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Intimal hyperplasia (IH), the thickening of the tunica intima of a blood vessel, is a universal response of an artery to injury. The occlusion of an artery due to IH can occur in a stenosed artery that has been treated with balloon angioplasty or that has been injured, and is a significant reason for late bypass failure. In an artery which illustrates IH, vascular smooth muscle cells migrate from the tunica media (middle) to the tunica intima (inner) layer of the artery wall in response to biochemical gradients of various cytokines, such as platelet derived growth factor (PDGF). IH may be stimulated, enhanced, or inhibited by cytokines released from invading inflammatory cells and by cellular signaling after the denudation of endothelial cells lining an artery. In order to predict which patients will develop IH following vascular injury and to understand the conditions under which estrogen and hormone replacement therapy have beneficial and/or detrimental influence on the development of IH, my project is to develop a conceptual theoretical model that describes the processes involved in IH using ArgoUML, a Unified Modeling Language tool for diagrammatically modeling object-oriented system designs. This conceptual model will be integrated into a mathematical model of vascular remodeling under development by Oak Ridge National Laboratory in collaboration with the Vascular Research Laboratory at the University of Tennessee Medical Center in Knoxville.

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