Progressive attenuation of the longitudinal kinetics in the common carotid artery: preliminary *in vivo* assessment

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Abstract

Longitudinal kinetics (LOKI) of the arterial wall consists in the shearing motion of the intima-media complex over the adventitia layer in the direction parallel to the blood flow during the cardiac cycle. The aim of this study is to investigate the local variability of LOKI amplitude along the length of the vessel. Using a previously validated motion-estimation framework, 35 *in vivo* longitudinal B-mode ultrasound cine-loops of healthy common carotid arteries were analyzed. Results demonstrate that LOKI amplitude is progressively attenuated along the length of the artery, as it is larger in regions located on the proximal side of the image (*i.e.* toward the heart), and smaller in regions located on the distal side of the image (*i.e.* toward the head), with an average attenuation coefficient of $-2.5 \pm 2.0\ \% \cdot \mathrm{mm}^{-1}$. Reported for the first time in this study, this phenomenon

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is likely to be of great importance for improved understanding of atherosclerosis mechanisms, and has potential to constitute a novel index of arterial stiffness.
Keywords: Longitudinal kinetics, Ultrasound imaging, Common carotid artery, Motion tracking, Atherosclerosis, Arterial stiffness

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Introduction

Cardiovascular diseases represent the leading cause of human mortality and morbidity (WHO, 2013). To assess cardiovascular risk, the common carotid artery (CCA) has been extensively analyzed *in vivo* using B-mode ultrasound (US) imaging. A few pioneering studies have investigated the deformation of the arterial wall tissues in the direction parallel to the blood flow during the cardiac cycle (Persson et al., 2003). This phenomenon, hereafter referred to as *"longitudinal kinetics"* (LOKI), corresponds to the cyclic shearing motion of the intima-media complex with respect to the tunica adventitia (Figure 1).

Recent findings contributed to elucidate the association between LOKI and vascular pathophysiology. Namely, LOKI has been reported to induce a wall shear strain (WSS) reflecting arterial stiffness (Cinthio et al., 2006; Nilsson et al., 2010; Zahnd et al., 2011b; Idzenga et al., 2012), and has been associated with the presence of cardiovascular risk factors (Ahlgren et al., 2009; Zahnd et al., 2011a, 2012; Ahlgren et al., 2012) as well as with atherosclerotic plaque burden (Svedlund and Gan, 2011; Soleimani et al., 2012; Gastounioti et al., 2013). Also, it was reported to predict 1-year cardiovascular outcome in patients with suspected coronary artery disease (Svedlund et al., 2011). These findings strongly suggest that LOKI constitutes a solid candidate to become a novel valuable image-based biomarker for improved cardiovascular risk prediction.

Various advanced techniques have been proposed to evaluate LOKI in B-mode US cine-loops. An echo-tracking approach, based on a careful imaging protocol to track a tiny region of interest (ROI) encompassing a well contrasted speckle pattern, provided a detailed characterization of LOKI (Cinthio et al., 2005). Towards accurate LOKI evaluation in routinely-acquired data, robust motion-tracking approaches have been proposed, based on Kalman filtering (Gastounioti et al., 2011; Zahnd et al., 2013), weighted least-squares optical flow (Golemati et al., 2012), and finite impulse response filtering (Gastounioti et al., 2013). Radio-frequency US was also used to asses the wall shear strain by means of a coarse-to-fine cross-correlation based strain algorithm (Idzenga et al., 2012).

One of the main parameters derived from LOKI analysis is its maximal peak-to-peak amplitude ΔX, (*i.e.* the total amplitude of the longitudinal motion of the intima and media layers along the direction parallel to the blood flow), which is significantly reduced in patients with presumably stiffer arteries (Zahnd et al., 2011a; Svedlund et al., 2011; Zahnd et al., 2012). Yet, the influence of the location of the assessed ROI onto the resulting motion amplitude ΔX is still unclear. Indeed, it has previously been reported by our team that different resulting trajectories could be observed when tracking points in different regions (*i.e.* on the right, center, and left side of the image) (Zahnd et al., 2011a).

The aim of this pilot study is to characterize such local variability of LOKI amplitude ΔX in longitudinal B-mode US cine-loops of healthy CCA. For each cine-loop analyzed, the motion of the far wall at different locations within the intima-media complex has been evaluated on a collection of points by means of robust speckle tracking, using a previously validated framework (Zahnd et al., 2013). Results obtained on 35 healthy volunteers not only confirm the presence

of a systematic local variability, but also demonstrate that ΔX is lower in ROIs located on the *"distal"* side of the image (*i.e.* toward the head) compared to ROIs located on the *"proximal"* side (*i.e.* toward the heart). This breakthrough suggests that LOKI-inducing forces are progressively attenuated as they further propagate from the heart to peripheral organs. To the best of our knowledge, the present study is the first to report this finding.

Methods

Study population

Fifty-seven healthy volunteers (mean age: 37.9 ± 14.1 y.o., 24 males) have been involved in this study. All participants were cardiovascular risk factor-free (tobacco use, hypercholesterolemia, diabetes, hypertension, or particular family history), as assessed by an oral questionnaire. Written informed consent was obtained from all participants. The study fulfilled the requirements of our institutional review board and the ethics committee.

Acquisition of carotid artery ultrasound sequences

The image acquisition protocol has been previously described (Zahnd et al., 2013). Briefly, longitudinal B-mode US cine-loops of the left CCA were obtained using a clinical scanner (Antares, Siemens Erlangen), equipped with a 7.5- to 10-MHz linear array transducer. Acquisition was performed by an expert physician during at least two full cardiac cycles, with the probe centered approximately 2 cm from the carotid bifurcation. The probe was systematically oriented in such way that the left border of the image corresponded to the distal side, and the right border to the proximal side, as pictured in Figure 1a. The frame rate was 26 fps,

and the width of the acquired US image was 21 mm, with a pixel size in both longitudinal and radial directions was 30 μm.

Quantification of LOKI amplitude ΔX

For each participant, a collection of salient points located within the intima-media complex of the far wall were manually selected to be tracked (Fig. 2a). Such salient echo scatterers are defined as well contrasted speckle patterns that remain clearly perceptible during the entire cine-loop (Cinthio et al., 2005; Zahnd et al., 2013). In order to assess the local variability of LOKI, motion tracking has to be performed on at least two different sites in the image. Therefore, participants presenting either none or only one salient echo were rejected from the study. For all remaining participants, the bi-dimensional (2D, *i.e.* radial and longitudinal) temporal trajectory of each salient echo during the entire cine-loop was extracted, using a framework previously developed and validated by our team (Zahnd et al., 2013). Finally, LOKI amplitude ΔX was measured from the resulting trajectory of each tracked point. The amplitude ΔX was determined as the peak-to-peak amplitude of the longitudinal component of the 2D trajectory, averaged over two cardiac cycles (Fig. 2b).

Attenuation coefficient Z

We define the attenuation coefficient Z as the slope of the linear regression that fits, for a given cine-loop, the set of points whose x-coordinate is the distance X from the right border of the image, and whose y-coordinate is the corresponding measured amplitude ΔX (Fig. 2c).

Statistical analysis

The Pearson correlation coefficient R was used to evaluate the correlation between LOKI amplitude ΔX and the attenuation coefficient Z. Statistical analysis was performed using MATLAB (MATLAB 7.13, The MathWorks Inc., Natick, MA, USA, 2011).

Results

For each participant, at least one salient point could be observed. Twenty-two participants were rejected from the study, as they presented only a single salient point to be tracked. In the remaining 35 participants, the average number of tracked points was 3.7 ± 1.5 (the number of cine-loops presenting 2, 3, 4, 5, 6, and 7 measurement points was 9, 9, 7, 5, 3, and 2, respectively). For those analyzed participants, the average width of the total assessed region, defined as the distance between the two extreme proximal and distal measurement points, was 15.3 ± 5.4 mm (range: $[3.2, 25.1]$ mm), corresponding to 56 ± 19 % of the entire image width, which was 21 mm. Within the analyzed region, the average distance between two neighbor measurement points was 5.3 ± 3.6 mm (range: $[1.0, 18.7]$ mm).

The average LOKI amplitude ΔX of all individual tracked points was 722 ± 284 μm. As the number of tracked points is subject-dependent, we also define $\widehat{\Delta X}$ as the mean value of all ΔX measurements for a given participant. The average amplitude $\widehat{\Delta X}$ for all participants was 717 ± 260 μm. For the majority of the analyzed subjects (*i.e.* 33 out of 35), we made the following observation: LOKI amplitude ΔX is systematically larger for points located on the proximal side of the

image, and smaller for points located on the distal side of the image, as depicted in Figure 2a,b.

Examples of the attenuation coefficient Z estimated in different participants are displayed in Figure 3. Calculating the average attenuation coefficient Z of all 35 participants, we confirmed the presence of a progressive attenuation of LOKI amplitude towards the distal direction, with an average Z value of -17 ± 16 μm $\cdot$ mm^{-1} (range $[-91, 17]$ μm $\cdot$ mm^{-1}, as displayed in Figure 4. This attenuation can also be quantified as follow: within a given CCA segment, the motion between two points separated by 1 mm undergoes a decrease corresponding to -2.5 ± 2.0 % of the average LOKI amplitude $\widehat{\Delta X}$ of the vessel. Arteries with a larger LOKI amplitude were not found to also undergo a stronger attenuation, as no significant correlation could be observed between the mean LOKI amplitude per subject $\widehat{\Delta X}$ and the corresponding attenuation coefficient Z (R=0.22).

The average dispersion error with respect to the linear regression model (*i.e.* the mean absolute vertical distance between the estimated points and the regression line) as a function of the number of tracked points is displayed in Table 1. The overall average absolute error of fitting between all the measurement points and the linear regression model was 34 ± 36 μm. By definition, the dispersion error generated by linear regression is null in cine-loops with exactly 2 tracked points. For this reason, we also calculated the average absolute error of fitting in cine-loops with at least 3 measurement points, which was 39 ± 36 μm. Such dispersion remains small in comparison with the average LOKI amplitude $\widehat{\Delta X}$ (*i.e.* 717 ± 260 μm), as depicted in Figure 3. The dispersion was also nearly twice smaller than the tracking accuracy of our framework in the longitudinal direction, previously validated in (Zahnd et al., 2013). Indeed, with respect to the reference that was

generated from the manual tracings of three experienced observers, the average absolute error of our framework for ΔX quantification was 74 ± 68 μm, while the inter- and intra-observer variability was 79 ± 103 μm and 62 ± 103 μm, respectively.

Discussion

The principal finding of this study is the presence of a systematic local variability of LOKI amplitude ΔX, and more specifically a decreasing trend in ΔX within the imaged region (Fig. 2). That is to say, tissues located on the proximal side of the image (*i.e.* towards the heart, right border of the image) present an overall larger motion amplitude than tissues located on the distal side (*i.e.* towards the head, left border of the image).

Fitting the measured amplitudes with linear regression, we observed an average attenuation coefficient Z equal to -17 ± 16 μm/mm (Fig. 4), which corroborates the presence of a decreasing trend along the arterial length. We observed an increase of the fitting error with the number of measurement points. This can be explained by the fact that a larger amount of roughly aligned points would increase the average distance between each point and its vertical projection on the linear regression.

To perform the measurements, we used a Kalman-based tracking framework, previously developed by our team and validated on a dataset of 82 subjects (Zahnd et al., 2013). The average absolute dispersion error of the measured amplitudes ΔX against a linear regression model was roughly twice smaller than the previously validated *i)* tracking accuracy of our framework, and *ii)* inter- and intra-observer variability. This strengthens the validity of the observed negative trend in ΔX across the width of the imaged regions.

This study implies a major caveat that should be taken into account when investigating LOKI in future work. This recommendation concerns the quantification of LOKI amplitude ΔX (*i.e.* the quantity of tissue deformation in the longitudinal direction) in a given subject. This parameter is relevant, as previous studies have demonstrated a negative association between ΔX and the presence of cardiovascular risk factors (Zahnd et al., 2011a; Svedlund et al., 2011; Zahnd et al., 2012; Ahlgren et al., 2012; Golemati et al., 2012; Gastounioti et al., 2013). Due to the attenuation phenomenon described in this study, LOKI amplitude ΔX is dependent on the initial location of the tracked point, and is thus subject to variability within the imaged region. Therefore, to thoroughly assess ΔX in a cine-loop, it would be more insightful to systematically evaluate LOKI at a calibrated location. From our experience however, a standardized horizontal origin cannot be fixed to determine the point to be tracked for the entire dataset, due to a variability in image local quality between different cine-loops. An alternative solution could be to evaluate LOKI in a set of different locations where the image quality is optimal, in the aim to determine the average value $\widehat{\Delta \mathrm{X}}$.

The attenuation phenomenon observed in this study can be explained by a number of factors contributing to arterial fixation *via* tethering to surrounding tissues: *i)* side branches, *ii)* perivascular connective tissues, *iii)* vessel three-dimensional tortuosity (Nichols and O'Rourke, 2005). Furthermore, LOKI attenuation can also be explained by the fact that arterial tissues have viscoelastic properties and play a cushioning function to dissipate high blood pressure provoked by pulsatile flow (Benetos et al., 1997). Such damping properties are likely to be associated with wall shear strain, which plays a fundamental role in vasa vasorum circulation as well as endothelial function (Cunningham and Gotlieb, 2004; Chatzizisis et al.,

2007).

The question of characterizing the actual forces at the origin of LOKI is still unanswered at the moment. Blood friction, creating a tangential force at the surface of the wall, is unlikely to constitute a significant contribution to LOKI. Instead, it is hypothesized that LOKI is induced by a large retrograde wall motion (*i.e.* in the direction opposite to blood flow), provoked by the apical traction of the aortic valve annulus in late systole (Cinthio et al., 2006). The amplitude of this pulling force, referred to as tricuspid annular plane systolic excursion, was measured in echocardiography and reported to be superior to 17 mm in healthy subjects (Tamborini et al., 2007; Rudski et al., 2010). Accordingly, as the average LOKI amplitude $\widehat{\Delta X}$ in the CCA was found to correspond to 717 ± 260 μm, the initial pulling force is likely to be dissipated along the way, which is confirmed by the results of this study. We formulate the hypothesis that LOKI would eventually become null (*viz.*: $\Delta X \approx 0$) at the point where the internal carotid artery enters the skull *via* the carotid canal, through the petrous portion of the temporal bone.

Although LOKI amplitude ΔX is associated with cardiovascular risk factors (Zahnd et al., 2011a; Svedlund et al., 2011; Zahnd et al., 2012; Ahlgren et al., 2012; Golemati et al., 2012; Gastounioti et al., 2013), the actual clinical relevance of the attenuation coefficient Z remains to be investigated. Since this pilot study involved healthy middle-aged participants, it is still unclear whether the measured LOKI attenuation coefficient Z reflects vascular health. Future studies, involving patients at cardiovascular risk, are necessary to investigate the association between Z and cardiovascular risk factors (*e.g.* diabetes, hypertension, metabolic syndrome, tobacco use, age). Further work, focusing on the association between the attenuation coefficient Z and surrogate markers of atherosclerosis (*e.g.* LOKI amplitude ΔX,

intima-media thickness, cross-sectional distensibility, pulse wave velocity), would determine whether Z actually constitutes a reliable and independent biomarker of vascular health. Besides, *in vivo* measurement of the attenuation coefficient Z would enable tissue stiffness to be quantified *via* a patient-specific biomechanical model.

This study does present several limitations that open the field to multiple perspectives. First, our framework requires the presence of several (at least two) bright echo scatterers in the intima-media complex, to accurately track the wall motion during the cardiac cycle and quantify the attenuation coefficient Z. For this reason, 22 subjects out of 57 were rejected from the study as they did not meet this condition. Second, although the actual wall pulsatility corresponds to a three-dimensional motion, LOKI was approximated by considering the projection of the motion along the horizontal axis. In this study, we assumed that both radial and azimuthal (*i.e.* along the axis perpendicular to the image plane) motion components can be neglected, since *i)* limited out-of-plane motion could be obtained at the location of the far wall *via* a careful positioning of the probe, and *ii)* the curvature of the wall was small w.r.t. the amplitude of the motion. These issues will be investigated in future work. Third, we have made the approximation that the variation of ΔX was linear within the imaged region. However, the actual attenuation is most likely to be non-linear along the total arterial tree. Further studies, involving data acquired both upstream (*i.e.* aortic valve annulus) and downstream (*i.e.* carotid bulb, as well as internal and external carotids), would provide valuable information about the local attenuation coefficient Z along the vascular tree. Finally, we have focused on the analysis of the left CCA, which is directly connected to the aorta. It would be insightful to realize a compari-

son with results obtained on the right CCA, which is connected to the aorta *via* the S-shaped brachiocephalic trunk. Accordingly, the initial pulling force is likely to be further attenuated in the right CCA, and could potentially be reflected by smaller LOKI amplitude ΔX and attenuation coefficient Z. Extending the scope of investigation to other main arteries (*e.g.* popliteal, humeral) would also benefit to a more comprehensive analysis.

Conclusion

In this pilot study, we analyzed B-mode US cine-loops of healthy CCA to assess the local variability of LOKI amplitude ΔX (*i.e.* the quantity of tissue deformation parallel to the blood flow) within the imaged region during the cardiac cycle. We observed that LOKI amplitude ΔX does undergo a progressive attenuation along the arterial segment, namely ΔX is larger on the proximal side of the image, and smaller on the distal side, with an average attenuation coefficient of -2.5 ± 2.0 % $\cdot$ mm^{-1}. To the best of our knowledge, the observation of this phenomenon *in vivo* has not previously been reported. We hypothesize that this gradual decrease reflects a damping role in the circulatory system, and could potentially be associated with cardiovascular risk. Further studies are required to investigate this finding.

Figure captions

Figure 1: Common carotid artery (CCA). (a) Longitudinal B-mode ultrasound image of a healthy CCA *in vivo*. The direction of the blood flow in the lumen is indicated by the white arrow. (b) Illustration of the longitudinal kinetics (LOKI), occurring within the concentric layers of the wall during the heart beat. Please note that the tissues motion obey to a multiphasic and bidirectional pattern, towards both left and right directions during the cardiac cycle.

Figure 2: Example of LOKI amplitude ΔX, evaluated on a healthy subject. (a) Location of three regions of interest (ROIs), encompassing the tracked points within the intima-media complex of the far wall. The resulting LOKI amplitude ΔX during the cine-loop is represented by the double arrows. (b) Longitudinal component of the estimated trajectory, for the left (dotted red), middle (dashed green), and right (solid blue) tracked points. The peak-to-peak amplitude ΔX of each tracked point is represented by the vertical bar of the corresponding color. (c) Representation of the attenuation coefficient Z for this specific cine-loop, determined by linear regression (solid line) of the three tracked points (circles). For each tracked point, the x- and y-coordinates correspond to the abscissa of the ROI in (a), and to the estimated amplitude ΔX in (b), respectively.

Figure 3: Examples of the attenuation coefficient Z in six participants, evaluated by means of linear regression (solid line) of the estimated LOKI amplitude ΔX (circles) over the length of the imaged region. The x-axis is oriented accordingly to the blood flow direction, from the right border of the

image ($x = 0$) towards the left border. The fitting error (err) is indicated, and the corresponding standard deviation is represented by the dashed lines. (a-e) Representative examples of a progressive attenuation of ΔX. (f) Outlier result, presenting an increasing trend, which is likely to be due to an insufficient number of points and a relatively small distance between them.

Figure 4: Histogram representing the distribution of the attenuation coefficient Z for the 35 analyzed healthy subjects. A negative trend is clearly visible. Please note that the two outliers (Z=-91 $\mu\text{m} \cdot \text{mm}^{-1}$ and Z=17 $\mu\text{m} \cdot \text{mm}^{-1}$, respectively) correspond to cine-loops where only two measurement points were available, thus leading to a potential estimation error.

References

Ahlgren ÅR, Cinthio M, Steen S, Nilsson T, Sjöberg T, Persson HW, Lindström K. Longitudinal displacement and intramural shear strain of the porcine carotid artery undergo profound changes in response to catecholamines. American Journal of Physiology - Heart and Circulatory Physiology, 2012;302:H1102–H1115.

Ahlgren ÅR, Cinthio M, Steen S, Persson HW, Sjöberg T, Lindström K. Effects of adrenaline on longitudinal arterial wall movements and resulting intramural shear strain: a first report. Clinical Physiology and Functional Imaging, 2009;29:353–359.

Benetos A, Laurent S, Asmar RG, Lacolley P. Large artery stiffness in hypertension. Journal of Hypertension, 1997;15:S89–S97.

Chatzizisis YS, Coskun AU, Jonas M, Edelman ER, Feldman CL, Stone PH. Role of endothelial shear stress in the natural history of coronary atherosclerosis and vascular remodeling: Molecular, cellular, and vascular behavior. Journal of the American College of Cardiology, 2007;49:2379–2393.

Cinthio M, Ahlgren ÅR, Bergkvist J, Jansson T, Persson HW, Lindström K. Longitudinal movements and resulting shear strain of the arterial wall. American Journal of Physiology, 2006;291:H394–H402.

Cinthio M, Ahlgren ÅR, Jansson T, Eriksson A, Persson HW, Lindström K. Evaluation of an ultrasonic echo-tracking method for measurements of arterial wall movements in two dimensions. IEEE Transactions on Ultrasonics, Ferroelectrics, and Frequency Control, 2005;52:1300–1311.

312 Cunningham KS, Gotlieb AI. The role of shear stress in the pathogenesis of
313 atherosclerosis. Laboratory investigation, 2004;85:9–23.

314 Gastounioti A, Golemati S, Stoitsis J, Nikita KS. Comparison of Kalman-filter-
315 based approaches for block matching in arterial wall motion analysis from B-
316 mode ultrasound. Measurement Science and Technology, 2011;22:114008.

317 Gastounioti A, Golemati S, Stoitsis JS, Nikita KS. Carotid artery wall motion
318 analysis from B-mode ultrasound using adaptive block matching: in silico eval-
319 uation and in vivo application. Physics in Medicine and Biology, 2013;58:8647.

320 Golemati S, Stoitsis JS, Gastounioti A, Dimopoulos AC, Koropouli V, Nikita KS.
321 Comparison of block matching and differential methods for motion analysis of
322 the carotid artery wall from ultrasound images. IEEE Transactions on Informa-
323 tion Technology in Biomedicine, 2012;16:852–858.

324 Idzenga T, Holewijn S, Hansen HHG, de Korte CL. Estimating cyclic shear strain
325 in the common carotid artery using radiofrequency ultrasound. Ultrasound in
326 Medicine & Biology, 2012;38:2229–2237.

327 Nichols WW, O'Rourke MF. McDonald's blood flow in arteries: Theoretic, exper-
328 imental, and clinical principles. Fifth Edition. Oxford University Press, 2005.

329 Nilsson T, Ahlgren ÅR, Jansson T, Persson H, Nilsson J, Lindström K, Cinthio M.
330 A method to measure shear strain with high spatial resolution in the arterial wall
331 non-invasively in vivo by tracking zero-crossings of B-mode intensity gradients.
332 In: IEEE Ultrasonics Symposium, San Diego, California (USA), 2010. pp. 491–
333 494.

Persson M, Ahlgren ÅR, Jansson T, Eriksson A, Persson HW, Lindström K. A new non-invasive ultrasonic method for simultaneous measurements of longitudinal and radial arterial wall movements: first in vivo trial. Clinical Physiology and Functional Imaging, 2003;23:247–251.

Rudski LG, Lai WW, Afilalo J, Hua L, Handschumacher MD, Chandrasekaran K, Solomon SD, Louie EK, Schiller NB. Guidelines for the echocardiographic assessment of the right heart in adults: A report from the american society of echocardiography. Journal of the American Society of Echocardiography, 2010;23:685–713.

Soleimani E, Manijhe MD, Hajir S, Shahram SH, R. S. Kinematics parameter extraction of longitudinal movement of common carotid arterial wall in healthy and atherosclerotic subjects based on consecutive ultrasonic image processing. Physiology and Pharmacology, 2012;16:165–178.

Svedlund S, Eklund C, Robertsson P, Lomsky M, Gan LM. Carotid artery longitudinal displacement predicts 1-year cardiovascular outcome in patients with suspected coronary artery disease. Arteriosclerosis, Thrombosis, and Vascular Biology, 2011;31:1668–1674.

Svedlund S, Gan L. Longitudinal common carotid artery wall motion is associated with plaque burden in man and mouse. Atherosclerosis, 2011;217:120–124.

Tamborini G, Pepi M, Galli CA, Maltagliati A, Celeste F, Muratori M, Rezvanieh S, Veglia F. Feasibility and accuracy of a routine echocardiographic assessment of right ventricular function. International journal of cardiology, 2007;115:86–89.

WHO. World Health Organization, Cardiovascular diseases (CVDs), Fact sheet number 317. http://www.who.int/mediacentre/factsheets/fs317/en/index.html, 2013.

Zahnd G, Boussel L, Marion A, Durand M, Moulin P, Sérusclat A, Vray D. Measurement of two-dimensional movement parameters of the carotid artery wall for early detection of arteriosclerosis: a preliminary clinical study. Ultrasound in Medecine & Biology, 2011a;37:1421–1429.

Zahnd G, Boussel L, Sérusclat A, Vray D. Intramural shear strain can highlight the presence of atherosclerosis: a clinical in vivo study. In: IEEE Ultrasonics Symposium, Orlando, Florida (USA), 2011b. pp. 1770–1773.

Zahnd G, Orkisz M, Sérusclat A, Moulin P, Vray D. Evaluation of a Kalman-based block matching method to assess the bi-dimensional motion of the carotid artery wall in B-mode ultrasound sequences. Medical Image Analysis, 2013;17:573–585.

Zahnd G, Vray D, Sérusclat A, Alibay D, Bartold M, Brown A, Durand M, Jamieson LM, Kapellas K, Maple-Brown LJ, O'Dea K, Moulin P, Celermajer DS, Skilton MR. Longitudinal displacement of the carotid wall and cardiovascular risk factors: associations with aging, adiposity, blood pressure and periodontal disease independent of cross-sectional distensibility and intima-media thickness. Ultrasound in Medecine & Biology, 2012;38:1705–1715.