



## LECTURE

# Post-stroke cerebral reorganization

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### Abstract

In 1898 the Scottish neurologist Byron Bramwell described “A Unusual Case of Aphasia” an autopsy-documented infarct affecting Broca’s area in the hemisphere dominant for speech and language. His case had aphasic syndrome of brief duration. Few examples were reported prior to 1973. Thereafter, the short clinical course has become well-recognized, especially with modern brain imaging. At first considered exception to the rule, then as examples that Broca aphasia is less important than the Wernicke syndrome, criticisms have slowly faded as a similar short-lived syndrome can occur from focal infarcts for Wernicke aphasia as well.

Sensory and motor syndromes from circumscribed infarcts are also now well-known. In large clinical trials the sensory component frequently has a short clinical course. Focal motor syndromes may present with a functionless limb, appearing normal on examination weeks later. For larger Rolandic infarcts or from war wounds, the classical syndrome of dystonic hemiparesis slowly emerges regardless of whether the injury is high or low in the rolandic system. These disparities in outcomes suggest a certain layering function in the Rolandic motor system, the major injuries allowing more primitive motor function to emerge, the perilesional functions or functional reorganization being prevented by the large size of the injury. By hemispherical functional magnetic resonance imaging of unilateral rolandic infarcts provides further support for perilesional activation: a focus of activation predicts functional improvement within 30 days in some patients; why only in some remains unknown.

Arguments still exist whether focal infarction in the calcarine cortex itself can be associated with improvement in the

visual fields, but there is little argument that a short clinical course may follow anopia from precuneus infarction. The classical disconnection model for alexia now seems established as a volumetric effect, by disruptions of trans-callosal and ipsi-hemispherical pathways.

Formerly-acceptable simplistic explanations of peri-lesional edema may have been satisfactory when autopsy documentation was the limited data source. But high-quality modern imaging now also prevents speculation of extremely superficial cortical infarction, or absence of penetration of the lesion into the white matter. In retrospect many of von Monakow’s cases described as ‘diaschisis’ were among these syndromes. If by diaschisis he meant disturbance and remote structures from those damaged, he may have underemphasized the importance of functional reorganization tissue surviving a focal infarct.

Focal tissue disruption from infarction or trauma is but one model testing functional reorganization: Transcranial magnetic stimulation maps of Rolandic convexity areas serving a limb to be surgically excised has demonstrated the area post-surgery to have shifted its function serve adjacent body parts. Ingenious studies of local limb anesthesia from arterial blood pressure cuff causing temporary ischemia has demonstrated the cortical reorganization begins almost immediately, and re-establishes its prior organization after blood flow is restored and the anesthesia fades.

Not merely perilesional function, but regional cerebral blood flow (rCBF) alterations may create the appearance of focal syndromes. Infarcts in the capsular genu has interrupted

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neurotransmitter pathways important for frontal lobe responses, creating a blunting of behavior characterized as "strategic infarct dementia". A dysphasic syndrome has also occurred from pulvinar infarction, blunting rCBF responses over the ipsilateral parietal lobe.

For many generations, clinicians have been taking credit for the improvement following focal lesions, assuming it related to our interventional efforts. More attention to the occur-

rence of what appeared to be compensatory mechanisms could allow insights into their control and augmentation. We could take our inspiration from the knowledge by designers of Voyager 2, allowing them to reprogram some of the

hardware with the spacecraft well beyond Pluto. We can look forward to the day when we may achieve similar restoration of performances to allow the brain to achieve its maximum potential after focal injury.