PFOs

See my separate presentation #1 on my stroke PFOs

My MRIs show I have had 3 strokes and the heart imaging showed a 13x3mm hole (PFO) that was likely the cause of those strokes (since plugged by a 30mm disk on Dec 9, 2025), allowing the unfiltered desaturated blood to cross over to the arteries that supply my brain.

About 25% of people have a PFO – usually small and not clinically significant. My PFO is considered 'large' though. Some people get migraines from their PFOs as even small ones can allow little clumps of blood clotting junk to get to the brain, temporarily blocking the blood supply to areas and then dissolving without leaving a permanent scar for an MRI to detect. I wonder if some of my episodes of suddenly feeling dizzy/crappy were caused by my PFO deep in my past.

Unfortunately PFOs cannot be diagnosed by the popular new MRI preventative health scanners that are all the rage with the wealthy 1%ers – the only way you can detect a PFO is via a complex echocardiogram with bubble injection via IV under the guidance of a cardiologist and then it needs to be confirmed and further defined by a nasty TEE imaging procedure taken by an ultrasound from inside your stomach, and then finally via a 3rd imaging procedure via catheter from inside your heart.

Motor Cortex Stroke Impairments

[Note: generally the right side of the brain controls the left side of the body, and so on]

I had my October 10, 2025 stroke in my right pre and post central gyri (motor and sensory cortex regions of the mid-brain). After the initial left side and facial paralysis abated I was left with a numb left foot, weakness in my left quadricep, some left hand coordination issues, some speech issues, a few visual impairments, and some right side spasticity issues with my arm and hand (see section below regarding Disinhibition and Interhemispheric Imbalance). My balance (my body's ability to maintain equilibrium using proprioception and vestibular input) is now relatively poor overall.

None of these impairments have gone away at the 5 month point but they generally have reduced over time, although some of these issues appeared weeks and even months after the stroke and that is my main point: expect the unexpected and don't think recovery will be a linear progression back to 100% functionality.

My left foot gets better and worse depending on the day. When it is bad I try and ignore it – it is not painful, it just feels strange like when you pull off a tight ski boot at the end of a big ski day.

My left hand is pretty much 100% normal now so piano playing is fine.

My left quad doesn't fire smoothly so I have a floppy left foot that has a delayed extension and then a delayed retraction. I just need to be mindful that I can trip more easily and can't walk as fast as I used to. But I can bike fine.

My poor balance doesn't affect me much on flat, smooth surfaces but when I have to walk on a sloping surface I can really struggle. When I close my eyes I can't really orient myself well – these are all things I am working on with my rehab program. But again, with biking I seem fine.

I have some speech related issues that can impact my ability to say some words and complete sentences – nothing major but I notice it and sometimes other people do as well. When my face is cold it gets worse and I start to slur my words so there is something going in my brain/mouth connection that we have not diagnosed yet.

In the corner of my vision I sometimes see small objects that are not really there that appear as dark blocks and move out of view quickly. I think something is there that isn't. It doesn't happen too often though and shouldn't impact my driving as it is a subtle impairment.

Nerve Disinhibition After Stroke

My right arm and hand are most likely impacted by the issue of 'disinhibition' as described technically here. The issues started several weeks post stroke which aligns well with this description of the underlying mechanisms …

[Note: This cut and paste from the internet describes my right arm/hand issues]

- Mechanism: Stroke causes a reduction in inhibitory GABAergic signaling in both the ipsilesional (damaged) and contralesional (undamaged) hemispheres. This imbalance between excitation (too much) and inhibition (too little) allows the brain to "re-wire," but also causes "disinhibition" of circuits.
- Motor Disinhibition: In the early, acute phase (days after stroke), reduced inhibition (specifically, a decrease in short-interval intracortical inhibition, or SICI) is common in both hemispheres. This is believed to be a protective mechanism that facilitates early motor learning.

- Maladaptive vs. Adaptive:
    - Adaptive: In the early stage, temporary disinhibition allows for the unmasking of dormant neuronal pathways, facilitating motor recovery.
    - Maladaptive: If this disinhibition persists, it can lead to poor recovery and spasticity (hyper-excitability of the stretch reflex) due to the loss of descending inhibition on the spinal cord.
- Interhemispheric Imbalance: The unaffected hemisphere can become hyper-excitable, leading to an abnormal, high inhibitory drive towards the damaged hemisphere, which impairs recovery.

The Mechanism of Interhemispheric Imbalance:

In a healthy brain, both hemispheres maintain a balanced, mutual inhibition of each other.

Following a stroke, this equilibrium breaks down:

- Reduced Inhibition from Damaged Side: The lesioned, affected hemisphere (AH) loses its ability to inhibit the healthy, unaffected hemisphere (UH).
- Hyperexcitability of Unaffected Hemisphere: Due to the loss of inhibition from the damaged side, the unaffected hemisphere becomes overactive or "hyperexcitable".
- Excessive Inhibition of Damaged Side: The hyperactive unaffected hemisphere exerts an abnormally strong inhibitory influence (transcallosal inhibition) on the damaged hemisphere.
- "Double-Disabled" Damaged Hemisphere: The affected hemisphere suffers from both its intrinsic stroke damage and the excessive inhibition from the healthy side, hindering recovery.

Impact on Stroke Recovery

- Maladaptive Plasticity: This mechanism is often viewed as maladaptive, meaning the brain's attempt to reorganize actually hinders recovery.
- Chronic Motor Impairment: The persistence of this high inhibitory drive (specifically in the chronic stage) is associated with poor motor recovery and weakness in the affected, paretic limb.
- State-Dependent Effects: Studies show this imbalance may be particularly damaging during the preparation and execution of movements, as the healthy side fails to "release" its inhibition, disrupting the initiation of voluntary movement.

I deal with my spasticity with massage and relaxation. The Keppra also seems to supress it. It is getting a bit better over time ...