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EDITORIAL

Arterial stiffness on a different scale

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This editorial refers to 'Home monitoring of arterial pulse-wave velocity during COVID-19 total or partial lockdown using connected smart scales', by R.M. Bruno *et al*., https://doi.org/10.1093/ehjdh/ztac027.

Introduction

Arterial stiffness is a known predictor of cardiovascular disease[1] and also a measure of target organ damage.[2] Although carotid–femoral pulse wave velocity (PWV) is the current gold standard for assessing arterial stiffness,[3] its measurement (i) requires the individual to be in a supine position, (ii) is complicated in overweight individuals, and (iii) requires a femoral measurement in the groin area, which in some countries is deemed inappropriate. Several devices have been proposed to overcome (some of) these limitations. These include devices measuring cardio-ankle vascular index (not requiring a femoral measurement)[4] and single-cuff-based devices.[5] All these devices aim at PWV measurement in the clinic.

In 2017, Campo *et al*.[6] presented a different type of device: a connected bathroom scale able to measure PWV at home. This device works by estimating the heart–foot pulse transit time interval. The start of the transit time interval is estimated using ballistocardiography: when the left ventricle contracts, it accelerates blood upwards into the ascending aorta, which (Newton's third law) causes a downward acceleration of the heart and of the body as a whole.[7] This acceleration is measured as a minute weight increase on the scale. The end of the transit time interval is estimated using impedance plethysmography at the foot: by applying a small electrical current through the foot, impedance is measured. As soon as the blood pressure wave reaches the foot, the arteries therein dilate, causing a temporal increase in blood volume in the foot. Since the impedance of blood is lower than that of the foot's tissue, it causes a small decrease in measured impedance.

In the present issue of the *European Heart Journal – Digital Health*, Bruno *et al*.[8] used this novel device to measure PWV in ∼53 000 individuals of which 50% resided in France and 50% in Germany. Measurements were collected during the first 18 weeks of 2020. During weeks 11–17 therein, France went into a full COVID-19 lockdown, whereas Germany enforced only a partial lockdown. In both countries, a significant decrease in PWV was observed during lockdown. However, the observed decrease in France (full lockdown) was twice as large as that in Germany (partial lockdown).

Discussion

Bruno *et al*.'s study highlights how connected smart technology enables at-home self-assessment of parameters that are normally measurable only clinically. The connectedness enabled them to collect and integrate data for scientific purposes—collecting a similar number of measurements (nearly 5 *million*) in a clinical setting would be virtually impossible. Several differences exist between PWV as measured by a scale and carotid–femoral PWV (*Table 1*). Three of these differences are highlighted in the following context.

First, the scale measures PWV along the heart–foot (not carotid–femoral) trajectory, which means that the more muscular arteries in the leg are also included. Pulse wave velocity in those distal arteries is typically higher than in the aorta, causing heart–foot PWV values to typically be higher than carotid–femoral values.[9] To correct for this, the scale was calibrated (and in the same study subsequently validated) against a device that measures carotid–femoral PWV.[6] Note that the scale's heart–foot PWV measurements taken in the standing position were calibrated to match carotid–femoral PWV measurements taken in the supine position[6] (also see below). Furthermore, this calibration was performed in young individuals (mean age 30 years). Besides the absolute differences between carotid–femoral and heart–foot PWV values, it is known that, for example, with age, proximal arteries stiffen more than distal arteries.[10] This implies that observed changes in heart–foot PWV may be of a different magnitude than changes in carotid–femoral PWV and, hence, raises the question of which reference values to use for the scale: the well-established carotid–femoral reference values[11] or reference values based on heart–foot PWV (e.g. Wohlfahrt *et al*.[12] after back-conversion to PWV).

Second, carotid–femoral PWV is typically measured in the supine position, ensuring that there is no hydrostatic blood pressure gradient along the arterial tree. When PWV is measured in the standing

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Table 1 Comparison of scale-based and carotid–femoral pulse wave velocity measurements

	Scale-based PWV	Carotid–femoral PWV
Feasibility to run a multi-million measurement study	Feasible	Not feasible
Device cost	$	$$$
Operator experience needed	No	Yes
Measurement setting	Home	Clinic
Arterial segments included in measurement		
Ascending aorta	•	
Descending thoracic and abdominal aorta	•	•
Femoral artery	•	•
Popliteal, tibial, and foot arteries	•	
Measurement position	Standing	Supine
Measurement complicated in obese individuals	No	Yes
Requires exposing femoral region	No	Yes
White-coat effect	No	Yes
Outcome data available	No	Yes
BP measurement typically available for PWV correction	No	Yes
Gold standard	No	Yes

BP, blood pressure; PWV, pulse wave velocity.

position, however, such gradient does exist and may influence the measured PWV. We observed this during a head-up tilt test: tilting induced only a modest increase in brachial blood pressure but induced a much larger increase in PWV, presumably due to a change in the blood pressure *gradient* along the arterial bed.[13] This observation is interesting in the light of a second validation study of the scale,[14] which also compared the scale-derived PWV to carotid–femoral PWV but observed scale-derived PWV to be lower than carotid–femoral PWV by 0.68 ± 0.43 m/s (mean $\pm$ standard deviation). A key difference between these validation studies is the position in which they measured carotid–femoral PWV (Campo *et al.*[6] supine; Collier *et al.*[14] standing). Standing up increases (carotid–femoral) PWV, causing Collier *et al.* to observe a scale PWV that was lower than the carotid–femoral PWV. Note that, if the scale would report a true, physical heart–foot PWV, Collier *et al.* (whom measured carotid–femoral and heart–foot PWV's in identical positions) would likely have observed the scale's PWV values to be higher (not lower) than carotid–femoral PWV values.[9]

Third, PWV is known to depend on blood pressure at the time of measurement.[15] When PWV is measured in the clinic, typically, a blood pressure measurement is also taken, enabling the interpretation of the measured PWV in the context of the current blood pressure, or conversion of PWV into a pressure-corrected metric.[15] When PWV is measured at home using a scale, a blood pressure measurement is typically not available, making it hard to pressure-correct such home PWV measurement. On a positive note, however, the blood pressure at which PWV is measured at home is not influenced by the white-coat effect that commonly occurs during clinical blood pressure and PWV measurements.[16] This implies that PWV measured at home may be more representative of the individual's 'typical' PWV during daily life and, hence, might correlate better with cardiovascular risk, even if uncorrected for blood pressure.

During their study period, Bruno *et al.*[8] observed an average decrease in PWV during lockdown. This finding is interesting given that, in general, arterial stiffness increases with age. Although the duration of ageing during lockdown is very modest herein (only 6 weeks), in a population of this size, one would expect an average increase in PWV over time.[11] The fact that the opposite was observed could be due to changes in active arterial smooth muscle tone, changing the functional stiffness of the arteries and, hence, the observed PWV.[17] In particular, as elaborated above, the arterial segment assessed by the scale PWV contains more muscular arteries than the segment assessed by carotid–femoral PWV and, hence, may be more prone to changes in vascular tone. Another explanation may lie in the change in blood pressure, which indeed in a subgroup was found to decrease modestly and would (all else equal) cause a decrease in PWV.[15] Finally, there could indeed be some reverse remodelling, causing the arteries to structurally de-stiffen in response to the new situation (of e.g. a reduced blood pressure). Another key observation by Bruno *et al.* was a difference in the PWV response to lockdown between France and Germany, with a bigger response observed in France. Bruno *et al.* speculate that this was due to the countries going into full and partial lockdown, respectively. While this differential PWV response could indeed relate to the lockdown difference and, for example, the difference in weight trajectories between the different countries, the differences between the French and German populations may reach beyond what is measurable with a (smart) scale.

In summary, Bruno *et al.* present an exciting study on PWV changes in a vast study population. Although differences exist between scale-based PWV and the archetypical carotid–femoral PWV, provided that these differences are recognized, scale-based PWV assessment poses a revolutionary step forward in the field of arterial stiffness research.

Conflict of interest: None declared.

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