Attenuation of Wave Reflection by Wave Entrapment Creates a "Horizon Effect" in the Human Aorta

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Abstract—Wave reflection is thought to be important in the augmentation of blood pressure. However, identification of distal reflections sites remains unclear. One possible explanation for this is that wave reflection is predominately determined by an amalgamation of multiple proximal small reflections rather than large discrete reflections originating from the distal peripheries. In 19 subjects (age, 35–73 years), sensor-tipped intra-arterial wires were used to measure pressure and Doppler velocity at 10-cm intervals along the aorta, starting at the aortic root. Incident and reflected waves were identified and timings and magnitudes quantified using wave intensity analysis. Mean wave speed increased along the length of the aorta (proximal, 6.8±0.9 m/s; distal, 10.7±1.5 m/s). The incident wave was tracked moving along the aorta, taking 55±4 ms to travel from the aortic root to the distal aorta. However, the timing to the refection site distance did not differ between proximal and distal aortic measurement sites (proximal aorta, 48±5 ms versus distal aorta, 42±4 ms; P=0.3). We performed a second analysis using aortic waveforms in a nonlinear model of pulse-wave propagation. This demonstrated very similar results to those observed in vivo and also an exponential attenuation in reflection magnitude. There is no single dominant refection site in or near the distal aorta. Rather, there are multiple reflection sites along the aorta, for which the contributions are attenuated with distance. We hypothesize that rereflection of reflected waves leads to wave entrapment, preventing distal waves being seen in the proximal aorta. **(*Hypertension*. 2012;60:778-785.)** • **Online Data Supplement**

Key Words: pressure augmentation ■ pulse wave propagation ■ wave reflection ■ aging and disease ■ 1D modeling ■ wave tracking ■ pulse wave velocity ■ augmentation index

Wave reflection is thought to be an important mechanism of augmentation of blood pressure with aging and in disease. This hypothesis assumes that the forward-traveling (incident) wave from cardiac ejection is reflected back toward the heart at sites of impedance mismatch. Several investigators have tried to identify the principal reflection locations, based on estimates of wave speed and time of return of the reflected wave, usually arriving at differing conclusions.[1–4] Others have taken an alternative approach, instead, identifying changes in aortic composition or aortic diameter as being most important in the determination of reflection sites. Furthermore, a recent meta-analysis has found that reflection timing changes little with aging (despite the expected increases in pulse wave velocity), supporting the findings from the Framingham Study, which showed a lengthening of distance to an apparent reflection site with aging.[5,6]

One explanation for these findings is that the reflection site is not fixed but is dynamically determined by sites of impedance mismatch, tapering, and composition of the aorta. We set out to explore this by measuring incident and reflected waves along the aorta to quantify how the reflected wave timing and magnitude change at differing locations along the aorta. In addition, we used numerical waveforms obtained in a validated nonlinear model of pulse wave propagation to study the mechanics underlying wave reflections. We hypothesized that if major sites of reflection are present, wave reflection should become progressively earlier and increase in magnitude toward the distal aorta.

Methods

Subjects

Nineteen volunteers (age, 54 years [range, 35–73 years]; 13 women) were recruited from patients scheduled for coronary angiography in whom coronary artery disease was considered a relatively low probability. Patient characteristics are shown in Table 1. Exclusion criteria included previous coronary intervention, valvular pathology, regional wall motion abnormality, and rhythm other than sinus. All of the subjects gave written informed consent for participation in this study, which was approved by the local ethics committee.

Received August 18, 2011; first decision September 2, 2011; revision accepted June 18, 2012.

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The online-only Data Supplement is available with this article at http://hyper.ahajournals.org/lookup/suppl/doi:10.1161/HYPERTENSIONAHA.111.180604/-/DC1.

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***Hypertension* is available at http://hyper.ahajournals.org** **DOI: 10.1161/HYPERTENSIONAHA.111.180604**

Table 1. Patient Characteristics

Variable	Value
Age, y	54.0±10.3
Women, n (%)	13 (68)
Height, cm	164.0±8.9
Weight, kg	75±15
Blood pressure, mm Hg	
Systolic	151±22
Diastolic	82±12
Total cholesterol, mmol/L	5.0±1.1
HDL cholesterol, mmol/L	1.6±0.9
Triglycerides, mmol/L	1.5±0.9
History of diabetes mellitus, n (%)	0 (0)
History of ischemic heart disease, n (%)	4 (21)
History of hypertension, n (%)	11 (58)
History of smoking, n (%)	9 (47)
Drugs, n (%)	
Aspirin	9 (47)
Statin	8 (42)
Calcium antagonist	2 (11)
β-blocker	4 (21)
Angiotensin II blocker	1 (5)
α-blocker	1 (5)

Data are mean±SD for scalars or n (%) for categories. HDL indicates high-density lipoprotein.

Precatheterization

Although structural composition is considered to be the principal determinant of arterial physiology, many nonstructural physiological parameters have important regulatory roles. These may be influenced by physical, psychological, and pharmacological factors. To minimize the effects of physical exertion, all of the subjects rested in bed for 1 hour before angiography. Smokers were asked to refrain from smoking for 24 hours.[7] Similarly, subjects were required to refrain from coffee[7,8] and alcohol[9] for ≥12 hours before study and fasted for ≥9 hours before the study. To minimize psychological stress, all of the subjects had careful explanation of the procedure during the consent phase and ample opportunity for further clarification and reassurance.

Study Protocol

In all of the subjects, after coronary angiography, a Judkins right coronary guide catheter was positioned in the proximal aorta. A 0.014-in diameter pressure (Wavewire) and a Doppler flow wire (Flowwire; Volcano Corporation, San Diego, CA) were advanced just beyond the end of the catheter and carefully positioned to ensure that their sensor tips were aligned. Once in situ, small rotational movements were made to the Flowwire to obtain the peak Doppler blood velocity. Simultaneous recordings of pressure and velocity were made at 10-cm intervals along the length of the aorta for 1 minute at each location. Analog output feeds were taken from the ECG and Wavewire and Flowwire consoles into a National Instruments DAQ-Card AI-16E-4 and acquired at 1 kHz using Labview. The recorded data were analyzed offline using customized Matlab software (Mathworks, Natick, MA). The blood pressure and Doppler velocity recordings were filtered using a Savitzky-Golay filter[10] and ensembled using the ECG R wave as a fiducial point. All of the subjects received 5000 U of heparin IV before the hemodynamic measurement phase. No other drugs were administered.

The timing and magnitude or incident and reflected waves were identified using wave intensity from simultaneous measures of pressure and velocity as described previously.[11] Wave speed was calculated at each location using the single-point method and over the entire length of the aorta using the gold standard foot-to-foot transit time methodology.[12]

Statistical Analysis

The statistical package Statview 5.0 (SAS Institute Inc, Cary, NC) was used for analyses. Continuous variables are reported as mean±SE at each location. Comparisons were made using Student *t* test. Reproducibility of hemodynamic measurements was assessed using the method of Bland and Altman.[13] $P<0.05$ was taken as statistically significant.

Reproducibility

Reproducibility of hemodynamic measurements was calculated by examining separate 30-second recordings of blood pressure and velocity for each patient. The SD of the difference between these replicate recordings in the aorta was 5.9 mm Hg for mean blood pressure (5.1% of mean value) and 0.052 m/s for mean Doppler velocity (16% of mean value).

Numerical Model

Pressure and flow waveforms were simulated using a nonlinear one-dimensional model of pulse wave propagation in the 55 larger systemic arteries in the human.[14,15] The connectivity and properties of the simulated arterial segments (Tables S1 and S2, available in the online-only Data Supplement, and Figure 1) were based on validated data.[16] The flow rate prescribed at the root of the network was based on our in vivo measurements at the aortic root (Figure 2). Arteries were simulated as thin, homogeneous, incompressible, and elastic tubes, in which each section is independent of the others, and the blood as a homogeneous, incompressible, and Newtonian fluid, with a density of 1050 kg/m^3 and a viscosity of 4 mPa s. Examples of the modeled pressure waveforms are presented in Figure 2. Incident and reflected wave timings were calculated at 1-cm intervals along the aorta using a similar wave intensity methodology, as applied to the in vivo aorta. Local wave speeds were calculated using the parameters of the model at mean pressure (Table S1).

A "wave-tracking" algorithm was used to describe all of the waves reflected at the internal junctions of the 55-artery network by a single pressure pulse impulse starting at the root.[17] The exponential curves in Figure 1 (dashed lines) were calculated by fitting an exponential function of the form $A*\exp(-\lambda*t)$, with A and λ the fitting constants, to the positive and negative peaks.

Results

A Consistent Sequence of Waves in the Aorta

In all of the subjects, the first wave seen was a large forward-traveling compression wave caused by ventricular contraction (the "incident" wave). Very soon afterward (mean delay, 92.6±9 ms), a reflected wave was seen (Figure 3). This sequence was consistent in all of the subjects, although the intensity and timing of the individual waves differed between subjects and at different locations along the aorta.

Wave Speed Along the Aorta

Wave speed was found to be higher in the distal rather than the proximal aorta (6.8±0.9 m/s at aortic root to 10.7±1.5 m/s at 50 cm; $P<0.004$; Table 2). The time taken for the incident wave to travel from the aortic root to the distal aorta (50 cm) was 55±4 ms. Using this transit time, the estimated mean aortic wave speed over the 50-cm section of aorta was 9.9±0.9 m/s, which was similar to the average of the wave speed at all of the sites along the aorta (9.0±1.2 m/s) calculated by the single-point technique.[12] Mean wave speed was found to increase with age ($r=0.77$; $P<0.001$).

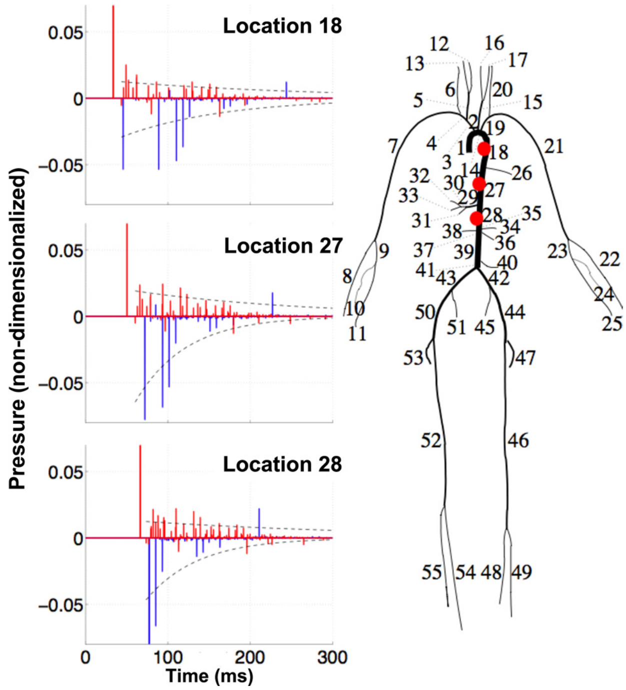

Figure 1. Theoretical evidence for the moving horizon effect in the human aorta. Pressure signals in the midpoint of the thoracic aorta A (location 18), thoracic aorta B (location 27), and abdominal aorta A (location 28) generated by a single wave starting at the inlet of the ascending aorta (location 1) at time t=0. They were calculated by solving the linear one-dimensional equations of pulse wave propagation in the elastic vessels of the 55-artery network using a wave tracking algorithm.[17] All of the pressures were nondimensionalized by the initial pressure in location 1, and only waves with a pressure >0.0001 of the initial pressure were computed. Forward-traveling waves are shown in red and backward-traveling waves in blue. After the arrival of the initial wave (which is truncated in the plots), there is an exponential increase in the number of reflected waves and an exponential decrease in their magnitude (highlighted with dashed lines). Reflection sites close to the measurement site contribute greatly to the magnitude of the measured reflected waves and those further away less so. This behavior is maintained as the measurement location is advanced along the aorta, creating an effect like a horizon that appears to move away as the measurement location approaches it. The properties of the 55 larger systemic arteries are shown in Tables S1 and S2.

Arrival of Incident and Reflected Waves in the Aorta

The incident (forward-traveling pushing) and reflected (backward-traveling pushing) waves were identified at each measurement site in the aorta. At each location, the mean time for the incident wave to travel to and return from the effective reflection site differed little between measurement sites along the aorta (92.6±9 ms; Table 2). There was no evidence of a single effective reflection site present in or near to the distal aorta. By using estimates of wave speed at each measured location in the aorta, the average distance to the reflection site was calculated as 41.8±2.1 cm. The timing to the refection site (in relation to the ECG R wave) did not differ, even between proximal and distal aortic measurement sites (proximal aorta, 48±5 ms versus distal aorta, 42±4 ms; P=0.3; Figure 4 and Table 2).

Magnitude of Wave Reflection in the Proximal Aorta

The reflection coefficient was calculated by dividing the magnitude of the reflected wave by the magnitude of the incident wave. The reflection coefficient was similar (20.3% to 16.9%) along the length of the aorta (Table 2). In the proximal aorta it was found to increase with age (r=0.51; P<0.03; Figure 5), systolic blood pressure (r=0.46; P<0.05), pulse pressure (r=0.61; P<0.005), and aortic wave speed (r=0.56; P<0.01), but was not related to diastolic blood pressure (r=−0.15; P=0.53). Overall mean reflection coefficient was not found to increase with age (r=0.05; P=0.8) or by separating patients according to hypertensive status.

Reflected Waves in the Numerical Model

The incident wave is seen to propagate along the length of the aorta in the numerical model (Figure 1) in a similar manner to the in vivo data (Figure 4). The speed of this propagation increases over the length of the aorta (Table S1), reflecting the change in aortic composition from a predominantly elastic vessel (proximal aorta, lower wave speed) to a more muscular vessel (distal aorta, higher wave speed). The mean reflected wave timings (Figure 4) are approximately parallel to the incident timings, again similar to the observations from the in vivo study.

At 3 sites in the modeled aorta, we calculated the magnitude and timing of reflected waves produced by a single pressure impulse propagated from the aortic root (Figure 1). We observed an exponential increase in the number of reflected waves at each site (3^m, with m being the number of reflection sites encountered), which corresponded with an exponential decline in reflected wave magnitude. Reflection sites close to the measurement site contribute greatly to the magnitude of the measured reflected waves and those further away less so. This behavior is maintained as the measurement location is advanced along the aorta, creating an effect like a "horizon" that appears to move away as the measurement location approaches it.

Discussion

In this study we have found that, despite changes in pulse wave velocity, the timing and estimated distance to reflection sites appear constant (with reference to the incident wave) along the length of the aorta.

Paradox of Reflection Sites in the Human Aorta

The established hypothesis of arterial wave reflection is that there are major sites of reflection in the distal vasculature, responsible for backward-traveling reflected waves. Although having a fixed anatomic reflection site is not essential to the generation of reflected waves per se,[18] it is thought to be important when attributing the change in shape in pressure waveforms, which occur with increasing aging and disease as pulse wave velocity increases.[19–21]

At each position along the aorta, we calculated the time interval between the incident initial contraction wave and the reflected wave, which represented the time taken for the wave to travel to the reflection site and return. Under the estab-

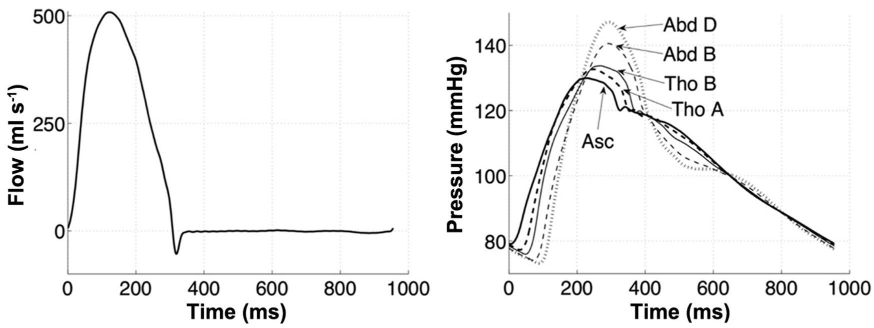

Figure 2. The modeled aortic pressure waveform in the human aorta using a nonlinear numerical model of pulse wave propagation in the larger 55 systemic arteries in the human (Figure 1). The pressure waveform is shown in the midpoint of the ascending aorta (Asc; segment 1), thoracic aorta A (Tho A; segment 18), thoracic aorta B (Tho B; segment 27), abdominal aorta B (Abd B; segment 35), and abdominal aorta D (Abd D; segment 39; bottom). At the inlet of the ascending aorta, the in vivo flow rate in the left panel was prescribed as a periodic inflow boundary condition.

lished hypothesis, the time for this "return trip" should shorten progressively as the point of examination moves distally toward the effective site of reflection. However, our findings show that the delay between the incident and reflected wave remained approximately the same (when referenced to the incident wave time) along the length of the aorta. Assuming a constant local wave speed, this distance was ≈45 cm at all sites in all of the subjects.

This finding suggests that the presence of either an anatomic or effective reflection site may be an over simplification and instead supports the observations of other authors who have identified multiple reflections sites along the course of the aorta, for which the contributions vary according to the site at which measurements are made.[1–3] In addition, this result is supported by the numerical data (Figure 1).

Why Does the Arterial Reflection Site Appear to Move?

Our interpretation of the data are that there are numerous minor reflection sites along the length of the aorta. Each individual reflection site makes a contribution to the summated reflected wave that is observed. Such reflections sites occur whenever a site of impedance mismatching is encountered, for example, at a bifurcation. At sites with greater impedance mismatching the magnitude of the reflected wave is larger and at sites with less impedance mismatching the magnitude of the reflected wave is smaller. Although it is tempting to consider wave reflection as a single entity, wave reflection is not a single wave but instead the combination of multiple tiny reflected waves. This is important when trying to rationalize how conditions of varying impedance mismatch

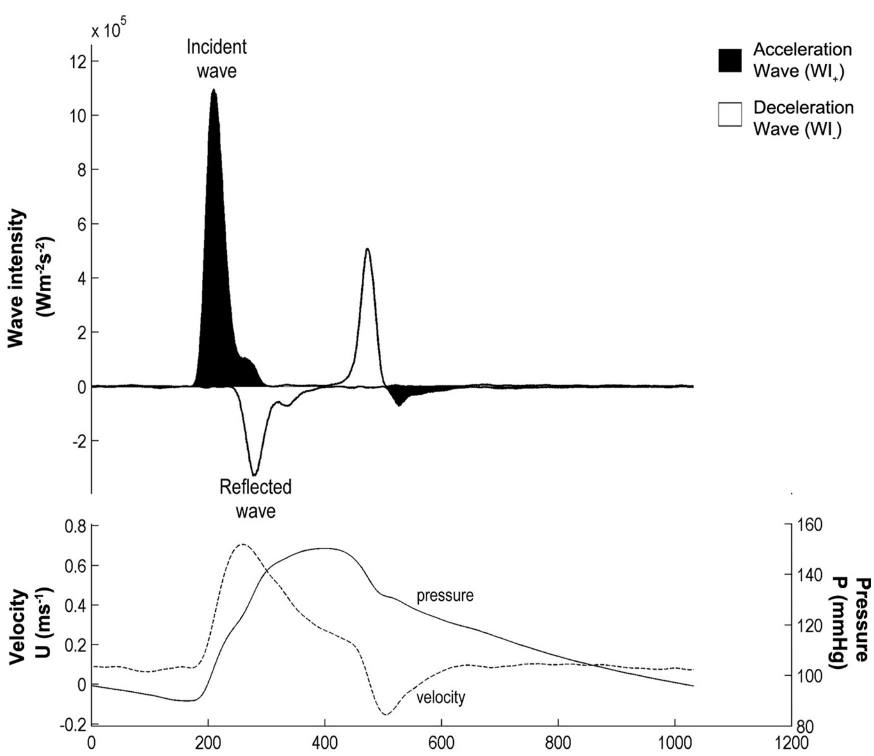

Figure 3. Identification of incident and reflected waves in the proximal aorta using wave intensity analysis. Wave intensity analysis is used to identify the incident and reflected waves in the proximal aorta (**top panel**). Corresponding pressure and velocity recordings are also shown (**bottom panel**). Using wave intensity, it is possible to determine the timing and magnitude of each wave in the cardiac cycle.

Table 2. Wave Speed, Timings of Incident and Reflected Waves, Estimates of Time to Reflection Site, and Reflection Coefficient at Each Measurement Location in the Human Aorta

Parameter	Aortic Root	10 cm	20 cm	30 cm	40 cm	50 cm
Wave speed, m/s±SE	6.8±0.9	9.1±1.3	9.2±1.1	9.4±1.3	9.1±1.3	10.7±1.5
Foot of incident wave, ms±SE	81±4.6	81.3±5.4	96.7±5.2	103.3±5.5	114.6±5.3	127.7±6.1
Time of inflection point, ms±SE	160.9±6.1	173.2±6.6	183.0±9.2	192.1±8.9	203.5±10.8	235±9.9
Median time of incident wave, ms±SE	115.3±3.4	123.9±4.6	133.9±4.3	146.3±5.5	154.0±4.4	170.9±4.8
Median time of reflected wave, ms±SE	211.4±9.6	209.8±9.2	235.3±7.3	239.4±7.3	250.9±7.2	254.1±9.7
Time to reflection site, ms±SE	48±5	43±4	51±4	47±3	49±3	42±4
Reflection coefficient, %	20.3±2	19.9±2	18.6±3	19.9±2	18.8±2	16.9±2

Data are mean±SE of n=19. No significant difference was found in either the time or distance to reflection site along the aorta.

effect forward and backward wave travel along the course of the aorta (Figure 1).

Horizon Effect From Attenuation of Reflected Waves

The arterial system has evolved to facilitate the forward passage of waves, minimizing cardiac workload while reducing the passage of backward-traveling waves.[22] This is possible because the degree of impedance mismatching (the ability of one system to accommodate input from another) at a bifurcation depends on the direction in which the wave is traveling when it encounters a bifurcation. When a forward-traveling wave encounters a bifurcation, impedance mismatching is low, and so most of the energy is transmitted forward with little reflection. In contrast, a backward-traveling wave encountering the same arterial branch from a reverse direction experiences a much greater impedance mismatch, and so most of the energy is reflected back distally with little transmitted proximally.

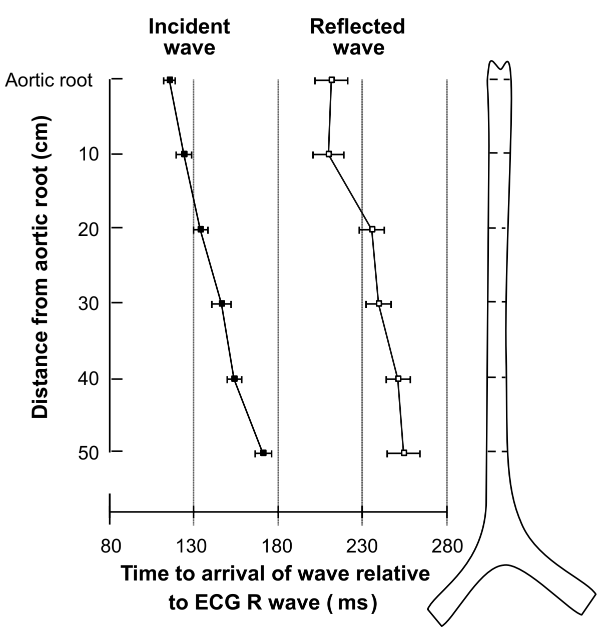

Figure 4. Time interval between incident and reflected wave remains approximately constant along the length of the aorta. The time of arrival of the incident (■) and reflected (□) waves were calculated at each measurement site in the human aorta. All of the timings are in milliseconds and relative to peak ECG R wave. The incident wave timing was delayed as measurement sites became progressively more distant from the heart. The reflected wave timing moved forward with the advancing incident wave and was not seen to shorten as expected as measurement sites approached major reflection sites, such as the renal arteries or the aortic bifurcation.

Thus, as waves travel progressively further along the arterial tree, they effectively become trapped with an ever diminishing proportion of their energy returning proximally to contribute to the observed reflected wave (Figure 6). The result resembles a horizon beyond which the magnitude of the reflected components is too small to contribute significantly to the observed reflected wave. This phenomenon is supported by the "wave tracking" analysis of our numerical waveforms (Figure 1). In addition to these mechanisms, it is also possible that transmission of backward-traveling waves could be further attenuated after accounting for the aortic reservoir pressure.[23]

Can Reflection and Rereflection Explain the Apparently Moving Reflection Site?

At each site of impedance mismatch, a proportion of the forward-traveling wave is reflected back. However, the backward travel of this reflected wave continues only as far as the next site of impedance mismatch where its intensity will be attenuated as described above. The residual backward-traveling wave experiences this attenuation at each of these reflection sites, because a relatively large proportion of wave energy is rereflected forward. Thus, the size of backward-traveling waves rapidly becomes diminished until in the proximal aorta it is no longer possible to identify contributions from the distal vasculature. This trapping by reflection and rereflection would explain why the net reflected wave in the aorta always appears to be reflected from a similar distance away, because the observed net reflected wave is preferentially composed of contributions from reflection sites relatively nearby. A similar horizon effect has also been identified in invasive animal experiments, where total occlusion of the aorta below the diaphragm made no difference to the timing of wave reflections measured in the proximal aorta.[24]

We suggest that aortic reflected waves are an amalgam of individual reflections from multiple reflection sites, with the nearer site contribution at full strength and sites further away having their contributions attenuated (Figure 6). The net effect of this distance-dependent attenuation is to create an effect like a horizon, which appears to move away as the

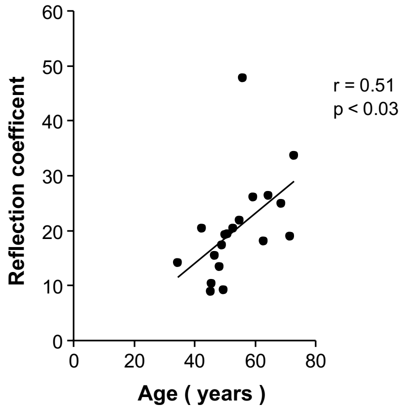

Figure 5. Increase in reflection coefficient in the proximal aorta with age.

measurement location approaches it. We hypothesize that this is because the configuration of impedance characteristics at bifurcations in the arterial tree preferentially transmit forward-traveling waves while preferentially reflecting backward-traveling waves.

Theoretical Evidence for the Existence of Multiple Reflection Sites

The nonlinear model used in this study is based on validated physiological data[16] and produces highly representative pressure waveforms (Figure 2). Although in humans we are frequently limited by the measurement constraints of the acquisition tools that we use, by using a numerical model it is possible to overcome these constraints and to further explore the mechanisms underlying our observations. We have used this model to track the passage of the incident wave along the length of the aorta to observe the timings of these reflections using a similar methodology to that applied to the in vivo data. Incident and reflected waves were observed traveling approximately in parallel along the aorta (Figure 7) similar to what was observed in the in vivo human data (Figure 4). Again, reflection timings were not found to shorten toward the distal end of the aorta.

In addition to tracking the incident and reflected waves using wave intensity analysis, we track the behavior of incident and reflected waves at bifurcations sites using the "wave-tracking algorithm" in Alastruey et al[17] (Figure 1). This algorithm demonstrates that a wave arriving at a bifurcation will generate 3 new waves; a reflected wave in the same segment and 2 transmitted waves in the other 2 segments connected to that bifurcation. Thus, the number of waves will increase exponentially with the number of bifurcations. After arrival of the incident wave, there is an exponential increase in the number of reflected waves but an exponential decrease in their magnitude (Figure 1).

Reflected waves are generated at multiple reflections sites along the course of the aorta; those close to the measurement site contribute greatly to the magnitude of the measured reflected waves and those further away less so. We hypothesize that this mechanism of wave trapping may explain the horizon effect by attenuation of reflected waves traveling back (toward the heart) in the arterial circulation. Our findings also support the observations of Segers et al[25,26] with regard to the limitations of using a pressure-derived inflection point as a method of identification of the timing of the reflected waves in comparison with the gold standard using pressure and flow.

Study Limitations

This study had power to show large differences in timing between waves arriving in systole and those in diastole but not to identify small changes in timing within a narrow time range in systole.

All of the hemodynamic measurements in the aorta used the aortic root as a starting landmark. Measurements were then repeated after successive withdrawal by 10-cm intervals. Although measurements in the aorta were made at precise locations, interpatient variation in aortic length will lead to

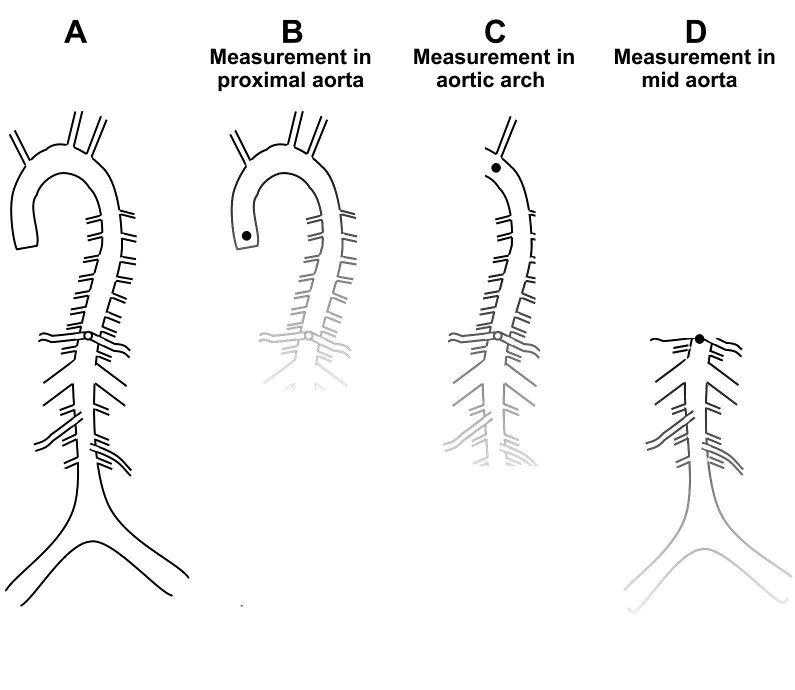

Figure 6. Illustration of the moving horizon effect in the human aorta. The arterial system is designed to facilitate the passage of forward-traveling waves. However, the properties that facilitate the passage of forward-traveling waves limit the magnitude of backward-traveling reflected waves. As a consequence, reflection sites close to the measurement site contribute greatly to the measured reflected waves and those further away less so. As the measurement location is advanced along the aorta (**B**, **C**, and **D**), the reflection site moves distally, creating an effect like a horizon, which appears to move away as the measurement location approaches it.

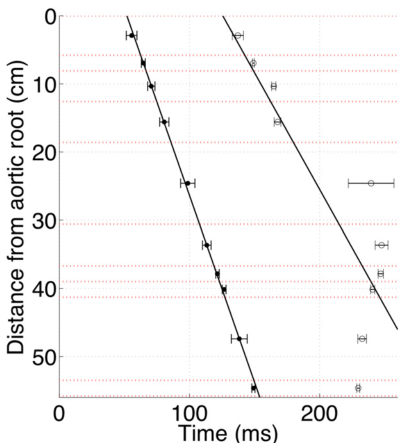

Figure 7. Modeled timing of incident and reflected waves along the aorta. The timing of arrival of the incident (filled circles) and reflected (unfilled circles) waves was calculated with distance (in 1-cm increments) along the aorta using the numerical data. The horizontal doted lines indicate the position of the inlet and outlet of each aortic segment in Figure 1. The linear fit of the reflected timings shows they are approximately parallel to the linear fit of the incident timings.

anatomic structures being located at different distances from the aortic root.

Estimates of wave speed are approximate for the calculation of distance to effective reflection site (we only know the local wave speed); wave speeds are likely to increase as vessels get smaller, so at each bifurcation the wave speed will probably increase, altering the estimate of distance to reflection site. We have used the single-point technique in our derivation of wave speed.[12] This determines wave speed locally rather than considering a long section of artery. Although this provides an accurate estimate of wave speed, there is a theoretical risk that, at very proximal reflection sites, wave canceling may produce higher estimates of wave speed than may otherwise be expected.

Perspectives

Wave reflection theory has been at the center of arterial hypertension research over the past 30 to 40 years. Although appealing from a simplicity perspective, it is increasingly being recognized that wave reflection theory alone cannot account for the underlying mechanisms of blood pressure augmentation.[5] This is perhaps most evident by the observation that, even to this day, only a single study has ever found a link between augmentation index and cardiovascular mortality. Identification of a horizon effect attenuating the return of the reflected wave casts doubt on the central tenets of wave reflection theory and suggests the presence of other mechanisms to explain pressure augmentation.

Although the effects of arterial stiffness on longitudinal pulse wave transmission have been extensively studied in wave reflection analyses, less attention has been paid to the loss of radial compliance (particularly in the elastic proximal aorta). Our findings and those of others suggest that this may be more important than previously envisaged and, together with new techniques that can measure its hemodynamic sequel (eg, the arterial reservoir[23]), may provide further mechanistic insight to account for increasing aortic pressure augmentation with aging and disease.

Conclusions

Using wave intensity analysis it is possible to accurately measure the timing and magnitude of the incident and reflected waves in the human aorta. There is no dominant reflection site along the aorta, and instead the timings of reflected waves are closely linked to those of the incident waves. Similar results were identified in both in vivo and numerical data. The intensity of reflected backward-traveling waves falls quickly in the aorta because of rereflection at sites of impedance mismatch. We hypothesize that this leads to wave entrapment, preventing distal waves from ever arriving back in the proximal aorta.

Sources of Funding

J.E.D. (FS/05/006), D.P.F., (FS/04/079), Z.I.W. (FS/05/068), and J.A. (FS/09/030/27812) are British Heart Foundation fellows. C.H.M. (077049/Z/05/Z) is funded by the Welcome Trust.

Disclosures

None.

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Novelty and Significance

What Is New?

- We conducted a serial analysis of separated forward and backward-traveling waves along the length of the human aorta.
- We compared the effects of branching on reflected wave propagation in a mathematical simulation and from in vivo human data.
- We measured the amount of wave energy that travels back from each measurements site.

What Is Relevant?

- Reflected waves are thought to be central to augmentation of central blood pressure, but studies are conflicting.
- This study increases the understanding of the mechanisms between the augmentation index and wave reflection.
- We assessed how the timing of waves relates to pressure augmentation.

Summary

There is no single dominant reflection site in the human aorta. Waves only travel back a short distance from their reflection sites. Wave energy falls rapidly, becoming negligible, shortly after being reflected. Wave reflection only has a small role in blood pressure augmentation. Wave reflection from the distal aorta cannot explain the inflection point in the systolic upstroke of the pressure waveform. Loss of aortic buffering capacity (or the aortic reservoir) may play a far more significant role in augmentation of blood pressure than wave reflection.