# 8. SUMMARY

**Establishment and characterization of a cardiac ischemia reperfusion model: The isolated normothermic hemoperfused working porcine heart**

The principal aim of this study was to establish and characterize the model of an isolated normothermic, hemoperfused working porcine heart. Hemodynamic and clinical chemical parameters were investigated for a period of seven hours. Moreover, we induced acute ischemia (2h), followed by reperfusion (4h) to investigate perivascular and interstitial collagen content using electron microscopy, western blot analysis and sirius red staining. The hearts were prepared and connected to a special perfusion system. After 1 hour of perfusion (adaptation time) the hearts were randomized to several groups:

1. working hearts (control),
2. infarcted hearts (MI; R. circumflexus),
3. infarcted hearts treated with Angiotensin I (Ang I-MI),
4. infarcted hearts treated with Ang I and Quinaprilat (Q-Ang I-MI) and
5. infarcted hearts treated with Quinaprilat (Q-MI)

Blood gas and oxymetric analysis were checked at the beginning of the experiment and all 30 min. Coronary perfusion, perfusion pressure, right ventricular pressure, heart rate, left ventricular systolic and end-diastolic pressure were measured online using a personal computer. In all groups and all media pH, $pO_2$, $pCO_2$, bicarbonate, glucose, natrium, calcium and lactate remained within the normal range. Potassium significantly increased at the end of the experiment but remained within the normal range. Heart rate, LVEDP and perfusion pressure of infarcted hearts elevated significantly ($P<0.005$). In the infarct area total collagen content was early increased after ischemia-reperfusion. Early application of Quinaprilat attenuated collagen type III accumulation.

**Conclusion:** We have established and characterized a new isolated porcine ischemia reperfusion model. Our data indicate that the early physiological and morphological effects of a myocardial infarction can be investigated *ex vivo*. In parallel, we have the possibility to perform new pharmacological strategies to prevent heart failure and pathologic cardiac remodeling post-MI.