Dynamics of language reorganization after stroke
Cornelius Weiller

The concept of brain plasticity may have arisen through the observation of recovery of lost function in patients with a persisting lesion in the brain, e.g.; an infarct implying the substitution or support of the altered function by other brain regions. This view is supported through another clinical observation of deterioration of the recovered function by another infarct, e.g. in the contralateral hemisphere. In principle, this hypothesis is assuming a more or less strict functio-anatomic correlation of brain regions. Von Monakow rather took the holistic standpoint, i.e. the brain functions as a dynamic organising entity, when formulating his theory of diaschisis. Diaschisis means a temporary dysfunction of a widespread network through a lesion at one part and recovery without substitutional plasticity. In this theory, the initial symptoms (e.g.; of stroke) are the consequence of dysfunction of the entire network with its infarcted and spared parts. Recovery of function is mediated by normalisation of brain activity in the spared parts of the network. The symptoms remaining after completion of the recovery process von Monakow referred to as the core- lesion. These two opposing basic mechanisms of functional loss and recovery may not completely contradictory but can be assessed nowadays with brain imaging.

In a recent longitudinal study on patients with aphasia, who were serially scanned at least three times from the first days after stroke until up to one year, we derived a model with three phases of reorganisation afterstroke (see Figure 1).
**Figure 1:** Three phases of brain reorganisation in the language system in 14 patients with aphasia. Patients were scanned three times (Ex1, Ex2, Ex3) with a comprehension task. The language effect is displayed (sentences with or without violations contrasted with reversed speech). Note the different time activity curves in both hemispheres, with a biphasic pattern in the right and a monophasic increase in the left. C denotes the normal pattern in 14 healthy controls.

In an initial period of “shock”, with almost complete abolishment of language function, there is little if any activation in language areas despite complete recanalisation of the feeding middle cerebral artery. The exact length of this period varies and has to be precisely assessed (in our current data it lasts from hours to a couple of days). In the second, subacute stage (around 8-12 days after the insult), there was massive activation in the entire (remaining) language network, with a temporary most pronounced activation in the right hemisphere. Activation in this phase included areas, which were not activated initially. During this stage language function starts to recover, sometimes suddenly and to a remarkable degree. This is followed by a third phase of consolidation, with steady but subtler further improvement of function. During this last period, the activation pattern remains qualitatively the same but activation is reduced in the individual knots of the network, returning to a more normal pattern, with peak activation returning to the left hemisphere. Not all patients transit from stage 2 to 3, some keeping their abnormally increased activation pattern. We assume that repetitive bouts of stages 2 and 3 may occur during the further course perhaps treatment induced.