Feeding artery of vascular accesses for hemodialysis:
model of arterial adaptation to high blood flow

PhD thesis summary

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1 Introduction

Chronic haemodialysis treatment requires repeated entries into the bloodstream. So-called permanent vascular access is used for this purpose. Creation of the permanent vascular access leads to significant haemodynamic changes in both the limb and the whole body. Moreover, despite called “permanent”, the lifespan of the accesses is very limited. Native arterio-venous fistula (AVF) is usually the access of the first choice because of longer patency and lower complication rates. However, when superficial veins are not suitable for AVF creation, a polytetrafluoroethylene (PTFE) graft (AVG) is used.

Both types of vascular accesses and especially the latter have limited patencies. Besides others, haemodynamic factors are involved in the pathogenesis of access complications. Blood flow increases in the feeding artery just after access creation because of a sudden decrease of peripheral vascular resistance. Increased blood flow (and velocity) leads to arteriodilatation. Endothelial cells possess special receptors, which detect physical forces. The leading force is the wall shear stress (WSS), having the vector tangential (and not perpendicular as in the case of blood pressure) to arterial lumen. WSS is directly related to the whole blood viscosity and to the wall shear rate (WSR). WSR is defined as the difference between adjacent velocities in the vascular lumen. The ratio between the maximum velocity at the center of the artery and the vessel radius is a common approximation of WSR. WSR is often used as an approximation of WSS. High values of WSR and WSS play a role in endothelial wall injury, von Willebrand factor activation and platelet aggregation, which are implicated in thrombus formation.
Altered haemodynamics plays also an important role in the induction and progression of diabetic vascular complications\textsuperscript{7}. There is increasing proportion of diabetics in the haemodialysis population. They suffer from considerably more frequent access complications, such as shorter patency, lower access flow and hand ischemia on the access side\textsuperscript{8}. Diabetic peripheral arteriopathy, in contrast to common atherosclerosis, affects also upper extremities, which are used for access creation. These structural changes in the arterial wall limit the regulation of vessel diameter, but consequently, because of higher peripheral vascular resistance, also the access flow, two determinants of WSR. We hypothesized that the vascular accesses of diabetic patients have higher values of WSR than that of non-diabetics.

The vessel responses to short-term and to chronic changes in WSS are different. The acute adaptation occurs within minutes is mediated via changes in smooth muscle cells tone, e.g. in case of vessels supplying working muscles\textsuperscript{9} or in flow mediated vasodilatation, a method of the endothelial function assessment\textsuperscript{10}. Chronic changes of arterial lumen occur within days and months and encompass wall structural changes named vascular remodeling, e.g. in peripheral arteries supplying muscles in athletes\textsuperscript{11} and in the feeding artery supplying vascular accesses for haemodialysis\textsuperscript{3,12}.

Creation of a dialysis vascular access offers a unique human model of vessel adaptation to chronic increase in blood flow. Access creation leads to a sudden decrease in peripheral vascular resistance. It is followed by blood flow rise in the feeding artery with simultaneous increase in WSS to supra-normal levels. Endothelial cells sense high WSS, and release more vasodilating nitric
oxide. The consequent vasodilatation is targeted to lower WSS via the feedback mechanism described above.

It has been shown that not only acute, but also long-term structural adaptation of mature arteries is endothelium dependent. Diabetes mellitus and dyslipidemia are both associated with endothelial dysfunction. Therefore, we expected that diabetes mellitus and dyslipidemia could limit further dilatation of the feeding artery.

2 Hypothesis

The adaptation to WSS is endothelium dependent. ESRD is associated with many co-morbidities representing risk factors for endothelial dysfunction: hypertension, hypercholesterolaemia, diabetes. We conducted two studies with the aim to:

1) Describe the haemodynamic profile in the feeding arteries of various access types shortly after access creation

2) Describe the haemodynamic profile in the feeding arteries in a 2 year course.

The hypothesis we tested are as follows:

A) Feeding artery shortly after access creation:

1) WSR values in radial and brachial arteries are comparable as WSS is the leading value

2) Diabetic patients have higher WSR than non-diabetics.

B) Long-term adaptation of the feeding artery
3) The internal diameter of the feeding artery after 1 and 2 years is higher that at baseline, with a decrease in WSR

4) The internal diameter of the feeding artery is lower in patients with risk factors of endothelial dysfunction, i.e. diabetes mellitus and hypercholesterolemia.

3  Feeding artery shortly after access creation

3.1 Population and Methods

3.1.1 Population

During a four-year period (2001 - 2005) we consecutively included patients with a newly created, well functioning upper extremity PTFE-graft in General University Hospital, Prague. Basic demographic data and diabetic status were recorded (Table 1).

3.1.2 Ultrasonography

It is known that AVG maturates approximately within 14 days, so we examined vascular accesses 14-180 days after their creation. Linear-array 3-11 MHz probe of SONOS 5500 device (Phillips, USA) was used. After careful examination of the whole access by duplex Doppler ultrasonography, as described earlier the attention turned to recording the artery. Ultrasound measurements were performed at the feeding artery 1-2 cm proximal to arterial anastomosis (Figure 2). Centerline peak, mean and minimal velocities, and internal artery diameter (ID) were measured.

Patients with huge arterial wall calcifications making the exact assessment impossible were excluded from the study. Similarly, subjects with access
complications (stenosis, thrombosis, inflammation, clinically apparent peripheral ischemia) were also excluded.

WSR was calculated using Poiseuillian parabolic model of velocity distribution across the arterial lumen based on the assumption of laminar blood flow, according to the following formula $^{19-21}$:

$$WSR = 4 \cdot \frac{V}{ID}$$

where $WSR$ is the wall shear rate (s$^{-1}$), $V$ is the blood velocity (m·s$^{-1}$), and $ID$ is the artery diameter (m). $WSR$ was calculated separately for peak (systolic), mean and minimal (end-diastolic) blood velocity.

### 3.1.3 Statistical analysis

Recorded values were compared according to access types (radial vs. brachial artery) and according to diabetic status using unpaired $t$-test. Data are expressed as mean $\pm$ standard deviation.

### 3.2 Results

A total of 106 patients was included into this study, 58 of them were non-diabetics and 48 diabetics. Basic demographic characteristics are listed in Table 1.

#### 3.2.1 Brachial vs. radial AVG comparison

Mean and minimal velocities were higher in radial artery, but this difference was significant only in diabetic patients. Distal (radial) accesses were characterized by significantly lower feeding artery diameter in both diabetic and
non-diabetic patients. Peak, mean and minimal WSR were significantly higher in distal accesses arteries.

3.2.2 Diabetics vs. non-diabetics

Diabetic subjects had significantly higher peak and mean arterial blood velocities in radial, but not brachial AVGs. Arterial diameter was significantly lower in diabetic patients in both access types. Arterial WSR was significantly higher in diabetic patients in both radial and brachial AVGs.

These results are summarized in Table 2.

4 Long term adaptation of the feeding artery

4.1 Population and Methods

4.1.1 Population

During a 5-year period (2001 – 2006) we consecutively selected patients with a newly created, well-functioning upper extremity PTFE graft in the General University Hospital, Prague. Informed consent was obtained from each patient before examination. The study was held in accordance with the Declaration of Helsinki. Together with the ultrasonography (see below), we recorded feeding artery type (brachial vs. radial), diabetic status, basic demographic and laboratory data.

4.1.2 Ultrasonography

The patients were examined within 3 months after access creation (baseline), then after 1 and 2 years. Linear-array 3-11 MHz probe of
SONOS 5500 device (Phillips, USA) was used (see Paragraph 3.1.2 for more details)

We report the values of ID, pWSR, and mWSR for baseline, first year and second year with appendices 0, 1, 2, respectively. The difference in ID between baseline and second year ΔID_{2:0}; between baseline and first year is reported as ΔID_{1:0}, and between first and second year as ΔID_{2:1}. We use in the same manner ΔWSR_{2:0}; ΔWSR_{1:0} and ΔWSR_{2:1}, separately for pWSR and mWSR.

### 4.1.3 Statistical analysis

WSR was transformed using the natural logarithm to obtain its normal distribution. ID and WSR changes during the study period were tested using paired t-test. It was performed first for all subjects, then in subgroups defined by the presence/absence of diabetes and by the type of the inflow artery (radial vs. brachial). Pearson’s correlation coefficients were calculated between cholesterol, triglycerides and arterial diameter and its changes.

The value of p<0.05 was considered significant. All calculations were performed using statistical software (STATISTICA Cz 6, StatSoft, Inc. 2003).

### 4.2 Results

We included 75 patients aged 61 ± 2 years, 20 were males and 33 had diabetes mellitus.

Values of ID, pWSR and mWSR are presented in Table 3. Internal diameter increased significantly during the study, with a concomitant decrease in pWSR and mWSR.
4.2.1 **Brachial vs. radial AVG**

Mean and minimal velocities were higher in radial artery, but this difference was significant only in diabetic patients. Distal (radial) accesses were characterized by significantly lower feeding artery diameter in both diabetic and non-diabetic patients. Peak, mean and minimal WSR were significantly higher in distal accesses arteries.

4.2.2 **Diabetes**

Patients with diabetes mellitus had smaller diameters than non-diabetics. During the first year, the feeding artery of non-diabetics dilated significantly by 19% compared to 7% in diabetics, despite the latter had higher WSR. In diabetics, WSR begun to decrease significantly later than in non-diabetics: during the second year of access age. These results are in *Table 3* and *Figure 3*.

4.2.3 **Cholesterol**

In the whole group of subjects, cholesterol correlated with ID0 \( (r=-0.24, p=0.043) \), ID1 \( (r=-0.30, p=0.009) \), ID2 \( (r=-0.34, p=0.028) \) but not with ΔID. Triglycerides correlated significantly only with ΔID1-0 \( (r=-0.23, p=0.046) \). Interestingly, these correlations were similar in the subgroup of radial artery accesses, but disappeared in the subgroup of brachial artery accesses.
5 Discussion

5.1 Feeding artery shortly after access creation

The first study has shown that feeding arteries of vascular accesses are exposed to unusually high WSR. WSR is even higher in arteries of distal accesses and in diabetic subjects.

Physiologically, WSS controls the relation between arterial diameter and blood flow. An increase in the blood flow leads to higher WSS, which, in turn, is followed by arteriodilatation and decrease of WSS. Similarly a decrease in blood flow leads to vasoconstriction. The adaptation of the vessel diameter represents an important feedback mechanism to keep WSS within a narrow, so-called physiological range. The adaptation has two steps: 1) rapid dilation within minutes as seen in flow-mediated vasodilatation and 2) long-term structural adaptation of vessel wall. Rapid adaptation to the blood flow changes is mediated by changes in vascular smooth muscle tone, which is endothelium-dependent (e.g. flow mediated vasodilatation). Arterial remodelling is a long-term, partly endothelium-dependent structural adaptation of the vessel wall.

Little differences in blood velocities do not explain substantial differences in arterial WSR in radial and brachial AVGs; this is rather the result of different internal diameter of these arteries and their capability to dilate. Brachial artery has a greater diameter than radial artery already before access creation. Furthermore the vessel wall response to increased blood flow could be limited by structural changes of the vessel wall, such as calcification. Arterial medial
calcification is more pronounced in diabetic patients, but uraemia per se also contributes to their development.\textsuperscript{26}

Konner reported that the arterial calcification was less pronounced in the elbow than in the wrist region.\textsuperscript{25} In diabetic patients atherosclerotic and calcified radial arteries do not undergo adaptive flow-mediated vasodilatation to deliver sufficient fistula blood flow.\textsuperscript{25} In these patients decreased vasodilatation ability is probably a result of both endothelium dysfunction linked to hyperglycemia\textsuperscript{27} and structural vessel wall changes.\textsuperscript{26} Distal arterial diabetic involvement probably explains higher WSR in feeding arteries of diabetic subjects.

Early thrombosis and low access flow are major causes of graft failure in haemodialysed patients and especially in diabetics.\textsuperscript{25} Subjects with diabetes mellitus have increased levels of von Willebrand factor (vWf) and decreased levels of tissue plasminogen activator.\textsuperscript{27} vWf plays a critical role in thrombus formation on PTFE surfaces. This is particularly efficient under conditions of high shear rate.\textsuperscript{28} At such conditions vWf is directly activated, it binds to an exposed subendothelium and activates platelet accumulation. Pathologically high shear rates (around 8000 s\textsuperscript{-1}), such as those at atherosclerotic stenosis, may directly activate platelet aggregation.\textsuperscript{5} Fibrin deposition increases also with increasing shear rate.\textsuperscript{28} All these pathological events lead to a local hypercoagulable state. High WSR in accesses of haemodialysed patients, and diabetic in particular, may play a role in access thrombosis and possibly also in hand ischemia.
5.2 Long term adaptation of the feeding artery

The main findings of the second study are as follows: 1) The dilatation of the feeding artery of vascular accesses continues at least 2 years after access creation with a continuous decrease in WSR, which however, remains highly supra-physiological. 2) Patients with diabetes have lower internal diameter and the vasodilatation of the feeding artery is delayed compared to non-diabetics. 3) Cholesterol is associated with lower internal diameter of the feeding artery however it does not influence its annual changes.

During the observation period, internal diameter continuously increased with a simultaneous decrease in WSR. These findings are in accordance with the theory of constant WSS, that states that the vessels tend to maintain WSS in physiological range by means of changes of internal diameter. However, even after 2 years neither pWSR nor mWSR reached normal values. The response to chronic blood flow increase requires structural adaptation associated with vascular cells growth and modification of vessel wall matrix, metalloproteinase’s activation being necessary for this process. Moreover, the feeding artery is subjected to supra-physiologically high WSR and the time to its normalization may be much longer than two years. However both acute and chronic increase of arterial diameter has probably its limits and it is possible that WSR will not normalize either after longer period.

The role of the feeding artery on the internal diameter results in naturally smaller internal diameter of the radial artery and higher normal values of pWSR and mWSR than brachial artery already before access creation and also shortly after access creation. Nevertheless, the annual percentual diameter...
changes were comparable in radial and brachial arteries, suggesting that the ability to dilate of both arteries are similar.

Already in our previous study we have reported lower internal diameter and higher WSR in diabetic patients shortly after access creation \(^31\). Vasodilatation and long-term vessel wall adaptation to increased blood flow is endothelium dependent \(^{13}\) and can be thus prevented by factors associated with endothelial dysfunction, such as diabetes. The magnitude of vasodilatation is negatively related to the degree of vessel wall calcification \(^{32}\), which is more pronounced in diabetic patients and known as mediocalcinosis \(^{33}\). Moreover, hyperglycemia in diabetic patients leads to changes in endothelial cells phenotype, which is associated with vasoconstriction \(^{15}\) and impaired vasodilatation \(^{18}\). Yet, the vessel adaptation to increased blood flow in diabetic patients is preserved, nevertheless progresses in a slower fashion that in patients without diabetes.

Higher WSR in the feeding artery of patients with diabetes may be both the cause and the consequence of lower diameter. The cause, if we assume that high WSR causes direct injury to endothelial cells \(^{27}\) and that high WSR is the reason for impaired vasodilatation and thus lower diameter. High WSR may be thus the consequence of lower diameter; and the lower diameter the consequence of impaired vasodilatation related to endothelial dysfunction, which is the consequence of diabetes. On the other hand, WSR is inversely related to internal diameter and the value of internal diameter was used to WSR calculation.

Our results suggest that the internal diameter is inversely related to cholesterolemia in the radial artery but not in the brachial artery. On the other hand the relative annual change of internal diameter does not depend on
cholesterolemia. Hypercholesterolemia by itself causes endothelial dysfunction by decreasing nitric oxide synthase activity and thus the vessel potential to dilate both acutely and chronically via NO-dependent metalloproteinases activation. The first measurement at baseline is already under the high flow load conditions suggesting that the first acute adaptation has already taken place. The subsequent chronic dilatation under such high WSR does not further depend on cholesterol.

5.3 Limitations

A possible limitation of our study is the use of WSR measurement as an estimation of WSS, because endothelial cells sense directly WSS and not WSR. WSS depends linearly on WSR and on blood viscosity. Some papers focus on WSS as a more accurate index of blood flow influence on endothelial cells. Nevertheless, to obtain the exact value of WSS one needs to know blood viscosity. Some authors use an arbitrary value for blood viscosity to estimate shear stress or use viscosimetry to measure it. It was shown that even actual measurements of viscosity must not lead to real values of shear stress. The use of an arbitrary value of blood viscosity would not change statistical significance of the results. Moreover, the variables affecting whole blood viscosity, such as haemoglobin level, haematocrit, plasma proteins, fibrinogen where not significantly different between groups (data not shown), suggesting that also viscosity was similar between groups. Thus, the comparison of WSR between groups can give the same results as for WSS with computed whole blood viscosity. For these reasons, we used WSR measurement.
Conclusions

We can conclude that feeding arteries of dialysis vascular accesses are exposed to supra-physiological values of WSR shortly after access creation. High WSR may play a role in development of access complications. Further research should reveal if long time exposition to high WSS is able do normalize or at least lower WSR.

The feeding artery continues to dilate to the second year after access creation, with a simultaneous WSR decrease. This process is dampened in patients with diabetes mellitus and hypercholesterolemia.
6 Abbreviations

AVF – arteriovenous fistula
AVG – arteriovenous graft
BA – brachial artery
CWS – circumferential wall stress
ESRD – end-stage renal disease
pO₂ – partial tension of oxygen
pCO₂ – partial tension of carbon dioxide
PTFE – polytetrafluoroethylene
RA – radial artery
VSMC – vascular smooth muscle cell
vWf – von Willebrand factor
WBV – whole blood viscosity
WSR – wall shear rate
WSS – wall shear stress

7 Tables

Table 1: Group characteristics at baseline

<table>
<thead>
<tr>
<th></th>
<th>DMᵃ</th>
<th></th>
<th>Non DM</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Radial artery</td>
<td>Brachial artery</td>
<td>Radial artery</td>
</tr>
<tr>
<td>Number (%)</td>
<td>27 (47%)</td>
<td>21 (43%)</td>
<td>30 (53%)</td>
</tr>
<tr>
<td>Age (mean ± SD) (years)</td>
<td>68 ± 10</td>
<td>66 ± 10</td>
<td>62 ± 17</td>
</tr>
<tr>
<td>Men/women</td>
<td>6/21</td>
<td>7/14</td>
<td>10/20</td>
</tr>
<tr>
<td>Number of previous accesses (median)</td>
<td>0</td>
<td>2</td>
<td>1</td>
</tr>
</tbody>
</table>

ᵃ DM = diabetes mellitus There were no significant differences in age and sex distribution between groups.
Table 2: Haemodynamic parameters in feeding arteries

<table>
<thead>
<tr>
<th></th>
<th>Radial artery</th>
<th>Brachial artery</th>
<th>Radial artery</th>
<th>Brachial artery</th>
<th>p-value DM vs. Non DM</th>
<th>p-value DM vs. Non DM</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number</td>
<td>27</td>
<td>21</td>
<td>30</td>
<td>28</td>
<td></td>
<td></td>
</tr>
<tr>
<td>$V_{\text{peak}}^b$ (cm·s$^{-1}$)</td>
<td>235 ± 73</td>
<td>206 ± 57</td>
<td>181 ± 70</td>
<td>178 ± 48</td>
<td>0.006</td>
<td>0.062</td>
</tr>
<tr>
<td>$V_{\text{mean}}$ (cm·s$^{-1}$)</td>
<td>179 ± 55</td>
<td>149 ± 46*)</td>
<td>145 ± 55</td>
<td>133 ± 33</td>
<td>0.021</td>
<td>0.167</td>
</tr>
<tr>
<td>$V_{\text{min}}$ (cm·s$^{-1}$)</td>
<td>139 ± 46</td>
<td>108 ± 40*)</td>
<td>118 ± 46</td>
<td>103 ± 23</td>
<td>0.100</td>
<td>0.537</td>
</tr>
<tr>
<td>ID$^c$ (mm)</td>
<td>2.7 ± 0.8</td>
<td>4.1 ± 0.8***</td>
<td>3.3 ± 0.8</td>
<td>5.0 ± 0.8***</td>
<td>0.012</td>
<td>0.0004</td>
</tr>
<tr>
<td>$\text{WSR}_{\text{peak}}^d$ (s$^{-1}$)</td>
<td>4040 ± 2889</td>
<td>2070 ± 670***</td>
<td>2512 ± 1623</td>
<td>1477 ± 582**</td>
<td>0.002</td>
<td>0.001</td>
</tr>
<tr>
<td>$\text{WSR}_{\text{mean}}$ (s$^{-1}$)</td>
<td>3043 ± 2041</td>
<td>1490 ± 528***</td>
<td>1985 ± 1169</td>
<td>1103 ± 401***</td>
<td>0.003</td>
<td>0.009</td>
</tr>
<tr>
<td>$\text{WSR}_{\text{min}}$ (s$^{-1}$)</td>
<td>2313 ± 1384</td>
<td>1075 ± 422***</td>
<td>1606 ± 913</td>
<td>848 ± 278***</td>
<td>0.008</td>
<td>0.069</td>
</tr>
</tbody>
</table>

Values are means ± SD.
Statistical significance of the difference between radial and brachial arteries is expressed by the stars in the particular column: *)$p < 0.05$, **) $p<0.001$, ***$p < 10^{-4}$

P-values comparing subjects with and without diabetes are listed at the right of the table separately for radial and brachial artery.

$^a$ DM = diabetes mellitus, $^b$ V = blood velocity (peak, mean, minimal), $^c$ ID = internal diameter of the artery, $^d$ WSR = wall shear rate (peak, mean, minimal)
Table 3. Haemodynamic variables at baseline, first and second year after access creation, according to the feeding artery type and diabetic status.

<table>
<thead>
<tr>
<th></th>
<th>All patients</th>
<th>Feeding artery</th>
<th>Diabetes mellitus</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Brachial</td>
<td>Radial</td>
</tr>
<tr>
<td>Number of subjects</td>
<td>75/41</td>
<td>33/19</td>
<td>42/22</td>
</tr>
<tr>
<td>(year 1/year 2)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ID0</td>
<td>3.9 ± 0.1</td>
<td>4.7 ± 0.2***</td>
<td>3.3 ± 0.1***</td>
</tr>
<tr>
<td>ID1</td>
<td>4.4 ± 0.2 (14%)</td>
<td>5.3 ± 0.2 (14%)***</td>
<td>3.6 ± 0.2 (14%)***</td>
</tr>
<tr>
<td>ID2</td>
<td>4.6 ± 0.2 (5%)</td>
<td>5.4 ± 0.3 (-1%)***</td>
<td>4.0 ± 0.3 (11%)***</td>
</tr>
<tr>
<td>pWSR0</td>
<td>2366±1290</td>
<td>1860±785**</td>
<td>2764±1468**</td>
</tr>
<tr>
<td>pWSR1</td>
<td>2108±1389</td>
<td>1584±842**</td>
<td>2521±1592**</td>
</tr>
<tr>
<td>pWSR2</td>
<td>1517±955</td>
<td>1279±715</td>
<td>1723±1096</td>
</tr>
<tr>
<td>mWSR0</td>
<td>1806 ± 113</td>
<td>1382 ± 99***</td>
<td>2140 ± 170***</td>
</tr>
<tr>
<td>mWSR1</td>
<td>1589 ± 118</td>
<td>1144 ± 95 ***</td>
<td>1939 ± 180 ***</td>
</tr>
<tr>
<td>mWSR2</td>
<td>1148 ± 107</td>
<td>963 ± 116</td>
<td>1308 ± 168</td>
</tr>
</tbody>
</table>

ID for internal diameter (mm), pWSR for peak wall shear rate (s\(^{-1}\)), mWSR for mean wall shear rate (s\(^{-1}\)), data are expressed as mean ± SE.

In parenthesis percents of change between baseline and first year and between first and second year are shown. Statistical significance (unpaired t-test) is shown for the comparison between feeding artery type and between patients with and without diabetes. *for p<0.05; ** for p<0.01; *** for p<0.001
8 Figures

Figure 1: Schematic representation of haemodynamic stresses in blood vessels. (from London et. al\textsuperscript{34})

\[
\sigma = \frac{P \times R}{h}
\]

Circumferential wall stress

\[
\tau = 4 \frac{\mu \cdot Q}{\pi R^3}
\]

Fluid shear stress

Figure 2: Ultrasonography assessment of internal diameter and velocity profile in the feeding artery
Figure 3: Mean wall shear rate and internal diameter in Radial and Brachial arteries separately for diabetic and no-diabetic patients

DM – diabetes mellitus; meanWSR – mean wall shear rate
References