Results: Neutrophils were three times increased after 30 minutes until 4 hours of reperfusion as well as after flushing. Doubling of macrophages and a four times increase of T-cells were observed after 30 minutes until 1 and 2 hours of reperfusion respectively. Apoptosis with important oedema in absence of necrosis was seen during the whole study period.

Conclusion: After 1 hour of warm ischemia followed by 30 minutes of reperfusion a significant increase of neutrophils, T-cells and macrophages was observed in this study. One hour of warm ischemia and 4 hours of reperfusion followed by flushing also resulted in a significant increase in infiltration of neutrophils. Finally, this study showed apoptosis with serious oedema in absence of necrosis after all periods of reperfusion.

P97

Experimental and Numerical Simulations of the Hydrodynamics in PTFE-Grafts

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Introduction: End stage renal disease (ESRD) patients who are referred for hemodialysis treatment need a vascular access able to deliver blood to the artificial kidney at a continuous flow rate of ±300 ml/min. If the vascular access type is a polytetrafluorethyleen (PTFE)-graft, stenosis development at the venous anastomosis is at high risk, leading to decreased flow rate and access thrombosis. The aim of the study is investigation of the hydrodynamics in two different PTFE-graft geometries.

Methods: A straight (6 mm) and a tapered (4–7 mm) PTFE-graft are each sewed onto a silicon artery and vein model having 4% relative distensibility to create the experimental vascular access models. A cardiac simulator pumps a water-glycerine mixture (60/40%) through this model and generates physiologic flow and pressure wave contours. Flow is varied between 500 and 1500 ml/min and two pressure scenarios are followed. 'Normal' scenario means mean arterial inlet pressure fixed at 100 mm Hg. Mean venous outlet pressure is fixed at 20 mm Hg in the 'low resistance' scenario. Pressure and velocity are continuously measured using a fluid filled catheter connected to a membrane pressure transducer and an ultrasound machine respectively. Numerical simulations of flow and shear stress distribution are performed using a rigid wall geometry. A set of measured pressure and velocity are utilized as boundary conditions.

Results: The results of the experiments show that flow in the vein is still unsteady. In the 'normal' scenario, mean venous pressure remains above 20 mm Hg in the tapered graft, where it is ±20 mm Hg in the straight graft. Pulse pressure in the vein in the 'normal' scenario is ±20 mm Hg where it is ±5 mm Hg in the 'low resistance' scenario. When flow rate goes down, mean velocity goes down, but velocity signal amplitude (= maximum – minimum velocity) increases. The results of the numerical model and the experiments still differ. Mean arterial pressure in the numerical simulations is 19% lower than the experimental results while pulse pressure (= maximum – minimum pressure) is 23.7% and maximum velocity is 30% higher in the numerical simulations. Nevertheless, the simulations clearly indicate that the maximum velocity increases at the anastomoses and the stagnation point moves in the venous anastomosis region, where stenoses tend to develop.

Conclusion: Our experiments clearly show that both flow pulsatility and mean pressure, in the vein in case of the PTFE-graft, are higher than normal values. This may introduce stenosis. The influence of stenosis and wall compliance will be part of further study.

P98

Pretreatment with Antioxidants Decreases Damage Due to Reperfusion of Isolated Intestines in the Absence of Leukocytes

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Introduction: Leukocytes are said to be the agent mainly responsible for intestinal reperfusion damage. In the 'in vitro without blood cells' model we have been testing for the last two years, we checked the scarce benefit of antioxidant drugs in reversing the damage due to ischemia. Now we wanted to investigate whether the same antioxidants (AO) might prove to be efficient as damage blockers when used before and after the ischemic period.

Methods: Male Wag rats have been used. The superior mesenteric artery and the portal vein were canulated, and both the jejunum and ileum were excised, cleansed with heparinized Ringer solution, and perfused with 10 ml of either Ringer (groups 1 and 2) or Ringer supplemented with AO (folinic acid 0.21 μ/ml, alfatocopherol 1.6 microl/ml and SOD 0.58 μ/ml). The organ was placed in a bath at 40°C for 60 minutes, and then it was perfused for 30 minutes with our acellular reperfusion solution (ARS) (group 1) or ARS + antioxidants (groups 2 and 3). Samples from the perfusate and portal drainage were collected at 0, 5, 15 and 30 minutes of perfusion, and electrolytes, pH, glucose, CPK, LDH and PA were determined. Once the perfusion was completed, six consecutive fragments of distal ileum (1 cm long) were obtained and embedded in paraffin.

Results: CPK and LDH were not increased during reperfusion. PA showed a mild elevation, which was blocked when adding AO to the perfusate (group 2). Oxygen extraction was significantly decreased between 5 and 15 minutes, reaching normal values at 30 minutes; the addition of AO to the perfusate could not prevent this effect (group 2). However, AO-pretreatment (group 3) induced an earlier recovery, reaching normal values within 15 minutes. In the control group, hyperkalemia and acidosis were found in the portal effluent during the first minutes, progressively declining until 15 minutes. AO added to the perfusate had no effect on these parameters. However, AO-pretreatment significantly reduced both hyperkalemia and acidosis throughout the whole experiment.

Conclusions: The slight improvement reported when adding AO to the perfusate (group 2) was significantly increased by AO-pretreatment of the intestine (group 3). Enzymatic studies proved that tissue damage in this model is very small, and completely reverted by AO.