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ABSTRACT
Although blood flows are mostly laminar, transition to turbulence and flow separations are observed at curved vessels, bifurcations, or constrictions. It is known that wall-shear stress plays an important role in the development of atherosclerosis as well as in arteriovenous grafts. In order to help understand the behavior of flow separation and transition to turbulence in post-stenotic blood flows, an experimental study of transitional pulsatile flow with stenosis was carried out using time-resolved particle image velocimetry and a microelectromechanical systems wall-shear stress sensor at the mean Reynolds number of 1750 with the Womersley number of 6.15. At the start of the pulsatile cycle, a strong shear layer develops from the tip of the stenosis, increasing the flow separation region. The flow at the throat of the stenosis is always laminar due to acceleration, which quickly becomes turbulent through a shear-layer instability under a strong adverse pressure gradient. At the same time, a recirculation region appears over the wall opposite to the stenosis, moving downstream in sync with the movement of the reattachment point. These flow behaviors observed in a two-dimensional channel flow are very similar to the results obtained previously in a pipe flow. We also found that the behavior in a pulsating channel flow during the acceleration phase of both 25% and 50% stenosis cases is similar to that of the steady flow, including the location and size of post-stenotic flow separation regions. This is because the peak Reynolds number of the pulsatile flow is similar to that of the steady flow that is investigated. The transition to turbulence is more dominant for the 50% stenosis as compared to the 25% stenosis, as the wavelet spectra show a greater broadening of turbulence energy. With an increase in stenosis to 75%, the accelerating flow is directed toward the opposite wall, creating a wall jet. The shear layer from the stenosis bifurcates as a result of this, one moving with the flow separation region toward the upper wall and the other with the wall jet toward the bottom wall. Low wall-shear stress fluctuations are found at two post-stenotic locations in the channel flow—one immediately downstream of the stenosis over the top wall (stenosis side) inside the flow separation region, and the other in the recirculation region on the bottom wall (opposite side of the stenosis).

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I. INTRODUCTION
Blood flows are mostly laminar, which helps maintain stable hemodynamic environments and normal physiological activities. However, conditions can be easily disturbed by curved vessels, bifurcations, or constrictions, where vascular diseases are usually found. Atherosclerosis develops where wall-shear stresses are low or change rapidly in time or space (Berger and Jou, 2000 and Caro, 2009). Ku (1997) also expressed the importance of wall-shear stress and unsteady phenomena in the cardiovascular system, believing that they play important roles in the diagnosis, understanding, and treatment of cardiovascular diseases. The diseases are localized to specific sites where fluid mechanical parameters deviate from their normal patterns (Farb et al., 2014). This may be due to the fact that turbulent wall-shear stress fluctuations greatly contribute to the endothelial cell turnover (Davies et al., 1986). In other words, the endothelium that lines the interior surface of blood vessels is an important participant in biology and pathobiology in the cardiovascular system (Nerem, 1993), where the wall-shear stress regulates physiologic and pathologic aspects of endothelial function (Davies, 2009). High wall-shear stress induces the atheroprotective gene
expression profile, while low wall-shear stress stimulates the atherosclerotic phenotype. Endothelial responses are also sensitive to oscillation and reversal of the wall-shear stress (Malek et al., 1999).

More than 50 years ago, Caro et al. (1969) showed that the fatty streaking and early plaques can be found at the arterial wall with low wall-shear stress, while it was demonstrated that the development of lesions can be inhibited in areas where the local wall-shear stress is high (Caro et al., 1971). These observations led these researchers to conclude that the fluid mechanics in these locations has a controlling as well as inhibiting effect of atherogenesis. Using laser Doppler anemometry (LDA) Friedman et al. (1981) measured a realistic pulsatile flow through a cast of aortic bifurcation from a patient with atherosclerosis, where they were able to show a strong negative correlation between the wall-shear rate and the intimal thickness of the diseased vessel wall. A similar negative correlation between the wall-shear stress and the atherosclerotic intimal thickness was also observed by Wootton and Ku (1999). Using quantitative angiography Gibson et al. (1993) found that low wall-shear stress was significantly correlated with an increased rate of atherosclerosis progression. Positive correlation between the low and oscillating wall-shear stress and the plaque location was obtained by Zarin et al. (1983) and Ku et al. (1985) who measured pulsatile flow velocities using LDA in a human carotid bifurcation model. More recently, Tada and Tarbell (2005) carried out a numerical simulation of a pulsatile flow through a compliant model of a bifurcation at the common carotid arteries. They revealed a large negative stress phase angle (SPA) between the circumferential stress of the blood vessel and the wall-shear stress where atherosclerotic plaques are localized.

Existence of turbulence in large arteries has been confirmed through in vivo tests. For example, velocity waveform in descending thoracic aorta of an adult dog was measured using hot-film anemometry, which showed developing turbulence (Seed and Wood, 1971). However, the duration of supercritical Reynolds number flow in peak systole was rather short, therefore turbulent disturbances damped out during diastole. This study was followed by Nerem and Seed (1972) and Nerem et al. (1972) who concluded that flow instabilities in the pulsatile aortic flow were responsible for the observed flow disturbances. The turbulence in blood flows can also contribute to a formation of thrombi (Stein and Sabbah, 1974). Here, two arteriovenous shunts were established in dogs from the femoral artery to the contralateral femoral vein, one of which contained a turbulence-producing device. The results suggested that the turbulent motion seemed have made the blood cells in contact with the walls for a longer period of time to accelerate the formation of thrombi. Stein and Sabbah (1976) also measured the blood flow in the ascending aorta of humans using a catheter-tip hot-film probe. For normal humans, the flow was highly disturbed during peak ejection, while those with aortic valvular disease showed turbulent flow during nearly the entire period of ejection. However, turbulence was observed throughout the ascending aorta and in the innominate artery in humans with aortic stenosis. In vivo blood flow measurements were also made by Giddens et al. (1976) in the descending thoracic aortas of anesthetized dogs using a hot-film anemometer, where flow disturbances in the velocity waveforms that are indicative of turbulence were found in the artery only with very minor constrictions. These data were analyzed further by Khalifa and Giddens (1978), who concluded that mild to moderate localized vascular disease can be detected by turbulence in blood flow. Falsetti et al. (1983) also conducted a hot-film study in ascending aorta of an open-chest dog. They observed turbulence-like disturbances at the peak velocity, which existed throughout the deceleration phase. Lieber and Giddens (1990) used LDA to examine the post-stenotic flow in an aorta model of the cyromogulus monkey and found significant velocity disturbances due to turbulence during acceleration, peak, and deceleration cycles. Particle image velocimetry (PIV) measurement of diseased carotid-artery models by Katayati et al. (2014) showed an increased level of turbulent intensity with an increasing degree of stenosis, where an eccentricity in stenosis gave a further increase in turbulent intensity. Using high-resolution numerical simulations Grinberg et al. (2009) observed a transitional and intermittent flow between laminar and turbulent states in a stenosed internal carotid artery (ICA). Here, the model was reconstructed using MRI images, while the boundary conditions were obtained by in vivo velocity measurements. High-frequency velocity fluctuations associated with transition to turbulence were observed by Mancini et al. (2019) who carried out large-eddy simulation (LES) of flow in the post-stenotic, patient-specific internal carotid artery (ICA). The flow was already unstable upstream of the stenosis, which broke down as the flow accelerated through the stenosis.

Wall-shear stress also plays an important role in arteriovenous (AV) grafts, a surgical connection between an artery and a vein for hemodialysis treatments (Haruguchi and Teraoka, 2003). Due to high pressure gradient across the two vessels, AV grafts have high flow rates with the mean Reynolds number up to 1800 (Loth et al., 2008). The turbulence created during flow transition and its fluctuating wall-shear stress in AV grafts have pronounced effects on the morphology and biochemical signaling of endothelial cells (ECs) (Chiu and Chien, 2011 and McCormick et al., 2012). This leads to intimal thickening which is a frequent cause of graft failure due to the blockage of blood flow (Rittgers et al., 1978, Loth et al. (2002) examined the relative contribution of wall-shear stress to the induction of intimal hyperplastic thickening in AV grafts. It was shown that the intimal hyperplastic pannus migrating from the graft’s suture line was enhanced by reduced levels of wall-shear stress at the graft/host artery interface, agreeing with the findings of Bassiony et al. (1992). An ultimate cause of graft failure, however, is the thrombus formation. It was demonstrated by Binns et al. (1989) that oversized grafts can create low wall-shear stress, leading to intimal thickening. On the other hand, small grafts produced high wall-shear stress, leading to poor graft patency due to thrombosis. Lei et al. (1997) demonstrated a possibility in designing grafts that can significantly reduce wall-shear stress gradient, thereby reducing the propensity for thrombosis. Late restenosis and stent placement are linked with neoointimal hyperplasia (Duraiswamy et al., 2007), where the low wall-shear stress regions in the arteries are associated with maximal intimal thickness (Cunningham and Gotlieb, 2003). This is because the re-establishment of the endothelium is strongly affected by the stent configuration (Sprague et al., 1997).

The aim of this study is to improve our understanding of the behavior of flow separation and transition to turbulence in post-stenotic blood flows including those involving grafts. We also want to know how the intermittent turbulence in a pulsatile flow affects the wall-shear stress distribution and fluctuations in such flow configurations. In order to attain this aim we designed and executed an experimental study of a generic (non-patient specific) flow configuration based on a two-dimensional channel with an asymmetric stenosis of a
sinusoidal profile. Time-resolved PIV technique was used to obtain flow velocity and vorticity fields at different phases of pulsation. We also used a microelectromechanical systems (MEMS) based floating element wall-shear stress sensor to directly measure wall-shear stress distribution and fluctuations around the stenosis to complement the velocity measurements to understand the unsteady flow field created by the stenosis. This paper begins with a review of relevant work, which is followed by experimental results and discussions on (1) the steady flow, (2) pulsatile flows without stenosis and (3) pulsatile flows with stenosis through a two-dimensional channel. Concluding remarks will then follow with a short remark on physiological implications.

II. BACKGROUND
A. Transitional behavior of pulsating flows

The behavior of transition from laminar to turbulent flow in a pipe or channel changes dramatically when the flow becomes unsteady. The laminar-to-turbulent transition behavior in a sinusoidally pulsatile flow through a circular tube was experimentally studied by Yellin (1966). At the maximum velocity, the pulsatile flow was laminar and undisturbed, suggesting that the large instantaneous Reynolds numbers during systole do not necessarily result in transition to turbulence. A similar study was carried out by Stettler and Hussain (1986) using LDA. Here, periodic flow modulation produced longitudinally periodic turbulent cells, called patches, whose length increased with an increase in the modulation magnitude. Shermer et al. (1985) investigated into the laminar pulsating flows in a straight pipe at the mean Reynolds number of 4000, where it was concluded that RMS values of velocity fluctuations are not affected by pulsations in both laminar- and turbulent-flow regimes. Lodahl et al. (1998) observed transition to turbulence at a mean Reynolds number that was greater than the critical Reynolds number for steady pipe flows. The wall-shear stress increased by four times when the flow was wave-dominated. Peacock et al. (1998) conducted an experiment in a pipe flow to show that the increase in the critical Reynolds number due to flow pulsation was not only due an increase in the Womersley number, but was also influenced by a reduction in the Strouhal number. Numerical simulation of pulsatile turbulent channel flow was carried out by Scotti and Piomelli (2001), who showed that the mean velocity profile within twice the Stokes layer thickness from the wall remains unaltered, while the logarithmic layer shifted up- and down-wards during the flow pulsation. Trip et al. (2012) carried out an experimental study of a transitional, sinusoidal pulsatile flow in a straight pipe using PIV technique. The transition occurred in the region of $Re = 2250–3000$, where isolated turbulent structures similar to puls in steady flow transition were observed. The turbulence intensity was reduced during the accelerating phase while it increased during deceleration. Xu et al. (2017) carried out experiments in a sinusoidal-shaped pulsatile pipe flow to observe that the critical Reynolds number increased with an increase in the Womersley number until $W_o = 2.5$. Then the critical Reynolds number reduced with a further increase in the Womersley number until it reached to $W_o = 12$. These results were confirmed by Xu and Avila (2018) who carried out a numerical simulation with a low pulsation amplitude. The effect of the pulsatile waveform in a pulsatile pipe flow was investigated by Brindise and Vlachos (2018), who demonstrated that the waveform with a longer deceleration induced an earlier onset of transition, while a longer acceleration delayed the transition.

B. Pulsatile flows with stenosis

The transition to turbulence is accelerated by the presence of severe stenosis, but its behavior is influenced by the size, shape, and location of the stenosis. Young and Tsai (1973) used a hot-film sensor to find that the pulsating flow was more stable than the steady flow for mild (56%) constrictions. For severely constricted (89%) cases, the pulsatile flow through a tube was slightly less stable. A pulsatile poststenotic flow through a straight tube was investigated using LDA (Ahmed and Giddens, 1984) at the mean Reynolds number of 600 and the Womersley number of 7.5, where a strong turbulence was observed in a flow separation region downstream of the 75% stenosis. Pulsatile flow through constricted tubes was experimentally investigated by Ojha et al. (1989) at the mean Reynolds number of 575, where vortical and helical structures were observed during the deceleration phase of the flow cycle close to the reattachment point, where the wall-shear stress fluctuated intensely. Experiments in a two-dimensional stenotic channel were conducted by Asimina (2004) to investigate flow instabilities associated with pulsatile flows, where a central vortex was created during the deceleration phase, resulting in a turbulent burst downstream. Sherwin and Blackburn (2005) and Griffith (2009) examined the behavior of transition to turbulence of an idealized vascular pulsatile flow through a tube with a 75% stenosis at the Reynolds number up to 750. The pipe flow became unstable through a subcritical period-doubling bifurcation involving alternating tilting of the vortex rings ejected from the throat. Varghese et al. (2007) carried out a direct numerical simulation (DNS) study of pulsatile flows through a 75% stenotic tube. The starting vortex formed during the acceleration phase broke down at the peak of the pulsation, forming a turbulent spot downstream. In the early part of deceleration there was intense turbulent activity within the spot. A DNS study of transitional flow in a stenosed, subject-specific carotid bifurcation was carried out by Lee et al. (2008), who confirmed the existence of transitional flow during systole and laminar flow during diastole. Significant turbulence with high spatial gradients of wall-shear stress was also observed in the flow separation region. Molla et al. (2012) observed that the energy spectra of pulsatile channel flows with stenosis deviated significantly from the $-5/3$ slope expected for turbulent flows to reflect its transition status. Jain (2016) carried out a numerical study of transitional pulsatile stenotic flow using Lattice Boltzmann method at the mean Reynolds number of 600, where coherent vortices in the post-stenotic regions were observed, which evolved into turbulence during the deceleration phase of the pulsatile cycle. The effect of viscoelastic wall in blood flows was numerically investigated by Nejad et al. (2018), who showed that flow fields remain unchanged for the stenosis less than 50%. For the 75% stenosis, however, the axial velocity is increased by 2% over the viscoelastic artery. Choi et al. (2018) studied pulsatile flows with deformable stenosis models, where they found that the deformed stenosis enhanced the jet deflection, increasing the turbulent kinetic energy production near the opposite vessel wall. In a CFD study of a pulsatile pipe flow, Freidoonimehr et al. (2020) showed that the flow separation starts from the asymmetric stenosis side and spreads to the opposite side.
C. Wall-shear stress in pulsatile flows

Direct measurements of local wall-shear stress and its fluctuations are extremely difficult especially for unsteady conditions. As such, wall-shear stresses are often obtained through numerical simulation or estimated from indirect measurements (Katritsis et al. 2007). Tuttty and Pedley (1993) numerically studied a two-dimensional unsteady flow in a stepped channel at the Reynolds number of up to 750, where they found that the wall-shear stress was always low immediate lee of the step, although it depended on the flow parameters and the step geometry. A pulsatile flow through a circular tube with 75% stenosis was studied by Deplano and Siouffi (1999) using pulsed Doppler ultrasonic velocimetry. They observed low and oscillating wall-shear stress in the downstream of stenosis, while high wall-shear stress value was found at the throat. Long et al. (2001) used a numerical simulation to study the physiologically realistic pulsatile laminar pipe flow at a mean Reynolds number of 300. They found a positive peak wall-shear stress at the non-stenotic side of the pipe as a result of skewed jet flow over the asymmetric stenosis. Large wall-shear stress oscillations were found near the reattachment locations of the separated flow downstream the stenosis. A similar study was conducted by Razavi et al. (2011), who observed a reduction in wall-shear stress in a large recirculation region downstream of the stenosis. The maximum value of oscillatory shear index was found at the reattachment point. Stroud et al. (2002) carried out a CFD study of a flow through a severely stenotic carotid artery bifurcation at the Reynolds number of up to 900, where it was found that both dynamics pressure and wall-shear stress were very high at the stenosis throat. The wall-shear stress gradient was also strong along the throat. A pulsatile flow in a two-dimensional channel with an asymmetric stenosis was numerically investigated by Tian et al. (2013) at the Reynolds number of up to 400, where they found that the flow separation region behind the 80% stenosis was elongated to nearly 12 times the channel half-width. Immediately downstream, another recirculation region was observed over the non-stenotic wall. Wall-shear stress was very low in these recirculation regions. Usmani and Muralidhar (2016) studied a pulsatile flow through a compliant stenosed tube using PIV technique at the peak Reynolds number of between 300 and 800. During the deceleration phase of the flow cycle the flow separation point shifted upstream of an asymmetric stenosis, whereas the reattachment points moved downstream. The wall-shear stress was maximum at the throat, reducing downstream in the recirculation region. Mahalingam et al. (2016) carried out a CFD study of pulsatile flow through a coronary artery model with varying degree of stenosis. As a result of transition to turbulence, the wall-shear stress and oscillatory shear index increased from the laminar value by more than 10% for the 50% stenosis case.

III. EXPERIMENTAL SETUP AND PROCEDURE

A. Experimental facility

Experimental setup for this investigation is shown in Fig. 1. The test channel is 9 mm high, 100 mm wide and 5000 mm long, whose measuring section is located at 3000 mm from the inlet, ensuring that the channel flow is fully developed. The channel is made of clear cast Perspex for PIV measurements. The surface roughness of the channel is less than Ra = 0.05 μm (Lepore et al., 2008), which corresponds to the roughness Reynolds number of 0.01. Therefore, it has a negligible influence on the channel flow. The effect of the finite aspect ratio (11.1) of the channel is less than 0.6% in both the mean velocity and the turbulence intensity (Vinuesa et al., 2014). Dry airflow inside the channel is driven by a compressed air supply system and measured by a flow meter (Tokyo Meter QFS-400 with a measurement accuracy of ± 3% FS). A honeycomb and fine screens are installed at the inlet to reduce flow disturbance and to improve the flow uniformity. A programmable servo valve (FESTO MPYE-5–1/8LF) controlled by
LabVIEW software is used to generate pulsatile airflow, simulating the blood flow, see Fig. 1. Here, 10 phases (A to J in Fig. 1) in the pulsatile waveform are chosen for detailed PIV measurements. This waveform is close to a patient-specific waveform that was derived from MRI by Li et al. (2009). The maximum velocity is found at $t/T = 0.06$, where the wall-shear stress also reaches to a maximum value. The mean Reynolds number of the pulsatile flow based on the channel height and the bulk velocity is 1750, while the peak Reynolds number is 3200, which is close to that of blood flow in aorta during ventricular systole (Manna et al., 2015). The Womersley number, a dimensionless pulsatile flow frequency, is set to $W_0 = h \sqrt{\omega/\nu} = 6.15$ (1.12 Hz) based on the channel height in the present investigation. Here, $\nu = 15.1 \times 10^{-6}$ m$^2$/s is the kinematic viscosity of air at the room temperature of 20 °C.

Stenoses are modeled by a sinusoidal function, which is representative of pathology specimens (Ahmed and Giddens, 1984). The profile is given by

$$\frac{y(x)}{h} = \delta_s \cos \frac{x \pi}{2h}, \quad -h < x < h,$$

where $h$ is the channel height and $\delta_s$ is the maximum channel-height reduction, representing mild (25%), intermediate (50%), and severe (75%) cases. Stenoses are 2$h$ long and are manufactured by a 3D printer (Zortrax M300). They are polished using fine (P1000) sandpapers before attaching to the top wall of the channel, as indicated in Fig. 1.

**B. Wall-shear stress sensor**

A MEMS-based floating element, wall-shear stress sensor with capacitive sensing is commonly used for aerodynamic applications due to its high sensitivity and measurement precision (Chandrasekharan et al., 2011 and Lv et al., 2013). Small size and high dynamic performance of the sensor make it possible to access local wall-shear stress and its fluctuations. The wall-shear stress sensor used in the current investigation consists of a floating element for sensing the shear-force, four folded tethers for storing elastic energy to keep the floating element in balance by resisting the applied shear force, and comb-like capacitors for sensing the position of the floating element [Fig. 2(a)]. The working principle of the sensor can be described as follows. When the sensor is exposed to a flow, the floating element moves along the direction of wall-shear stress, changing the distance between comb fingers together with the capacitance of capacitors. One can use an external circuit to read the capacitance difference, which is linearly proportional to the applied wall-shear stress.

We have used this floating element wall-shear stress sensor in the linear operating range of −15 Pa to +15 Pa, whose resolution is better than 0.03 Pa. The designed natural frequency of the sensor is 3500 Hz, indicating that the response frequency can reach to at least 1500 Hz (Caruntu and Knecht, 2011). The sensor chip was fabricated by a 4-inch SOI (silicon on isolation) wafer with a device layer of 40 μm thickness. The total upper surface area of floating element and movable fingers is about 1 mm$^2$, which determines the spatial resolution of the sensor. Details of the sensor’s structural design, fabrication process, and high-robust package can be found in Ding et al. (2017 and 2018).

The floating element, wall-shear stress sensor was calibrated in a fully developed channel flow without stenosis. Here, wall-shear stress was obtained from the pressure gradient along the channel (Hussain and Reynolds, 1975),

$$\tau_w = -\frac{h dp}{2 dx},$$

where $h$ is the channel height and $dp/dx$ is the pressure gradients along the channel. The wall pressure measurements along the channel were conducted by using a digital manometer (Furness Controls FCOS520) with a resolution of 0.001 Pa. By changing the direction of installation, both positive and negative outputs of the sensor were obtained with a range of wall-shear stress from 0 to 0.333 Pa. Figure 3 gives the result of static calibration of the sensor and the corresponding pressure gradients. This gives the static sensitivity of the sensor of 13 mV/Pa
with the $R^2$ value for a straight-line fit greater than 0.99. The MEMS floating element, wall-shear stress sensor was flush-mounted to the inside wall of the channel (Fig. 1) for both steady and pulsatile wall-shear stress measurements. Wall-shear stress was sampled at 1 kHz using a 12-bit analogue-to-digital converter.

### C. Particle image velocimetry

Velocity fields in the channel were obtained by using a time-resolved Dantec PIV system, which consists of a Litron LDY 302-PIV Nd:YLF laser with 15 mJ per pulse, a Vision Research Phantom V12 high-speed camera with a QUESTAR QM1 short mount long-distance microscope, a National Instrument 80N77 Timer box and a dedicated PC. Olive oil seeding particles with a nominal diameter of $1 \mu m$ were introduced at the inlet of the channel using a TSI 9307 oil droplet generator. The PIV measurements were synchronized with a wave-form generator in order to take the first image at the beginning of each period of the pulsatile flow.

PIV measurements were made on the streamwise ($x-y$) plane through the central line of the channel where the laser light sheet was around $1 \text{ mm}$ in thickness. 70% of the full laser power was used to generate light sheet with the field of view of $80 \text{ mm} \times 32 \text{ mm}$, which was captured by the high-speed camera. However, the useful field of view was only about $80 \text{ mm} \times 9 \text{ mm}$ because of the size of the channel. The repetition rate of PIV camera was 10 Hz for steady flows and 100 Hz for pulsatile flows, respectively. The sampling time was 10 s for steady flow and 22.3 s (25 cycles) for pulsatile flows, thus obtaining 2500 image pairs in each measurement with a time delay of $50 \mu s$ between image pairs. The number of ensemble-averaged images used in our experimental investigation is 25, which is similar to that of Vaghese et al. (2007) and Jain (2016) in their DNS studies.

PIV data processing was carried out by using Dantec DynamicStudio 4.10. Adaptive PIV was used to calculate velocity field, where the minimum and maximum interrogation areas were set to 16 and 32 pixels in $x$-direction, and 8 and 16 pixels in $y$-direction, respectively, both with a 50% overlap. Assuming that the primary error of PIV measurements comes from the shift distance $D$ of seeding particles and the interval time $T$ between image pairs, the uncertainty of velocity measurements can be given by the following expression (Kline and McClintock, 1953):

$$U_e = \sqrt{\frac{D_e^2}{T^2} + \left( \frac{D}{T} \right)^2 T^2},$$

where $D_e$ is the uncertainty in particle shift distance, which is about 0.35 pixel in our measurements (with a corresponding length of 0.02 mm). The uncertainty in the interval time $T_e$ between image pairs is negligible. This gives $U_e = 0.4 \text{ m/s}$, providing an uncertainty of PIV measurements of $U_e/U = 4.8\%$, which is slightly lower than that estimated by Westerweel (1997).

### IV. RESULTS AND DISCUSSIONS

#### A. Steady flows with and without stenosis

In order to establish the accuracy of our PIV measurements in a two-dimensional channel flow, we examine the results of velocity profiles and flow patterns under steady flow conditions with and without stenosis. For a non-stenotic case, the Reynolds number based on the mean bulk velocity and the channel height is 4000, with a corresponding wall-shear stress of 0.21 Pa measured by a MEMS sensor. This result is about 23% lower than the value estimated from the empirical formula $\tau_w = 0.0376 Re^{1/6}$ by Patel and Head (1969) for fully developed turbulent channel flows, suggesting that the channel flow is still under transition at this Reynolds number. Dean (1978) has carried out a comprehensive survey of skin-friction correlations to conclude that this empirical formula is only valid for the Reynolds number greater than 6000. When the Reynolds number is less than 6000, the channel flow is still in a stage of transition to turbulence, therefore the skin-friction coefficient is overestimated (Davies and White, 1928).

Figures 4 and 5 show the mean velocity and turbulence intensity profiles for steady flows without stenosis, which are presented in inner and outer scale, respectively. Our logarithmic velocity profile is shifted slightly upwards [see Fig. 4(a)], reflecting the late transitional flow at the Reynolds number of 4000 (Patel and Head, 1969 and Seki and Matsubara, 2012). The transitional behavior of the mean velocity profile is also evident in Fig. 5(a) which is presented in outer scale.
Turbulent intensity profiles are slightly confusing, however. When $u'$ and $v'$ are presented in inner scale [see Fig. 4(b)], our results are slightly lower than DNS results of Kim et al. (1987) obtained at the Reynolds number of 6600. When our results are presented in outer scale [see Fig. 5(b)], however, they give a reasonable agreement with Kim et al. (1987). Here, we must consider the effect of the Reynolds number in addition to the transitional effect in order to explain these results (Gad-el-Hak and Bandyopadhyay, 1994 and Fernholz and Finley, 1996). Indeed, Wei and Willmarth (1989) demonstrated that the turbulence intensities presented in inner scale are increased with an increase in the Reynolds number, as shown in Fig. 4(b). However, the Reynolds number effect on turbulence intensities is negligible if they are presented in outer scale, see Figure 5(b).

The main characteristics of steady flows around a stenosis are shown in Figure 6, where we present streamlines, velocity profiles, streamwise turbulence intensity contours and vorticity contours at the Reynolds number of 3200. Here, flow separation regions, which can be identified by the shear layer with vorticity concentration, are marked by thick blue lines, following a streamline from the separation point over the stenosis to the reattachment point on the top wall. To mark recirculation regions over the bottom wall, which can be identified by negative velocity profiles close to the wall, we follow a streamline from the upstream separation point to the reattachment point downstream. Mean bulk velocity at the throat of stenosis reaches to 1.45, 1.90, and 3.88 times of the corresponding upstream values for the 25%, 50%, and 75% stenosis, respectively. It is interesting to note that the maximum velocity is found at a short distance downstream of the stenosis, rather than at the throat. This is due to the inclined velocity vector at the throat, expanding the flow separation region immediately downstream of the stenosis. The reattachment length on the upper wall increases with an increase in stenosis, as shown in Fig. 6(a), qualitatively agreeing with the result of Ghali et al. (1998). The reattachment point is found at $x/h = 1.9$ and $x/h = 4.2$ for the 25% and 50% stenosis, respectively. For the 75% stenosis, however, the reattachment point is outside the field of view of PIV measurements, i.e., $x/h > 5$.

A small recirculation region is found for the 25% stenosis over the bottom wall at $x/h = 1.5–3$ for the 25% stenosis. A similar recirculation region was observed by Armaly et al. (1983) in a channel flow on the opposite side of backward-facing step, which is due to strong adverse pressure gradient created by the step. However, the size of this recirculation region is reduced when the stenosis is increased to 50%, and completely disappears at 75% stenosis. This is due to the stenosis-

![Figure 4](image1.png)

**FIG. 4.** Mean velocity profile (a), and RMS velocity fluctuations (b) normalized by friction velocity $u^*$ at $Re = 4000$ for non-stenotic steady flow. The DNS results by Kim et al. (1987) are also plotted for comparison.

![Figure 5](image2.png)

**FIG. 5.** Mean velocity profile (a), and RMS velocity fluctuations (b) normalized by mean bulk velocity $U_0$ at $Re = 4000$ for non-stenosis steady flow. The DNS results by Kim et al. (1987) are also plotted for comparison.
induced accelerating flow (jet-like flow) becomes more influential in the post-stenotic flow.

For the 75% stenosis, the accelerating flow is directed toward the bottom of the channel to create a wall jet, interacting with the shear layer. This increases the turbulence level downstream of the stenosis. The maximum streamwise turbulence intensity \((u'/u_0)\) for the 75% stenosis is 2.5 and 5 times higher than that for the 50% and 25% stenosis, respectively.

B. Pulsatile flows without stenosis

To validate our PIV measurements in pulsatile flows, we examine the results in a two-dimensional channel without stenosis. Velocity profiles across the channel at different \(y\) locations and the wall-shear stress during one cycle of the pulsatile flow without stenosis are shown in Figs. 7(a) and 7(b), respectively. Here, the mean Reynolds number is 1750, while the peak Reynolds number is 3200 at \(t/T = 0.06\). It is shown here that the center velocity lags the near-wall velocity as well as the wall-shear stress, indicating that flow is dominated by inertia. It is also noted that wall-shear stress becomes close to zero or even slightly negative during the pulsatile cycle.

Mean velocity profiles and RMS intensity profiles of streamwise velocity fluctuation at selected phases of pulsation are shown in Figs. 8(a) and 8(b) in inner and outer scale, respectively. The pulsatile frequency in wall units is \(\omega^+ = \omega u_0/\nu^2 = 0.0017\), which corresponds to the time-averaged Stokes layer thickness \(\ell^+ = l\nu' / \nu = 34\). It should be noted that the Stokes-layer thickness is not constant as the pulsatile profile in this study is not sinusoidal. It reduces at the start of pulsatile cycle (points A to C in Fig. 1) when the flow is sharply accelerated. After that, the pulsatile flow decelerates until the end of the cycle, so that the Stokes layer thickness gradually increases. The mean velocity profile as shown in Fig. 8 responds to the change of the Stokes layer thickness (Scotti and Piomelli, 2001). For example, a large part of central channel flow is not affected by the pulsation at the start of the cycle \((t/T = 0.02\) to 0.06), but the pulsatile effects gradually extend to nearly the entire channel for the rest of the pulsatile cycle \((t/T = 0.18\) to 0.82). The channel flow at this Reynolds number is transitional (Seki and Matsubara, 2012), therefore the time-averaged velocity profile is not expected to fit to the logarithmic profile in Fig. 8(a) or the Blasius profile.
However, the pulsatile flow becomes momentarily turbulent in phase C ($t/T = 0.06$) as shown by an increase in the turbulence intensity near wall. This is due partly to a reduction in the Stokes layer thickness and partly to an increase in the Reynolds number associated with the accelerated flow in this phase (Lodahl et al., 1998). A similar phenomenon has been observed in a weakly turbulent blood flow inside the internal carotid artery during the systolic phase (Lee et al., 2008).

### C. Pulsatile flows with stenosis

Pulsatile flows with stenosis are studied at the mean Reynolds number of 1750 and the peak Reynolds number of 3200 (at $t/T = 0.06$), where the 25% stenotic case is discussed first. Figure 9 shows velocity profiles, streamwise turbulence intensity contours and vorticity contours of selected phases of interest. As the channel flow accelerates at the start of the pulsatile cycle in phase A ($t/T = 0.02$), a strong shear layer with concentrated positive vorticity develops from the tip of the stenosis, as shown in Fig. 9(b), increasing the flow separation region. At the same time, a recirculation region appears over the bottom wall at $x/h = 1.5$, see Fig. 9(a), which moves downstream together with the reattachment point. The recirculation region over the bottom wall is located immediately downstream of the reattachment point on the opposite wall (stenosis side), where the adverse pressure gradient becomes greater due to the expansion of flow. The development of the shear layer continues until phase F ($t/T = 0.35$), moving the reattachment point downstream. It is shown that the reattachment point moves from $x/h = 1.8$ in phase A to around $x/h = 4.2$ in phase F and then reduces back to $x/h = 3$ toward the end of the pulsatile cycle. The flow through the stenosis is always laminarized due to acceleration (Ahmed and Giddens, 1984; Sherwin and Blackburn, 2005; and Jain, 2016), but will become turbulent as it approaches to the reattachment point (Ojha et al., 1989). This is evident from the high turbulence intensity shown in Fig. 9(a). The shear layer weakens as the flow decelerates in phase E ($t/T = 0.25$). The flow behavior in a pulsating channel flow during acceleration ($t/T = 0.02–0.06$) is similar to that of the steady flow (see Fig. 6), including the location and size of post-stenotic flow separation regions. This is because the peak Reynolds number of the pulsatile flow ($Re = 3200$) which takes place at the end of acceleration ($t/T = 0.06$) is similar to the Reynolds number of the steady flow.

Figure 10 show the temporal change in wall-shear stress during the pulsatile cycle at selected locations of interest in a channel flow. Here, the ensemble-averaged wall-shear stress is indicated by red, while five individual signals (out of 25 sets of signals acquired) are shown in different colors, indicating the degree of fluctuations in wall-shear stress. Wavelet spectra are also given in Fig. 10 to show the turbulence energy distribution of wall-shear stress fluctuations at $U_1–U_3$ and $L_2–L_4$. Here, we used generalized Morse wavelets defined by $\Psi_{P, \gamma}(\omega) = U(\omega)a_P\omega^P e^{-\omega^\gamma}$ in the frequency domain $\omega$, where $U(\omega)$ is the unit step function and $a_P$ is a normalizing constant (Lilly and Olhede, 2012). The symmetry parameter and the time-bandwidth product were set to $\gamma = 3$ and $P^2 = 60$, respectively.
The negative wall-shear stress fluctuation can be seen in Fig. 10 at $U_1 (x/h = 1.5)$ and $U_2 (x/h = 2)$, which are within the flow separation region behind the stenosis (Deplano and Siouffi, 1999 and Long et al., 2001). The wall-shear stress fluctuation at $U_3 (x/h = 2.5)$ is initially positive, but will become negative later in the pulsatile cycle ($t/T = 0.45–0.6$), corresponding to the movement of flow reattachment point with an increase in $t/T$. Broadening of turbulence energy up to 80 Hz is observed in wavelet spectra between phase $B$ ($t/T = 0.03$) and phase $D$ ($t/T = 0.18$) at $U_1$, $U_2$ and $U_3$, suggesting that transition to turbulence is taking place near the flow reattachment points (Ojha et al., 1989). Transition to turbulence is also seen in wavelet spectra in the later phase $H$ ($t/T = 0.55$) of pulsatile cycle when there is a second flow acceleration, see Fig. 1. The increase in turbulence intensity during these phases of pulsatile cycle ($t/T = 0.03–0.18$) is clearly seen in PIV measurements, see Fig. 9(a).

Ku et al. (1985) introduced a concept of oscillatory shear index (OSI) to account for changing wall-shear stress direction during a flow pulsation, which can be expressed as

$$\text{OSI} = \frac{1}{2} \left( \frac{\int_0^T \tau_w(t) \, dt}{\int_0^T |\tau_w(t)| \, dt} \right).$$

The OSI takes a value between 0 to 0.5, where a larger value means a greater change in wall-shear stress direction. The maximum OSI values tend to be found at the flow separation or reattachment point, where transient effects are more significant (Razavi et al., 2011). For the 25% stenosis case, the location $U_3 (x/h = 2.5)$ is close to the mean reattachment point, where the OSI becomes as much as 0.3. The wall-shear stress at $U_1 (x/h = 0)$, at the throat of stenosis on the...
bottom wall, does not exhibit any turbulent fluctuations due to laminarized flow through the stenosis (Ahmed and Giddens, 1984; Sherwin and Blackburn, 2005; and Jain, 2016), whose temporal profile is similar to that of the bulk velocity profile as shown in Fig. 1. A similar wall-shear stress profile is observed at $L_2$ ($x/h = 1$) and $L_3$ ($x/h = 1.5$), although the wall-shear stress magnitude is reduced at these locations. The wall-shear stress at $L_4$ ($x/h = 2.5$) is nearly zero throughout the pulsatile cycle. This is due to the recirculation region being formed at this location (see Fig. 9) moving downstream with an increase in $t/T$. There is no indication of turbulence in wavelet spectra (see Fig. 10) at these locations over the bottom wall. These observations are supported by the wavelet spectra, which only shows low frequency contents below 10 Hz during the acceleration phase ($t/T = 0.03–0.18$).

The behavior of post-stenotic flow for the 50% stenosis is similar to that for the 25% stenosis case, except that the recirculation region that appear on the bottom wall is much greater, see Fig. 11(a). This is due to a greater adverse pressure gradient created behind the stenosis. A laminar shear layer developing from the tip of the stenosis immediately becomes turbulent, as shown in Fig. 11. This is due partly to high local Reynolds number and partly to a greater adverse pressure gradient, making the velocity profile more inflectional that is susceptible to flow instability. Figure 11 shows that the reattachment point moves from $x/h = 3$ in phase A to $x/h = 4.2$ in phase C and remains there for...
the rest of the pulsatile cycle, although it temporarily moves back to \(x/h = 3.6\) as the post-stenotic flow is laminarized at phase \(F\). At the same time, a large recirculation region appears over the bottom wall at \(x/h = 2.8\), see Fig. 11(a), which moves downstream together with the reattachment point over the top wall.

The wall-shear stress distribution for the 50% stenosis is also similar to that for the 25% stenosis case, except at \(U_3\) over the top wall, see Figure 12. There, the wall-shear stress is negative throughout the pulsatile cycle, confirming that the MEMS sensor positioned at \(x/h = 2.5\) is always inside the flow separation region downstream of the stenosis. The transition to turbulence is more dominant for the 50% stenosis as compared to the 25% stenosis, as the wavelet spectra indicate a greater broadening of turbulence energy up to 120 Hz during the initial acceleration phase \((t/T = 0.03–0.18)\) at \(U_1, U_2,\) and \(U_3\). The wavelet spectra also indicate the presence of turbulence at \(U_2\) during phase \(H\) \((t/T = 0.55)\) when there is the second flow acceleration. Again, there is no sign of turbulence at \(L_1\), indicating that the flow is laminarized at the throat of the stenosis \((x/h = 0)\) due to the flow acceleration (Ahmed and Giddens, 1984; Sherwin and Blackburn, 2005; and Jain, 2016). The fluctuations of wall-shear stress on the bottom wall at \(L_2\) and \(L_3\) are much less than those on the top wall \((U_1, U_2,\) and \(U_3)\), suggesting that the flow through the stenosis remains laminar in these locations. These are confirmed by the wavelet spectra, which only show low frequency contents below 10 Hz. Although the turbulence energy level is rather low, there is some indication of broadening in the wavelet spectrum at \(L_4\), where the recirculation region appears during the acceleration phase \((t/T = 0.03–0.18)\) of pulsatile cycle.

At 75% stenosis (see Fig. 13), the accelerating flow through the stenosis is directed toward the bottom wall at \(t/T = 0.02\), separating the flow upstream of the tip the stenosis (Tian et al., 2013). This pushes the shear layer down toward the bottom wall, creating a wall jet there. Interestingly, the shear layer with strong positive vorticity concentration [see Fig. 13(b)] seems to bifurcate, one along the wall jet toward the bottom wall and the other along the flow separation region toward the top wall. This behavior can be seen in the vorticity map, see Fig. 13(b), in phase \(A\) \((t/T = 0.02)\) to phase \(C\) \((t/T = 0.06)\). Figure 13(a) shows that the shear-layer bifurcating is accompanied by a large increase in turbulence intensity. With an increase in \(t/T\) during the pulsatile cycle, the upper shear layer comes down toward the center of

![Image](https://example.com/image.png)
the channel with an increase in the size of flow separation region, while the lower shear layer moves upwards as the wall-jet thickness increases. The two shear layers merge in phase \( F (t/T = 0.25) \), when the turbulence intensity is significantly reduced, see Fig. 13(a), as the flow decelerates. The flow reattachment point for the 75% stenosis always stays outside the field of view of the PIV measurement \((x/h > 5)\).

Increasing stenosis generates a higher velocity through the throat of stenosis, which creates a larger velocity gradient near the wall, resulting in a higher wall-shear stress at \( L1 \) \((x/h = 0)\) (Long et al., 2001; Razavi et al., 2011; Tian et al., 2013; and Usmani and Muralidhar, 2016), see Fig. 14. After going through the 75% stenosis, however, the wall-shear stress at the bottom wall is momentarily reduced at \( L2 \) \((x/h = 1)\) and \( L3 \) \((x/h = 1.5)\) as the accelerated flow is entrained into the flow separation region over the top wall. The flow expansion leads to an adverse pressure gradient, which accelerates transition to turbulence as seen in Fig. 13(a), resulting in the increase in turbulence at \( L2 \) and \( L3 \). Corresponding broadening of the wavelet spectra is observed in Fig. 14, although the turbulence energy of wall-shear stress fluctuation is mostly contained within 60 Hz. The wall jet over the bottom wall will then accelerate, giving rise to an increased wall-shear stress at \( L4 \) \((x/h = 2.5)\). At 75% stenosis MEMS sensors at \( U1 \) \((x/h = 1.5)\), \( U2 \) \((x/h = 2)\), and \( U3 \) \((x/h = 2.5)\) over the top wall are always in the flow separation region, therefore the wall-shear stress stays negative.
throughout the pulsatile cycle. Wall-shear stress spikes and associated wavelet spectra at $U_2$ and $U_3$ are the result of large flow acceleration at the start of pulsation in phase $A$ ($t/T = 0.02$) to $C$ ($t/T = 0.06$).

V. CONCLUDING REMARKS

An experimental study of transitional pulsatile flow with stenosis was carried out using time-resolved PIV and a MEMS wall-shear stress sensor at the mean Reynolds number of 1750 with the Womersley number of 6.15. The stenosis is modeled by a sinusoidal function, which is representative of pathology specimens (Ahmed and Giddens, 1984). The pulsatile velocity profile used is close to a patient-specific velocity profile derived from MRI by Li et al. (2009), where the maximum velocity is found at $t/T = 0.06$. Here, we have shown that the most of basic elements, if not all, of blood flow that are associated with the vascular disease can be found in such a simple flow configuration, which include the transition to turbulence, relaminarization, unsteady flow separation, shear layers, flow reattachment, recirculation regions and wall-shear stress fluctuations. We have carefully studied these basic flow elements to extract their underlying mechanisms with a view to help improve our understanding of the flow behavior in poststenotic blood flows including those involving grafts. Particular efforts were made to directly measure the wall-shear stress fluctuations during the pulsatile cycle and to analyze their turbulent energy contents using wavelet spectra.

At the start of the pulsatile cycle, a strong shear layer develops from the tip of the stenosis, increasing the flow separation region. The flow at the throat of the stenosis is always laminar due to acceleration, which quickly becomes turbulent through a shear-layer instability under strong adverse pressure gradient. At the same time, a recirculation region appears over the wall opposite to the stenosis, moving downstream in sync with the movement of the reattachment point. The shear-layer weakens as the flow decelerates later in the cycle, which is accompanied by an upstream shift of the reattachment point. These flow behaviors observed in a two-dimensional channel flow with the 25% and 50% stenosis are very similar to the results in a pipe flow obtained by Armaly et al. (1983), Ahmed and Giddens (1984), Ojha et al. (1989), Sherwin and Blackburn (2005), Razavi et al. (2011), and Jain (2016).
We also found that the behavior of a pulsating channel flow during the acceleration phase ($t/T = 0.02 - 0.06$) of both 25% and 50% stenosis cases is similar to that of the steady flow, including the location and size of post-stenotic flow separation regions. This is because the peak Reynolds number of the pulsatile flow is similar to that of the Reynolds number of the steady flow that is investigated. However, the transition to turbulence is more dominant for the 50% stenosis as compared to the 25% stenosis, as the wavelet spectra show a greater broadening of turbulence energy up to 120 Hz during the initial acceleration phase at $U_1(x/h = 1.5)$, $U_2(x/h = 2)$ and $U_3(x/h = 2.5)$. The wavelet spectra also indicate the presence of turbulence at $U_2$ during the second flow acceleration.

With an increase in stenosis to 75%, the accelerating flow is directed toward the opposite wall, creating a wall jet. The shear layer from the stenosis bifurcates as a result of this, one moving with the flow separation region toward the upper wall and the other with the wall jet toward the bottom wall. Later in the pulsatile cycle, the upper shear layer comes down toward the center of the channel with an increase in size of the flow separation region, while the lower shear layer moves upwards as the wall jet develops. Eventually, these two shear layers merge together creating a region of large turbulent intensity. The wall-shear stress always takes the maximum value at the throat of stenosis, although no fluctuations are observed there due to local flow laminarization. Immediate downstream of stenosis,
however, the wall-shear stress is temporarily reduced as the stenotic flow is entrained into the flow separation region. With a development of the jet over the bottom wall, the wall-shear stress starts increasing again.

Low wall-shear stress fluctuations are found at two post-stenotic locations – one immediately downstream of the stenosis over the top wall (stenosis side) inside the flow separation region (Deplano and Siouffi, 1999 and Long et al., 2001), and the other in the recirculation region on the bottom wall (opposite side of the stenosis) which appears during the acceleration phase of pulsatile cycle. The latter can only be found for the 25% and 50% stenosis. No recirculation region on the bottom wall is found for the 75% stenosis, at least within the field of view of our PIV measurements. The movement of reattachment point and the recirculation region over the top wall during the pulsatile cycle set up not only spatial, but also temporal gradient in the wall-shear stress distribution in a channel flow. As a result, the oscillatory shear index of the wall-shear stress becomes as much as 0.3 in the downstream of stenosis.

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DATA AVAILABILITY

The data that support the findings of this study are available within the article.

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