Ultrasound in Med. & Biol., Vol. ■, No. ■, pp. 1–9, 2016

0301-5629/$ - see front matter

http://dx.doi.org/10.1016/j.ultrasmedbio.2016.04.014

● *Original Contribution*

# CAROTID ATHEROSCLEROTIC PLAQUE ALTERS THE DIRECTION OF LONGITUDINAL MOTION IN THE ARTERY WALL

JIMMY TAT,* IOANNIS N. PSAROMILIGKOS,† and STELLA S. DASKALOPOULOU*
*Department of Medicine, McGill University, Montreal, Quebec, Canada; and †Department of Electrical and Computer Engineering, McGill University, Montreal, Quebec, Canada

(*Received* 8 *November* 2015; *revised* 21 *April* 2016; *in final form* 25 *April* 2016)

**Abstract—Longitudinal motion of the artery, a cyclical, bidirectional movement of the wall in the long axis of the artery, has recently gained interest in the characterization of artery function. The aim of this study was to evaluate longitudinal motion in patients with internal carotid atherosclerotic plaques. Speckle tracking ultrasound was used to assess common carotid artery wall motion in 12 patients with carotid plaque causing either moderate (50%–79%) or severe (80%–99%) stenosis based on the North American Carotid Endarterectomy Trial, and 23 healthy participants. Although healthy individuals were found to have a retrograde wall motion pattern, a distinct anterograde pattern was noted with plaque presence. Importantly, patients with severe plaque stenosis had greater anterograde motion (0.53 ± 0.36 mm) than those with moderate stenosis (0.17 ± 0.15 mm) ($p < 0.05$), likely owing to high wall shear stresses associated with greater peak systolic velocities at the site of stenosis (severe: 342.0 ± 99.4 cm/s, moderate: 177.5 ± 31.2 cm/s, $p < 0.01$). There were no differences in peak systolic velocities at plaque-free segments between plaque groups (severe: 80.2 ± 24.8 cm/s, moderate: 92.7 ± 23.0 cm/s). Blood flow at stenotic areas better predicted motion than plaque-free segments. We conclude that the presence of carotid plaque can have significant influence on longitudinal motion, with significantly greater anterograde displacements with increased stenosis. Future studies are needed to further investigate carotid artery wall mechanics. (E-mail: stella.daskalopoulou@mcgill.ca)** 

*Key Words:* **Speckle tracking, Ultrasound, Carotid artery, Arterial wall, Longitudinal motion, Peak systolic velocity, Plaque, Atherosclerosis.**

## INTRODUCTION

Ultrasound imaging provides a non-invasive approach to visualization of the morphology and function of the arterial system. Layers of the artery are seen as double-line layers of hyper-echoic tissue (appear bright white on ultrasound) that represent the boundary between the lumen–intima and media–adventitia. This distinct pattern allows for easy and reliable tracking of motion of the arterial wall in the radial direction. Radial direction movements indicate distension of the artery and can be used to assess functional changes in the arterial wall, such as arterial stiffness (Failla et al. 1997; Gotschy et al. 2013; Laurent et al. 2001). Evaluation of such mechanical properties is important because functional changes occur early in vascular remodeling, often before structural changes occur, and therefore can be used for the early detection of vascular disease (Alva et al. 1993).

In recent years, advances in ultrasound imaging have permitted capture of the motion in the arterial walls with higher spatial resolution to elucidate motion in the long axis of the artery, parallel to the direction of blood flow. Speckle tracking techniques can now measure longitudinal motion, indicating a pattern that is cyclical, bidirectional and stable over time, with magnitudes equal to or greater than those of radial distension (Ahlgren et al. 2012; Cinthio et al. 2006; Tat et al. 2015). Current evidence suggests that the bidirectional pattern could be the result of two opposing pulsatile forces acting on the arterial wall: blood flow shear stresses and ventricular coupling. In healthy individuals, there is an initial displacement of the wall in the retrograde direction (opposite the blood flow direction), which may be caused by the descent of the ventricular base in systole, pulling the aorta inferior and its artery branches retrograde (Simonson and Schiller 1989). By late systole to early diastole, the wall reverses in direction and moves

Address correspondence to: Stella S. Daskalopoulou, Department of Medicine, McGill University, McGill University Health Centre, Montreal General Hospital, 1650 Cedar Avenue, C2.101.4, Montreal, Quebec, H3G 1A4, Canada. E-mail: stella.daskalopoulou@mcgill.ca

in the anterograde direction of blood flow, back to its resting position. A combination of forward blood flow shear stress and elastic recoil may be responsible for this axial stress (Cinthio et al. 2006).

Previous studies have reported a reduction in longitudinal motion of the carotid artery wall in populations at risk of cardiovascular disease (*e.g.,* with periodontal disease, diabetes and spinal cord injury) compared with healthy individuals (Tat et al. 2015; Zahnd et al. 2011, 2012). This reduction in longitudinal motion was independent of traditional cardiovascular risk factors, such as pulse wave velocity, local distensibility or carotid intima–media thickness (cIMT) (Tat et al. 2015; Zahnd et al. 2012). However, no evidence currently exists on the effect of atherosclerosis on local longitudinal motion of the carotid artery wall.

Atherosclerosis involves narrowing of the artery lumen from the buildup of fat and cholesterol, which forms a plaque. Ultrasound imaging has widely been used to examine the effect of plaques on artery flow mechanics and has considerably advanced our understanding of the pathogenesis of atherosclerotic plaque (Akkus and Ramnarine 2010; Makris et al. 2010; Malek et al. 1999; Sui et al. 2015). For example, plaque increases blood flow velocities and alters flow patterns upstream and downstream of the plaque site (Grant et al. 2000; Hunink et al. 1993; Nicolaides et al. 1996; Robinson et al. 1988; Sui et al. 2015). Upstream segments are associated with high peak velocities and large wall shear stresses that may promote plaque instability, leading to cap rupture and hemorrhage (Lovett and Rothwell 2002). Conversely, downstream, low, oscillatory flow may provide an "atherogenic" environment necessary for plaque growth (Davies et al. 2013). Given that plaque presence has such a profound effect on hemodynamics, a principal force in the axial direction, it is important to investigate whether plaque presence can also influence longitudinal motion.

Using ultrasound speckle tracking, the present study aimed to assess the longitudinal motion of the common carotid artery (CCA) wall in healthy patients and patients with carotid atherosclerotic plaques. We hypothesize that the longitudinal motion of the CCA in patients with plaques will differ from that in healthy patients and that plaques will disrupt longitudinal motion patterns according to the severity of stenosis.

## METHODS

*Participants*

Patients with carotid atherosclerotic plaques in the internal carotid artery causing ≥50% stenosis (hemodynamically significant) were recruited from the Vascular Surgery Division at the Royal Victoria Hospital and Jewish General hospital, McGill University, Montreal, Canada. A plaque was defined as a focal structure encroaching into the arterial lumen and meeting any of three criteria: (i) size ≥0.5 mm; (ii) thickness value 50% of the surrounding cIMT; (iii) thickness >1.5 mm as measured from the media–adventitia interface to the intima–lumen interface (Touboul et al. 2006). Based on the North American Carotid Endarterectomy Trial (NASCET) criteria, carotid stenosis of the plaques was categorized as moderate (50%–79%) or severe (80%–99%) using blood flow velocities measured at the plaque site with Doppler ultrasound (Ferguson et al. 1999; Nicolaides et al. 1996; Yan et al. 2009); peak systolic velocities between 150 and 249 cm/s were included in the moderate stenosis group, and velocities ≥250 cm/s were considered severe stenosis.

A medical history was obtained from each participant through questionnaires and medical records. Information was collected on cardiovascular risk factors, such as smoking history, diabetes, hypertension, lipid profile and medication use. Additionally, anthropometric variables (height, weight, body mass index) and sitting blood pressure in triplicate were obtained according to guidelines (Daskalopoulou et al. 2015).

Patients selected for our healthy group did not have a history of diabetes, hypertension or hypercholesterolemia; were not taking any medications associated with these clinical entities; and had no evidence of atherosclerosis. Healthy participants were not matched to the patients with carotid plaques, as our purpose was not to compare the two groups, but rather to study the longitudinal motion pattern of the CCA wall in the healthy state and in the disease state using these distinct populations. This study was approved by the institutional review board of McGill University, and informed consent was obtained from all participants.

*Image acquisition*

The iu22 (Philips, Andover, MA, USA) echocardiography console and linear 9- to 3-MHz probe were used to image the carotid artery based on a standardized protocol, as previously described (Doonan et al. 2013, 2014). An experienced vascular ultrasonographer performed a bilateral carotid examination of the far wall using duplex ultrasound (with color images). Cine loops were synchronized with electrocardiographic recordings of the cardiac cycle to define systole and diastole.

The probe was positioned near the carotid bifurcation in both population groups (healthy and diseased) to approximately represent the same segment of the artery in our speckle tracking analyses. For healthy patients, the probe was positioned on the long axis of the left carotid artery 2 cm proximal to the bifurcation site. In