Ventricular-arterial coupling

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Cardiff University
Ventricular–arterial coupling

- Increased loading triggers LV hypertrophy
- Ventricular response is asymmetrical
- Central arterial pressure
- Conduit arterial stiffness
- Ventricular and vascular elastance
- Arterial waves and energy
Central arterial pressure changes with age

- Central pressure augmentation
- Peripheral pressure amplification

McDonald’s Blood Flow in Arteries
Stiff conduit arteries cause increased central aortic pressure, isolated systolic hypertension, and increased arterial pulse pressure.

Wave speed is a powerful prognostic indicator.
Imaging arterial stiffness

**BETA INDEX**
- Pressure-independent Young’s modulus of stiffness
- Adjusted for BP
- No units

\[ \beta = \frac{\log_n \left( \frac{P_s}{P_d} \right)}{\left( \frac{D_{s} - D_{d}}{D_{d}} \right)} \]

**EPSILON**
- Pressure-strain elastic modulus
- Measured in kPa

\[ \Ep = \frac{(P_{s} - P_{d}) \ D_{d}}{\left( D_{s} - D_{d} \right)} \]
Pooled observations in 144 subjects – Age

Conduit arterial stiffness
Central pressure waveforms can be derived from tissue Doppler.

Haluska B et al, Cardiovasc Ultrasound 2007;5:6
Tissue velocity of aortic wall motion
Aortic wall motion in Marfan syndrome
Prospective, double-blind, randomised cross-over

Time to peak systolic velocity of anterior aortic wall motion

Williams A et al
Arterial stiffness is a determinant of $\text{VO}_2 \text{ max}$

$\text{VO}_2 \text{ max} (\text{ml/min})$

$10^{-3} \text{ mmHg}^{-1}$

Hundley, JACC 2001; 38: 796
Arterial compliance & peak exercise capacity are related & both decline with risk factors & severity of the metabolic syndrome

Wong C et al, AJC 2005;96:1686
Myocardial dysfunction in metabolic syndrome correlates with number of diagnostic features

393 subjects with negative stress echocardiography

Wong C et al, Am J Cardiol 2005; 96: 1686-91
Left ventricular pressure-volume loop

Pressure

End-systolic pressure volume relationship ≡ contractility

Isovolumic contraction

Ejection

Isovolumic relaxation

End-diastolic pressure volume relationship

Volume

ESP

DP

ESV

EDV
The traditional concept of ventricular-arterial coupling

**Elastance** = change in pressure for a given change in volume
Elastance

Change in pressure for a unit change in volume (mmHg/ml)

- **Arterial elastance**
  \[ E_A = \frac{ESP}{SV} \] (SV stroke volume)
  Higher elastance = greater sensitivity to volume change, more variable pressure

- **Ventricular elastance**
  \[ E_{LV} = \frac{ESP}{ESV} \] (ESV LV end-systolic volume)
  Net arterial load exerted on the ventricle
Coupling ratio

\[ \text{VAC} = \frac{E_A}{E_{LV}} \text{ with volumes indexed for body surface area} \]

- Greatest efficiency when elastances are matched
- Optimal transfer of blood from LV to aorta
- BP, LV pressure, and CO are maintained in a physiological range
- **Normal ratio** \( \sim 1.0 \pm 0.36 \) \((\text{VAC} = \frac{1}{\text{EF}} - 1)\)
- Normal \( E_A \) 2.2 \( \pm \) 0.8 mmHg/ml
- Normal \( E_{LV} \) 2.3 \( \pm \) 1.0 mmHg/ml

*Chantler, 2008*
M aged 30, triathlete

SV = 49ml
VAC = ESV / SV
= 41/49 = 0.84
Combined Ventricular Systolic and Arterial Stiffening in Patients With Heart Failure and Preserved Ejection Fraction

Implications for Systolic and Diastolic Reserve Limitations

Miho Kawaguchi, MD; Ilan Hay, MD; Barry Fetics, MSE; David A. Kass, MD

Circulation 2003; 107: 714-20
Myocardial contractility in the left ventricle is inversely related to arterial compliance

Kawaguchi M et al, Circulation 2003; 107: 714-20
Forward and Backward Running Waves in the Arteries: Analysis Using the Method of Characteristics

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C. J. H. Jones

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London, SW7 2AZ England

Wave intensity = \( \frac{dP}{dt} \cdot \frac{dU}{dt} \)
There are 4 types of arterial waves

<table>
<thead>
<tr>
<th>Wave Type</th>
<th>Velocity</th>
<th>Pressure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Forward compression wave</td>
<td>↑</td>
<td>↑</td>
</tr>
<tr>
<td>Forward expansion wave</td>
<td>↓</td>
<td>↓</td>
</tr>
<tr>
<td>Backward compression wave</td>
<td>↓</td>
<td>↑</td>
</tr>
<tr>
<td>Backward expansion wave</td>
<td>↑</td>
<td>↓</td>
</tr>
</tbody>
</table>

The integral of wave intensity is energy
Wave intensity and reflection

Control

Abdominal Aorta

Diaphragm

Descending Aorta

Ramsey, Jones & Sugawara
Wall tracking of common carotid artery

Diameter / distension waveform, calibrated as estimate of pressure

Simultaneous measurement of blood velocity at same site

→ Non-invasive wave intensity
Aloka SSD 5500  7.5 MHz linear array transducer

Anterior wall

Posterior wall

Pressure
Velocity
Compression wave
Expansion wave
Reflections
dP/dt·dU/dt
Ventricular-arterial coupling

Conduit arterial stiffness impairs *long-axis* function

<table>
<thead>
<tr>
<th></th>
<th>Beta index</th>
<th>Pressure-strain elastic modulus</th>
<th>Integral of mid-systolic wave reflections</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Longitudinal S</strong></td>
<td>-0.45**</td>
<td>-0.37**</td>
<td>-0.37**</td>
</tr>
<tr>
<td><strong>Longitudinal E</strong></td>
<td>-0.53**</td>
<td>-0.47**</td>
<td>-0.50**</td>
</tr>
<tr>
<td><strong>Radial S</strong></td>
<td>-0.07</td>
<td>-0.02</td>
<td>-0.06</td>
</tr>
<tr>
<td><strong>Radial E</strong></td>
<td>-0.28*</td>
<td>-0.25*</td>
<td>-0.13</td>
</tr>
<tr>
<td><strong>Ejection fraction</strong></td>
<td>-0.21</td>
<td>-0.16</td>
<td>-0.15</td>
</tr>
<tr>
<td><strong>E/Ea</strong></td>
<td>0.48**</td>
<td>0.50**</td>
<td>0.34**</td>
</tr>
</tbody>
</table>

1. Peak velocity of radial shortening of the left ventricle coincides with arrival of reflected waves.

2. Generation of longitudinal shortening persists against reflections.

Page C et al, Int J Cardiol 2009 e pub
The major “determinant” of reflected wave energy is left ventricular systolic function.

\[ r = 0.81, \ p < 0.0001 \]

\(\text{Log}_n\ \text{FCW integral}\)

\(\text{MALE}\)

\(\text{FEMALE}\)

\(n = 106, \ 57\ M, \ \text{aged} \ 46 \pm 12 \ (22-71) \ \text{years}\)

The heart does not operate independently ..

.. it’s part of an integrated cardiovascular symptom

**Why do we have to understand V-A coupling?**

- to understand mechanisms of LV dysfunction
- because conduit arterial stiffness, central arterial pressure, and wave reflections can be important new therapeutic targets