Topic 15 - Recovery from Stroke

Motor Recovery Post-Stroke - Spontaneous

Recovery Post-Stroke
At least 3 separate but interactive processes associated with recovery

1) Resolution of diaschisis, inflammation etc..
2) Behavioural compensation
3) Neuroplasticity

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Diaschisis

- Loss of function in remote areas anatomically connected to region of lesion

-Possibly from disruption of afferent excitatory input from lesioned area to other brain regions.
Some initial improvement also likely corresponds to resolution of tissue inflammation.

- early stage inflammation starts a few hours after onset of ischemia
- microglia and leukocyte involvement

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Recovery Post-Stroke
At least 3 separate but interactive processes associated with recovery

1) Resolution of diaschisis, inflammation etc..

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Behavioural compensation

- Use unaffected arm
- Learn to walk with a cane

Recovery Post-Stroke
At least 3 separate but interactive processes associated with recovery

1) Resolution of diaschisis, inflammation etc..

2) Behavioural compensation

3) Neuroplasticity
Mechanisms of Neuroplasticity Implicated in Stroke Recovery

1) Redundancy – alternate pathways take over lost function

2) Unmasking - activation of normally inhibited pathways

3) Long-term potentiation - increasing efficiency of synaptic connections and formation of new synapses.

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1) Redundancy – alternate pathways take over lost function

![Brain diagram showing compensatory pathways and site of lesion](image)

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1) Redundancy – alternate pathways take over lost function

Cortical Activity During Index Finger Tapping

![Brain images showing control and stroke subjects](image)

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2) Unmasking – activation of normally inhibited pathways

Model of intracortical connections in M1 -

Inhibitory interneuron prevents excitatory connections from whisker area from activating forelimb area.


Adjacent cortical regions expand when preexisting lateral excitatory connections are unmasked by decreased intracortical inhibition.

GABA antagonist


Reduction in GABA Receptors throughout cortex after focal ischemic lesion.

3) Long-term potentiation

- Increasing efficiency of synaptic connections.

- Formation of new synapses.

- A means of modifying neural circuitry

Increase in Ca2+ in post-synaptic cell is critical trigger for induction of LTP

Ca2+ ions activate postsynaptic protein kinases

---> result is increased synaptic strength

LTP may arise from rapid insertion of AMPA receptors

NMDA-R  AMPA-R

LTP may increase size and number of synaptic contacts


New dendritic spines begin to form approx 1 hour after induction of LTP

Long-term depression
- weakening of a synaptic connection