Central Arterial Pressure and Arterial Pressure Pulse: New Views Entering the Second Century After Korotkov

MICHAEL F. O’ROURKE, MD, AND JAMES B. SEWARD, MD

The ubiquitous brachial cuff method gained widespread clinical acceptance for blood pressure recording after confirmation of its prognostic value in 1917. This method displaced radial pulse waveform analysis by sphygmography, which also gave prognostic information but was difficult to use. Since that time, brachial cuff sphygmomanometry has migrated from the physician’s office to 24-hour monitoring and home use, with electronic methods replacing the Korotkov sound technique for determining systolic and diastolic pressure. Detailed instrumental studies, required by regulatory bodies, revealed inaccuracies of all cuff methods for recording true intra-arterial pressure. A major source of inaccuracy in assessing left ventricular load is the amplification of the pressure wave in its transit from the central aorta to upper limb arteries, as extensively studied by Earl H. Wood at the Mayo Clinic in Rochester, Minn, in the 1950s. This limitation can be overcome by combining newer methods using radial artery waveform analysis in conjunction with conventional cuff sphygmomanometry to noninvasively measure the central aortic pressure waveforms. Recent studies using radial tonometry have proved that this is more effective than conventional manometry in predicting cardiovascular events and gauging response to therapy. Measurement of central as well as peripheral arterial pressure and physiology is becoming increasingly used as an office practice and a laboratory procedure.

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The brachial cuff methods introduced by Riva-Rocci1 in 1896 (systolic pressure by palpation) and Korotkov2 in 1905 (systolic and diastolic pressure by auscultation) were landmarks in clinical medicine, enabling clinicians to detect an important medical condition in asymptomatic or minimally symptomatic persons whose health was at serious risk. After Fischer’s presentation to the US Institute of Actuaries in 1917,3 blood pressure determination became routine part of the medical examination in general practice for life insurance and entry into the military service. The most graphic example of the importance of the sphygmomanometer cuff was the record of Franklin Delano Roosevelt’s arterial pressure before his stroke in 1945, which at the time was described in the press as coming “out of a clear sky” (Figure 1).4 Nonetheless, the method of measuring and interpreting arterial pressure as used 100 years ago must be reevaluated and revised if we are to assess patients adequately, make the most of antihypertensive drugs, and avoid inappropriate assumptions and problems with new and established drug therapy.5,6

The primary issue is a pervasive misunderstanding of diastolic pressure. There is no doubt about its importance in the case of President Franklin Delano Roosevelt, since diastolic pressure increased in conjunction with systolic pressure. Eighty years ago, systolic pressure was dismissed by the father of modern cardiology, Sir James Mackenzie, as giving a measure of cardiac force, whereas diastolic pressure was related exclusively to peripheral resistance and thus the assumed cause of the disease.5,7 Mackenzie7 stated, “As regards the relative importance of systolic and diastolic pressure, it may be said that systolic pressure represents the maximum force of the heart while the diastolic pressure measures the resistance the heart has to overcome.” This simplistic notion is attributed to James Orr, Mackenzie’s literary executor and editor of the third (1926) edition of his book.5 Such a view persisted despite initial doubts raised by Kannel et al8 in the Framingham study and Dustan9 until the results of the Systolic Hypertension in the Elderly Program were published in 199110; this study confirmed the sinister and dominant role of elevated systolic pressure in elderly persons and the benefits of lowering systolic pressure even if diastolic pressure was normal or low. Until that time, the diagnosis of hypertension and entry into hypertension trials were based on the level of diastolic pressure. Hypertension did not officially exist unless diastolic pressure was elevated. Further studies of arterial pressure continued to clarify the understanding of systolic and diastolic blood pressure. The Framingham study and other studies in Europe and the United States showed that with aging, systolic pressure in adults remains relatively constant up to approximately age 40 years, then rises progressively (Figure 2, top), whereas diastolic pressure rises progressively to age 50 years, then decreases so that pulse pressure (systolic minus diastolic), which is constant to age 40 years, begins to markedly rise

From the St. Vincent’s Clinic/VCCRI, University of New South Wales, Sydney, Australia (M.F.O.); and Division of Cardiovascular Diseases, Mayo Clinic College of Medicine, Rochester, Minn (J.B.S.).

Dr O’Rourke is a founding director of AtCor Medical Pty Ltd, manufacturer of systems for pulse waveform analysis.

Address reprint requests and correspondence to Michael F. O’Rourke, MD, Suite 810, St. Vincent’s Clinic, 438 Victoria St, Darlinghurst, NSW 2010, Australia (e-mail: M.ROrourke@unsw.edu.au).

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after age 50 years11-19 (Figure 2, bottom). Further studies by Franklin et al20,21 in the Framingham Heart studies showed that coronary risk was best predicted by diastolic pressure up to only age 40 years, by systolic pressure between 40 and 60 years, and by pulse pressure after 60 years (Figure 3). These studies indicated that low diastolic pressure in elderly persons had a more unfavorable prognosis. Results of these studies have been repeatedly confirmed and endorsed,22 although not uniformly accepted. For such dissenting work,23 methodological and other issues have been raised in previously published work.5,24,25

TECHNOLOGICAL INNOVATIONS

The changing views regarding arterial pressure during the past 15 years have been accompanied by a number of technological innovations that have and will substantially change clinical practice and are a focus of the remainder of this article.

The earliest innovation was introduction of the oscillometric device for clinic, home, and 24-hour blood pressure recording. One of the incentives to introduce such a device was to eliminate use of mercury because of environmental concerns. The oscillometric devices provide mean pressure from the level of maximal cuff fluctuation, with systolic and diastolic pressure determined by different proprietary algorithms. The pressure values usually conform to those obtained with the Korotkov method and must do so within specified limits for Food and Drug Administration acceptance.26 However, these limits are far wider than most clinicians appreciate (mean, ≤5 mm Hg; SD, ≤8 mm Hg) and raise doubts about the accuracy not only of the various devices but also of the conventional Korotkov method for measuring intra-arterial pressure27 since the same generous limits (mean, ≤5 mm Hg; SD, ≤8 mm Hg) are accepted for comparisons of the Korotkov method with intra-arterial pressure.5 A corollary of this issue27 is a warning to physicians not to trust the sphygmomanometer numbers alone and to seek confirmation with other means, such as by assessing target organ damage, so that abnormality is not accepted as normality or vice versa. Regrettably, the seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure28,29 appeared to stress this narrow “numbers” approach, whereas the European Societies of Cardiology and Hypertension favored a wider view.30 However “target organ damage” of retinopathy, albuminuria, elevated creatinine value, and left ventricular hypertrophy are usually late signs of hypertensive disease. Hence, other earlier evidence of hypertensive injury can be sought, such as increased left atrial size on echocardiography, microalbuminuria, and abnormalities of the pulse waveform or pulse transit time, indicating increased arterial stiffness or evidence of impaired endothelial function.31,32 None of these innovations has generally been accepted for routine use in cardiovascular assessment, but they should be understood and considered. However, use of home or 24-hour blood pressure measurement has gained widespread endorsement and
The major value is not just to overcome the well-known elevation of blood pressure on visiting a physician’s office, the “white coat” effect, but to obtain and average a large number of recordings and thus decrease the random error and limitations of the cuff process. The brachial method is the standard, but other noninvasive devices have been developed to measure pressure at the wrist or in the finger; a finger-based device (Portapres) has been used in the space program.

Ever since blood pressure recordings have been obtained invasively from experimental animals and humans, it has been known that the pressure pulse is amplified as it travels from the ascending aorta to peripheral arteries. This is accepted as being a consequence of pressure augmentation by wave reflection in peripheral arterioles. The brachial method is the standard, but other noninvasive devices have been developed to measure pressure at the wrist or in the finger; a finger-based device (Portapres) has been used in the space program.

**MEASUREMENT OF CENTRAL PRESSURE**

Blood pressure is customarily measured in the upper limb as a matter of convenience and available technology. The
phenomenon underwent detailed study at the Mayo Clinic by Earl H. Wood and colleagues, when cardiac catheterization was established more than 50 years ago. They confirmed that the mean pressure decreases by just 1 mm Hg or so between the ascending aorta and brachial or radial artery and that the diastolic pressure decreases by just a little more. However, the amplification in pulse pressure is principally due to an increase in systolic pressure, which is normally represented by a sharp narrow peak on the peripheral pressure wave. Further studies by Wood and others confirmed that this amplification of the systolic pressure wave between the aorta and peripheral (brachial or radial) arteries is greater in young vs old adults (the pulse wave of elderly adults is similar in amplitude in the aorta and upper limb). Amplification of systolic pressure varies with physiological maneuvers, such as change in heart rate, in body position, or with Valsalva maneuver, and with vasodilator drugs. The latter is particularly impressive for nitrates, in which a 20 mm Hg decrease in aortic and left ventricular systolic pressure may not be appreciated if reliance is on pressure recorded at the brachial artery (Figure 4).

The pressure augmentation phenomenon is of practical importance in the interpretation of community blood pressures. The apparent plateau of systolic and pulse pressure between 20 and 40 years is principally due to exaggerated augmentation of brachial systolic and pulse pressures in younger adults (Figure 2). When calculated indirectly for the aorta (see subsequent discussion) or when measured directly (Figure 2) aortic, systolic, and pulse pressure actually rise progressively with age.
The best method for measuring aortic and left ventricular systolic pressure is with a catheter threaded from a peripheral artery. However, this method cannot be justified except during a diagnostic cardiac or vascular procedure. Even during such a procedure, it is rare for the physician to take the opportunity to check aortic against brachial pressure or to perform any analysis on the contour of the recorded aortic pressure waveform.

Three procedures can be used to noninvasively estimate central aortic systolic pressure. For pressure calibration, all require accurate recording of the pressure waveform in the brachial, carotid, or radial artery. Thus, each noninvasive method is subject to the calibration inaccuracies of the cuff blood pressure measurement, as referred to previously. The first method uses a generalized mathematical transfer function process (see subsequent discussion) to correct for distortion of the pressure wave in travel to the brachial or radial site (Figure 5). The second method is based on the aforementioned near equality of mean pressure and of diastolic pressure in the aorta and upper limb. The third method is based on the ability to identify the peak of the secondary, reflected wave in the upper limb waveform.

The second method requires noninvasive measurement of central (usually carotid) pressure or diameter waveform, whereas the other 2 methods require only the calibrated upper limb pressure pulse. In ideal circumstances in which waveform quality is good and the same cuff values are used, correspondence between the different techniques is good. Each method is described subsequently in more detail.

**Transfer Function Process**

This process requires measurement of a pressure waveform invasively in the radial artery or preferably noninvasively from that artery by applanation tonometry. The 2 waveforms are virtually identical when requirements for applanation tonometry are met. The noninvasively recorded wave is calibrated to brachial (or radial) systolic and diastolic pressure. Usually, there is little amplification of the pulse between brachial and radial arteries. The peripheral pressure waveform is then convolved into the ascending aortic wave (Figure 5). The same mean pressure is assumed at the 2 sites. This process averages a series of waves during at least 1 respiratory cycle and provides a

![FIGURE 5. Top, Difference between simultaneously recorded central aortic and radial pressure waveforms. Bottom, Spectral plots of generalized transfer functions (shaded area defines 95% confidence interval) generated from the steady-state (left) or hemodynamic transient data (right). Modulus above; phase below. Adapted from *Circulation*, with permission.](image-url)
calibrated ascending aortic pressure waveform (Figure 6). The process has been shown prospectively to be applicable with changes in heart rate and rhythm, with vasoactive drugs, at different ages, and during the Valsalva maneuver.\(^5,39,41,43,44\) and it is accepted as equivalent to direct manometry by the US Food and Drug Administration. The process provides additional information regarding the aortic waveform (Figure 6) and pressure. Mayo Clinic cardiovascular investigators have used this process during the past 3 years.\(^45-47\)

**METHOD BASED ON EQUIVALENCE OF CENTRAL AND PERIPHERAL MEAN AND DIASTOLIC PRESSURES**

This method was first proposed by Kelly and Fitchett\(^40\) to calibrate carotid pressure waveforms for noninvasive calculation of systemic vascular impedance. It requires measurement of pressure waves in the carotid and radial or brachial arteries, then determination of diastolic and mean pressure in the peripheral artery. The same values are then applied to the central waveform, and the systolic pressure is calculated by extrapolation (Figure 7). One of us (M.F.O.) was originally skeptical, but the method has been confirmed by other colleagues\(^5\) and is now widely used.\(^5,48,49\) It has been applied to the carotid diameter waveform (which is almost identical to pressure).\(^50\)

**METHOD BASED ON IDENTITY OF BROAD ROUNDED REFLECTED WAVE IN CENTRAL AND PERIPHERAL ARTERIES**

The late systolic peak of the pressure waveform, rather than the sharp primary peak, almost always represents the systolic peak pressure in the ascending aorta and left ventricle (Figure 7). The transfer function process\(^5,39\) (Figure 5) confirms that the lower frequency components (ie, those <2 Hz) of the pulse are minimally amplified between the aorta and upper limb, whereas sharper spikes (around 3-6 Hz) are...
maximally amplified. If the peak of the secondary wave can be identified in the radial or brachial pressure pulse, it can give a good representation of peak aortic pressure (Figure 7). Even if identification of the peak of the secondary wave is not done formally, the decrease in central systolic pressure can be estimated with a drug such as nitrate based on the decrease in the late secondary systolic pulse in the brachial or radial artery.

**VALUE OF CENTRAL AORTIC PRESSURE MEASUREMENT**

As previously discussed, there is little difference in mean and diastolic pressure between central and peripheral arteries, however, any value of this knowledge relates to the ability of the examiner to confidently measure systolic or pulse pressure in the ascending aorta.

The most obvious benefits pertain to situations in which there can be significant differences between central and peripheral pressure, which may influence medical diagnosis or treatment or provide interpretation of mechanisms.

**CHANGE IN ARTERIAL PRESSURE WITH AGE**

As already discussed, there are age-associated changes in systolic and pulse pressure in the brachial artery (Figure 2). Systolic pressure increases progressively up to age 17 years (when full body height is attained), then plateaus up to age 40 years before again rising steeply from age 45 to 50 years (Figure 2, top). Brachial pulse pressure consequently remains relatively constant or decreases from age 17 to 40 years (Figure 2, bottom). Knowledge of wave transmission in the upper limb largely explains this phenomenon.

The pressure pulse is maximally amplified in the arm when the body is fully grown, during which time the large arteries still have the elastic properties of childhood. However, when aortic systolic and pulse pressures are considered, these rise progressively and linearly with age (Figure 2).

**Spurious Systolic Hypertension of Youth**

This phenomenon has been described as occurring principally in tall young males and represents an extreme of the amplification phenomenon (referred to previously). Such individuals usually come to a physician’s attention during a medical examination, the results of which show no abnormality except for high brachial systolic and pulse pressure. Unfortunately, these patients may be characterized as having isolated systolic hypertension, and barriers are placed on their careers and life opportunities. Multiple investigations are often undertaken, and therapy is prescribed. Hypotensive responses to therapy are relatively common.

When aortic pressure is measured with the invasive or noninvasive techniques, central aortic systolic and pulse pressures are within or close to the normal range (Figure 6). Diastolic and mean pressures are normal. When the radial pulse waveform is measured, the high peak systolic value is due to a narrowed apex of the wave in the arteries of the upper limbs. A narrowed apex is not seen in the aorta or in the carotid trace when the aortic pressure is measured independently.

Spurious elevation of systolic pressure is a relatively common finding; in a study by Mahmud and Feely, it was seen in about 12% of young male medical students. There is every reason to believe on mechanistic grounds that such persons can be reassured, therapy avoided, and medical restraints lifted. No definite outcome studies have confirmed this, but reassurance comes from French data that showed a low risk of cardiovascular complications for decades in such persons younger than 40 years and from Framingham Heart studies that showed, for the offspring cohort, that coronary events were inversely related to brachial pulse pressure for men younger than 40 years in contrast to a strong positive direct relationship for those older than 50 years (Figure 3).

**OUTCOME STUDIES**

The clinician is often concerned with outcome studies, which may not relate to individual patient management. Problems in the interpretation of peripheral brachial systolic and diastolic pressure have already been alluded to. The first published data on this subject was Fischer’s presentation to the American Society of Actuaries in 1917. This established the relationship between systolic pressure and mortality in asymptomatic persons who were seeking to obtain life insurance policies. However, diastolic pressure was subsequently preferred, probably because most persons assessed were in their teens or early 20s and systolic pressure could be variable and misleading (Figure 2). Strong views still pertain to the prognostic value of systolic, diastolic, and mean pressure, with a notable large meta-analysis championing the superiority of diastolic pressure at any age and disparaging the value of pulse pressure. That study, although comprising almost 1 million subjects, runs against state-of-the-art studies such as the Framingham Heart Study and has been criticized on many accounts, including the age of subjects and the therapy offered. Such group analysis is unsuitable for application to patients consulting a physician in the 21st century.

Before Fischer’s presentation in 1917, life insurance examiners without cuff sphygmomanometers paid attention to the radial pressure wave and recognized premature arterial senility as prominence of the late systolic part of
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TABLE 1. Summary of Published Studies With Small Numbers of Persons (40-1337), Showing Relationship Between Central (Aortic or Carotid) Pressure and Cardiovascular Disease

<table>
<thead>
<tr>
<th>Measure of aortic/carotid pressure</th>
<th>Reference</th>
<th>Country</th>
<th>Measure of outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pulse pressure</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Invasive</td>
<td>Lu et al⁶⁰</td>
<td>China</td>
<td>Restenosis after coronary angioplasty</td>
</tr>
<tr>
<td>Nishijima et al⁶¹</td>
<td>Japan</td>
<td>Severity of coronary disease</td>
<td></td>
</tr>
<tr>
<td>Chemla et al⁶²</td>
<td>France</td>
<td>Relationship to concentric left ventricular hypertrophy</td>
<td></td>
</tr>
<tr>
<td>Philippe et al⁶³</td>
<td>France</td>
<td>Extent of coronary disease</td>
<td></td>
</tr>
<tr>
<td>Nakayama et al⁶⁴</td>
<td>Japan</td>
<td>Restenosis after coronary angioplasty</td>
<td></td>
</tr>
<tr>
<td>Danchin et al⁶⁵</td>
<td>France</td>
<td>Presence and extent of coronary disease</td>
<td></td>
</tr>
<tr>
<td>Jankowski et al⁶⁶</td>
<td>Poland</td>
<td>Extent of coronary disease</td>
<td></td>
</tr>
<tr>
<td>Noninvasive</td>
<td>Waddell et al⁶⁷</td>
<td>Australia</td>
<td>All-cause and cardiovascular mortality</td>
</tr>
<tr>
<td>Safar et al⁶⁸</td>
<td>France</td>
<td>Carotid intima-media thickness</td>
<td></td>
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<tr>
<td>Boutouyrie et al⁶⁹</td>
<td>France</td>
<td>Regression of carotid intima-media thickness with change in carotid pulse pressure</td>
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<tr>
<td>Jondeau et al⁷⁰</td>
<td>France</td>
<td>Primary coronary events</td>
<td></td>
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<tr>
<td>Roman et al⁷¹</td>
<td>United States</td>
<td>Aortic dilation in Marfan syndrome</td>
<td></td>
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<tr>
<td>Augmentation index</td>
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<td></td>
<td></td>
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<tr>
<td>Invasive</td>
<td>Hayashi et al⁷²</td>
<td>Japan</td>
<td>Severity of coronary disease</td>
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<tr>
<td>Ueda et al⁷³</td>
<td>Japan</td>
<td>Restenosis after coronary angioplasty</td>
<td></td>
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<tr>
<td>Chirinos et al⁷⁴</td>
<td>United States</td>
<td>Major adverse cardiovascular events</td>
<td></td>
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<tr>
<td>Noninvasive</td>
<td>London et al⁷⁵</td>
<td>France</td>
<td>All-cause and cardiovascular mortality</td>
</tr>
<tr>
<td>Saba et al⁷⁶</td>
<td>Italy</td>
<td>Left ventricular hypertrophy, carotid remodeling</td>
<td></td>
</tr>
<tr>
<td>Weber et al⁷⁷</td>
<td>Austria</td>
<td>Extent and severity of coronary disease</td>
<td></td>
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<tr>
<td>Nurnberger et al⁷⁸</td>
<td>Germany</td>
<td>Cardiovascular risk</td>
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<tr>
<td>Weber et al⁷⁹</td>
<td>Austria</td>
<td>Severe cardiovascular events</td>
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<tr>
<td>Weber et al⁸⁰</td>
<td>Austria</td>
<td>Diastolic dysfunction</td>
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<tr>
<td>Fractional systolic/diastolic</td>
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<tr>
<td>pressure</td>
<td>Jankowski et al⁸¹</td>
<td>Poland</td>
<td>Extent of coronary disease</td>
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<tr>
<td>Nakayama et al⁸²</td>
<td>Japan</td>
<td>Risk of coronary disease</td>
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<tr>
<td>Nishijima et al⁸³</td>
<td>Japan</td>
<td>Risk of coronary disease</td>
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<tr>
<td>Fractional pulse pressure,</td>
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<tr>
<td>pulsatility index</td>
<td>Jankowski et al⁸¹</td>
<td>Poland</td>
<td>Extent of coronary disease</td>
</tr>
<tr>
<td>Systolic pressure</td>
<td>de Luca et al⁸²</td>
<td>Europe/Australia</td>
<td>Regression of left ventricular hypertrophy</td>
</tr>
<tr>
<td>Ejection time</td>
<td>Weber et al⁸³</td>
<td>Austria</td>
<td>Diastolic dysfunction</td>
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</table>

The radial pulse.³ Osler’s textbook referred to the systolic part of the radial arterial pulse as “enduring.”⁵⁷ The phenomenon could be recognized from the radial sphygmographic tracings of that time and indeed had initially been described by Marey⁵⁸ in 1863 and by Mahomed in 1872.⁵⁹ Modern pulse waveform analysis allows such information to be added to the physician’s assessment through generation of central aortic waveforms and consideration of central aortic systolic and pulse pressure through the noninvasive techniques previously described.³

Because the aforementioned noninvasive techniques are relatively new, fewer data are available compared to data on brachial cuff pressures. However, central pressure data are actually less contentious than data obtained with the brachial cuff and uniformly show a strong relationship between cardiovascular events and the amplitude and contour of the central or aortic pressure wave. Such a relationship is independent of peripheral brachial cuff pressures and other conventionally recorded indices. The data,⁶⁰-⁸⁵ up to late 2005, are summarized in Table 1 and are complemented by findings in a large prospective study, Strong Heart Study, of American Indians.⁷³ The noninvasive studies of central pressure have prompted comparisons of invasive central aortic pressure recorded at cardiac catheterization in patients with disease and events. These studies have shown an incremental benefit of central pressure over conventional peripheral brachial cuff pressures³⁹ (Table 1). Such data have been confirmed by noninvasive studies of the carotid pressure pulse that show that carotid pressure is a better predictor of coronary disease than brachial pressure⁶⁷ (Table 1). These outcome studies are now complemented by drug studies that show how improvement in outcome can be explained by reduction of central but not peripheral brachial blood pressure.

**Drug Studies**

It is well known that nitroglycerin is a powerful drug for relief of angina pectoris and for dyspnea in acute heart failure. Since brachial systolic pressure and calculated peripheral resistance are usually maintained, effects of this
Drug on the arterial system are usually discounted, and benefits are attributed to venodilation and/or to coronary artery dilation alone. Such views are highlighted in most recent textbooks and journal articles. However, nitroglycerin has been shown to have a powerful effect on the reduction in wave reflection from the lower body when measured directly (Figures 4 and 8) or indirectly and in doses (0.1 mg/h) that have little effect on venous tone (Figure 9). Such an effect reduces central aortic and left ventricular systolic pressure while having no effect or a smaller effect on brachial or radial systolic pressure (Figure 4). The anomalous effect on central and peripheral upper limb pressure is readily explained on the position of the reflected wave at the 2 sites. In mature adults, the

![Central Arterial Pressure and Arterial Pressure Pulse](image_url)
reflected wave constitutes the peak of aortic and left ventricular pressure, so that reduction in reflection decreases central pressure substantially. However, in the upper limb, the reflected wave constitutes the late systolic shoulder, so that its reduction has no effect on the measured pressure at peak (Figure 4).

In many recent drug studies, notably the Heart Outcomes Prevention Evaluation (HOPE) study and Losartan Intervention for Endpoint Reduction in Hypertension (LIFE) trial, improvements in outcome with ramipril (HOPE) and losartan (LIFE) occurred with what appears to be a trivial reduction in brachial pressure compared to that with standard therapy. Both studies claimed that benefits of the drugs were “pleiotropic” or “beyond blood pressure lowering.” Such views were challenged on the basis of explanations given previously. An acute study of ramipril compared to atenolol showed a 5 mm Hg lower reduction in aortic vs brachial pressure and queried whether benefits found in the HOPE study were “beyond the brachial artery” within the central aorta rather than “beyond blood pressure.”

This issue was conceded by LIFE investigators, who urged that central pressure be measured in future anti-hypertensive trials.

Søgaard et al have shown the general relationship between blood pressure decrease and reduction of events in a series of clinical trials. However these relationships are not linear, with considerable variability when different trials are compared. At least for stroke, however, the relationship is almost linear, and variability is markedly reduced, when an estimation is made for greater reduction in central aortic systolic pressure by 3 mm Hg with drugs such as angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, and calcium channel blockers, which reduce wave reflection similar to that with nitrates.

The value of central over peripheral pressure was confirmed in the Conduit Artery Function Evaluation (CAFÉ) substudy of the large Anglo-Scandinavian Cardiac Outcomes Trial (ASCOT). ASCOT showed long-term superiority of new (amlodipine with or without perindopril) compared to old (atenolol with or without diuretic) therapy with respect to cardiovascular events. The modest reduction in brachial systolic pressure of less than 1 mm Hg with “new” therapy was 4.3 mm Hg more when estimated for the central ascending aorta and similar to that found by Hirata et al in their acute study. CAFE extends results of the pReTerax in regression of Arterial Stiffness in a controlled double-blind study (REASON), which showed similar benefit of a perindopril/diuretic combination compared to atenolol during a 12-month period. REASON showed a much greater decrease in calculated central (aortic and carotid) systolic pressure than in brachial pressure. A greater reduction in left ventricular mass with the combination therapy correlated with the difference in central but not brachial systolic pressure in that study.

CONCLUSION

Views on the prognostic value of arterial pressure have evolved during the past 2 centuries. The description of high tension of the pulse at the wrist by Richard Bright led to identification of a characteristic radial pulse waveform in sphygmograms. This was used clinically by physicians and life insurance examiners during the next century. The introduction of the brachial cuff for measurement of systolic and diastolic pressure drew attention to the value of systolic, then diastolic pressure, then back again to systolic pressure at the end of the 20th century. The most recent studies combine brachial cuff values and radial pulse contour to noninvasively generate the central aortic pressure waveform. This new technological advance has the potential to be more useful than the peripheral blood pressure techniques. Noninvasive measurement of central arterial pressure can be used for individual patients and is simple enough to be suitable for office practice.

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