Introduction

#1 cause of death in western society is heart disease, especially atherosclerosis of the coronary arteries. This disease, characterized by a narrowed arterial lumen can be treated by medication or catheter-based surgical interventions called percutaneous transluminal coronary angioplasty (PTCA): balloon dilation (fig.1A) followed by stenting (fig.1C). The mechanical loads applied on an artery wall during an intervention differ from the physiological situation: They are relatively high and are induced locally. Clinical trials showed wall injury after PTCA resulting in renarrowing of the lumen. This restenosis process is mostly located at transition sites between PTCA-loaded and not PTCA-loaded cells (fig.1D). Clinical trials showed wall injury after PTCA resulting in renarrowing of the lumen. To prevent restenosis the vascular cells (VCs) undergo intraluminal coronary radiation therapy (ICRT): a catheter-based $\beta$-radiation treatment (fig.1B).

![fig.1 Consecutive intervention techniques](image)

Objective

The aim of this project is to study morphological and biochemical responses to interventions and the effects of $\beta$-radiation on (damaged) VCs.

Method

To investigate the intervention responses an experimental setup must be built in which arterial segments can be conditioned and perfused in a physiological environment. Physiological mechanical loads, shear stress $\tau_w$ and circumferential stretch $\varepsilon_{\phi\phi}$, are necessary for VCs to maintain their functionality [1]. Furthermore it must be possible to do interventions.

![fig.3 Perfusion system](image)
![fig.4 Physiological loads](image)

To determine cellular responses biochemical and morphological changes are monitored. Extreme proliferation of VCs, resulting in restenosis, after PTCA is a reaction to the high mechanical loads and may be prevented by ICRT. $\beta$-irradiation from a source placed near the lesion site may change their functionality. In vitro experiments showed that the most important consequence of ionizing radiation is a cell killing effect [2]. Cells that stay alive show a decrease in enzyme activity besides changes in the cell cycle. In this cycle the cell doubles its contents and divides. Irradiation discontinuous the cycle so the proliferation reaction is stopped. First results in clinical trials show a positive effect [3].

![fig.4 Reproductive cell cycle: irradiation discontinuous the cycle at 'red arrow'-locations](image)

References: