Weekly Colloquium
Tuesday, 03/22/2016, Billings Building – Rosedale Conference Room


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Research Abstract:
Whereas t-PA revolutionized treatment for acute stroke, a similar revolution is needed in stroke rehabilitation. Improved acute stroke care, including recent endovascular clot removal has resulted in fewer deaths but ~ 50% of survivors have chronic disabilities and nearly a third are institutionalized. The brain has tremendous capacity to adapt to injury through recently identified structural and molecular changes collectively referred to as neuroplasticity. Exploiting these neuroplastic processes is thought to be key to developing more effective rehabilitation protocols. Animal studies have dramatically increased our understanding of neuroplasticity and its potential to reorganize and restore function to the stroke-damaged brain. Clearly, more effective therapies require a better understanding of the basic mechanisms through which rehabilitation and injury factors (e.g. stroke location) interact to affect plasticity and recovery. In addition, there is an urgent need to extend the window of efficacy and to develop adjunct therapies to potentiate the effects of rehabilitation since mono-therapeutic approaches have not been successful in stroke or other neurological diseases. In humans and in animal models there is evidence of spontaneous post-stroke recovery but little is known about the location and type of neuroplastic changes that underlie this recovery. Such information is essential in order to develop new treatments and make rehabilitation more effective. Findings from animal studies concerning the type, timing and intensity of rehabilitation to better optimize clinical rehabilitation will be discussed and related to molecular events that may limit or put “brakes” on post-stroke recovery.

Relevant publications:
