motor-evoked potentials (MEPs) and immediate effects of focal cortical TMS inactivation

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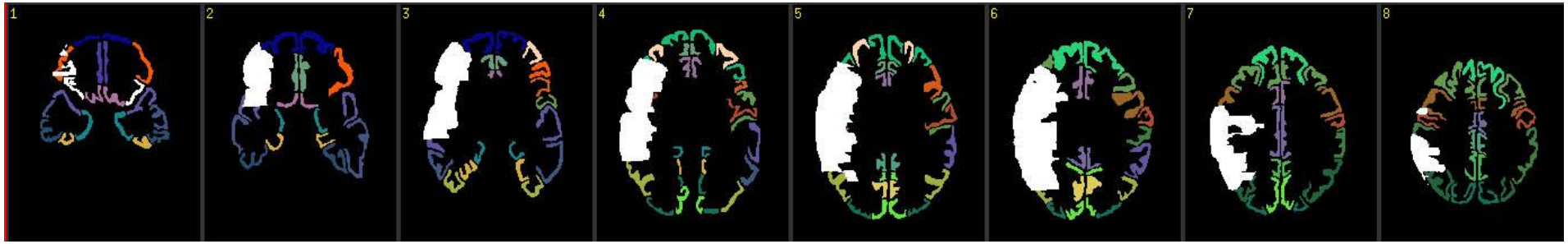


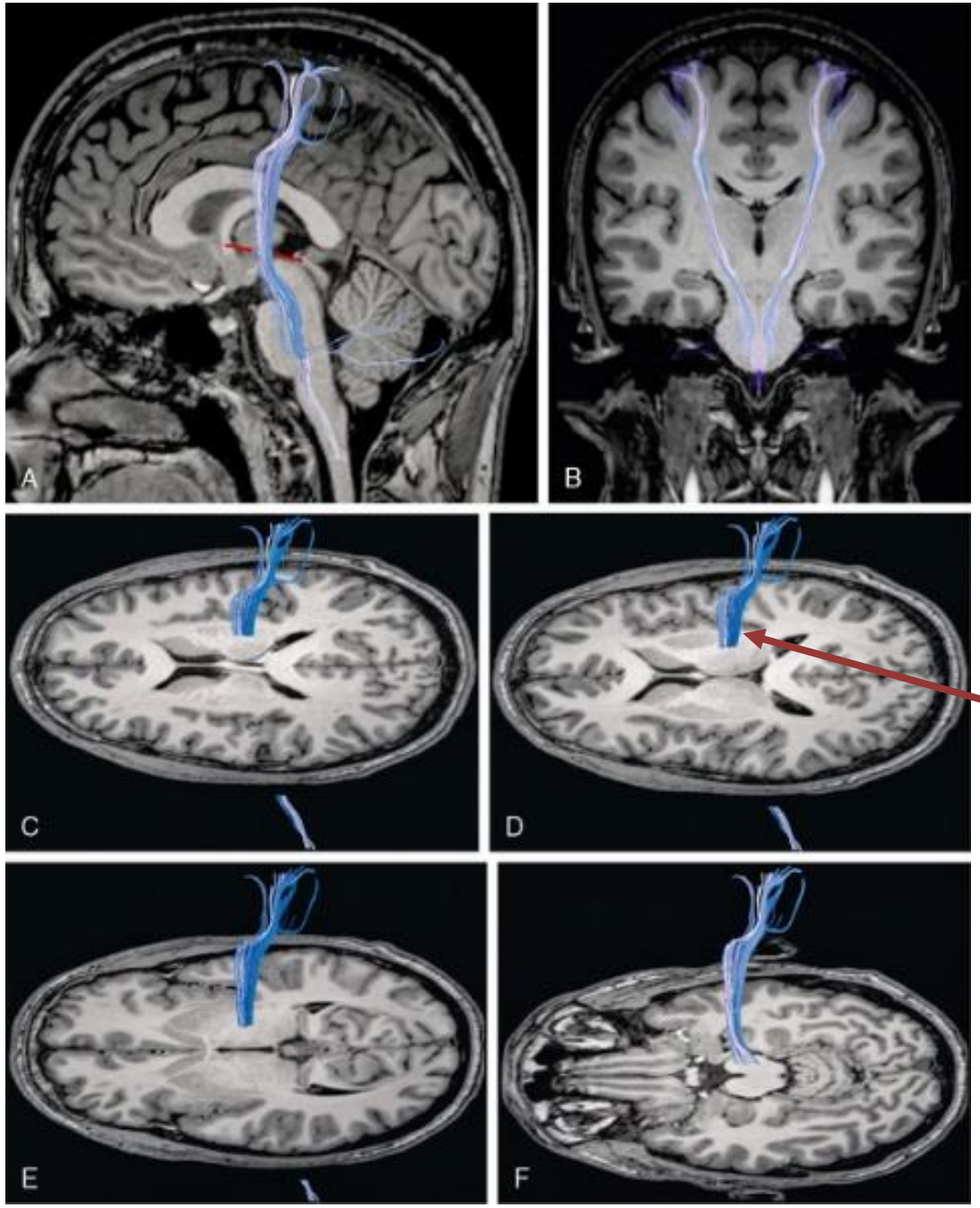
IAPRM Annual Meeting 2022 – Pre-Congress WS

lecture overview

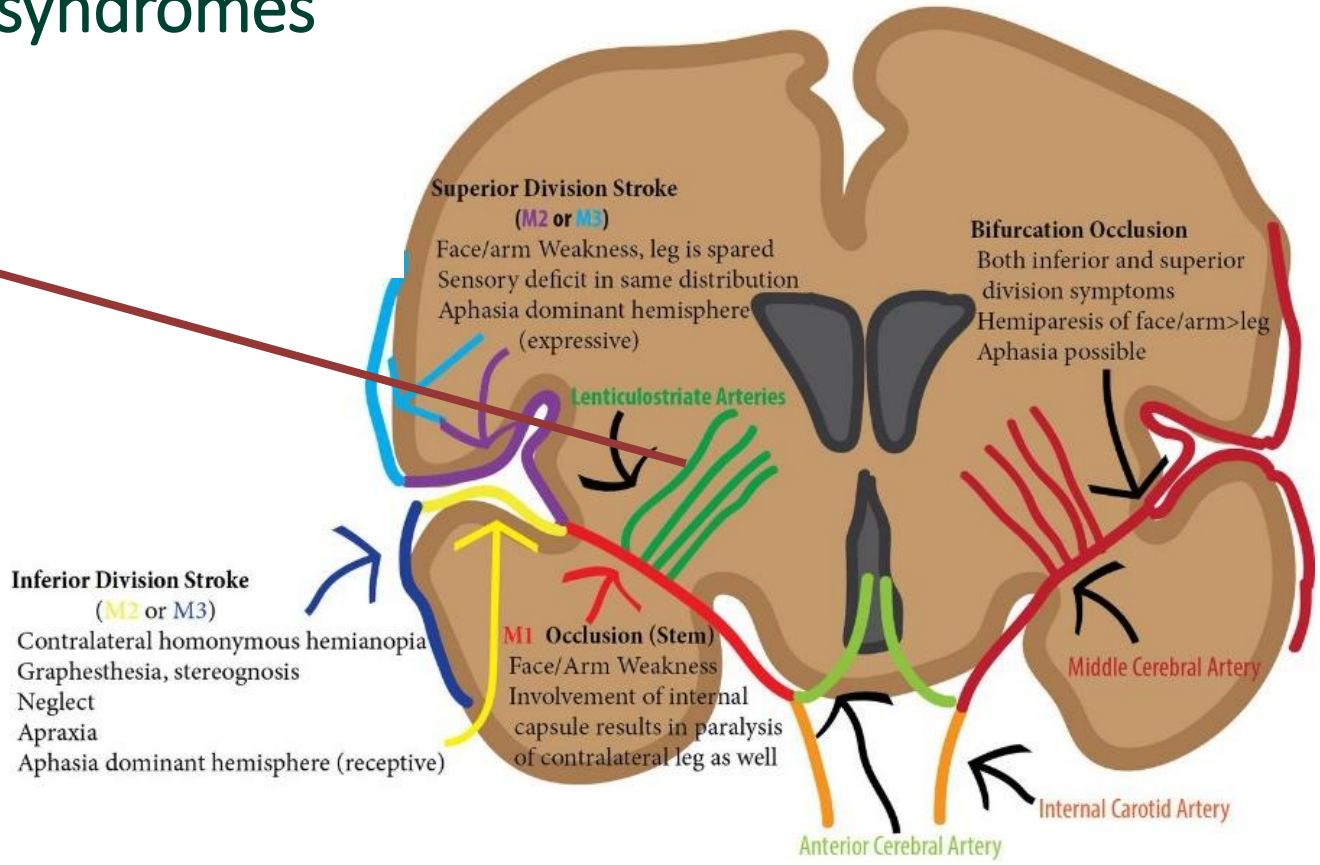
1. MEPs contribution to outcome prediction (PREP2)
2. MEPs contribution to clinical decision making in light of the bimodal hypothesis of plasticity
3. focal cortical TMS inactivation as a biomarker

Brain Rehab Lab
Department of Neurological Rehabilitation, LRMC

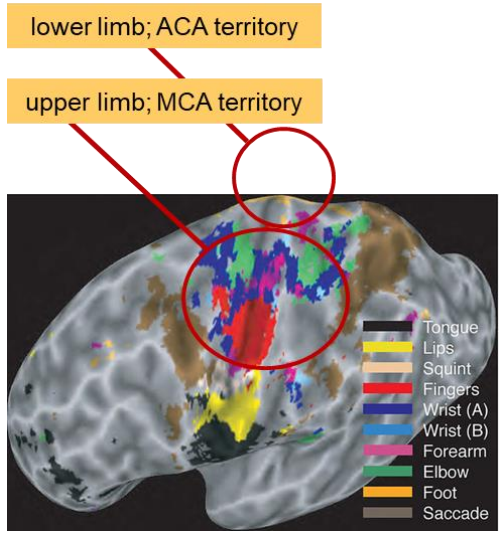
among the descending motor pathways the CST is of crucial importance



MCA syndromes



what factors affect recovery ?



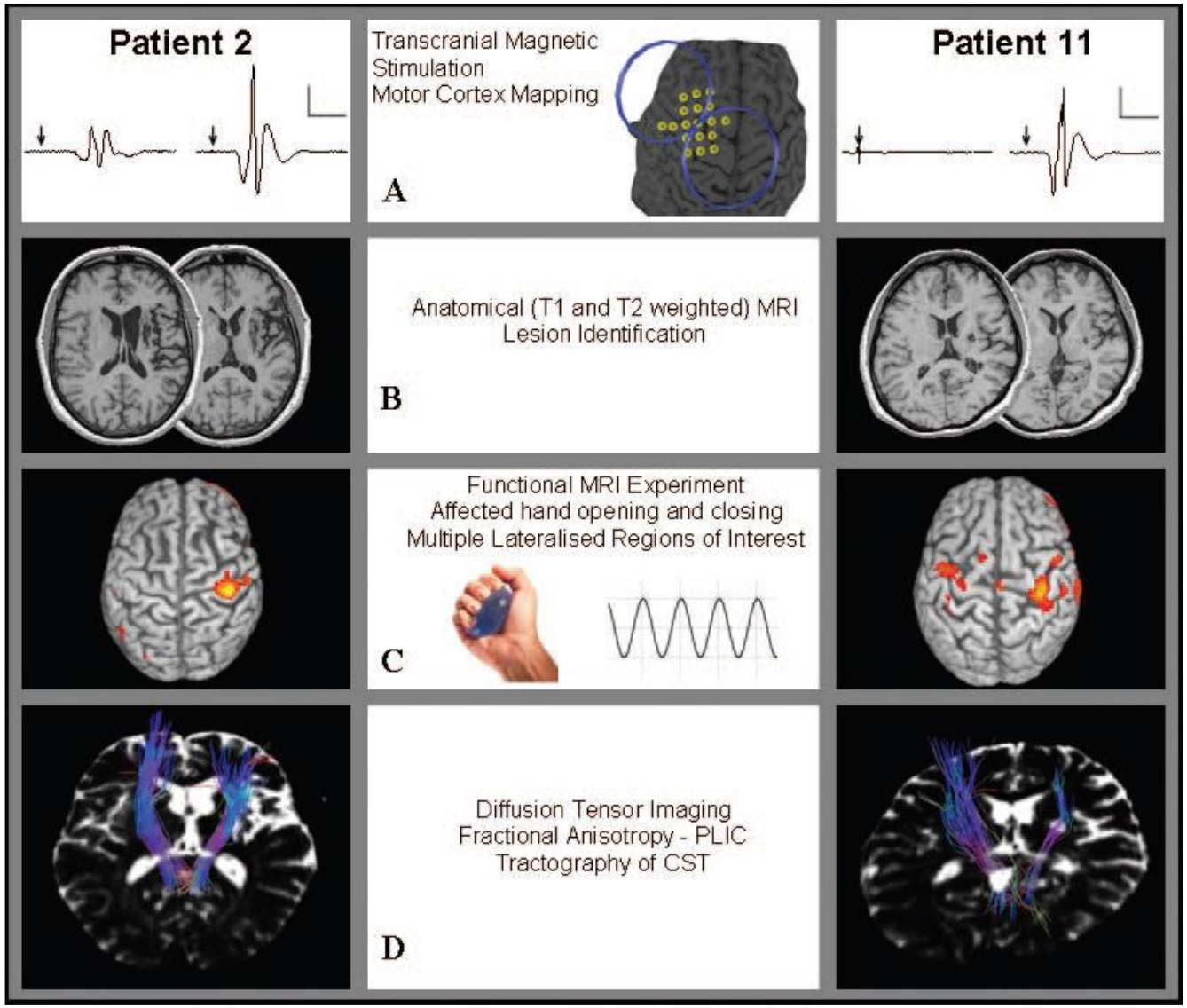
- past research found that most recovery at the impairment level (as judged by the FMA-UE test) is obtained in the first 4-6 weeks after stroke onset.

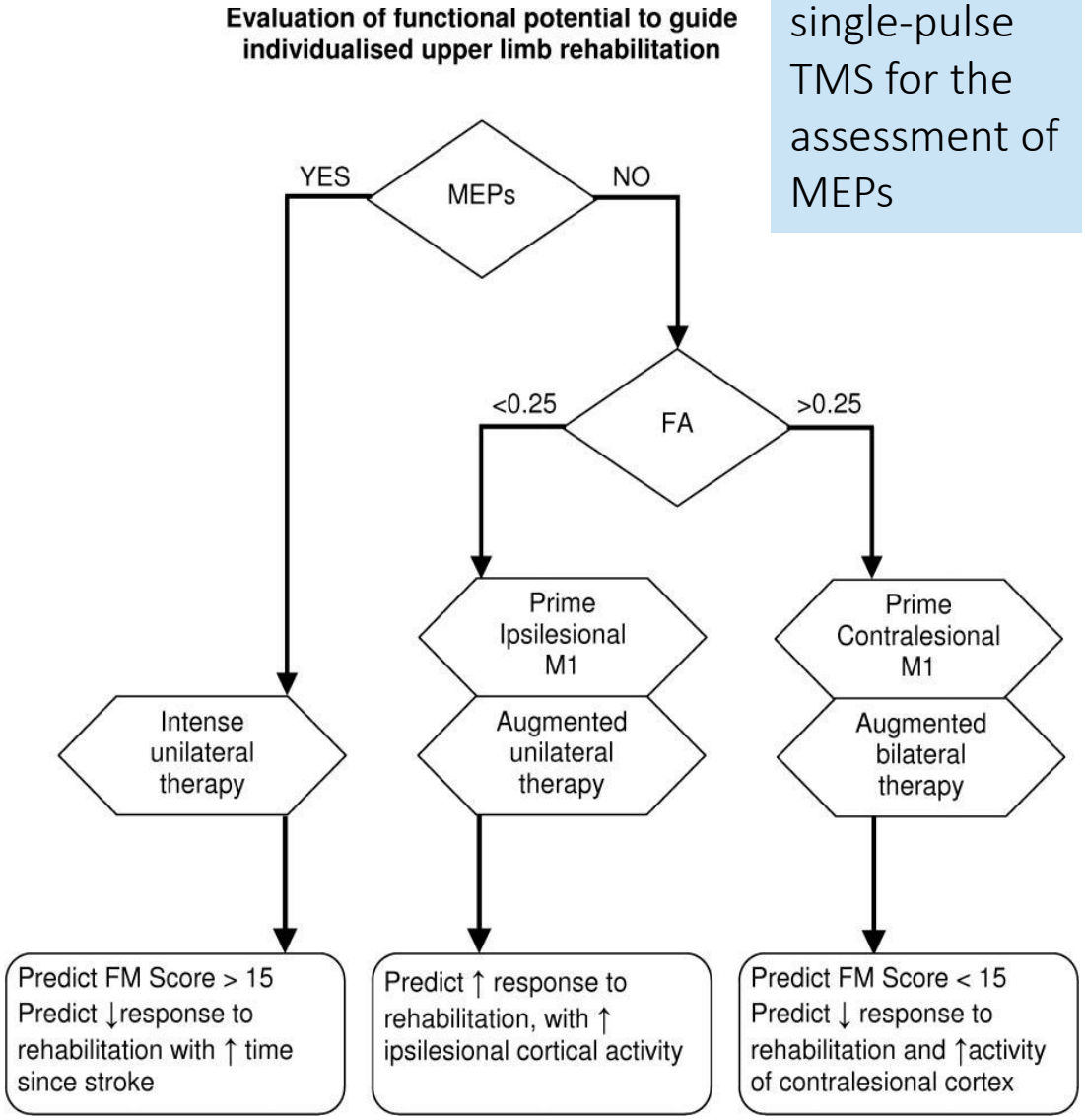
- claims have been made that this recovery depends on biological factors operating in a short time window, with little or no impact to the rehabilitation patients receive in current clinical practice.

- support for this claim is said to derive from the PRR, as it enables calculation, shortly after stroke onset (before active rehab starts), of the final outcome. $\Delta FMA = FMA_{3m} - FMA_{1\text{-}3d} = 0.7*(66\text{-}FMA_{1\text{-}3d})$

  (Prabhakaran,…, Krakauer, NNR 2008; Winters,…, Kwakkel, NNR 2015: $\beta = 0.78$)

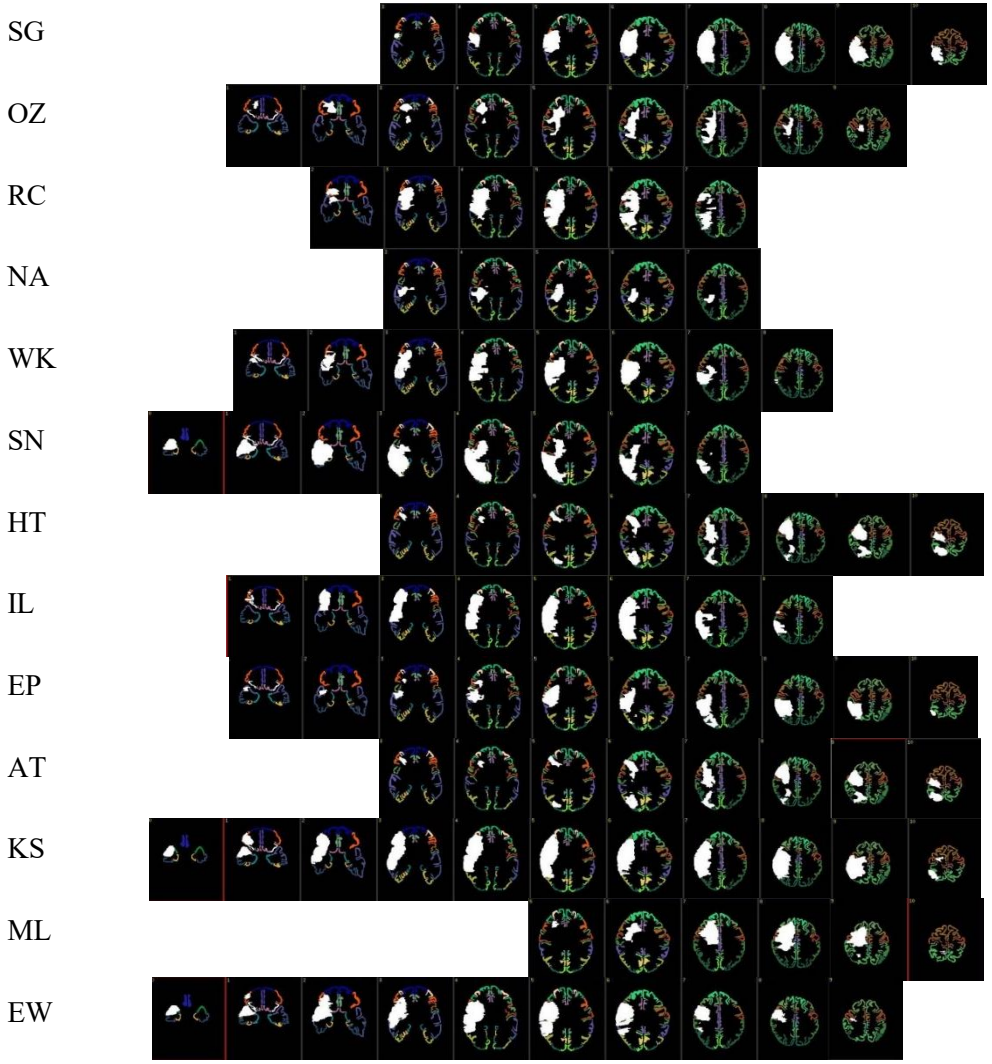
functional potential in chronic stroke patients depends on CST (Stinear et al., Brain 2007)





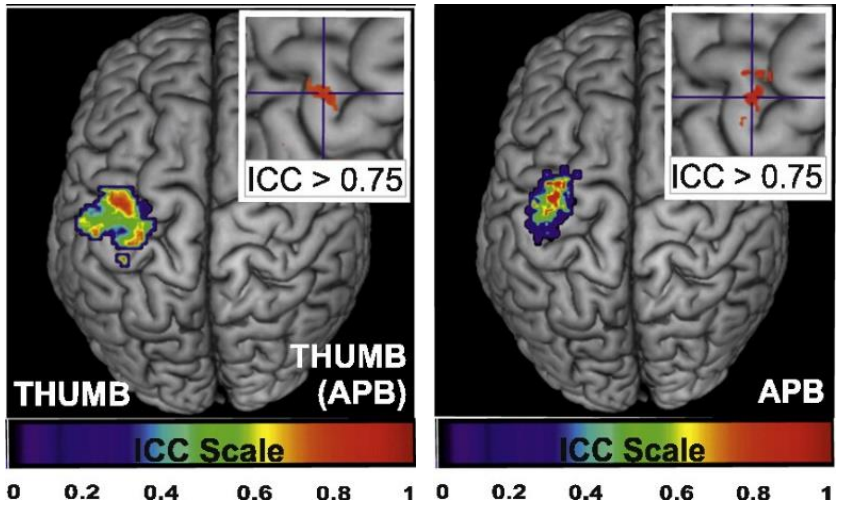
single-pulse TMS for the assessment of MEPs

a note on the importance of lesion analysis



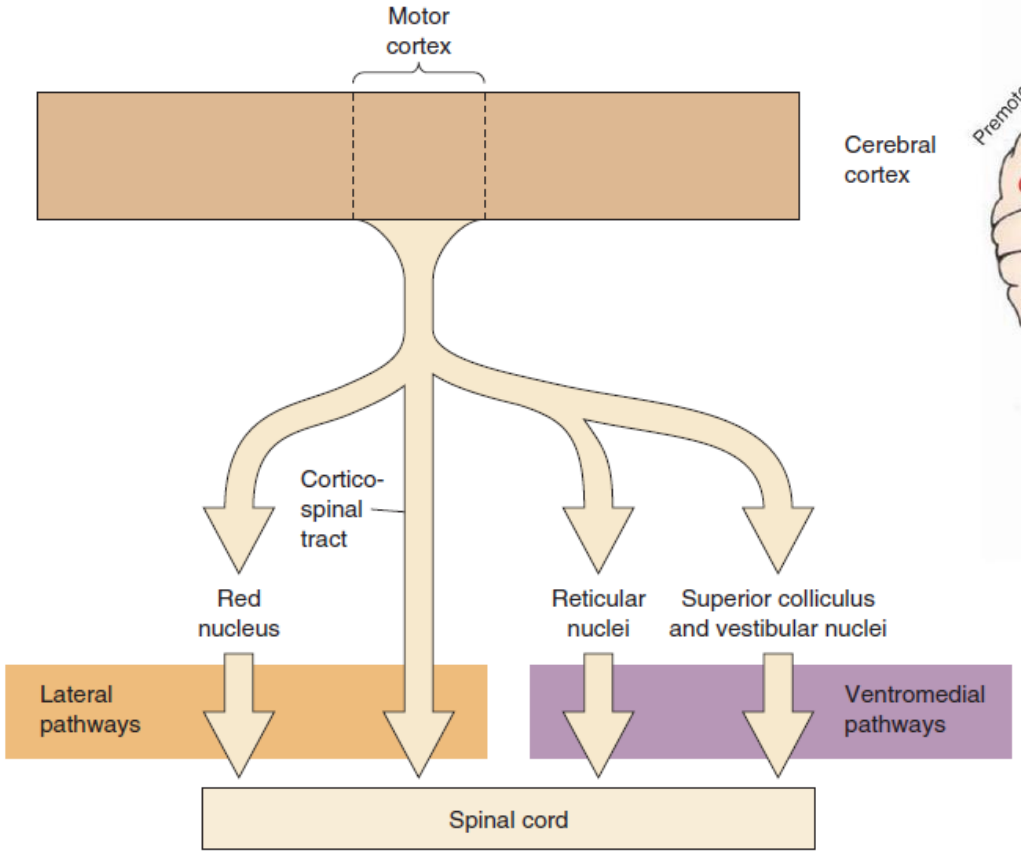
fMRI

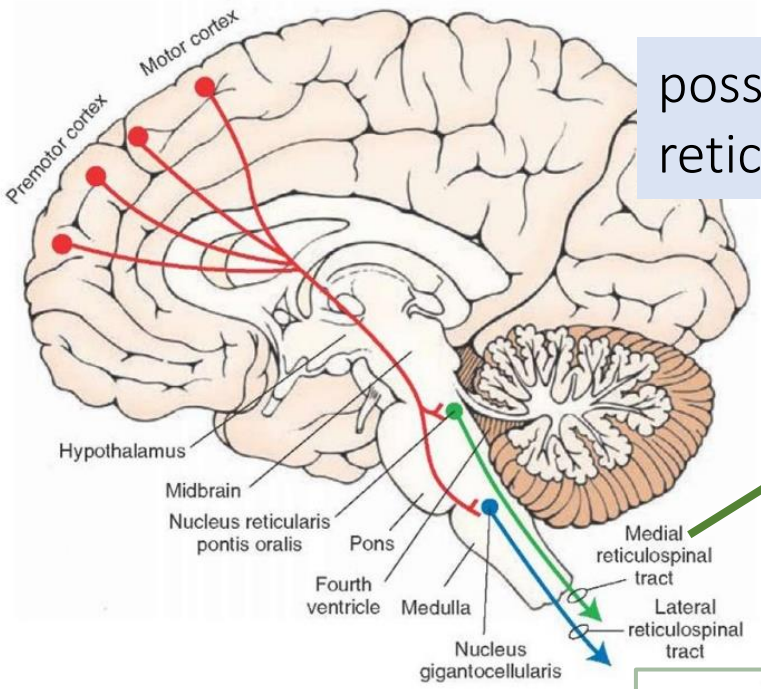
navigated TMS



- lesion studies point to causal relationships between focal damage and patients' symptoms and disclose important info on the functional architecture of the brain
- lesion characteristics exert a dominant effect on the severity of motor impairment following stroke and on the likelihood of obtaining natural and treatment-induced recovery
- information obtained from lesion studies and functional imaging paves the way to development of novel NIBS / EEG-BF rehab therapies

can other descending tracts replace the damaged CST ?

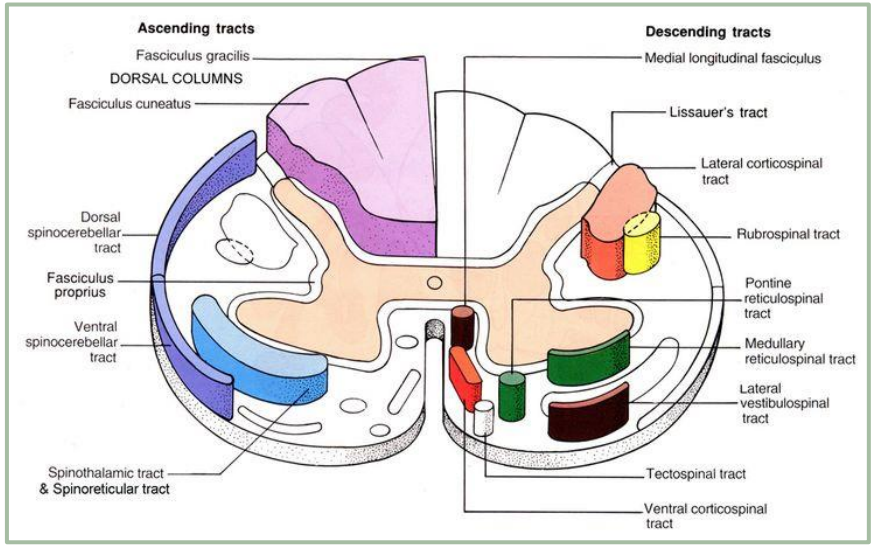




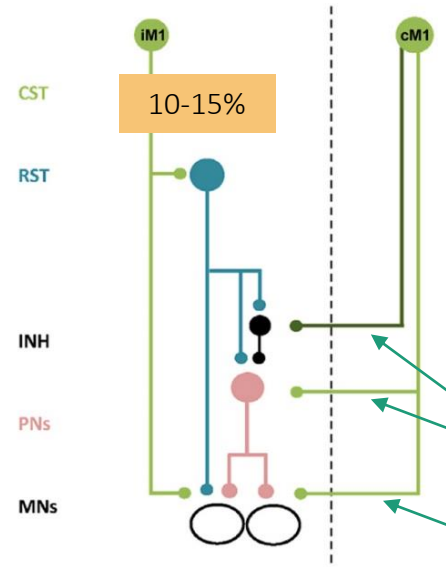
possible role for cortico-reticulo-propriospinal tracts?

ipsilateral

bilateral



Ipsilateral motor pathways after stroke: implications for NIBS (Bradnam, Stinear & Byblow, *Front Hum Neurosci* 2013)



(!) stratification based on CST lesion load and MEPs

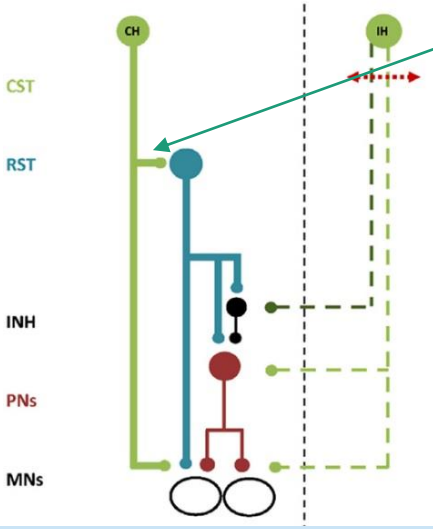
bilateral neural control model

commands to PNs, INHs via i-RST and c-CST
→ balanced input to spinal αMNs

disynaptic (indirect) CST; rubrospinal, tectospinal

monosynaptic CST - direct activation of αMNs

cortico-reticulo-propriospinal pathway (CRPP)



severe stroke → disruption of facilitatory and inhibitory descending inputs from lesioned hemisphere + up-regulation of c-hemisphere excitability → hyper-excited PNs → impaired motor control of the paretic upper limb

baseline
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contralesional inhibitory NIBS

mild impairment
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severe impairment
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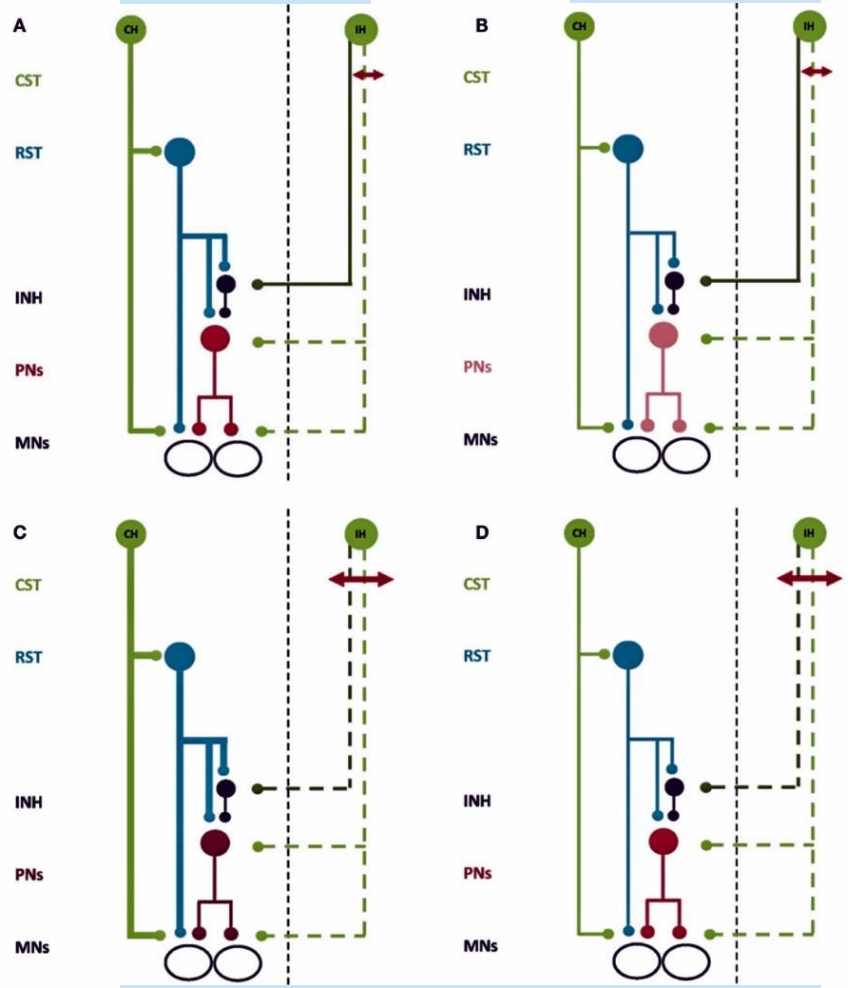

CST- corticospinal tract
RST - reticulospinal tract
i/c - ipsi/contra-lateral

INH - inhibitory interneurons
PNs - propriospinal neurons
MNs - alpha motoneurons

- line thickness - relative excitability
- all projections except INH - facilitatory

mild impairment – positive effect
severe impairment – negative effect

thanks for your attention

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link to downloadable pdfs: Nachum Soroker (researchgate.net)