**Background:** Arteries adapt their diameter to changing haemodynamic conditions to maintain constant wall shear stress, the force generated by flowing blood on endothelial cells. The feeding artery of haemodialysis vascular accesses is a human model of arterial adaptation to chronically high blood flow and thus to high wall shear stress. The process of arterial adaptation is endothelium dependent. Endothelial dysfunction related to End-Stage Renal Failure, diabetes mellitus, dyslipidemia may impair also the dilatation of the feeding artery of vascular accesses.

First the review of the literature presents in three parts different aspects of arterial adaptation: 1) arterial adaptation with focusing on the role of haemodynamic factors, 2) the influence of end-stage renal disease on arterial adaptation, 3) feeding artery of vascular accesses as a model of arterial response to chronic increase in blood flow.

**Methods:** We examined the feeding artery of radial and brachial polytetrafluoroethylene grafts shortly after and one and two years after access creation. We used duplex ultrasonography to obtain internal diameter and blood velocity in the feeding arteries. We calculated wall shear rate as $4 \times \text{blood velocity} / \text{internal diameter}$ and used it as approximation of wall shear stress.

**Results:** In the first study we included 106 patients (58 non-diabetics and 48 diabetic). WSR was significantly higher in radial compared to brachial arteries independently of diabetes status. Diabetic subjects had significantly higher WSR in both radial and brachial arteries compared to non-diabetics.

In the second study we examined 75 patients. Internal diameter raise from $3.9 \pm 0.1 \text{ mm}$ after access creation to $4.4 \pm 0.2 \text{ mm}$ in the first year and to $4.6 \pm 0.2 \text{ mm}$ at the second year. Mean WSR decreased from $1806 \pm 113 \text{ s}^{-1}$ after access creation to $1589 \pm 118 \text{ s}^{-1}$ in the first year and to $1148 \pm 107 \text{ s}^{-1}$ at the second year. Internal diameter was negatively correlated to diabetes, cholesterol and WSR.

**Conclusions:**
1) The feeding arteries of vascular accesses are exposed to unusually high WSR shortly after access creation.
2) WSR is even higher in arteries of distal accesses and in diabetic subjects.
3) 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.
4) Patients with diabetes have lower internal diameter and the vasodilatation of the feeding artery is delayed compared to non-diabetics.
5) Higher levels of cholesterol are probably associated with thinner arterial lumen.