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"Loss of function during stroke is due partly to neuronal death in the infarcted tissue but also to cell dysfunction in the areas surrounding the infarct. These areas encompass the part of the underperfused penumbra that survives the insult, the non-ischemic peri-infarct tissue, and remote (including contralateral) brain areas that are connected to the area of tissue damage."

Wieloch T, Nikolich K. Mechanisms of neural plasticity following brain injury. *Current Opinion in Neurobiology* 2006, 16: 1-7.

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Stroke Educator, Inc. is committed to educating the wider public about stroke and the 50 state "Aim High for Aphasia!" Aphasia Awareness campaign.

Aphasia Insights!

Aphasia Recovery: The Illusion of *"Reaching One's Plateau"* for People with Aphasia (PWA).

By Tom Broussard, Ph.D.

I had a stroke and aphasia on September 26, 2011. I was an associate dean at The Heller School at Brandeis University when I fell down on Main Street, Walth am, MA.

I lost my langua ge and could not read, write or speak well.

From the start, my family assumed that I would get better at some point. They could tell that I was speaking relatively well and hoped that there was some pathway to recovery for all my language modalities (reading, writing and speaking). It takes a while to become aware of one's deficits, and people with aphasia spend a long time living unaware of those altered abilities.

My nurse had said that it would take at least a month for the brain to 'settle down' and heal its internal 'bruises', stopping inflammation and absorbing the dead tissues. In my case, I had had a hard time sleeping, thinking, talking, remembering, or doing almost anything other than trying to do those same things badly. But within a month, I was able to sleep better and think somewhat more clearly and more consciously about the problems I was experiencing.

That was the start of my recovery as my brain was in the process of repairing itself. I got better that month but without any therapeutic interventions. The natural healing helped reduce the swelling, and began the process of cell repair; restoring the flow of blood to the affected cortical areas.

This phase is known as a period of spontaneous recovery (6 to 12 months) with the reversal of diaschisis (Greek meaning 'shocked throughout'). The

primary mechanism of diaschisis is a loss of information (cell to cell transmission) from the part of the brain where the stroke occurred to distant cells.

The decrease in neural firing causes the cells' synaptic connections to weaken and lose the connectivity between the affected/damaged area and the distant parts of the brain that are connected but not receiving enough blood to operate (Wieloch & Nikolich, 2006).

The remaining phases of recovery include changes to the *existing* neuronal pathways and the process of neuronal reorganization with new dendrites and synapses.

These processes take much longer and require persistent language activities and personal effort to induce plasticity and help create *new* brain matter for long-term recovery and learning (Wieloch & Nikolich, 2006).

Recovery takes place using two completely different mechanisms: one by nature (and relatively fast) and the other by effort (and imperceptibly slow). The first (spontaneous) phase starts fast and provides more easily seen evidence of increased health and improvement in a (relative) short amount of time.

The second phase starts slowly and can overlap with the first phase. Once the first, spontaneous phase ends, it leaves observers (clinicians, family or friends) with the *impression* that progress is slowing and coming to the end, when is it in fact, a lifelong feature (not a bug) of the neural mechanism of rebuilding the networks of the brain.

When people with aphasia exit the first phase and enter the next, they are often labeled as having "reached their plateau" by *comparing* the faster, spontaneous and natural segment of healing to the slower (but indefinite) process of creating new brain matter at the cellular level.

Unfortunately, once language improvement has been seen (by clinicians and others) to be demonstrably slowed, it makes it even more difficult to continue with formal (or funded) therapy with the rules currently in place.

Too many people assume that slowed improvement after the initial thrust of the first stage is an indication that recovery and learning are stopping. But like a three-stage rocket ship, the first stage has the power to lift the entire ship only to separate at a higher altitude where the next stage begins; shifting to a slow and steady ascent towards a life-long journey of continued language improvement.

The biggest difference between stages is that the final push uses a fuel that is *self-sustainable* by people who can generate the fuel themselves using motivation, effort and purposive activities that create *more* activities, *more* neuroplasticity, and as a result, *more* fuel.

A person with aphasia who has learned a new game, a new story, a great joke, or met a new friend, is learning every day. It is an impossibility *not* to learn, given that every single neuronal impulse is transmitting new information to other (continuously growing) cells that have the ability to learn.

People with aphasia cannot *not* learn at any stage. That is the illusion of having "reached one's plateau" when there are still so many mountains able to climb.

Signed: *The Johnny Appleseed of Aphasia Awareness*

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