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The gonadotropins, whose members are human chorionic gonadotropin (hCG), luteinizing hormone (LH) and follicle-stimulating hormone (FSH) are a well characterized hormone family known to regulate reproductive functions in both females and males. Recent studies indicate that they can modulate the vascular system of reproductive organs. It was shown that gonadotropins not only influence the expression of vascular endothelial growth factor (VEGF) and both its receptors VEGFR-1 and -2, but also modulate other ubiquitously expressed angiogenic factors like the angiopoietins and their receptor Tie-2, basic fibroblast growth factor or placental-derived growth factor. Some recent data indicates a possible direct action of gonadotropins on endothelial cells. Thus, the gonadotropins act as tissue-specific angiogenic factors providing an optimal vascular supply during the menstrual cycle and early pregnancy in the female reproductive tract as well as in testis. In pathological conditions (e.g. preeclampsia, intrauterine growth restriction, ovarian hyperstimulation or endometriosis), these tightly regulated interactions between the gonadotropins and the ubiquitous angiogenic factors appear to be disturbed. The intent of this short manuscript is to review the current knowledge of the regulatory role of the gonadotropins in vasculo-and angiogenesis. We also review angiogenic actions of thyroid-stimulating hormone (TSH), a glycoprotein closely related to gonadotropins, which display strong gonadal actions.