Exercise and Dietary Influences on Arterial Stiffness in Cardiometabolic Disease

Julian W. Sacre, Garry L.R. Jennings, Bronwyn A. Kingwell

Brief Review

The association of aortic and proximal arterial stiffness with cardiovascular and all-cause mortality is independent of conventional risk factors and strongest in the setting of elevated cardiometabolic risk (including hypertensive, metabolic, atherosclerotic, and renal disease). These associations are consistent with the physiological consequences of proximal arterial stiffening. In particular, widening of central (ie, aortic) pulse pressure through elevation in systolic and a reduction in diastolic blood pressure (BP). Higher central systolic BP increases cardiac afterload, whereas lower diastolic BP reduces myocardial perfusion. Elevation in central pressure pulsatility has also been linked to peripheral vascular dysfunction and impaired blood flow to the brain, kidneys, and other organs, which may in turn promote cognitive decline, renal impairment, and other adverse outcomes.

Physical activity and diet habits are well-known determinants of the rate of arterial stiffening in healthy aging. However, it is controversial whether such lifestyle measures represent clinically useful interventions to modulate arterial biomechanical properties beyond their impact on BP and other conventional risk factors, as may be achieved with some pharmacological therapies. This review examines and other conventional risk factors, as may be achieved with some pharmacological therapies. This review examines and other conventional risk factors, as may be achieved with some pharmacological therapies.

Aerobic Exercise

Aerobic exercise has been attractive for reducing arterial stiffness since the demonstration of improved systemic arterial compliance and aortic β-stiffness index after as little as 4 weeks of training in healthy, sedentary young adults. Notably, arterial properties after training were similar to those in endurance athletes in spite of a relatively modest exercise dose (ie, cycling 3×30 minutes per week at 75% of maximum workload). These short-term changes may argue for exercise-mediated arterial adaptations being predominantly functional rather than structural in origin. Multiple mechanisms inducing vascular smooth muscle cell relaxation contribute and include increased arterial wall shear stress and nitric oxide bioactivity, and reductions in vasoconstrictor tone, oxidative stress, and inflammation. Although vascular functional adaptations with exercise are independent of conventional risk factors, concurrent BP lowering may contribute to early destiffening (Figure 1). Longer-term structural adaptations are suggested by exercise training studies in animals, but remain difficult to demonstrate in humans. Studies applying aortic impedance estimation indicate that age-related intrinsic wall stiffening is slower in individuals who habitually train throughout their lifespan, but is irreversible after sedentary aging.
Hypertension

Trials of aerobic exercise in hypertension have produced mixed findings in relation to arterial stiffness and central systolic loading (Table S1 in the online-only Data Supplement). Because BP lowering is common in this setting, it is difficult to distinguish whether arterial destiffening is an incipient contributor to this process or a favorable consequence of a shift in the operating point of the pressure–stiffness curve (Figure 1).

Figure 1. Theoretical mean arterial pressure–stiffness curves demonstrating the biomechanical implications of arterial functional vs structural responses to aerobic exercise training. (1) Functional destiffening: Lowered mean arterial pressure translates to a reduction in stiffness by virtue of a shift in the curve’s operating point. (2) Structural destiffening: A reduction in intrinsic stiffness of the arterial wall manifests in a downward shift of the entire pressure–stiffness curve (dashed line). Reduced stiffness is evident for a given mean arterial pressure. (3) Functional + structural destiffening: Combination of (1) and (2).

Some nonsequential elements of the cycle may also directly affect one another. This figure has been derived from Diourté et al.

Higher-intensity training interventions appeared most successful in eliciting positive changes in arterial properties. This is consistent with superior BP lowering and endothelial function improvement from high-intensity interval training compared with continuous, moderate-intensity training in essential hypertension. Although this contrasts with the low training threshold needed in healthy, young adults, arterial properties nor BP were changed in people with isolated systolic hypertension. Even where exercise training has been successful in lowering BP in other hypertension phenotypes, changes in arterial stiffness were not always observed (Table S1). It is possible that these divergent findings were a consequence of differences in the type or intensity of exercise.

Higher-intensity training interventions appeared most successful in eliciting positive changes in arterial properties. This is consistent with superior BP lowering and endothelial function improvement from high-intensity interval training compared with continuous, moderate-intensity training in essential hypertension. Although this contrasts with the low training threshold needed in healthy, young adults, arterial stiffness may involve a virtuous cycle of cardiac and vascular adaptations (Figure 2).

Coronary Artery Disease and Heart Failure

Arterial stiffening associated with coronary artery disease (CAD) may have greater implications for exercise capacity than in other disease states because it may determine the ischemic threshold independent of stenosis severity and other covariates. However, exercise training studies in CAD have not established whether arterial biomechanical adaptations act as mediators of functional improvement. Furthermore, inferences from studies in heart failure cannot be made because these are largely limited to nonischemic etiologies. In patients with heart failure and reduced ejection fraction (New York Heart Association class II to III, 71% nonischemic), 8 weeks of training (up to 5–7×60 minutes per week; 50% to 60% of maximum heart rate) increased systemic arterial compliance, but aortic PWV and central AIx were unchanged. Aside from these equivocal findings in heart failure and reduced ejection fraction, a key unanswered question relates to the capacity of exercise to modify arterial properties in heart failure with preserved ejection fraction. Arterial and myocardial stiffening are inherent to this phenotype and directly implicated in

There are few controlled studies of aerobic exercise for arterial destiffening in patients with risk factors other than hypertension. In a recent 12-month RCT in patients with moderate chronic kidney disease and ≥1 uncontrolled cardiovascular risk factor, lifestyle modification (including combined aerobic/resistance training) demonstrated no efficacy for reduction of aortic PWV, central AIx, or central BP, despite a 12% net increase in cardiorespiratory fitness. Preliminary findings in end-stage renal disease are mixed. Cycling at a self-selected intensity during dialysis sessions reduced aortic PWV over 3 months without altering BP. However, these positive findings were not replicated in a 6-month RCT of intradialytic cycling versus home-based walking versus usual care.

Coronary Artery Disease and Heart Failure

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exercise intolerance (probably by abnormal arterial–ventricular coupling). Similar to CAD, RCTs of exercise training have identified large gains in cardiorespiratory fitness in this setting, but have not established whether arterial destiffening is a contributing factor.

**Resistance Training**

Stiffened arteries in resistance-trained athletes have triggered concerns about potential harm. However, a recent meta-analysis of RCTs (general population) demonstrated that arterial stiffening is probably restricted to young people performing high-intensity training (ie, resistive loads >70% of the 1 repetition maximum). Moderate-intensity resistance training seems to have a neutral effect on arterial properties in healthy individuals, but given its possible benefits for cardiometabolic risk reduction (including BP lowering), further study is warranted in cardiometabolic disease populations.

**Dietary Interventions**

Previous reviews have highlighted numerous diet-related factors that may modify arterial properties in the general population. However, their relative importance is difficult to define in the face of multiple confounding variables (particularly genetic and other lifestyle factors) and by the limitations of dietary assessment. The current discussion will focus on areas other than calorie restriction/weight loss (which modifies arterial properties largely via BP-related mechanisms) with evidence specifically pertaining to cardiometabolic risk reduction. A wide variety of specific foods and nutrients have been investigated for their potential impact on arterial stiffness. A 2011 systematic review of RCT evidence in the general population identified positive influences of fish oils, polyphenols (mainly soy isoflavones), and fermented milk products. Conversely, sodium and caffeine supplements were found to promote arterial stiffening.

**Sodium Restriction**

Observational data suggest that the age-related increase in arterial stiffness is blunted in normotensive individuals who consume a sodium-restricted diet. However, it is unclear whether sodium restriction in hypertensive individuals destiffens arteries independently of BP lowering. Three months of sodium restriction studied as an active control reduced aortic PWV and central AIx more than by aerobic exercise training in pre-/stage 1 hypertensive patients, although these changes coincided with mean arterial pressure reduction. The other hand, marked improvement in the more BP-independent β-stiffness index occurred alongside systolic BP lowering (<12 mm Hg) in individuals with stage 1 hypertension within only 2 weeks of a sodium-restricted diet (57 versus 135 mmol/day). However, these results in medicated patients may not be generalizable to patients on pharmacotherapy. Indeed, aortic PWV and central AIx only tended lower in a resistant hypertension cohort after 1 week of sodium restriction (50 versus 250 mmol/day), despite substantial reduction in BP (<23/9 mm Hg). Further RCTs, preferably longer term, are clearly needed to resolve the extent to which arterial properties per se respond to sodium restriction.

**Dietary Approaches to Stop Hypertension**

Strong RCT evidence supports BP-lowering efficacy of the Dietary Approaches to Stop Hypertension (DASH) eating pattern, characterized by high fiber, protein, potassium, magnesium, and calcium content, and sometimes combined with sodium restriction. The 4-month Exercise and Nutrition interventions for Cardiovascular Health (ENCORE) trial demonstrated significant improvements in aortic PWV with a DASH diet conferring ≈30% reduction in sodium intake versus a control typical American diet (participants were nonmedicated, overweight/obese individuals with at least prehypertension). However, concurrent lowering of BP (net −7.7/−3.6 mm Hg) may argue for indirect modification of arterial stiffness. Greater BP and PWV lowering was derived from a cointervention arm involving the DASH diet, regular aerobic exercise, and weight loss.

**Specific Foods, Nutrients, and Supplements**

A meta-analysis of 10 RCTs up to September 2010 identified positive effects of omega-3 fatty acids (ie, eicosapentaenoic and docosahexaenoic acids) on PWV and systemic arterial compliance in the general population. Although none of the studies evaluated aortic PWV or central systolic loading markers, it was promising that arterial adaptations appeared unrelated to BP changes. This may reflect positive effects on other cardiovascular risk factors, particularly plasma lipids and regression of atherosclerosis (based on carotid intima-media thickness). Indeed, a trial subsequent to the meta-analysis identified a significant reduction in carotid β-stiffness index in statin-treated CAD patients after 48 weeks of eicosapentaenoic acid supplementation (1.8 g per day). The benefits of fish oil in relation to arterial destiffening may extend to hypertension and the metabolic syndrome/diabetes mellitus despite mixed effects on glucose metabolism (including potential worsening).

**Polyphenols**

An arterial destiffening effect of polyphenols in the general population, independent of BP, is supported by RCTs of isoflavone supplementation in postmenopausal women. However, except for 1 RCT of soy isoflavones in hypertensive patients (which indicated no benefit), studies in cardiometabolic disease groups (mainly atherosclerotic) have largely focused on polyphenol-rich berries, red wine, or cocoa, albeit with varying success. In patients with CAD, arterial stiffness was lowered by cranberry juice supplementation (reduction in aortic PWV over 4 weeks), but not by a chocolate bar/cocoa beverage intervention (no change in systemic arterial compliance over 6 weeks). Notably, neither of these RCTs reported improved BP or endothelial function, although an antihypertensive effect of some cocoa preparations is recognized.
**Fermented Milk Products**

Fermented milk product peptides may alter arterial stiffness by inhibiting the angiotensin-converting enzyme. Reductions in aortic PWV over 6 weeks, independent of systolic BP, have been reported in nonmedicated patients with pre-/stage 1 hypertension.

**Other Supplements**

Alternative approaches to arterial destiffening by dietary supplementation have used folic acid, minerals, and various antioxidants. All have demonstrated some preliminary evidence of benefit in cardiometabolic disease populations, except for folic acid, which had no effect in type 2 diabetes or kidney disease despite improving systemic arterial compliance in patients with normal or high-normal BP. Potassium has shown promise for arterial stiffness reduction beyond its capacity to lower BP. In a RCT of hypertensive patients, 4 weeks treatment lowered aortic PWV despite no change in office BP. Of antioxidant studies, combined vitamin C and vitamin E therapy for 8 weeks lowered aortic PWV in men with stage 1 hypertension. This was related to a reduction in oxidative stress independent of BP. In contrast, systemic arterial compliance was unchanged by 3 months of vitamin E treatment in patients with type 1 diabetes mellitus. As they become available, future antioxidant studies may be best directed to more potent agents (eg, astaxanthin).

**Conclusions**

Current evidence pertaining to the modification of arterial stiffness by exercise training and dietary interventions is summarized in the Table, stratified by disease state. It is important to recognize that this differentiation of nonpharmacological treatments by their effects on arterial stiffness is based on scarce and typically short-term (<12 months) RCTs. Distinguishing a direct structural versus indirect functional influence on stiffness (ie, independent of BP) is also difficult, and future studies in this field would be well served by complementing established, prognostic markers (eg, PWV) with measures more closely related to intrinsic structural characteristics (eg, aortic impedance).

Aerobic exercise reduces arterial stiffness in young, healthy individuals. However, in line with diminished capacity of older arteries to respond to exercise, efficacy beyond BP lowering has not been established in those at heightened cardiometabolic risk. Interventions targeting cardiorespiratory fitness (ie, higher-intensity or interval training) may be more effective, but require confirmation in larger RCTs. Moderate-intensity resistance training is advocated for risk reduction and prevention of functional decline in older individuals and some cardiometabolic disease states, but effects on arterial stiffness have not been well studied in these settings.

With the exception of the DASH eating pattern, data are scarce on comprehensive, whole-diet strategies for arterial destiffening. DASH trials have so far only demonstrated functional destiffening via BP lowering. However, its capacity to directly influence intrinsic arterial properties over the longer term should not be overlooked based on positive outcomes from increased potassium intake and sodium restriction per se (ie, inherent features of a low-sodium DASH diet). Of dietary supplements studied, fish oil so far has the most robust evidence of benefit. Fermented milk products, along with some polyphenols and antioxidants, also show promise for modification of arterial properties.

Since exercise and dietary interventions may reduce arterial stiffness by a variety of mechanisms, additive effects

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Table. Current Evidence for the Effects of Exercise and Dietary Interventions on Arterial Stiffness in Health and in Cardiometabolic Disease

<table>
<thead>
<tr>
<th>Effect on Arterial Stiffness</th>
<th>Healthy/General Population</th>
<th>Hypertension</th>
<th>Metabolic Syndrome/ Diabetes Mellitus</th>
<th>Dyslipidemia/CAD</th>
<th>Renal Disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>↓Arterial stiffness independent of BP</td>
<td>Aerobic exercise training&lt;sup&gt;48&lt;/sup&gt;</td>
<td>Fish oils&lt;sup&gt;60&lt;/sup&gt;; isoflavones&lt;sup&gt;61,62&lt;/sup&gt;†</td>
<td>High-intensity aerobic interval training&lt;sup&gt;63&lt;/sup&gt;; fermented milk products&lt;sup&gt;73&lt;/sup&gt;; vitamin C/E&lt;sup&gt;79&lt;/sup&gt;†</td>
<td>Moderate-high-intensity aerobic exercise training&lt;sup&gt;39&lt;/sup&gt;; fish oils&lt;sup&gt;67&lt;/sup&gt;</td>
<td>Cranberry juice polyphenols&lt;sup&gt;70&lt;/sup&gt;</td>
</tr>
<tr>
<td>↓Stiffness statistically independent of SBP (ie, SBP or an unspecified BP parameter†)</td>
<td>↓Stiffness with nonsignificant ΔSBP/ΔDBP</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>↓Arterial stiffness, possibly mediated by BP lowering</td>
<td>Fermented milk products&lt;sup&gt;72&lt;/sup&gt;</td>
<td>Resistance exercise training (intensity-dependent)&lt;sup&gt;35,47&lt;/sup&gt;; folic acid&lt;sup&gt;77&lt;/sup&gt;</td>
<td>Sodium restriction&lt;sup&gt;35,39&lt;/sup&gt;, moderate-intensity aerobic exercise training (Table S1)</td>
<td></td>
<td>Aerobic exercise training&lt;sup&gt;35,47&lt;/sup&gt;</td>
</tr>
<tr>
<td>Equivocal findings</td>
<td>DASH diet&lt;sup&gt;41&lt;/sup&gt;</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No evidence of benefit</td>
<td>Vitamins (C, E) and antioxidants (α-lipoic acid&lt;sup&gt;18&lt;/sup&gt;)</td>
<td>Isoflavones (polyphenols)&lt;sup&gt;69&lt;/sup&gt;</td>
<td>Folic acid&lt;sup&gt;44&lt;/sup&gt;; vitamin E&lt;sup&gt;80&lt;/sup&gt;</td>
<td>Cocoa polyphenols&lt;sup&gt;71&lt;/sup&gt;</td>
<td>Folic acid&lt;sup&gt;75,76&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

All references are to controlled trials or systematic reviews/meta-analyses. BP indicates blood pressure; CAD, coronary artery disease; DASH, Dietary Approaches to Stop Hypertension; DBP, diastolic BP; and SBP, systolic BP.

<sup>*</sup>Treatment effect on arterial stiffness after adjustment for SBP, or no association was observed between change in stiffness and change in SBP.

<sup>†</sup>Treatment effect on arterial stiffness after adjustment for an unspecified BP parameter (eg, SBP or mean arterial pressure), or no association was observed between change in stiffness and change in the unspecified BP parameter.
on arterial stiffness are possible, but evidence is scarce. The present gold-standard lifestyle intervention could arguably be based on the most successful intervention arm of the ENCORE trial; that is, aerobic exercise training, a DASH eating pattern, and weight loss.\(^{61}\) Although this combination should markedly reduce arterial stiffness, poor adherence is a major drawback to intensive lifestyle modification. It must also be acknowledged that even if we are able to modify arterial properties nonpharmacologically, it is unclear whether this will translate to improved prognosis. However, given its fundamental importance to arterial–ventricular coupling and cardiovascular function (Figure 2), reducing arterial stiffness seems highly desirable and worthy of further investigation.

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**Disclosures**

None.

**References**


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Exercise and Dietary Influences on Arterial Stiffness in Cardiometabolic Disease

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\textbf{Short title:} Exercise and diet for arterial stiffness

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Methodological considerations

The validity, reliability and confounders of methods characterizing both arterial stiffness (ie: systemic estimates derived from Windkessel models and regional/local markers such as pulse wave velocity [PWV] and $\beta$-stiffness index) and central systolic loading (ie: augmentation index [AIx]) have been described in detail elsewhere.\textsuperscript{1,2} Pertinent issues relating to arterial stiffness endpoint evaluation in exercise and diet-based intervention trials are discussed below.

Prognostic significance
Aortic (carotid-femoral) PWV has the largest evidence base,\textsuperscript{3,4} and typically out-performs central systolic loading markers (including AIx and central pulse pressure)\textsuperscript{5,6} in predicting adverse outcome. The prognostic relevance of measurements of local artery stiffness remains to be established. However, they are certainly valid and some may even hold advantages over PWV and AIx in informing mechanisms of change, as detailed below.

Physiological considerations, confounding factors and approaches to statistical analysis
Blood pressure (BP)-dependence of the method is a key factor given the antihypertensive effects of many exercise and dietary interventions. Indeed, this makes relatively BP-independent methods (eg: local $\beta$-stiffness index, aortic impedance estimation) particularly attractive for differentiating whether changes in stiffness reflect intrinsic adaptations in large artery properties or are secondary to changes in mean arterial pressure (MAP).\textsuperscript{7,8}

In the absence of a relatively BP-independent measure of arterial stiffness, statistical adjustment and/or a regression analysis characterizing change ($\Delta$) in stiffness vs. $\Delta$MAP should ideally be reported. MAP at the time of measurement is most relevant, so independence from changes in this parameter, rather than ambulatory BP, should be established. From a mechanistic perspective, changes in stiffness parameters mediated by MAP obviously do not reflect changes in the material properties of the large arteries.

Although MAP adjustment is preferred, many published studies have adjusted for systolic BP. This practice may actually diminish the observed effect of the intervention on arterial stiffness given its strong co-dependence with systolic BP.\textsuperscript{9} This is because reductions in pulse pressure (ie: reduced SBP and increased DBP) would be predicted as a consequence of reductions in arterial stiffness (Figure 2).

Where a reduction in arterial stiffness is observed in the absence of a statistically significant change in MAP, this may also indicate an independent, or direct, vascular effect. However, statistical adjustment should still be implemented in this scenario to account for small MAP changes.

Finally, consideration should also be given to concurrent changes in resting heart-rate (a major determinant of AIx in particular, given its dependence on ejection duration) and abdominal adiposity (a confounder of the carotid-femoral path length input to aortic PWV determination). However, short-term exercise studies are not usually associated with significant anthropometric changes and the current review focuses on dietary interventions other than weight loss.
Online Data Supplement References


### Table S1. Controlled studies of exercise training for large artery de-stiffening in hypertension

<table>
<thead>
<tr>
<th>First author, Year</th>
<th>Design</th>
<th>Population</th>
<th>Exercise training</th>
<th>Wks</th>
<th>( \Delta ) Office BP (mm Hg)</th>
<th>Effects on large artery properties / central systolic loading</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ferrier, 2001(^{10})</td>
<td>Randomized crossover</td>
<td>Isolated systolic hypertension (untreated) n=10: 64y, 50% men</td>
<td>Cycling: -3 × 30 min/week -65% HR reserve</td>
<td>8</td>
<td>No change (change not reported)</td>
<td>No change: - Aortic PWV - Systemic arterial compliance - Aortic input impedance</td>
</tr>
<tr>
<td>Westhoff, 2007(^{11})</td>
<td>Randomized, parallel</td>
<td>Isolated systolic hypertension (treated) n=51: 67–69y, 48% men -2.5 ± 0.5 mmol/L blood lactate ( \dagger )</td>
<td>Treadmill: -3 × 30–36 min/week</td>
<td>12</td>
<td>(+2/+2) (-8/-4) (sig. not reported)</td>
<td>No change: - Large artery compliance ( \dagger ) - Central AIx</td>
</tr>
<tr>
<td>Westhoff, 2008(^{12})</td>
<td>Randomized, parallel</td>
<td>Hypertension (treated and untreated) n=24: 66–68y, 46% men -2.0 ± 0.5 mmol/L blood lactate ( \dagger )</td>
<td>Upper limb ergometry: -3 × 30 min/week</td>
<td>12</td>
<td>(+1/+4) (-7^<em>/-6^</em>)</td>
<td>No change: - Large artery compliance ( \dagger )</td>
</tr>
<tr>
<td>Dimeo, 2012(^{13})</td>
<td>Randomized, parallel</td>
<td>Resistant hypertension n=47: 63–68y, 42% men -2.0 ± 0.5 mmol/L blood lactate ( \dagger )</td>
<td>Treadmill: -3 × 30–36 min/week</td>
<td>8–12</td>
<td>(+1/-1) (-7/-3 \text{ ns})</td>
<td>No change: - Large artery compliance ( \dagger )</td>
</tr>
<tr>
<td>Madden, 2009(^{14})</td>
<td>Randomized, parallel</td>
<td>Hypertension (treated and untreated); co-morbid T2DM &amp; hypercholesterolemia n=34: 71y, 53% men</td>
<td>Treadmill / cycle ergometry: -3 × 40 min/week -60–75% HR reserve</td>
<td>12</td>
<td>(-2/-1) (-10/-5 \text{ ns})</td>
<td>Improvement: - Aortic PWV</td>
</tr>
<tr>
<td>Guimaraes, Randomized, parallel: continuous vs. interval training vs. control</td>
<td>Hypertension controlled by medication n=43: 45–50y, 30% men -60% HR reserve</td>
<td>Continuous: Treadmill 3 × 40 min/week Interval (2 min / 1 min): -60% / 80% HR reserve</td>
<td>16</td>
<td>(-4/-3) (-7/-3 \text{ ns (cont.)})</td>
<td>Improvement: - Aortic PWV (interval training only)</td>
<td></td>
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<tr>
<td>First author, Year</td>
<td>Design</td>
<td>Population</td>
<td>Exercise training</td>
<td>Wks</td>
<td>Δ Office BP (mm Hg)</td>
<td>Effects on large artery properties / central systolic loading</td>
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<tr>
<td>Nualnim, 2012&lt;sup&gt;16&lt;/sup&gt;</td>
<td>Randomized, parallel</td>
<td>Pre-/ stage 1 hypertension (untreated) - 3–4 × 15–45 min/week n=43: 58–61y, 26% men - 60–75% maximum HR</td>
<td>Swimming:</td>
<td>12</td>
<td>0/−1</td>
<td>−9&lt;sup&gt;†&lt;/sup&gt;/−4</td>
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<td>Central AIx</td>
<td>- Carotid artery compliance</td>
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<td>- Central AIx</td>
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</table>

Studies listed in chronological order. Where an age range is given to describe the study population, this refers to the range of group means rather than the range of individual ages.

* Significant difference at p<0.05.
† Large artery compliance derived from radial pulse wave analysis (modified Windkessel model).
‡ Initial training included half-speed or rest intervals, with progression to continuous training toward the end of the intervention.
Δ indicates pre-/post-intervention change; AIx, augmentation index; BP, blood pressure; DBP, diastolic blood pressure; HR, heart-rate; min/week, minutes per week; ns, not significant; PWV, pulse wave velocity; SBP, systolic blood pressure; Wks, Weeks; y, years.