Abstract:

A regular tissue functioning requires the adequate supply of oxygen and nutrient via blood vessels. The sequences of formation and maturation of vessels are initiated and maintained by different growth factors. The VEGF growth factor plays an exceptional role in these mechanisms. The creation of sublethal ischemia as an angiogenic stimulus known as "Delay" is a well established procedure in plastic surgery, although the underlying molecular biological mechanisms still remain unknown. The important role of VEGF and its regulation depending on oxygen pressure suggest a strong connection between this growth factor and the delay phenomenon. The VEGF concentration in skin and underlying muscle was measured in overdimensioned random pattern flaps on 32 male Sprague-Dawley rats after either VEGF gene therapy or circumcision without elevation of the flap and compared to controls. Additional random pattern flaps were raised seven days post gene therapy or delay. The effect on the flap perfusion was measured postoperatively using Indocyanine green Laser Fluoroscopy and the size of the surviving and necrotic areas of the flaps were analysed. The skin of the random pattern flaps showed both in the Delay group and in the VEGF gene therapy group a significantly elevated VEGF concentration compared to the controls. The underlying rectus abdominis muscle showed no significant differences in
VEGF concentration between the groups. The flap perfusion postoperatively was significantly increased solely in the VEGF gene therapy group. The analysis of the surviving area of the flaps showed a significant increase over the controls in the gene therapy group. The Delay procedure results in a significantly and locally raised concentration of the VEGF growth factor. The gene therapeutical use of this growth factor allows us to raise flap perfusion and to reduce necrosis. Both VEGF gene therapy and Delay seem to promote similar mechanisms whereas the gene therapy produced superior results in this setting.