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Brachial Artery Differential Characteristic Impedance: Contributions from Changes in Young's Modulus and Diameter

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Abstract This examination of brachial artery (BA) differential characteristic impedance, ΔZ_c, illustrates that changes in Z_c can occur from changes in either BA wall stiffness (Young's modulus, E) and/or its diameter, D. Furthermore, we assessed how changes in both E and D combine in either an isolated, synergistic, or antagonistic manner to yield the net change in BA Z_c. The basis of this analysis is a partial differential equation which approximates ΔZ_c as a total differential. The effects on BA ΔZ_c of acetylcholine, atenolol, fenoldapine, nitroglycerin, hydrochlorothiazide and other medications are examined using data from previously published studies. Clinical situations which alter BA Z_c, such as congestive heart failure, hypertension, and hyperemia, are also analyzed. Results illustrate the usefulness of the present approach in differentiating how medications, hyperemia, and pathological conditions affect BA ΔZ_c by causing independent changes to E and/or D.

Keywords Brachial artery characteristic impedance · Elastic modulus · Vessel diameter · Modeling · Drug intervention

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Introduction

In the absence of wave reflections, characteristic impedance, Z_c, represents the ratio of change in pressure to change in flow in an artery (Milnor 1989; Li 1986, 2000):

$$Z_c = \frac{dP}{dQ}. \quad (1)$$

Therefore, characteristic impedance can be defined as the *slope* of the pressure-flow relationship. Thus, a vessel which displays a greater change in pressure, for a given change in flow, will have a greater characteristic impedance. This is illustrated in Fig. 1.

The brachial artery (BA) is typically where non-invasive blood pressure measurements are acquired using either auscultated Korotokoff sounds or with automated oscillometric techniques (Li 2000). BA, pulse wave velocity (V_{pw}), and diameter (D) have been examined under numerous clinical situations. These include changes in V_{pw} and D as a function of medications such as: acetylcholine in normal patients (Naka et al. 2006), atenolol in hypertensive (HTN) patients (Armentano et al. 2001), ramipril in HTN patients (Armentano et al. 2001), felodipine in HTN patients (Asmar et al. 1993), nitroglycerin (NTG) in normal patients (Bank et al. 1999), NTG in patients with congestive heart failure (CHF) (Naka et al. 2006), hydrochlorothiazide (HCTZ) in HTN patients (Asmar et al. 1993), and NG-monomethyl L-arginine (L-NMMA) in normal patients (Naka et al. 2006).

BA, V_{pw}, and D have also been studied under pathological states such as congestive heart failure (Arnold et al. 1991) and HTN (Armentano et al. 1991; Simon et al. 1985). The effect of hyperemia, following a tourniquet release, has also been examined in both normal patients and patients with CHF (Naka et al. 2006).

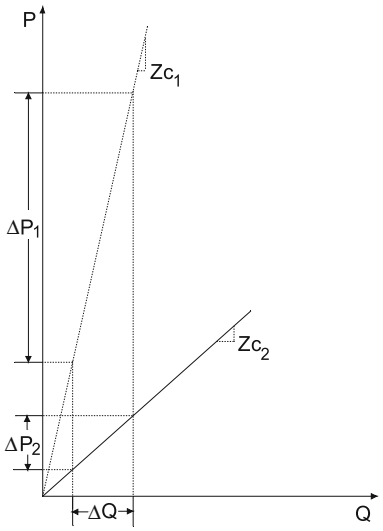

Fig. 1 Characteristic impedance, Z_c, represents the slope of the pressure (P) versus flow (Q) relationship. Note that Z_{c_1} is associated with a greater change in pressure, for a given change in flow, ΔQ, as compared to Z_{c_2}. This results in ΔP_1 being greater than ΔP_2

Since BA Z_c has contributions from both its elastic and geometric properties, we attempted to quantify these contributions by establishing a mathematical approach for their separation.

Methods I

Differing from vascular impedance, systemic vascular resistance (SVR) is defined as:

$$\mathrm{SVR} = \frac{(\mathrm{MAP} - \mathrm{CVP})}{\mathrm{CO}}. \tag{2}$$

where MAP refers to mean arterial blood pressure, CVP refers to central venous blood pressure, and CO is cardiac output. SVR is frequently used with invasive pulmonary arterial hemodynamic monitoring. Whereas total systemic vascular resistance (TSVR) is defined as:

$$\mathrm{TSVR} = \frac{\mathrm{MAP}}{\mathrm{CO}}. \tag{3}$$

TSVR is an approximation of SVR and is frequently used with minimally-invasive hemodynamic monitors such as the esophageal Doppler monitor (Atlas 2008).

Pulse wave velocity, V_{pw}, represents the "wave speed" or "propagating wave velocity" for the arterial pulse in a blood vessel. Z_c can also be determined using V_{pw} (Li 2000):

$$Z_c = \frac{\rho V_{pw}}{A}. \tag{4}$$

where ρ is blood density and A represents blood vessel cross sectional area. Equation 4 is derived using dP/dt from the water-hammer effect:

$$\frac{dP}{dt} = \rho V_{pw} \frac{dv}{dt} \tag{5}$$

and dQ/dt using bulk flow:

$$\frac{dQ}{dt} = A \frac{dv}{dt}. \tag{6}$$

Note that dv/dt, where v is blood flow velocity, represents the acceleration of blood flow. Dividing (5) by (6) yields Eq. 1:

$$Z_c = \frac{dP}{dt}\frac{dt}{dQ}. \tag{7}$$

The Moens–Korteweg (Milnor 1989; Li 2000) equation relates the physical characteristics of both blood and blood vessels to pulse wave velocity:

$$V_{pw} = \sqrt{\frac{Eh}{\rho D}}. \tag{8}$$

where D is vessel diameter, E is Young's modulus, and h is vessel wall thickness. Recall that E is defined as the ratio of stress to strain:

$$\text{stress} = \sigma = \frac{R\Delta P}{h} \tag{9}$$

and

$$\text{strain} = \epsilon = \frac{\Delta R}{R}. \tag{10}$$

Therefore:

$$E = \frac{\sigma}{\epsilon} = \frac{\Delta P R^2}{h \Delta R}. \tag{11}$$

It is assumed that the BA has a circular cross sectional area and that pressure is distributed equally in a circumferential manner.

Brachial Artery Differential Characteristic Impedance

Differential characteristic impedance, ΔZ_c, represents a *change* in slope of the pressure flow relationship:

$$\Delta Z_c = Z_{c_2} - Z_{c_1}. \tag{12}$$

Equation 12 can be expressed as (See "Appendix"):

$$\Delta Z_c = \frac{4\rho}{\pi}\left(\frac{V_{pw_2}}{D_2^2} - \frac{V_{pw_1}}{D_1^2}\right). \tag{13}$$

Where Z_{c_2} and Z_{c_1} represent final and initial characteristic impedances, respectively. Figure 1 illustrates this. These changes, in Z_c, can occur from disease states, medications, or a physiologic response such as hyperemia from the release of a tourniquet.

ΔZ_c from either Eqs.12 or 13 can be approximated using a partial differential equation (PDE) which represents the total differential (See "Appendix"):

$$\widetilde{\Delta Z_c} = \frac{\partial Z_c}{\partial E}\Delta E - \frac{\partial Z_c}{\partial D}\Delta D. \tag{14}$$

Thus:

$$\widetilde{\Delta Z_c} \approx \Delta Z_c. \tag{15}$$

Equation 14 demonstrates that independent contributions, to changes in Z_c, can occur from changes in either Young's modulus and/or vessel diameter. Where:

$$\Delta E = E_2 - E_1 \text{ and } \Delta D = D_2 - D_1. \tag{16}$$

The change in Z_c, as illustrated in Eq. 14, can be represented from contributions in either the elastic and/or diameter components, which are now separable:

$$\text{Elastic component} = \frac{\partial Z_c}{\partial E}\Delta E \tag{17}$$

$$\text{Diameter component} = -\frac{\partial Z_c}{\partial D}\Delta D. \tag{18}$$

Thus:

$$\widetilde{\Delta Z_c} = \text{Elastic component} + \text{Diameter component}. \tag{19}$$

This approximate total differential is assumed to be applicable in the range of normal and near-normal physiologic and pathologic conditions investigated. Furthermore, BA vessel wall thickness and blood density are assumed constant (See "Methods II").

As will be subsequently demonstrated, these changes, occurring in either the elastic and/or the diameter components, can behave in an isolated, synergistic, or antagonistic manner with respect to the *overall* change in BA Z_c. Thus, in the BA, Young's modulus and diameter, can change independently with the *net* effect being a change in BA Z_c.

Furthermore, the absolute percentage contribution, to the total change in BA Z_c, can be determined for both the elastic and the diameter components:

$$\text{Absolute percent change in } \Delta Z_c \text{ from the elastic component} = \left|\frac{\text{Elastic component}}{\Delta Z_c}\right| \times 100. \tag{20}$$

$$\text{Absolute percent change in } \Delta Z_c \text{ from the diameter component} = \left|\frac{\text{Diameter component}}{\Delta Z_c}\right| \times 100. \tag{21}$$

These absolute percentage changes are then assigned either a positive or negative value depending on the overall net change in BA Z_c and whether they combined in a synergistic or antagonist manner.

Tables 1 and 2 illustrate how synergistic or antagonist changes in Young's modulus and diameter can potentially contribute to changes in Z_c.

Methods II

Using ultrasound and non-invasive pressure measurements, numerous studies have examined simultaneous changes in BA V_{pw} and D during various clinical conditions. These are summarized in Table 3. The reader can refer to each study for detailed information regarding the specific methodology used and associated statistical information.

Utilizing the Moens–Korteweg Eq. 8, Young's modulus, E, can be subsequently estimated from Table 3:

$$\frac{(V_{pw_1})^2 \rho D_1}{h} = E_1 \text{ and } \frac{(V_{pw_2})^2 \rho D_2}{h} = E_2. \tag{22}$$

Note that subscripts 1 and 2 refer to the different clinical situations, or conditions, which are being compared. Table 4 demonstrates the values for Young's modulus obtained from each clinical study.

It is assumed that vessel wall thickness, h, is equal to a constant which is a fraction of the initial diameter of the BA:

$$h = 0.11 \times D_1. \tag{23}$$

Furthermore, blood density, ρ, is also assumed constant and is equal to 1.06 g/cm^3.

The partial derivatives: $\partial Z_c/\partial E$ and $\partial Z_c/\partial D$ are then approximated (See "Appendix"):

Table 1 When brachial artery vessel wall stiffness (Young's modulus), E, increases, with a simultaneous decrease in vessel diameter, D, these effects act synergistically to increase characteristic impedance

Net change in Z_c	Change in E	Change in D	Effect
$\uparrow Z_c$	$\uparrow E$	$\downarrow D$	Synergistic
$\downarrow Z_c$	$\downarrow E$	$\uparrow D$	Synergistic

Whereas a decrease in Young's modulus, with a simultaneous increase in vessel diameter, leads to a synergistic decrease in characteristic impedance

Table 2 Changes in brachial artery Young's modulus (E) and diameter (D) can behave in an antagonistic manner with respect to the overall change in characteristic impedance

Net change in Z_c	Change in E	Change in D	Effect
$\uparrow Z_c$	$\downarrow E$	$\downarrow\downarrow D$	Antagonistic
$\uparrow Z_c$	$\uparrow\uparrow E$	$\uparrow$D	Antagonistic
$\downarrow Z_c$	$\downarrow\downarrow E$	$\downarrow D$	Antagonistic
$\downarrow Z_c$	$\uparrow E$	$\uparrow\uparrow D$	Antagonistic

Table 3 Brachial artery (BA) pulse wave velocity, V_{pw}, and diameter, D, have been simultaneously examined in various clinical situations

Medication or condition	Initial data						Reference
	V_{pw1}	V_{pw2}	ΔV_{pw}	D_1	D_2	ΔD	
Ach in normals	9.4	8.5	−0.9	4.2	4.5	0.4	Naka et al. (2006)
L-NMMA in normals	9.4	10.9	1.5	4.2	4.1	−0.1	Naka et al. (2006)
Atenolol in HTN	11.7	10.4	−1.3	4.2	4.1	−0.1	Armentano et al. (2001)
Ramipril in HTN	11.6	9.5	−2.1	4.1	4.1	0	Armentano et al. (2001)
Felodipine in HTN	12.0	10.0	−2.0	4.37	4.49	0.12	Asmar et al. (1993)
HCTZ in HTN	12.0	11.0	−1.0	4.37	4.31	−0.06	Asmar et al. (1993)
NTG in normals	7.1	6.532	−0.568	4.2	4.494	0.294	Bank et al. (1999)
NTG in CHF	7.0	6.3	−0.7	4.07	4.36	0.29	Naka et al. (2006)
Hyperemia in normals	7.1	6.2	−0.9	4.2	4.536	0.336	Naka et al. (2006)
Hyperemia in CHF	6.7	6.4	−0.3	4.1	4.13	0.03	Naka et al. (2006)
CHF vs. normals	9.2	10.6	1.4	4.53	4.07	−0.46	Arnold et al. (1991)
HTN vs. normals	8.8	11.6	2.8	4.63	5.03	0.4	Armentano et al. (1991)
HTN vs. normals	8.8	11.5	2.7	4.63	5.0	0.37	Simon et al. (1985)

V_{pw} and D are expressed with dimensions of m/s and mm, respectively

Table 4 Determination of Young's modulus, E, based on the information from Table 3 and using Eqs. 22 and 23

Medication or condition	E_1	E_2	ΔE
Ach in normals	1.703×10^6	1.492×10^6	-2.11×10^5
L-NMMA in normals	1.703×10^6	2.24×10^6	5.32×10^5
Atenolol in HTN	2.64×10^6	2.03×10^6	-6.03×10^5
Ramipril in HTN	2.59×10^6	1.74×10^6	-8.54×10^5
Felodipine in HTN	2.78×10^6	1.98×10^6	-7.95×10^5
HCTZ in HTN	2.78×10^6	2.30×10^6	-4.75×10^5
NTG in normals	9.72×10^5	8.80×10^5	-9.17×10^4
NTG in CHF	9.44×10^5	8.19×10^5	-1.25×10^5
Hyperemia in normals	9.72×10^5	8.00×10^5	-1.71×10^5
Hyperemia in CHF	8.65×10^5	7.95×10^5	-6.99×10^4
CHF vs. normals	1.63×10^6	1.95×10^6	3.14×10^5
HTN vs. normals	1.49×10^6	2.82×10^6	1.32×10^6
HTN vs. normals	1.49×10^6	2.75×10^6	1.26×10^6

The dimensions for Young's modulus are expressed in Pascals or N/m^2

$$\frac{\partial Z_c}{\partial E} \approx \frac{8\rho h}{\bar{Z}_c \pi^2 \bar{D}^5} \tag{24}$$

and

$$\frac{\partial Z_c}{\partial D} \approx \frac{40\rho \bar{E} h}{\bar{Z}_c \pi^2 \bar{D}^6}. \tag{25}$$

Note the following terms refer to mean values which aid in the determination of PDEs (24) and (25):

$$\bar{E} = (E_1 + E_2)/2 \text{ and } \bar{D} = (D_1 + D_2)/2 \text{ and } \bar{Z}_c = (Z_{c_1} + Z_{c_2})/2. \tag{26}$$

Values for $\widetilde{\Delta Z_c}$ are then determined using Eq. 14. Furthermore, an error term can be defined as:

$$\% \text{ error} = \frac{|\widetilde{\Delta Z_c} - \Delta Z_c|}{|\Delta Z_c|} \times 100. \tag{27}$$

It should be noted that data from CHF versus normals and HTN versus normals were based upon analyses of unpaired patients whereas all other comparisons were based upon paired data. Thus, with paired data, each patient functioned as his or her own control.

Results

Table 5 shows how the PDE model (14) is able to accurately assess how each change in BA Z_c is composed of changes from its elastic and/or diameter components. Furthermore, these changes can be evaluated as a percentage of the *total* change in Z_c.

Acetylcholine, in normal patients, yielded a net decrease in BA Z_c. Specifically, this change was due to a 27.4% reduction in ΔZ_c, from the elastic component of ΔZ_c, and a 71.5% reduction from the diameter component of ΔZ_c. In addition, these changes combined in a *synergistic* manner.

Synergistic reductions in BA Z_c were also noted following the administration of felodipine and NTG to hypertensive patients. It should be noted that this effect of NTG occurred in both normal patients as well as those with CHF. Hyperemia, following the release of a tourniquet, also yielded a synergistic decrease in both the elastic and diameter components of ΔZ_c. Interestingly, normal patients had a much greater decrease, in their BA Z_c, resulting from hyperemia, than those patients with CHF.

A synergistic increase in BA Z_c was noted following the administration of NG-monomethyl L-arginine (L-NMMA),

Table 5 Characteristic impedance, Z_c, and change in characteristic impedance, ΔZ_c, are calculated from preexisting data

Medication or condition	Z_{c1}	Z_{c2}	ΔZ_c	Elastic component (**% change in ΔZ_c**)	Diameter component (**% change in ΔZ_c**)	Effect: isolated, synergistic or antagonist
Ach in normals	7.19×10^8	5.67×10^8	-1.53×10^8	-4.18×10^7 (**−27.4**)	-1.092×10^8 (**−71.5**)	Synergistic
L-NMMA in normals	7.19×10^8	8.75×10^8	1.56×10^8	1.08×10^8 (**69**)	4.8×10^7 (**31**)	Synergistic
Atenolol in HTN	8.95×10^8	8.35×10^8	-6.02×10^7	-1.12×10^8 (**−186**)	5.25×10^7 (**86**)	Antagonistic
Ramipril in HTN	9.31×10^8	8.95×10^8	-1.69×10^8	-1.69×10^8 (**−100**)	(**0**)	Isolated
Felodipine in HTN	8.48×10^8	6.69×10^8	-1.79×10^8	-1.27×10^8 (**−71**)	-5.14×10^7 (**−29**)	Synergistic
HCTZ in HTN	8.48×10^8	7.99×10^8	-4.89×10^7	-7.74×10^7 (**−158**)	2.86×10^7 (**58**)	Antagonistic
NTG in normals	5.43×10^8	4.37×10^8	-1.07×10^8	-2.39×10^7 (**−22.4**)	-8.17×10^7 (**−76.6**)	Synergistic
NTG in CHF	5.70×10^8	4.47×10^8	-1.23×10^8	-3.55×10^7 (**−28.9**)	-8.62×10^7 (**−70.1**)	Synergistic
Hyperemia in normals	5.43×10^8	4.07×10^8	-1.37×10^8	-4.51×10^7 (**−33.0**)	-8.95×10^7 (**−65.6**)	Synergistic
Hyperemia in CHF	5.38×10^8	5.06×10^8	-3.15×10^7	-2.20×10^7 (**−69.0**)	-9.52×10^6 (**−30.2**)	Synergistic
CHF vs. normals	6.05×10^8	8.64×10^8	2.59×10^8	6.23×10^7 (**24.1**)	1.90×10^8 (**73.4**)	Synergistic
HTN vs. normals	5.54×10^8	6.19×10^8	6.48×10^7	1.88×10^8 (**290.4**)	-1.27×10^8 (**−195.6**)	Antagonistic
HTN vs. normals	5.54×10^8	6.21×10^8	6.68×10^7	1.81×10^8 (**271.5**)	-1.17×10^8 (**−175.7**)	Antagonistic

Use of the PDE model allows for the determination of the amount of contribution, to ΔZ_c, from changes in Young's modulus and/or diameter. These physical changes may combine in an isolated, synergistic, or antagonist manner with respect to the overall change in characteristic impedance. Z_c is expressed with dimensions of N s/m^5. Bold values refer to the percentage change in ΔZ_c

to normal patients. In addition, a synergistic increase in BA Z_c was also observed when comparing patients with CHF to normal patients. Thus, CHF and L-NMMA appear to increase Young's modulus, E, within the BA. Both CHF and L-NMMA also decrease BA diameter, D.

Atenolol, when administered to hypertensive patients, was noted to have an *antagonist* effect which resulted in a decrease in BA E as well as a slight decrease in diameter. The net effect resulted in an overall decrease in BA Z_c. HCTZ, when administered to hypertensive patients, also produced a similar antagonistic change in BA Z_c with an overall reduction in characteristic impedance. When comparing those patients with hypertension (HTN) to normals, an antagonist response was noted in that BA diameter was significantly dilated and E was increased.

Ramipril, when administered to hypertensive patients, demonstrated an *isolated* effect of only decreasing E within the BA and had no effect on D.

Discussion

Clinicians typically refer to vasoactive medications and conditions as simply being vasoconstricting or vasodilating. The present study has demonstrated that there are two distinctive physical properties, namely Young's modulus of elasticity (E) and blood vessel diameter (D) that can change independently within the brachial artery. Furthermore, these changes can occur in a synergistic, antagonist, or isolated manner and yield a net change in brachial artery characteristic impedance (BA ΔZ_c).

Specifically, synergistic changes, in both E and D, which resulted in an overall decrease in BA Z_c, occurred following the administration of acetylcholine to normal patients. Felodipine, and nitroglycerin, when given to hypertensive patients, also resulted in synergistic changes. Hyperemia, following the release of a tourniquet, yielded a similar synergistic response with respect to changes in E and D. These synergistic changes, in both of these physical parameters, may contribute to the observed acute decrease in blood pressure, from the decrease in BA Z_c, following the administration of these medications or from tourniquet release.

The decrease in BA Z_c, occurring from the administration of NTG, appeared slightly greater in patients with CHF than normal patients. Specifically, the greater change in BA Z_c was due to both a greater decrease in Young's modulus and an increase in diameter.

This effect of NTG on BA diameter and distensibility has been additionally examined in both CHF patients and controls although characteristic impedance was not calculated (Nakamura et al. 2004).

Hyperemia, after the release of a tourniquet, yielded a strikingly greater reduction in BA Z_c in normal patients as compared to those with CHF. Following this maneuver, normal patients also had a greater percentage reduction in the diameter component of their BA Z_c. Whereas CHF patients had a greater percentage reduction in their elastic

component. The utility of statins in treating endothelial dysfunction in CHF, as assessed by hyperemia, has been investigated (Tousoulis et al. 2005).

The administration of L-NMMA (NG-monomethyl L-arginine), as well as the presence of CHF, appears to be associated with synergistic changes in both E and D which lead to an overall increase in BA Z_c. It should be noted that L-NMMA inhibits nitric oxide synthetase (Schilling et al. 1993). Furthermore, nitric oxide-mediated vasodilation, within the forearm, is impaired in patients with CHF (Katz et al. 1994). In addition, those patients with CHF have a significantly diminished, or absent, response to L-NMMA when this medication is infused within the brachial artery (Katz et al. 1996).

Antagonistic responses were noted following the administration of HCTZ and atenolol to hypertensive patients. In both instances, a small reduction in the diameter of the BA occurred with a reduction in Young's modulus, E. The net effect, in each case, was a reduction in BA Z_c.

The presence of hypertension (HTN), as compared to normal patients, was associated with increases in BA Z_c. Furthermore, this antagonistic response was due to an increase in the elastic component in spite of a decrease from the diameter component of BA Z_c.

Ramipril, an angiotensin converting enzyme inhibitor, was the only medication or condition associated with a change in BA Z_c which was due to an isolated effect. Specifically, ramipril resulted in a decrease in E with no effect on diameter. This was noted in hypertensive patients.

Potential sources of error, for this retrospective analysis of existing data, include the assumptions that blood density and BA wall thickness are both constants. This may be particularly significant when examining the unpaired data associated with CHF versus normal patients and HTN versus normal patients. Another source of error is the use of the mean values for $\bar{E}$, $\bar{D}$, and $\bar{Z}_c$ from Eq. 26 when computing the approximate PDEs from Eqs. 24 and 25. Lastly, measurement errors, although small, could also contribute to inaccuracies. In spite of the above assumptions, Table 6 demonstrates that the approximate total differential $\widetilde{\Delta Z_c}$ and ΔZ_c yield numerically similar results.

In deriving contributing factors to brachial artery characteristic impedance, the water-hammer formula was used. In general, this formulation is accurate in large vessels. For smaller arteries, the effect of the wave reflections may become significant. Wave reflections can increase blood pressure and may alter both geometry and elastic behavior of blood vessels (Li 2004). This aspect needs to be investigated in future studies. The present study provides a potentially clinically useful methodology to quantify contributing factors to blood vessel properties as they are modified by different pathological conditions, hyperemia, and following drug treatment.

Table 6 A comparison between the measured change in characteristic impedance, ΔZ_c, and the approximate change in characteristic impedance, $\widetilde{\Delta Z_c}$, which is derived using the PDE model from Eq. 14. Note that % error is determined using Eq. 27

Medication or condition	ΔZ_c	$\widetilde{\Delta Z_c}$	% error
Ach in normals	-1.53×10^8	-1.51×10^8	1.09
L-NMMA in normals	1.559×10^8	1.556×10^8	0.18
Atenolol in HTN	-6.02×10^7	-5.99×10^7	0.32
Ramipril in HTN	-1.686×10^8	-1.686×10^8	0
Felodipine in HTN	-1.79×10^8	-1.78×10^8	0.23
HCTZ in HTN	-4.887×10^7	-4.883×10^7	0.09
NTG in normals	-1.07×10^8	-1.06×10^8	1.0
NTG in CHF	-1.23×10^8	-1.22×10^8	1.1
Hyperemia in normals	-1.37×10^8	-1.35×10^8	1.4
Hyperemia in CHF	-3.152×10^7	-3.15×10^7	0.02
CHF vs. normals	2.59×10^8	2.52×10^8	2.53
HTN vs. normals	6.48×10^7	6.14×10^7	5.17
HTN vs. normals	6.68×10^7	6.397×10^7	4.23

Conclusions

The present analysis has shown that changes in brachial artery characteristic impedance, BA ΔZ_c, can be modeled as a function of independent changes in both Young's modulus and diameter. Furthermore, medications and conditions may not act as strict vasodilators or vasoconstrictors. Rather, changes in vessel wall elastance and diameter can independently occur.

Further research and prospective analyses would be beneficial in evaluating the potential utility and limitations of this newly developed analysis of differential physical and geometric contributions to changes in characteristic impedance.

Appendix: Derivation of the Approximate Total Differential for Characteristic Impedance

The definition of characteristic impedance, based upon pulse wave velocity, from Eq. 4 is:

$$Z_c = \frac{\rho V_{pw}}{A}. \tag{28}$$

Substituting diameter (D), for area (A), where $A = \pi D^2/4$ yields:

$$Z_c = \frac{4\rho V_{pw}}{\pi D^2}. \tag{29}$$

Substituting the Moens–Korteweg equation (8), for pulse wave velocity, V_{pw}:

$$Z_c = \sqrt{\frac{16\rho E h}{\pi^2 D^5}}. \tag{30}$$

Squaring both sides of (30) yields:

$$Z_c^2 = \frac{16\rho Eh}{\pi^2 D^5}. \tag{31}$$

Using implicit differentiation:

$$2Z_c dZ_c = \frac{16\rho h}{\pi^2 D^5} dE - \frac{80\rho Eh}{\pi^2 D^6} dD. \tag{32}$$

The total differential is then approximated as:

$$\widetilde{\Delta Z_c} = \frac{\partial Z_c}{\partial E}\Delta E - \frac{\partial Z_c}{\partial D}\Delta D. \tag{33}$$

Where:

$$\frac{\partial Z_c}{\partial E} \approx \frac{8\rho h}{\bar{Z}_c \pi^2 \bar{D}^5} \tag{34}$$

and

$$\frac{\partial Z_c}{\partial D} \approx \frac{40\rho \bar{E} h}{\bar{Z}_c \pi^2 \bar{D}^6}. \tag{35}$$

Note that the following terms are also used:

$$\bar{E} = (E_1 + E_2)/2 \text{ and } \bar{D} = (D_1 + D_2)/2 \text{ and } \bar{Z}_c = (Z_{c_1} + Z_{c_2})/2. \tag{36}$$

References

Armentano R, Simon A, Levenson J, Chau NP, Megnien JL, Pichel R. Mechanical pressure versus intrinsic effects of hypertension on large arteries in humans. Hypertension. 1991;18(5):657–64.

Armentano RL, Graf S, Ramirez AJ, Espinosa JD, Brandani L, Baglivo H, et al. Mechanical vs intrinsic components in the improvement of brachial arterial compliance. Comparison of the effects of atenolol versus ramipril in hypertensive patients. Medicina. 2001;61(5 Pt 1):535–40.

Arnold JM, Marchiori GE, Imrie JR, Burton GL, Pflugfelder PW, Kostuk WJ. Large artery function in patients with chronic heart failure. Studies of brachial artery diameter and hemodynamics. Circulation. 1991;84(6):2418–25.

Asmar RG, Benetos A, Chaouche-Teyara K, Raveau-Landon CM, Safar ME. Comparison of effects of felodipine versus hydrochlorothiazide on arterial diameter and pulse-wave velocity in essential hypertension. Am J Cardiol. 1993;72(11):794–8.

Atlas GM. Development and application of a logistic-based systolic model for hemodynamic measurements using the esophageal Doppler monitor. Cardiovasc Eng. 2008;8(3):159–73.

Bank AJ, Kaiser DR, Rajala S, Cheng A. In vivo human brachial artery elastic mechanics: effects of smooth muscle relaxation. Circulation. 1999;100(1):41–7.

Katz SD, Rao R, Berman JW, Schwarz M, Demopoulos L, Bijou R, et al. Pathophysiological correlates of increased serum tumor necrosis factor in patients with congestive heart failure. Relation to nitric oxide-dependent vasodilation in the forearm circulation. Circulation. 1994;90(1):12–6.

Katz SD, Krum H, Khan T, Knecht M. Exercise-induced vasodilation in forearm circulation of normal subjects and patients with congestive heart failure: role of endothelium-derived nitric oxide. J Am Coll Cardiol. 1996;28(3):585–90.

Li JK-J. Time domain resolution of forward and reflected waves in the aorta. IEEE Trans Biomed Eng. 1986;BME-33:783–5.

Li JK-J. The arterial circulation. Physical principles and clinical applications. Totowa: Humana Press; 2000.

Li JK-J. Dynamics of the vascular system. Singapore: World Scientific; 2004.

Milnor WR. Hemodynamics. 2nd ed. Baltimore: Williams & Wilkens; 1989.

Naka KK, Tweddel AC, Doshi SN, Goodfellow J, Henderson AH. Flow-mediated changes in pulse wave velocity: a new clinical measure of endothelial function. Eur Heart J. 2006;27(3):302–9.

Nakamura M, Sugawara S, Arakawa N, Nagano M, Shizuka T, Shimoda Y, et al. Reduced vascular compliance is associated with impaired endothelium-dependent dilatation in the brachial artery of patients with congestive heart failure. J Card Fail. 2004;10(1):36–42.

Schilling J, Cakmakci M, Battig U, Geroulanos S. A new approach in the treatment of hypotension in human septic shock by NG-monomethyl-L-arginine, an inhibitor of the nitric oxide synthetase. Intensive Care Med. 1993;19(4):227–31.

Simon AC, Levenson J, Bouthier J, Safar ME, Avolio AP. Evidence of early degenerative changes in large arteries in human essential hypertension. Hypertension. 1985;7(5):675–80.

Tousoulis D, Antoniades C, Bosinakou E, Kotsopoulou M, Pitsavos C, Vlachopoulos C, et al. Effects of atorvastatin on reactive hyperemia and inflammatory process in patients with congestive heart failure. Atherosclerosis. 2005;178(2):359–63.