Neuroplasticity and recovery from stroke and aphasia

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What is neuroplasticity?

What is the role of neuroplasticity in recovery after stroke?

Are there parallels between recovery of movement and recovery of communication?

What are the big questions?
Neuroplasticity

Changes in the strength and number of synapses between neurons

Practice leads to strengthening and addition of synapses

Dis-use leads to weakening and removal of synapses
Verb generation

Verb generation after 15 min practice
Plasticity triggers

Input to the brain
- Sensation

Output from the brain
- Movement, speech

Plasticity is also affected by
- Medication
- Attention
- Diseases
- Genetics
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Functional plasticity
Structural plasticity
Cell death
Axonal growth inhibition
Metabolic depression

1d
1w
4w
3m
6m

Time since stroke

Percentage of patients who have achieved maximum recovery

25%
50%
75%
100%

Structural plasticity
Functional plasticity
Neurogenesis
Gliogenesis
Angiogenesis
Cell death
Metabolic depression
Axonal growth inhibition

Stinear & Byblow, *Current Opinion in Neurology*, 2014
Neuroplasticity

Is neuroplasticity super-charged after stroke?

At the chronic stage, neuroplasticity and learning appear to be similar in people with and without stroke.

Find out if neuroplasticity is enhanced during the first few weeks of recovery after stroke.
What is neuroplasticity?

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Current thinking

The stroke side is under-active
The other side is over-active

Recovery is associated with ‘balancing’
Interhemispheric inhibition model
A  INTACT CONNECTIONS

Active
Inhibited

Lateral view: LH  LH  RH

Spironelli & Angrelli, *Scientific Reports*, 2015
Interventions

Turn up the stroke side
Turn down the other side

Transcranial magnetic stimulation
Transcranial direct current stimulation

Over 850 publications about their use in stroke
But....

The vast majority of studies are of patients at the chronic stage

Only 5% of RCTs recruit all patients within 30 days of stroke

Stinear et al., Stroke, 2013

What’s happening in the brain during the first few weeks of recovery?
What do we know?

Recovery of motor function is related to increasing activity in the ipsilesional motor cortex.

No evidence for

- Over-activity in the contralesional motor cortex
- Unbalanced interhemispheric interactions

Find out if the interhemispheric inhibition model applies for communication at the sub-acute stage.
Recovery is predictable

We can predict an individual patient’s potential for
  recovery of motor function
  resolution of motor impairment
PREP algorithm

72h
Shoulder Abduction
Score each out of 5 and sum
Finger Extension

5 - 7 d
TMS
Presence of MEPs

10 - 14 d
MRI
Asymmetry Index

SAFE Score
8 - 10
Complete
Rehabilitation focus: Promote normal use

SAFE Score
5 - 7
Notable
Rehabilitation focus: Promote function

MEPs
< 0.15
Limited
Rehabilitation focus: Promote movement

No MEPs
> 0.15
None
Rehabilitation focus: Promote compensation

Stinear et al., *Brain*, 2012
www.prepforstroke.rehab.wikispaces.com
Impairment resolves by 70% of the maximum possible within 12 weeks

*For people with an intact corticospinal tract*

Byblow, Stinear et al., *Annals of Neurology*, 2015
Proportional Rule

Motor cortex activity also recovers by 70% over the same time course.

*Proportional resolution of impairment is not related to therapy dose*

Byblow, Stinear et al., *Annals of Neurology*, 2015
Spontaneous neurobiological recovery

Resolution of impairment is not affected by current clinical practice.

The role of therapy is to improve function in the face of the residual impairment.

Goal

Find out if the resolution of impairment and recovery of communication function are predictable, and affected by therapy.
A hint...

WAB scores improve by 70% of the maximum possible within 12 weeks

*Improvement is not related to therapy dose*
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The big questions

Is neuroplasticity enhanced at the sub-acute stage of stroke?

Does the interhemispheric inhibition model hold true at the sub-acute stage of stroke?

Are the recovery of function and resolution of impairment predictable after stroke?

Does impairment resolve spontaneously, while therapy drives recovery of function through experience-dependent neuroplasticity?

Answering these will help us devise more effective therapies.
Thankyou