RESEARCH ARTICLE

Pulse transmission time and amplitude of digital pulse wave determined by fingertip plethysmography as a surrogate marker of brachial artery flow-mediated dilatation

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Abstract

Objectives: To assess the changes in blood vessel stiffness and digital pulse wave amplitude because of flow-mediated dilatation, and to explore how these two variables change when endothelial dysfunction is experimentally induced.

Method: The experimental study was conducted at the departments of physiology at the College of Medicine, Mustansiriyah University, and the College of Medicine, Al-Iraqia University, Baghdad, Iraq, from October 14, 2021, to May 31, 2022, and comprised healthy young males who were subjected to the flow-mediated dilatation technique on the left brachial artery. Pulse transit time and the amplitude of the digital pulse wave were measured during reactive hyperaemia for 2.5 minutes from the left middle finger using a piezoelectric pressure sensor and a simultaneous Lead I electrocardiogram. Endothelial dysfunction (ED) was induced by oscillatory and retrograde shear rates. The correlation between variables was calculated in Excel running on the Windows operating system.

Results: There were 10 second-year medical students with mean age 22±0 years and mean body mass index 25.7±4.8kg/m2. During reactive hyperaemia, pulse transit time was significantly increased by 3-5% in both normal endothelium and experimentally induced endothelial dysfunction relative to the pre-occluded artery, and the difference was not significant ($p>0.05$). Digital pulse wave amplitude increased significantly in normal endothelium relative to the pre-occluded artery ($p<0.05$), but not in experimentally-induced endothelial dysfunction ($p>0.05$).

Conclusion: The pulse transit time and digital pulse wave amplitudes of the photo plethysmography signal may be used to detect changes in vessel wall diameter and tone throughout the reactive hyperaemia process. Digital pulse wave amplitude was better able to detect experimentally-induced endothelial dysfunction, as assessed by the flow-mediated dilatation protocol, than pulse transit time.

Key Words: Hyperemia, Brachial Artery, Dilatation, Plethysmography, Electrocardiography, Pulse Wave, Endothelium, Running

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Introduction

The pulse transit time (PTT) is the time interval between two arterial sites during which the pulse wave propagates and is influenced by the stiffness of the artery.[1] PTT has been measured using a variety of methods, including photo plethysmography (PPG), which is a non-invasive optical measuring method that can track the expansion and contraction of blood vessels by tracking changes in blood volume. Alternatively, a finger pulse transducer, which employs a piezoelectric device to transform the applied force on the active surface of the transducer into an electrical analogue signal, may be used in lieu of PPG. As both systolic blood pressure (SBP) and diastolic blood pressure (DBP) fluctuate, the finger blood flow changes, and, as a result, the volume of the finger changes in response. As a matter of fact, Sharma et al.[2] were able to demonstrate that the signals acquired from PPG were identical to the signals obtained from a piezoelectric finger pulse transducer. In general, PTT and pulse wave velocity (PWV) are inversely proportional to one another (PWV = distance/PTT). In fact, PWV may be referred to as the gold standard measure of arterial stiffness. Therefore, PTT is considered to be particularly beneficial in the investigation of cardiovascular problems.[3] The vascular endothelium controls platelet function, inflammation, smooth muscle cell proliferation, and vascular tone.[4] Reduced nitric oxide (NO) bioavailability is a marker of endothelial dysfunction (ED), which leads to the advancement of cardiovascular diseases, such as atherosclerosis and hypertension (HTN).[5] The endothelium-dependent release of NO is partly responsible for the vasodilatory response to increased shear stress during reactive hyperaemia (RH) after a brief period of ischaemia.[6] The flow-mediated dilatation (FMD) of major arteries[7] or variations in digital pulse wave (DPW)

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amplitude could be used to detect this adaptation in vascular tone non-invasively.[6] Recent research has suggested that a decrease in PWV during RH could be a reflection of arterial distensibility and endothelial function.[8]

The current study was planned to investigate the changes in blood vessel stiffness and DPW amplitude as a result of FMD, and to explore how these two variables change when ED is experimentally induced.

Subjects and Methods

The experimental study was conducted at the departments of Physiology at the College of Medicine, Mustansiriyah University, and the College of Medicine, Al-Iraqia University, Baghdad, Iraq, from Oct 14, 2021, to May 31, 2022. After approval from the ethics review committee of Mustansiriyah University, the sample was raised using consecutive nonprobability sampling technique. Those included were young healthy male subjects who gave informed consent prior to enrolment. The scope of the study was intentionally restricted to a sample of healthy young male participants to eliminate the potential influence of obesity or other health conditions tied to body mass index (BMI), enabling a more accurate investigation within the designated demographics. All procedures were carried out in a laboratory environment with temperature of 25°C.

Participants who were smokers, participated in intense physical activity during a 12-hour period before to the procedure, or had tea or coffee were not included in the study.

Prior to taking the measurements, the participants were advised to rest in a supine position for 10 min. During this time, repeated BP and heart rate (HR) measurements were taken with an automated sphygmomanometer (Omron 705IT upper arm BP monitor, Omron Healthcare, Japan) until stable BP and HR readings were obtained to ensure haemodynamic stability before the test.

A piezoelectric finger pulse transducer was used to record the fingertip DPW of the left middle finger signals and the Lead I electrocardiogram (ECG) through 3-surface

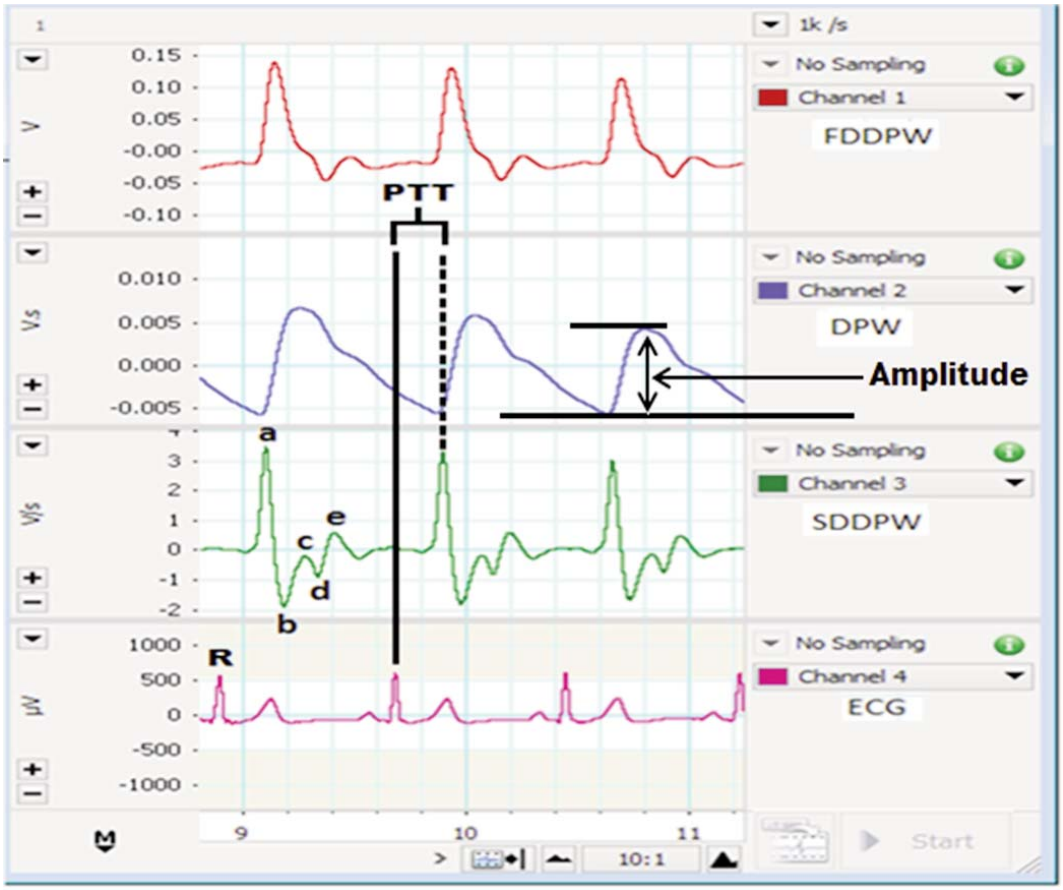

Figure-1: Measurement of pulse transit time (PTT), which is the time interval between each R wave peak and the maximum first peak of second derivative of the digital pulse wave (SDDPW) ('a' wave) that is located at a point at which signal voltage is 10% above the preceding baseline value at the foot of digital pulse wave (DPW)[9].

electrodes, all of which were connected to Power Lab Data Acquisition Unit 26T (AD Instruments Pty Ltd, New South Wales, Australia), which is an analogue-to-digital converter. A computer running Lab Chart Pro version 7.2 software was used for offline computer analysis of the signals. A low-pass filter and cut-off frequency of 35Hz were used for ECG recording.

BMI was calculated using the standard formula of weight(kg) / height^2 (m^2).Amplitude of PPG pulse waveform was the magnitude of the difference between the maximum signal voltage and the baseline signal voltage for each waveform. PTT represented the time interval between each R wave peak on the ECG and the maximum first peak of the second derivative of the DPW (SDDPW).[9] The endothelial function meant the health and function of the inner lining of blood vessels that were assessed using FMD.[7] induced ED meant a temporary reduction in endothelial function induced by inflating a tourniquet to a pressure of 75mmHg, causing arterial wave reflections and retrograde blood flow. RH showed the increase in blood flow following a period of arterial occlusion, which is a temporary blockage of blood flow in an artery induced by inflating a BP cuff to a pressure 50mmHg above the baseline SBP.

The most common procedure for determining the PTT is to measure the time difference between the R peak of the ECG and the feature point of the DPW (Figure 1). The period between each R wave's peak and the highest of the first peak of SDDPW 'a; wave, which is located in a place where the signal voltage is 10% higher than the preceding baseline value at the foot of DPW, was measured. The Lab Chart software's 'peak detection' module was used to identify ECG R wave and SDDPW peaks. There was an average of about 30-40 cardiac cycles for each time interval (30 sec).

After the subjects had rested in a supine position for at least 10 minutes with their eyes closed, the endothelial function was evaluated. The entire recording period included 30 sec of baseline recording, 5 min of arterial occlusion and 2.5 min of reactive hyperaemia with 30-sec intervals. The left brachial artery occlusion was achieved by elevating the cuff pressure to 50mm Hg above the baseline SBP after obtaining baseline ECG and DPW signals. The absence of a finger DPW signal from the monitor was used to verify the maintenance of artery occlusion in real time. After 5 min, the arterial blockage was relieved by completely deflating the cuff pressure, and recording was resumed at 30-sec intervals for 2.5 minutes during the RH phase. This was followed by a 3-min rest period before ED was induced by inflating the left mid-forearm tourniquet to 75mmHg. Although this moderate pressure does not limit arterial inflow, it does cause arterial wave reflections and retrograde blood flow .[9-11] The tourniquet was slowly deflated over 30 sec after 30 min of increased retrograde shear stress, and all measurements were promptly redone as specified in the FMD protocol.[7]

The correlation between variables was calculated in Excel running on the Windows operating system. The data was expressed as the mean and standard deviation. To compare data between variables, a paired student's t-test was used. The correlation between variables was calculated in Excel. P<0.05 was considered statistically significant.

Results

There were 10 second-year medical students with mean age 22±0 years and mean body mass index 25.7±4.8kg/m2. The mean SBP was 127.5±14.3mmHg, and mean DBP was 72.1±16.2mmHg.

The PTT during RH of the brachial artery with normal endothelium (NE) was significantly higher at 60 sec (5%, p<0.002), 90 sec (5%, p<0.003), 120 sec (4%, p<0.003) and 180 sec (3%, p<0.007) relative to the control PTT before

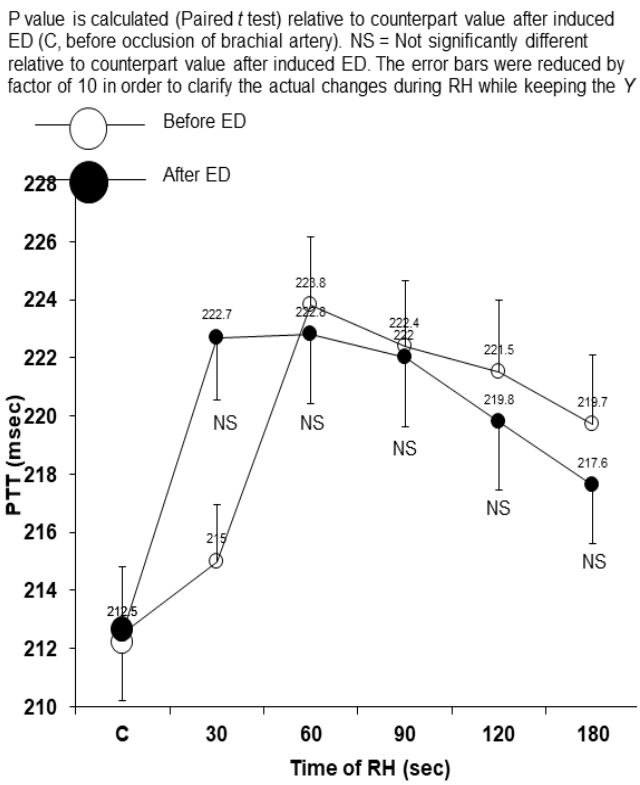

Figure-2: Pulse transit time (PTT) during reactive hyperaemia (RH) before and after induced endothelial dysfunction (ED) . N = 10.

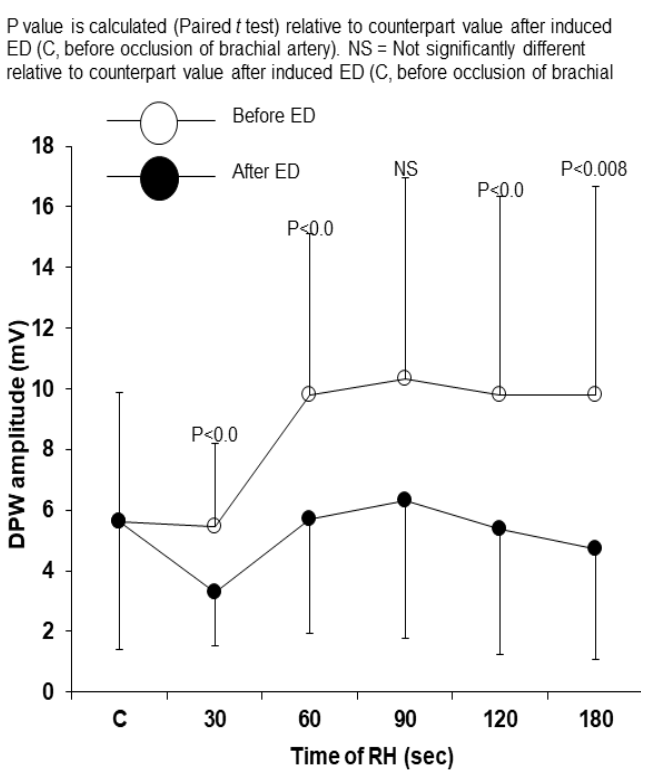

Figure-3: Digital pulse wave (DPW) amplitude during reactive hyperaemia (RH) before and after induced endothelial dysfunction (ED) . N = 10

occlusion of the artery. The PTT during RH of the brachial artery with induced ED was higher at 30 sec (5%, p<0.003), 60 sec (5%, p<0.003), 90 sec (4%, p<0.003), 120 sec (3%, p<0.007) and 180 sec (2%, p>0.05) relative to the control PTT before occlusion of the artery. There were no significant differences between the PTT of the brachial artery with NE or with induced ED throughout RH (Figure 2).

The DPW amplitude during RH of the brachial artery with NE was significantly higher at 60 sec (75%, p<0.02), 90 sec (84%, p<0.04), 120 sec (75%, p<0.02) and 180 sec (75%, p<0.03) relative to the control DPW amplitude before occlusion of the artery. The DPW amplitude during RH of the brachial artery with induced ED was lower by 40%, 42%, 45.4% and 52% than their NE counterparts at 30, 60, 120, and 180 sec of RH, respectively, while at 90 sec of RH, the DPW amplitude after induced ED was lower by 39% than its NE counterpart, but the difference was not significant level (p=0.05) (Figure 3).

Discussion

Several studies have been conducted on ED, which is regarded as an early atherogenic event.[12] A 'low-flow' state is widely acknowledged to promote atherosclerosis [13], with retrograde shear stress hypothesised to be a potent contributing factor.[14]

The current findings showed that the PTT during RH of the brachial artery with NE was significantly higher relative to the control PTT before occlusion of the artery. Similar behaviour was observed for the PTT during RH of the brachial artery with induced ED. This is in complete agreement with Sharma et al.[15] According to the Moens-Korteweg equation, the relationship between PTT and PWV is inversely proportional[16]. During RH, the PTT increases. As a result, the PWV decreases, meaning the stiffness of the blood vessels is reduced and the blood vessel becomes more compliant. However, when comparing the PTT of the brachial artery with NE or with induced ED, no significant differences were found throughout the whole time of the RH in the current study. Yet, by themselves and relative to the baseline, the responses were significant. This contradiction is possibly due to the temporary and weak stimulus used to induce ED. In contrast, the DPW amplitude had a different behaviour during RH after induced ED, in which the DPW amplitude was significantly lower than their NE counterparts.

The current findings further affirm that the DPW amplitude reflects brachial artery diameter changes[17] more sensitively than the measurement of PTT. The findings are in agreement with earlier studies[18,19]. Davignon et al.[20] described ED as a decrease in endothelium-dependent vasodilation resulting from reduced vasodilator bioactivity.

Limitation: The current study has a limitation as the sample size was not calculated, which could have affected the power of the study.

Conclusion

The PTT and the amplitude of the DPW signal may be used to measure the changes in the diameter and tone of the vessel wall that occur throughout the RH process.

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