreement between ascending aortic impedance in the model and results obtained from subjects (Fig. 3). There was a minimal value of modulus at 3.0 Hz and a maximal value at 7.8 Hz. Impedance at the origin of the descending aorta was similar to that in the ascending aorta; at the origin of the brachiocephalic artery, the minimum and maximum of the impedance modulus occurred at 4.8 and 9.0 Hz, respectively (Fig. 4A).

**FIGURE 4** A: Vascular impedance in the multibranched model for ascending aorta, (AA), DTA, and BCA. B: Ascending aortic impedance in the multibranched model and at the input of the asymmetric uniform T tube with the following characteristics—short limb, 26 cm; long limb, 40 cm; wave velocity in short limb, 514 cm/sec, and long limb, 478 cm/sec; attenuation, 60% per wavelength; reflection coefficient, 0.7 at each end.
tively, in the brachiocephalic artery and descending aorta. These values were used to calculate the distance to reflecting sites as 26 and 40 cm, respectively \([514 + (4 \times 4.8) \text{ and } 478 + (4 \times 3.0)]\), from the origin of the brachiocephalic artery and of the descending thoracic aorta.

Thus, these impedance studies suggested that the complex arterial network of the model behaves—to a first approximation—like an asymmetrical T tube with a short limb 26 cm long, which represents all arteries supplying the upper part of the body, and a long limb 40 cm long, which represents the aorta and the other arteries supplying the lower part of the body (Fig. 1). The input impedance to this simple asymmetric T model was similar to that of the multibranched anatomical model (Fig. 4B). Data from Mills et al. (1970) support this asymmetric T model with short-to-long limb ratio of approximately 1:1.5. They calculated the distance to upper body reflecting site to be 29 cm from the brachiocephalic root and the distance to lower body reflecting site to be 31 cm from the middescending thoracic aorta at T7 level. Allowing 10 cm around the aorta arch and down from T4 to T7 level, the lower body reflecting site would have been 41 cm from the origin of the descending aorta. (Mills et al., 1970 had calculated the distance to the peripheral reflecting site as 44 cm from the ascending aorta.) The asymmetric T tube suggested by data of Mills et al., thus, had a short limb of 29 cm and a long limb of about 41 cm.

Figure 5 shows waves synthesized at the origin of the brachiocephalic artery and descending thoracic aorta when the model was activated by an ascending aortic flow wave recorded in one subject. These brachiocephalic and descending aortic waves were almost identical to those recorded by us and others (Gabe et al., 1969; Mills et al., 1970) in man. The brachiocephalic flow wave showed the same shortening of systolic flow as is noted in man. The most likely explanation for the difference between brachiocephalic and descending aortic flow patterns is the different distance to arterial terminations—and so to functional reflecting sites—in the upper and lower parts of the body. To test this hypothesis, all arterial segments in the upper part of the body were doubled in length and, in this model, activated by the same ascending aortic flow wave. This resulted in a loss of the characteristic brachiocephalic flow contour so that it resembled that in the proximal descending aorta (Fig. 5).

The effect of change in pulse wave velocity was simulated in the model by altering elastic constants of the arterial segments while maintaining all other parameters constant. Such changes had little effect on the descending thoracic aortic flow patterns but led to considerable alteration in the brachiocephalic flow wave (Fig. 6). An increase in pulse wave velocity led to further shortening of the initial brachiocephalic flow peak, whereas a decrease in pulse wave velocity resulted in lengthening of the period of forward systolic flow and appearance of a prominent secondary flow wave in diastole. Changes similar to these were brought about by increasing and decreasing the duration of ventricular ejection with pulse wave velocity held constant (Fig. 7). These alterations in brachiocephalic flow contour with reduction in pulse wave velocity or ejection period are virtually identical to those noted in man when wave velocity was reduced and ejection duration shortened during the Valsalva maneuver. (Mills et al., 1970).

Pressure waves synthesized in the model for the same ascending aortic flow wave were very similar to those recorded in corresponding arteries in man. The changes brought about by altered pulse wave velocity and ejection period also were similar to those reported in man. Under normal circumstances, a diastolic wave was not apparent in the proximal aortic or brachiocephalic pressure pulse. However, when pulse wave velocity was reduced, a diastolic wave became apparent in these vessels. As wave velocity then was increased progressively, the diastolic wave became less obvious, whereas amplitude of the pressure wave increased in late systole. These changes in wave contour were attributable to early return of reflected waves before aortic valve closure under normal circumstances and late return after aortic valve closure when wave velocity was...
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FIGURE 6 Changes in brachiocephalic artery (above) and descending thoracic aortic (below) flow waves with increase in pulse wave velocity by $\sqrt{2}$ (left) and decrease in wave velocity by $\sqrt{2}$ (right). Control waves in center.

FIGURE 7 Effects of change in ejection period on pressure and flow waves in the brachiocephalic artery of the multibranched model with systolic ejection 355 msec (left) and 222 msec (right). Ascending aortic flow (top) was input and brachiocephalic pressure (center) and flow (bottom) were generated in the model. The line XX corresponds to the peak of ventricular ejection; the line YY corresponds to the calculated return of peak reflection from the upper part of the body (26 x 2/514 = 0.10 sec after peak flow); and the line ZZ to the calculated return of peak reflection from the lower body (40 x 2/478 = 0.17 sec after peak flow).

Discussion

The heart occupies an eccentric position in the mammalian body and so is closer to arterial terminations in the upper part of the body than to arterial terminations in the lower body. This arrangement in dogs, sheep, and rabbits has been shown to result in two functionally discrete reflecting sites being presented to the heart—one relatively close and representing the resultant of all arterial terminations in the upper part of the body, and the other further away and representing the resultant of all arterial terminations in the upper part of the body, and the other further away and representing the resultant of all arterial terminations in the lower part (O'Rourke, 1965; O'Rourke and Taylor, 1967; O'Rourke, 1967; McDonald, 1974, Avolio et al., 1976). The mammalian arterial system has been likened to an asymmetric T whose shorter limb represents the upper body vasculature and the longer limb the aorta and lower body vasculature (McDonald, 1974). In dogs, the ratio of short-to-long limbs of the T is approximately 1:2 (O'Rourke and Taylor, 1967; O'Rourke, 1967), whereas in rabbits and sheep, it is approximately 1:3 (O'Rourke, 1965; Avolio, 1976; Avolio et
Artifact appeared to be present in all our records catheters, which were prone to whip and artifact. Ascending aortic impedance patterns were explained on the basis of two reflecting sites in the upper and lower parts of the body, presented by brachiocephalic and subclavian arterial beds on the one hand and the descending aorta and lower body vasculature on the other. This explanation was supported by determinations of vascular impedance in these individual arteries and in the ascending aorta when they were occluded.

The concept of two separate reflecting sites in the systemic arterial tree explained not only ascending aortic impedance patterns, but also the reciprocal diastolic fluctuation of flow in the brachiocephalic artery and descending aorta. It also explained the reciprocal diastolic pressure fluctuation in the upper and lower parts of the body, about a "node" in the proximal descending aorta. This concept modified the thesis presented by Frank (1905) and by Hamilton and Dow (1939), who considered that wave reflection occurred between a single peripheral vascular reflecting site and the aortic valve; this, however, could not explain the reciprocal flow oscillations which were subsequently demonstrated in upper and lower body vessels or the proximal location of the thoracic pressure "node."

Since the human arterial system has the same general anatomical arrangement as other mammals, one would expect that the same general principles for wave reflection as found in animals also would apply in man. There is, however, no general agreement that this is the case. Pressure and flow data and impedance determinations in man have been interpreted to show definite wave reflection (Mills et al., 1970; Nichols et al., 1977; Pepine et al., 1978), minimal or no wave reflection (Muro et al., 1978a), or that wave reflection disappears under some conditions including the Valsalva maneuver (Muro et al., 1978b).

We believe that the general principles do apply and indeed provide the only comprehensive explanation for our own and other published data.

One of the sources of confusion in man has been the variability of published data—not only between different series but also in the same report. Some of the variability is attributable to technical problems, particularly difficulties in measuring flow in the proximal aorta with the earlier electromagnetic catheters, which were prone to whip and artifact. Artifact appeared to be present in all our records taken from the ascending aorta with a catheter-tip electromagnetic flowmeter and was the reason for these data not being used for impedance determination. However, most of the variability in published results appears to be real and is attributable to differences in aortic compliance between different subjects. Variability of aortic compliance in man is well known and indeed has thwarted attempts to measure stroke volume from the pressure pulse (Remington et al., 1948).

Vascular Impedance

In the human ascending aorta, there is usually more fluctuation of impedance modulus with frequency than in experimental animals. Most reported studies have shown a maximal value of impedance modulus at 6-10 Hz following an initial minimum at 3-5 Hz. This finding has been interpreted to indicate wave reflection from a single functionally discrete peripheral reflecting site. The fluctuations of impedance modulus with frequency have been most marked in patients with hypertension and known arterial disease (Nichols et al., 1977) and in patients with aortic pressure waves suggestive of arterial degeneration (Muro et al., 1978a; O'Rourke et al., 1968; Puls and Heizer, 1967; Freis et al., 1966). These fluctuations have been least marked in normal subjects (Nichols et al., 1977), in patients with a normal aortic diastolic pressure wave (Muro et al., 1978), and during the Valsalva maneuver (Mills et al., 1970; Muro et al., 1978b; Westerhohf et al., 1979).

The model suggests an explanation for these findings. As a consequence of relatively long arms and a relatively short trunk, there is in man a relatively greater distance to reflecting sites in the upper part of the body than in experimental animals. The ratio of short-to-long limbs in the asymmetrical T model proposed for man is approximately 1:1.5 as against 1:2 for dogs and approximately 1:3 for sheep and rabbits. Thus, in man, there is less interaction of reflected waves from both sites, greater fluctuations of impedance modulus in the ascending aorta, and a maximum of modulus closer to twice the frequency of the first minimum than in experimental animals.

The greater fluctuations in impedance modulus in patients with known or apparent arterial disease (Nichols et al., 1977; Muro et al., 1978a) may be due to relatively greater wave velocity in the aorta and lower limb arteries than in the upper limb vessels. Changes in wave velocity with age appear to be more marked in the lower body arteries (O'Rourke et al., 1968; O'Rourke, 1970a). Such differential changes in wave velocity would result in reflected waves returning almost simultaneously from the upper and lower parts of the body and mimicking a single peripheral reflecting site. Disappearance of impedance modulus fluctuations during the Valsalva maneuver (Mills et al., 1970; Muro
et al., 1978b) is attributable to lower wave velocity in the thoracic and abdominal aorta with later return of reflected waves from the lower body (Kroeker and Wood, 1956).

The concept of the systemic arterial system as an asymmetric T thus appears to provide an explanation for our own and all published results of ascending aortic impedance in man under all conditions. The explanation of different patterns on the basis of difference in interaction of reflected waves is the same as previously offered for different experimental animals and seems more logical than the alternative explanation of difference in intensity of peripheral reflection.

Flow Patterns

The most convincing evidence of differential reflection from upper and lower body sites comes from consideration of the flow patterns in major arteries supplying the upper and lower parts of the body. It is acknowledged that these differ markedly in man (Mills et al., 1970) as well as in experimental animals. Flow patterns at a vascular junction are more affected by wave reflection than pressure patterns, since reflected waves subtract from flow in one direction while adding to flow in the other, whereas reflection adds equally to pressure in all branches of the junction (Mills et al., 1970). When the junction is between two vessels of unequal diameter (as that of the brachiocephalic artery and aortic arch), reflected waves are more obvious in the narrow vessel (Mills et al., 1970).

Mills et al. described three types of flow patterns in the brachiocephalic and subclavian arteries and referred to these as type I, type II, and type III patterns. These three waves correspond to the three brachiocephalic flow waves generated in the model for the same ascending aortic wave when pulse wave velocity or ejection duration was altered (Figs. 6 and 7). The three wave patterns from Mills' paper are redrawn in Figure 8. The timing of reflected waves from upper and lower body is indicated by triple arrows and the incisura caused by aortic valve closure by a single arrow. The reflected wave from the upper part of the body decelerates flow in the brachiocephalic artery and tends to shorten the period of forward flow; the reflected wave from the lower part of the body accelerates brachiocephalic flow in late systole or early diastole. The type I wave is explained to result from early return of reflected waves from upper and lower body which precedes aortic valve closure, type II to result from the lower body reflected wave occurring during the incisura, and type III to result from this wave following the incisura. Mills et al. offered this explanation for the type III but did not go further to explain type I and type II which were the most commonly observed patterns. This appears strange, since they had shown that brachiocephalic impedance was similar irrespective of brachiocephalic flow contour. Mills and colleagues showed the transition of type I through type II to type III brachiocephalic flow patterns during the Valsalva maneuver, with return to type I afterward. Gault et al. (1966) had shown similar changes in brachial artery flow patterns when ejection duration was shortened after a premature ventricular extrasystole. All changes are easily explained on the basis of decreased wave velocity in the aorta and decreased ejection period during the Valsalva maneuver, with different timing of upper and lower body reflected waves in relation to aortic valve closure.

![Figure 8](http://circres.ahajournals.org/)

**Figure 8** Types I, II, and III brachiocephalic flow waves redrawn from the paper by Mills et al. with suggested mechanism outlined below. The single arrow represents aortic valve closure and the triple arrow represents the timing of wave reflection from the upper part of the body and later wave reflection from the lower part of the body. The dotted line indicates the expected wave pattern in the absence of an incisura caused by backflow through the aortic valve.
Pressure Patterns

In experimental animals, pressure wave contour in different arteries is readily explained on the basis of wave reflection between two functionally discrete reflecting sites in the upper and lower parts of the body. This explanation has not been as obvious in man, because man often does not show the same evidence of wave reflection—a prominent diastolic wave (Porjé, 1946; Salans et al., 1951; Kroeker and Wood, 1956; O'Rourke et al., 1968) as is regularly seen in dogs, rabbits, sheep, and other experimental animals (Hamilton and Dow, 1939; O'Rourke, 1967; McDonald, 1974), except the guinea pig (Avolio et al., 1976). Children regularly show the same patterns as these animals, but as age advances, the diastolic wave tends to disappear (O'Rourke et al., 1968). In the elderly, in patients with hypertension and arteriosclerosis, the pressure wave is similar in all arteries with a late systolic peak, and with pressure falling exponentially from this peak, interrupted only by the incisura which represents aortic valve closure (Salans et al., 1951; Freis et al., 1966; O'Rourke et al., 1968; O'Rourke, 1970b).

These changes with age and disease are attributable to high arterial wave velocity with reflected waves from upper and lower body fusing with each other and with the initial wave generated by ventricular ejection (O'Rourke, 1970b). Although reflected waves are not apparent as such in pressure waves recorded in human arteries under most conditions, they do become prominent when wave velocity falls or duration of ejection shortens (Kroeker and Wood, 1956; Greenfield et al., 1967; Mills et al., 1970). In other conditions in man, such as shock when hypotension and a short ejection period are combined, the diastolic waves become extremely prominent (O'Rourke, 1970a). Indeed, such exaggerated diastolic waves in patients with cardiogenic shock can cause difficulties in timing of arterial counterpulsation (personal observations).

The arterial pressure wave in man and the changes seen with ageing, with disease, and with alteration of blood pressure and ejection period thus are readily explained on the basis of wave reflection between two functionally discrete sites in upper and lower parts of the body.

We present here an explanation of arterial pulse contour in man on the basis of two functionally discrete reflecting sites in the upper and lower parts of the body. This elaborates the original concept of discrete reflecting sites in the upper and lower parts of the body. This explanation has not been as obvious in man, because man often does not show the same evidence of wave reflection—a prominent diastolic wave (Porjé, 1946; Salans et al., 1951; Kroeker and Wood, 1956; O'Rourke et al., 1968) as is regularly seen in dogs, rabbits, sheep, and other experimental animals (Hamilton and Dow, 1939; O'Rourke, 1967; McDonald, 1974), except the guinea pig (Avolio et al., 1976). Children regularly show the same patterns as these animals, but as age advances, the diastolic wave tends to disappear (O'Rourke et al., 1968). In the elderly, in patients with hypertension and arteriosclerosis, the pressure wave is similar in all arteries with a late systolic peak, and with pressure falling exponentially from this peak, interrupted only by the incisura which represents aortic valve closure (Salans et al., 1951; Freis et al., 1966; O'Rourke et al., 1968; O'Rourke, 1970b).

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We present here an explanation of arterial pulse contour in man on the basis of two functionally discrete reflecting sites in the upper and lower parts of the body. This elaborates the original concept of wave reflection propounded by Otto Frank over 70 years ago and subsequently refined by Hamilton, Dow, Remington, Wood, McDonald, and others.

We do not claim that such a concept explains all features of the pulse—effects of subsidiary reflection sites, including the aortic valve, obviously play some role as well. Further, other factors besides reflection also modify the arterial pulse, including nonuniform elasticity and attenuation and dispersion of the wave in travel. We simply draw attention to a mechanism which appears to be responsible for the major features of pulse wave contour hoping that, if this can be confirmed, further attention can be given to the other factors which are responsible for alterations of arterial pulse contour in cardiac and vascular disease.

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Membrane Electrical Effects of Histamine on Vascular Smooth Muscle of Canine Coronary Artery

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SUMMARY This study was undertaken to determine some of the electrophysiological effects of histamine on the coronary arterial vascular smooth muscle of the dog. Transmembrane potentials were recorded from small (<500 μm o.d.) isolated canine coronary arteries with glass microelectrodes filled with 3 mM KCl. Histamine (10^-6 M) increased the resting membrane potential (E_m) from -55 to -64 mV and reduced input resistance from 9.8 to 4.0 MΩ. These effects of histamine were abolished when Mn^2+ (1 mM) was added to block Ca^2+ influx. The amplitude, maximal rate of rise, and frequency of the Ca^2+-dependent action potential induced by tetraethylammonium ion (TEA) increased in the presence of histamine in a dose-dependent manner (10^-7 to 10^-6 M). Also, the effect of histamine on the TEA-induced action potential was inhibited by the H1 antagonist pyrilamine maleate (10^-7 M). When tension was recorded from helically cut strips of coronary arteries, variable results were obtained upon addition of histamine; i.e., some preparations showed no change in tension and others, a small increase. When histamine was added to this preparation in the presence of TEA, tension increased to 60% of the maximum contraction induced by K^+. These findings suggest that histamine increases the Ca^2+ inward current in coronary arterial smooth muscle. The hyperpolarization induced by histamine may be due to an increased K^+ conductance that is mediated by an increased Ca^2+ influx, since inhibition of Ca^2+ influx by Mn^2+ prevented the hyperpolarization. Circ Res 46: 372-377, 1980

THE effects of histamine on vascular smooth muscle (VSM) are highly variable and range from excitation to inhibition to no effect (Broadley, 1975; Carrier, 1965; Reite, 1972; Altura and Altura, 1974; Hudgins and Weiss, 1968). When injected into the beating dog heart, histamine causes a marked increase in coronary blood flow (Giles et al., 1977) suggesting a vasodilation. However, when added to the bathing solution of isolated coronary arteries of dog (Carrier, 1965), swine, and man (Smith et al., 1951), histamine causes a vasoconstriction. Thus, there is controversy as to the direct mechanism of action of histamine on coronary arterial smooth muscle.

There are only a few reports on the effect of histamine on electrophysiological characteristics of VSM. Histamine increases spike frequency in rabbit mesenteric vein (Cuthbert and Sutter, 1965; Somlyo and Somlyo, 1968) and depolarizes rabbit pulmonary artery (Somlyo and Somlyo, 1968). There are

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