

Ultrasonic Measurement of Tissue Motion for the Diagnosis of Disease

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Abstract

Ultrasonic pulsed Doppler velocimetry has become a standard international method of classifying carotid disease. Because the measured angle adjusted velocity increases as the Doppler angle increases, examinations should be performed at a convenient standard Doppler examination angle. An angle of 60 degrees is achievable throughout most examinations. Multiple Doppler viewing angles allow the acquisition of velocity vectors during the cardiac cycle, revealing the complex velocity patterns.

Ultrasonic velocimetry (whether Doppler or time domain) is based on changes in the phase of the ultrasound echo. Other examinations can be done based on the echo phase. Slow motions of organs such as the brain can be used to monitor changes in edema. Measurements of tissue strain due to the pulsatile filling of the arterioles. This plethysmographic imaging method can display differences in tissue perfusion because of different tissue types and changes in autonomic activity.

Key words: Doppler, Blood Velocity, Tissue Motion, Vibrometry, Plethysmography.

Introduction

Since the development of the first medical ultrasound instruments half a century ago, ultrasound has provided two complementary kinds of information about tissue: echogenicity and motion. Echogenicity of the tissue, manifested in the strength of the attenuated compensated echo, has been used for image displays. First, the RF-A-mode then the demodulated A-mode, followed by the B-mode, then the 2-dimensional B-mode and now the 3-dimensional B-mode. Although 2-D real-time B-mode imaging is now the most popular diagnostic imaging method in the world, the images have several limitations. The echogenicity of tissue is highly dependent on the angle between the ultrasound incidence and the angles of any linear or surface structures in the

tissue. Although we assume that ultrasound travels in straight lines in tissue, refraction and specular reflection of ultrasound causes lateral displacement and distortion of objects in images and causes duplication of objects. Deflection of the ultrasound beam patterns has an equal effect on motion imaging methods.

In 1959, Satamura (electrical engineer) and Koneko (neurologist) introduced an ultrasonic method of detecting blood velocity and generating a sound with intensity related to the amount of flowing blood and frequency related to the observed velocity component of flowing blood. Independently, Dean Franklin and Don Baker developed a similar system. In the following years Doppler methods were improved with the directional Doppler introduced by McCleod, spectrum analysis by Strandness, Duplex scanning by Barbar and Strandness and Color Doppler

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imaging by Baker and associates.

Strandness, Sumner and Hokanson became interested in careful measurements of arterial wall motion just after 1970. However, in spite of progress in this area by Phillips, by Bonnefous, and by Hoeks, who each developed instruments to measure the motion of solid tissues, the diagnostic utility of tissue motion measurements remains undemonstrated.

In this paper, I'll describe some of the problems that stimulated our interest in using advanced Doppler and other tissue monitoring methods to solve specific problems in the diagnosis and classification of disease. This paper is based on a talk given in Seoul, Korea, 29 November 2002 to the Biomedical Engineering Society for Circulatory Disorders (www.besco.or.kr).

Pulsed Doppler Methods

Duplex scanning is a popular method for identifying and classifying internal carotid artery stenosis. If the Doppler examination is done using an examination angle of 60 degrees and the Doppler signal is displayed as an angle adjusted velocity (Figure 1), then the degree of stenosis can be classified using the maximum peak systolic measurement and maximum end diastolic measurement compared to the empirically developed classification criteria (Figure 2). A carotid velocity measurement exceeding 1.25 m/s indicates the presence of a stenosis. Patients with carotid end-diastolic velocities greater than 1.40 m/s have a clinically significant stenosis. These patients have a 20% chance of having a carotid source embolic stroke or TIA in the next 2 years. In these latter patients, several studies have demonstrated the effectiveness of carotid endarterectomy in preventing those strokes or TIAs.

To use these criteria, it is necessary to take the Doppler measurement at an angle of 60 degrees to the artery axis. Measuring blood velocities with Doppler in most arterial segments using a lower Doppler examination angle will provide lower velocity measurements, using a higher angle will give higher measurements. This is not an error in the Doppler equation, it is a result of complex blood flow in normal arteries. Use of the Doppler equation is based

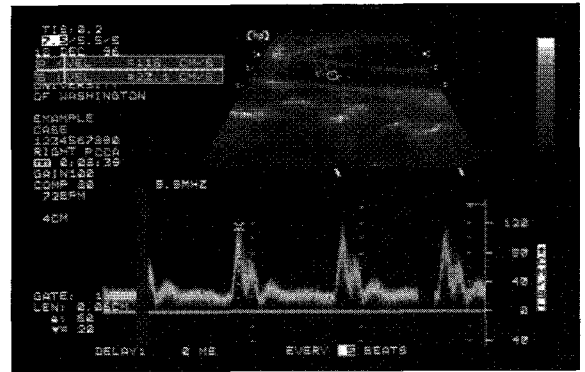


Figure 1 Carotid artery duplex scan.

Using the longitudinal view of the artery in the B-mode image (upper half) as a guide, the angle between the Doppler ultrasound beam pattern (dotted line running from upper left to lower right in the B-mode) and the vessel axis (dark uniform width band running from right to left at a depth of 1.2 cm in this 4 cm deep image). An arrow in the artery is adjusted by the examiner to indicate the direction and angle of the blood flow. One standard method of performing this examination is to set that angle to 60 degrees, as indicated in the lower left of the image ($\Theta = 60$). The peak systolic velocity is measured at the top of the initial acceleration. The end diastolic velocity is measured just before the onset of systole. As indicated by the two white marks at the bottom of the B-mode image, most of the ultrasound beam patterns forming the image are angled down to the right. However, on the left of the B-mode image, the ultrasound beam patterns form a "fan" pattern. At the left edge of the image, some of the ultrasound scan lines are perpendicular to the artery wall producing a bright echo at the wall due to specular back-reflection to the transducer.

Legend

A X Peak Systolic Velocity Measurement = 1.18 m/s

B + End Diastolic Velocity Measurement = 0.23 m/s

on the assumption that blood velocities are parallel to the axis of the artery. In normal arteries with laminar flow, the velocities are helical¹. In flow approaching a stenosis, the flow is converging, in flow after a stenosis, there is often a central jet with adjacent re-circulation zones.

To confirm this assertion that angle adjusted arterial Doppler blood velocity measurements take measurements in a variety of cases, not just low velocity normal volunteers where the effect is small. At the same location in an artery, take angle adjusted velocity measurements using several angles (40, 50 and 60 degrees).

Doppler methods measure only the velocity component along the ultrasound beam direction. Using geometry (Figure 3) it is easy to demonstrate that the magnitude of the net velocity vector can be determined by dividing the measured value by cosine of the angle between the ultrasound beam

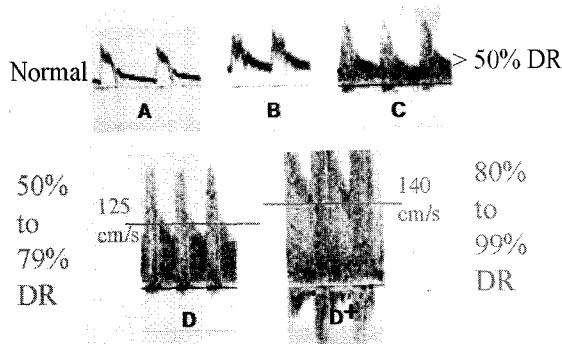


Figure 2 Carotid artery velocity criteria for classifying carotid stenoses.

If the carotid artery peak systolic velocity is less than 1.25 m/s (60 degree Doppler angle) then the stenosis seen on the corresponding arteriogram is likely to indicate a stenosis less than 50% diameter reduction. In such patients, the risk of embolic stroke from the carotid arteries is low. The use of increased spectral broadening (C, compared to A and B) to divide the classification into subgroups is not useful clinically and is subject to extreme inter-reader variability. If the peak of the early systolic acceleration at the location of highest value, during a regular heart cycle, is greater than 1.25 m/s, a > 50% diameter reduction carotid stenosis is likely. Such a stenosis is suspect as the source of emboli in a patient with symptoms of cerebral ischemia. If the end-diastolic velocity at the location of highest value, is greater than 1.4 m/s, a stenosis of greater than 80% diameter reduction is likely. The peak systolic velocity is often not measurable in such cases because of limitations on the sampling frequency of the Doppler. Maximum velocities exceeding 4.0 m/s are usual in such stenoses. The hydraulic forces on the plaque in such cases can disrupt the plaque causing emboli. These patients have a 20% chance of episodes of cerebral emboli if untreated.

Legend

DR = Diameter Reduction on arteriography. $DR [\%] = (NL - ML)/NL * 100 [\%]$

ML = minimum lumen diameter; NL = normal lumen diameter

There is controversy about the location where NL should be measured.

In European studies, NL is in the normal internal carotid artery distal to the stenosis.

In US studies, NL is the original diameter of the carotid bulb before the atherosclerotic lesion began to form. Since this cannot be seen, the radiologist must guess the location.

and the velocity vector. The velocity vector (magnitude and heading) in two dimensions or in 3 dimensions can be determined by making measurements of the Doppler sample volume along several viewing angles. The vector in a plane can be determined from two views, the vector in 3-dimensional space can be determined by 3 vectors.

Although an integrated 2-dimensional Doppler system was patented in 1981 which could acquire 2-dimensional Doppler data in real time, a practical system has not yet been developed for wide use². In the interim, Phillips et al³. (Figure 4) and others⁴ have attempted

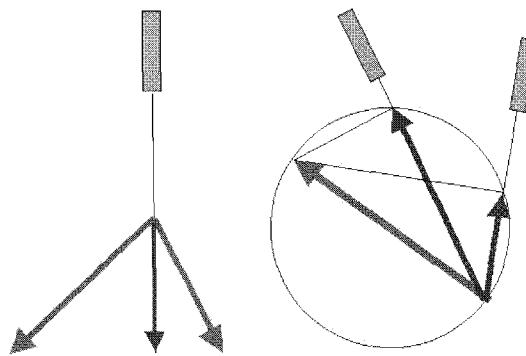


Figure 3A

Figure 3 Geometry of Doppler angle adjustment.

Doppler methods measure the component of velocity in the direction of the ultrasound beam pattern. Geometric constructions show the relationship between the measured component (blue) and the velocity vector (red): the velocity vector forms the hypotenuse of a right triangle, the Doppler measured component is the base of the triangle. The cosine of the angle between these values is the ratio of the measured component to the velocity vector. In all of the constructions, the origin of the velocity components and the velocity vector are at a common location, the construction shows the locus of the terminations of the vectors. It is important to remember that the heading of the velocity vector changes from moment to moment in normal blood flow.

Figure 3A. Left, the measured component (blue) can represent a variety of vectors (red). LEFT: The measured Doppler velocity component (blue) can be the vector component of any true velocity vector extending from the component origin to plane (in 3 dimensions) through the tip of the component which is perpendicular to the component. RIGHT: Geometrically, any triangle in a circle with one side along the diameter is a right triangle with the diameter as the hypotenuse. Thus, if the true velocity is known, all Doppler components form chords of a circle with the velocity as the diameter. The chords (component vectors) have the same origin as the velocity vector.

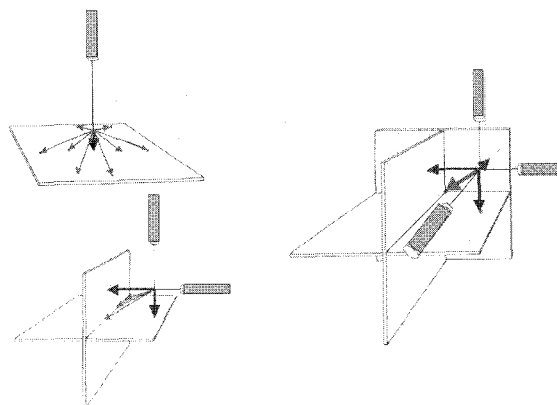


Figure 3B

Figure 3B. In 3 dimensions, three orthogonal components of the velocity vector can be projected together to determine the heading and magnitude of the velocity vector. This is a 3-dimensional extension of Figure 3A LEFT.

UPPER LEFT: Knowing one vector component of the velocity (blue) in 3 dimensions limits the true velocity to any vector (red) that terminates on a plane perpendicular to the component vector.

LOWER LEFT: Adding a second velocity vector component (blue) provides an additional constraint. The true velocity vector terminates along the line of intersection of the two planes. In this figure, the two component vectors are shown as perpendicular, but they don't need to be perpendicular, just not colinear.

LOWER RIGHT: Adding a third component, the true velocity vector terminates at the unique intersection of the three planes. In this figure, the three component vectors are shown as mutually perpendicular (orthogonal), but they don't need to be orthogonal, just not coplanar.

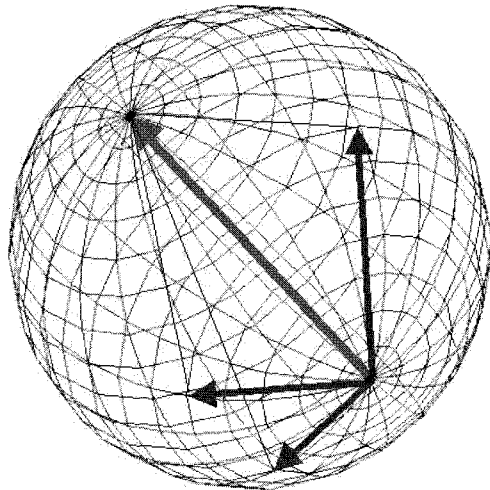


Figure 3C

Figure 3C. 3 velocity components do not have to be orthogonal, but only oriented so that they do not form a plane. By using the vector origins as a common point and the terminations as 3 additional points, these 4 points can be used to identify a sphere with the velocity vector forming the diameter. This is a 3-dimensional extension of Figure 3A RIGHT. Figure 3A RIGHT is true for any plane section through the sphere defined by the true velocity vector, passing through the origin of the true velocity vector. If the sectional circle does not include the diameter, of the sphere, then the diameter of the circle is a component of the true velocity vector.

to make usable systems. None has come into regular research or clinical use. The most convenient method of operating such a system is using a central transmitter with lateral receivers (Figure 5). Using such a 2-dimensional system, interesting velocity patterns can be measured (Figure 6). The velocity patterns that can be measured are complex, and their diagnostic utility is not immediately obvious. The correct measurement of arterial velocity is not necessary to properly classify arterial stenoses, but correct velocities are necessary to hemodynamic forces, such as the Bernoulli effect in a stenosis⁵.

In a typical case of carotid artery stenosis, the patient may have a blood pressure of 120/80 mmHg. In such a case, intra-stenotic velocities of 4.0 m/s systolic and 2.0 m/s diastolic are possible. In this example, a computation of the Bernoulli pressure depression leads to an interesting result. Substituting the density of blood into the Bernoulli equation and converting the pressure to mmHg yields the following equation:

$$\Delta p \text{ [mmHg]} = 4 \times [V \text{ m/s}]^2$$

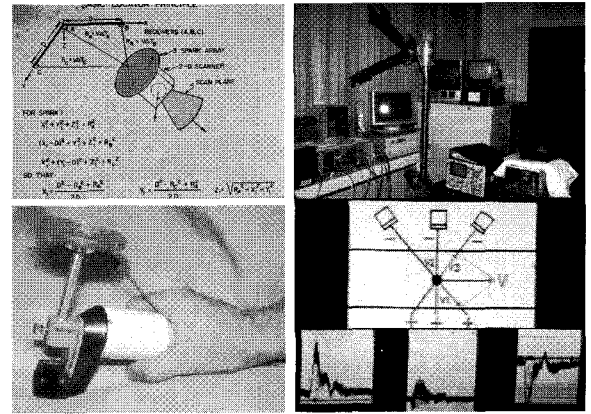
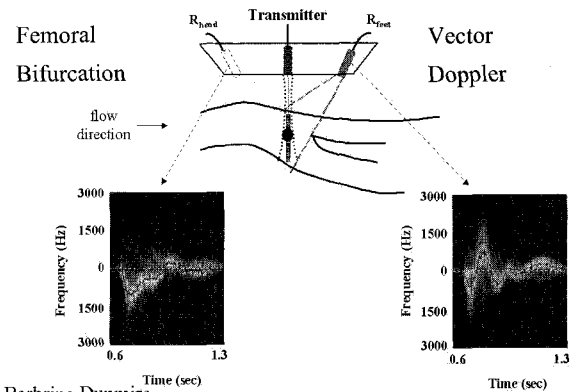


Figure 4 Historic "Spark Gap" 3-dimensional ultrasound scanhead tracking technique.

One method of obtaining the three dimensional data required is to track the location of the ultrasound scanhead. Pictured is a method developed around 1980 which uses an array of 3 spark sound sources mounted on the scanhead and an array of 3 microphones to detect the spark clicks and measure the time of flight. This system required an unobstructed path between the sparks and the microphones. It is also sensitive to temperature and breeze. Optical tracking systems have similar restrictions. A modern system using magnetic fields requires a non-metallic environment.



Barbrina Dummire

Figure 5 Vector Doppler scanhead.

Multiple views of the velocity vector can be obtained from a combination of a common ultrasound transmitter surrounded by an array of ultrasound receivers. The observed vector components are along bisectors of the angle between the transmitted beam pattern and the received beam pattern.

During systole, when the velocity is 4 m/s, the Bernoulli effect will depress the intrastenotic pressure by 64 mmHg during systole. During diastole, when the velocity is 2 m/s the Bernoulli effect will depress the intrastenotic pressure by 16 mmHg during diastole leaving a "paradoxical" intrastenotic blood pressure of 56/64 mmHg ((120-64)/(80-16)). This will cause the pulsation of the stenotic region of the artery to be inverted compared to the proximal normal region of the artery. If the pressure in the vasa-vasorum is 60 mmHg, then during systole, the

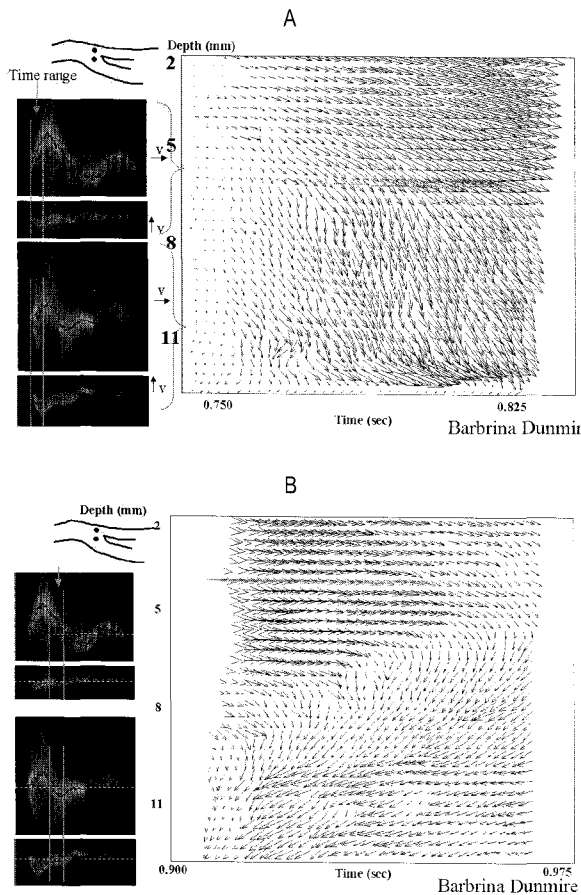


Figure 6 Vector velocity plots at the origin of the femoral bifurcation.

The plots show depth on the vertical axis and time along the horizontal axis.

A. During early systole, the difference in heading of the velocities entering the superficial femoral artery and the profunda femoris can be seen. Those entering the profunda are headed downwards.

B. In late systole, during deceleration, the velocity reversal in the profunda proceeds the reversal in the superficial femoral artery.

plaque can expand because the pressure compressing the plaque is lower than the pressure expanding the plaque. To predict the forces on the plaque, measurement of the correct velocity magnitude is critical. Although chronic hypertension does not increase carotid velocity, because the carotid artery flow rate is controlled by arteriolar auto-regulation, with an arrhythmia, the systolic velocity is occasionally critically elevated (Figure 7).

Note that Table 1 also sets a maximum on the possible blood velocity. The Bernoulli equation simply describes the relationship between potential energy density (related to pressure) and kinetic energy density (related to velocity). This velocity is the

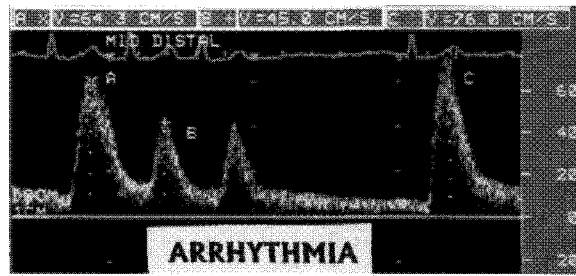


Figure 7 The Effect of ventricular filling on the carotid systolic velocity.

Over multiple cardiac cycles, the cerebral blood flow is regulated by the arteriolar sphincters to keep the peak systolic velocity in the carotid arteries constant. However, when an unexpected transient systolic pressure, either low or high, occurs due to inadequate or excess ventricular filling, the corresponding carotid velocity is correspondingly low or high. Thus, even if the usual peak systolic velocity is not high enough to reduce the intrastenotic pressure below the vasa vasorum pressure, occasional beats may result in such depression. This may explain why the control of heart rhythm reduces the chance of stroke.

Table 1 Bernoulli pressure depression.

Blood Velocity (m/s)	Bernoulli Pressure Depression (mmHg)
6	144
5	100
4	64
3	36
2	16
1	4

maximum exit velocity from a cylinder of liquid with a hole in the bottom. If the systolic pressure is 100 mmHg, it is not possible to get a blood velocity greater than 5 m/s, whether through a stenosis or through an arterial puncture from a bleeding artery.

Arterial Wall Motion and Vibration

An alternative to measuring the correct velocity is measuring the variation in arterial diameter compared to the cardiac cycle timing. In cases with very high systolic velocity causing a paradoxical pulse in the stenosis due to high systolic Bernoulli pressure depression can be detected. An early Doppler based arterial diameter measurement system was developed by Philips et al.(Figure 8)⁶. The arterial diameter waveform, seen in the center of the image has all of the characteristics of an arterial pressure pulse waveform or an AC coupled

plethysmographic waveform. This system digitized the RF with a 1 bit analog-to-digital converter operating at a 50 nanosecond interval at a selected depth to detect changes in the phase of the 5 MHz ultrasound echo of a quarter wave. This allowed a displacement of each arterial wall of 40 microns to be detected. With improved digitizers (12 bit rather than 1 bit), the displacement resolution can be improved by a factor of 1000 allowing the detection of 40 nanometer displacements.

Detection of carotid bruits with a stethoscope is a common method during physical examination. Around 1980, a number of investigators attempted to measure the amplitude, duration and frequency of bruits to assist in the classification of carotid artery stenoses (Figure 9). Because of attenuation, the amplitude of a bruit cannot be measured using a microphone, but the frequency and duration of a bruit can be measured. Dewey et al⁷, showed that the number 500 mm/Hz divided by the carotid artery bruit frequency yielded the minimal lumen diameter in a carotid stenosis^{7,8}. Unfortunately, the most important carotid stenoses have residual lumen diameters near 1 mm and the amplitude of the bruit is so low as to be undetectable in most cases. So, in the important cases, this signal is not available for diagnosis.

Using a method developed by Primozich, the frequency can be obtained from the spectral waveform of the

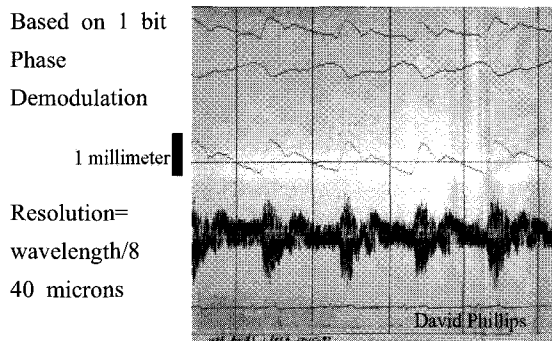


Figure 8 Tissue doppler tracking of arterial wall motion. If a pulsed Doppler beam is oriented nearly perpendicular to an artery axis, the motion of the artery walls can be tracked. In this image, the RF echo was digitized at 1 bit in quadrature to track the wall motion with a 40 micron resolution. The upper two tracings are the motions of the superficial and deep common carotid artery walls. The difference between these tracings is shown in the middle, the ECG is at the bottom under the spectral waveform.

Doppler signal (Figure 10). These wall vibrations are often associated with eddies which appear in the Doppler waveform if the time resolution of the waveform is adequate (Figure 11). Most current FFT spectrum analyzers require a millisecond for each FFT, so bruits near 500 Hz appear to be spectral broadening rather than waveform oscillations. For the harmonic pattern in Figure 10 to form, the bruit frequency must be greater than the inverse of the transform time. Using pulsed ultrasound phase demodulation methods and alternate analysis methods, the vibrations can often be measured at the arterial wall. These measurements at the arterial wall yield the amplitude, phase and duration of the bruit⁹. If the ultrasound beam is placed perpendicular to the vessel axis, the relationship between the eddy oscillations of blood in the artery and the wall motions can be seen. These motions are in phase.

By knowing the frequency and amplitude of the arterial wall vibration, the acoustic intensity of the bruit at the wall can be computed. By multiplying the area of the vibrating wall by the acoustic intensity, acoustic power dissipation can be computed. The hydraulic power of carotid flow is 50 mW; we have measured 5 mW of acoustic power dissipation in a bruit, but have not done a systematic study of this subject.

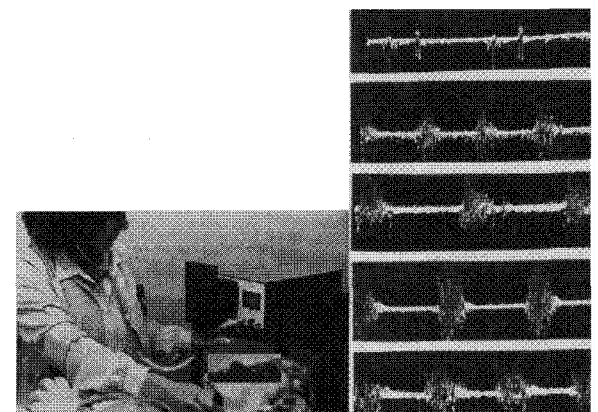


Figure 9 Carotid phono-angiography. Eddy flow, distal to the sudden expansion after a stenosis, causes wall vibrations called murmurs (by cardiologists) or bruits (by angiologists) which can give some information about arterial stenoses. The longer duration and louder sound of a bruit are generally considered to be more worrisome. The phono-angiography provides a visual display of the sounds heard through a stethoscope. Not shown here, quantitative phono-angiography provides a spectrum of the frequencies in the bruit.

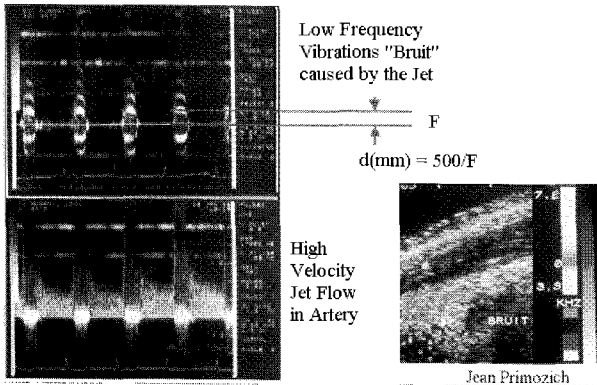


Figure 10 Bruits on spectral and color doppler displays.

Tissue vibrations range in frequency from a few Hertz to 1000 Hz. Because Doppler wall filters are typically set at 50 Hz, many bruits are passed through these filters and appear on the spectral waveform or color flow displays. On the spectral waveform, they are high amplitude, suppressing the blood signal (lower left) and when resolved by lowering the velocity (frequency) range of the display, form a double sideband harmonic pattern that is based on the Bessel Function. On color Doppler displays, the bruit usually appears as a checkerboard over solid tissue but can have other appearances depending on imaging processing methods.

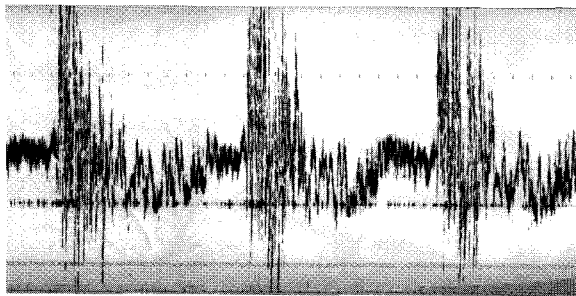


Figure 11 Spectral waveform of low frequency eddies.

By knowing the frequency and amplitude of the Spectral waveforms are formed with spectra computed from adjacent 10 millisecond windows providing 100 spectra per second. Therefore, eddy frequencies lower than 50 Hz (the Nyquist limit) can be resolved. Often the Doppler frequency shift caused by eddies is higher than the frequency shift caused by the normal blood flow. In this figure, the (had drawn) average velocity waveform (red) represents the blood ~0.1 sec average blood velocity. The oscillations around the average are eddies in the flow. This waveform was obtained at a Doppler angle of 60 degrees to the artery axis. If resolved into the velocity component parallel to the axis and the velocity component perpendicular to the axis, the component parallel to the axis probably shows little oscillation, but the component perpendicular to the axis contains large oscillations.

TransCranial Brain Velocimetry

Although it seems surprising, transcranial ultrasonic Doppler measurement of the blood velocity in the middle cerebral artery and other arteries of the Circle of Willis is now common. To penetrate the skull with ultrasound requires a low ultrasound

frequency, usually 1.5 or 2 MHz to reduce attenuation. Because of the high acoustic reflectivity of the bone surfaces, the bone may act as an interference filter, selectively rejecting the penetration of some frequencies. Because the erythrocytes in blood are Rayleigh scatterers, the echogenicity of blood increases by the fourth power of the frequency, favoring high frequencies for Doppler. 1.5-2 MHz represents a good compromise between ultrasound transmission through the skull (if it is not an interference filter) and echogenicity of blood.

Post-traumatic intra-cranial bleeding is a rare (1%) preventable cause of death. Detection with conventional CT imaging is expensive. Because the intra-cranial bleed is progressive, pushing the brain laterally and then posteriorly through the Foramen Magnum, we have been testing a Doppler ultrasound method of measuring the lateral speed of the brain. During a subdural bleed, the estimated lateral speed of the brain is 1 mm/hour or 15 microns per minute or 300 nanometers per second. By mounting an ultrasound transducer over the temporal bone on

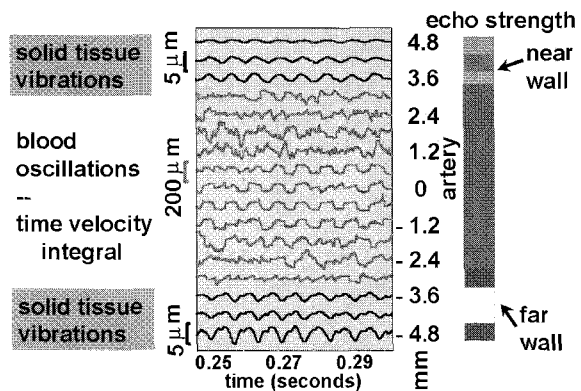


Figure 12 Eddy velocities and wall motion perpendicular to the arterial axis.

Wall vibrations that comprise bruits are caused by intraluminal eddies. These can be detected by Doppler ultrasound with the beam directed perpendicular to the vessel axis. In this case, the velocity was integrated to show blood displacement across the centerline of the artery. The coherent relationship between the intraluminal blood motion and the wall vibration can be seen.

VERTICAL AXIS depth through the vessel.

HORIZONTAL AXIS time during late systole, (50 milliseconds).

RED LINES Lateral blood motion in the lumen of the artery. Scale shows 200 microns of blood travel

BLACK LINES Lateral translation of the artery walls. Oscillation of the artery wall is a "Bruit" with frequency 160 Hz (8 cy/0.05 sec). This bruit frequency can be heard with a stethoscope near the vessel at end systole. Scale shows 5 microns of wall motion travel.

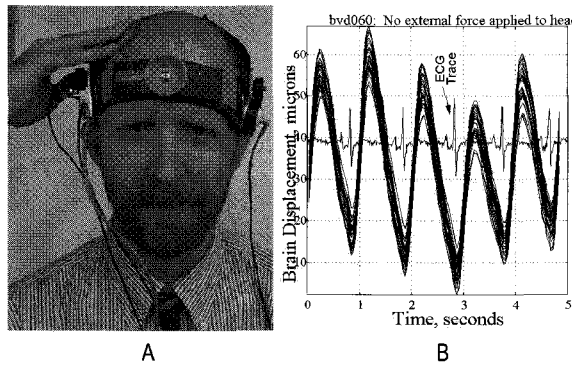


Figure 13 Brain motions by transcranial tissue doppler. By processing the transcranial ultrasound echoes from brain, the motions of the brain can be measured. In addition to the cardiac cycle, respiratory cycle motion can also be detected. The hand in this image serves to push the head sideways so that the resultant brain motion can be measured. The brain motion of 0.5 mm resulting from a hand push is not shown here. The head frame is instrumented with an accelerometer (patient right under hand) in addition to the ultrasound transducer (patient left).
A. Path of the ultrasound through the brain. The motion shown is from the contralateral parenchyma between the lateral ventricle and the cortex (D, A, C) and the motion is referenced to the contralateral skull (B).
B. Waveforms of 20 sample volumes from the contralateral brain parenchyma superimposed with ECG.

the side of the head, Doppler measurements can be made of the brain, demonstrating brain motion (Figure 13)¹⁰. The normal brain oscillates 60 micrometers laterally with each cardiac cycle and rocks an additional 15 micrometers with the respiratory cycle. Tilting the head causes the brain to float upwards 500 microns in the bathing cerebral spinal fluid.

Ultrasonic Plethysmography

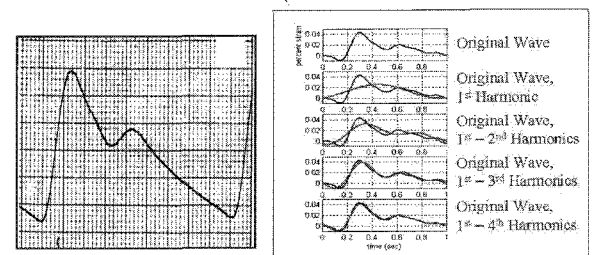
Plethysmography is a technique that measures the filling and emptying of blood in tissues with the cardiac and respiratory cycles. Normal tissues pulsate 0.1% by volume during the cardiac cycle swell 0.2% with the respiratory cycle and can expand by 2% with venous occlusion. The pulsatile expansion is due to the filling of arterioles, the slower expansions are due to the filling of the venules. This same effect is seen with a photoplethysmograph which can only look at the micro-circulation in the skin.

Because small tissue motions can be measured with ultrasound, a variety of methods of tissue motion measurement are under development by

others for tissue characterization. These methods, such as elastography, depend on an external mechanical drive to move the tissue under observation. Ultrasonic Plethysmography depends on the heart and the pulsations in the blood pressure to expand the tissue. Using tissue volumes (voxels) 1 mm thick and measuring 0.1 micrometer differential displacements, expansions of 0.01% can be resolved.

A typical plethysmographic waveform is a rounded triangular wave with a dicrotic wave interrupting the downslope (Figure 14). One of the tissues that we study is breast. For this examination, the breast space is divided into 500,000 voxels, each 3 mm by 3 mm by 1 mm deep. A wave like Figure 14 is measured in each of the voxels. The wave can be characterized by amplitude and phase. Then those parameters can be used to form an image. However, more information might be available if the waveform were represented by a 4 term Fourier analysis, with a magnitude and a phase for each harmonic. In this analysis, we use a 6 term Fourier series to represent the wave in each voxel. The phases, which characterize the waveform, are plotted as scattergrams, comparing each frequency with the other.

A simple tissue in this analysis is a muscle (Figure 15). We expected to see a uniform waveform representing



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Figure 14 Harmonic analysis of a plethysmographic waveform. Because there is a characteristic shape to a plethysmographic waveform, showing the arterioles filling with blood each cardiac cycle, the waveform can be decomposed into harmonic components. By combining the first 4 frequency components, the sharp up-slope and the dicrotic wave on the down-slope can be reproduced. This waveform decomposition method is used as a model of the waveforms obtained from tissue pulsatility measurements with ultrasound, by 3 mm by 1 mm deep. A wave like Figure 14 is measured in each of the voxels. The wave can be characterized by amplitude and phase. Then those parameters can be used to form an image.

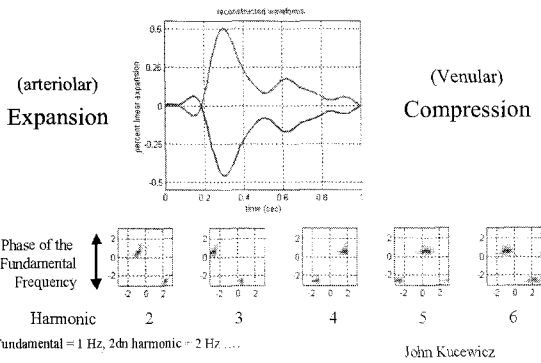


Figure 15 Tissue pulsatility waveforms from leg muscle.

The net expansion of leg muscles is composed of the combination of expansion and compression waveforms. The row of 5 scattergrams across the bottom of the image show the phase of the fundamental frequency (1 Hz) on the vertical axis and the phase of each of the harmonics (2 Hz to 6 Hz) along the horizontal axes. Each scattergram shows the data from all of the voxels present. The two spots on each scattergram verify that only two waveforms are present in the voxels in the tissue.

a 0.1% expansion. Astonishingly, the expansion amplitude was 0.5%, much greater than expected from measuring the whole limb volume expansion, however, two waveforms exist, an expansion waveform and a "compression" waveform that is the inverse of the expansion wave. The compression waveform was unexpected. Examining the phaseplots, only two waveforms exist. The same measurement in breast (Figure 16) provides a more complex result. In contrast to muscle, the two waveforms in breast have a continuum connection between them and there are more phase zones in the highest harmonic, suggesting more than one type of waveform.

Conclusion

Measurement and analysis of the motions of solid tissues can yield new information about the energetics of the tissue, and help the examiner to differentiate normal from abnormal tissues.

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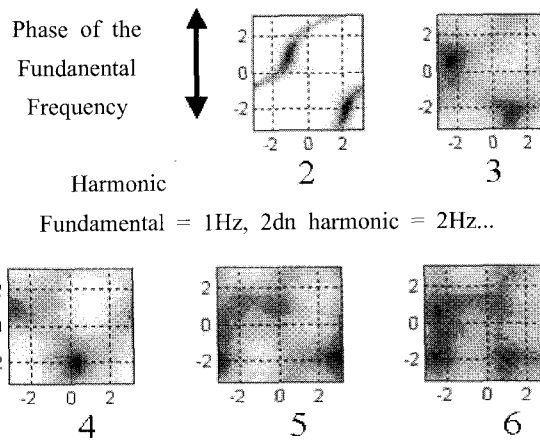


Figure 16 Tissue Pulsatility Waveforms from Breast.

The presence of multiple spots on the 5 Hz and 6 Hz plots show that there are at least 4 waveforms present, but they are different only in the higher harmonics, or fine structure of the waveforms.

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