

2010 Departmental Seminar

Thursday 20 May 2010

3.00 pm

Mechanical Engineering Seminar Room – E547

Computational Models of Neurovascular Coupling

Hannah Farr, Ph.D.-cand. UC

Abstract:

Functional hyperaemia is an important metabolic autoregulation mechanism by which increased neuronal activity is matched by a rapid and regional increase in blood supply. This mechanism is facilitated by a process known as “neurovascular coupling” - the orchestrated intercellular communication between neurons, astrocytes and microvessels. An important step in this process is the release of potassium into extracellular space by two potassium channels, BK and KIR. Previous models of neurovascular coupling have not included the mechanisms involving these channels. Here we provide such a model, which successfully accounts for the arteriolar dilation caused by the release of glutamate into the synaptic space between neurons. This model can achieve an approximate 20% vasodilation due to the rise in perivascular potassium concentration from 3 to 6mM. It also successfully emulates the experimental finding that vasoconstriction follows this vasodilation when potassium is increased further. We suggest that the interaction of the changing smooth muscle cell membrane potential and the changing potassium- dependent resting potential of the KIR channel are responsible for this paradoxical effect.