## **SUMMARY**

Renal ischemia-reperfusion injury can be an inevitable consequence of a number of clinical situations and operative procedures, and may result not only in the injury of the renal parenchyma but the clamped artery itself. These changes may impair the contractile-relaxant capacity of the vessel. Processes caused by oxidative stress may influence the rheological factors of the blood thereby altering microcirculation.

The aim of our study on mongrel dogs was to demonstrate the changes of renal artery clamping for 45 minutes, followed by reperfusion in the clamped artery applying functional (vascular reactivity) and morphological (light microscopy and apoptosis) examinations in the kidney using functional (serum urea and creatinin, urinary N-acetyl-β-D-glucosaminidase) tests. It was also our aim to show the alterations in systemic circulation (hematological and hemorheological examinations, determination of plasma antioxidant activity and endothelin level).

Since free radicals may play a key role in this process, their contribution was concluded indirectly by administering xanthin-oxidase inhibitor allopurinol.

In summary we can conclude that alterations may also occur in the clamped artery during ischemia-reperfusion, shifting the balance towards vasoconstriction. Further adverse effects are deterioration of renal function and considerable systemic changes such as deterioration of hemorheological parameters (red blood cell deformability), increased release of endothelin (vasoconstrictor mediator), reduced antioxidant activity in the body. These changes added together may influence the functioning of the whole organism.

The fact that these changes could partly be influenced by preischemic allopurinol treatment may indirectly indicate that, at least in part, the pathophysiological activation of xanthin-dehidrogenase/xanthin-oxidase is found in the background of the process.