**INCREASED ANGIOGENESIS IS NOT ABLE TO IMPROVE HEART FUNCTION AFTER CELL THERAPY**

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Cell transplantation therapy is considered a novel and potentially new strategy in regenerative medicine. Recent studies imply that paracrine effects and inflammatory modulations by transplanted cells are a key factor for improvement of myocardial function. Using apoptotic bodies which cannot induce inflammation, but transport a variety of factors, we tried in the presented study to differentiate between the paracrine effects and inflammatory reactions after cell transplantation. Myocardial infarction was induced by LAD ligation. 4 weeks later apoptotic bodies (1 x 106) from endothelial progenitor cells (EPC), fibroblasts, human endothelial vein endothelial cells (HUVEC) as well as medium in a control group were intramyocardially injected in 4 different groups into the infarct border zones. The functional results (echocardiography) showed no improvement of left ventricular function, 8 weeks after myocardial infarction. Furthermore, no differences in the infiltration amount of inflammatory cells were detectable, confirming our hypothesis that transplantation of apoptotic bodies is not able to alter inflammatory processes. Interestingly, histology revealed an increase in angiogenesis in HUVEC and EPC groups, showing that neovascularisation is not a condition for a better myocardial function. In conclusion, apoptotic bodies do not trigger inflammatory processes but induce paracrine effects, augmenting neovascularisation. It seems that, contrary to previously believed, these processes are not enough to improve myocardial infarction. Therefore, the modulations of inflammatory processes seem to be an important parameter which should be focus when developing new therapeutic strategies.