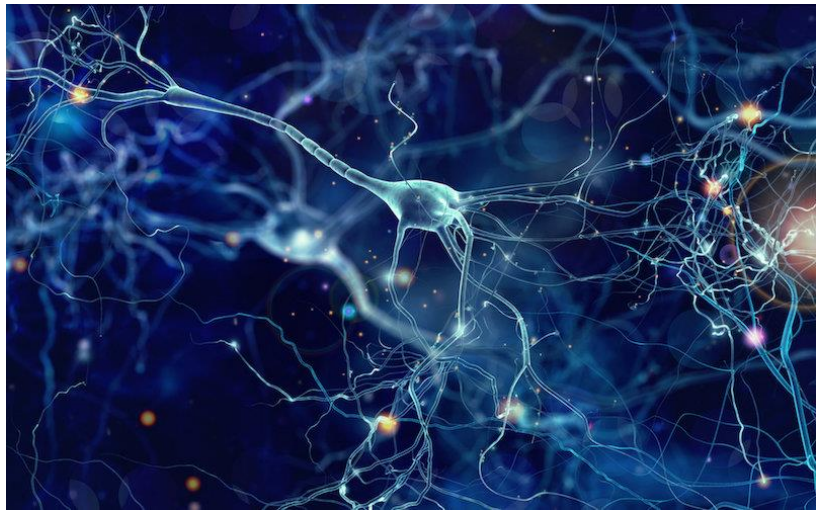


My Stroke Journey

Part 2 – Brain Impairment / Hyper-Excitability

Derek Spratt

Nov 14, 2025

Topics

- My stroke areas
- Functional areas of the brain
- What happens when a stroke occurs
- Hyper-excitability & focal seizures
- MRI imaging
- EEG monitoring

My strokes and resulting brain impairment

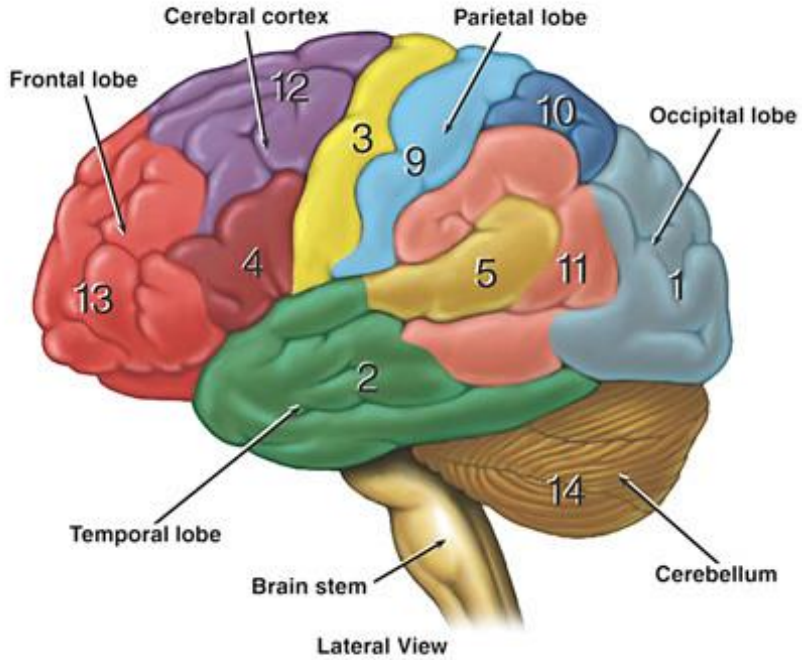
- My stroke areas:
- Precentral Gyrus (Oct 10/2025)
  - Initial left side paralysis and slurred speech, reducing to minor weakness after 36+ hrs
  - Sensory and motor issues (tingling, spasms, ‘fades’, Jacksonian focal seizures, balance, reduced mental acuity, some verbal impairment) 5+ weeks post stroke
- Cerebellum (Oct 4/2007)
  - Loss of vision & balance, nausea for several weeks (undiagnosed)
  - Long term minor balance impairment
- Up to 15% of stroke survivors experience focal seizures and/or hyper-excitability which typically originate at or near the location of the stroke. Most times this is temporary – only 1-2% end up with long term epilepsy

Functional Areas of the Cerebral Cortex

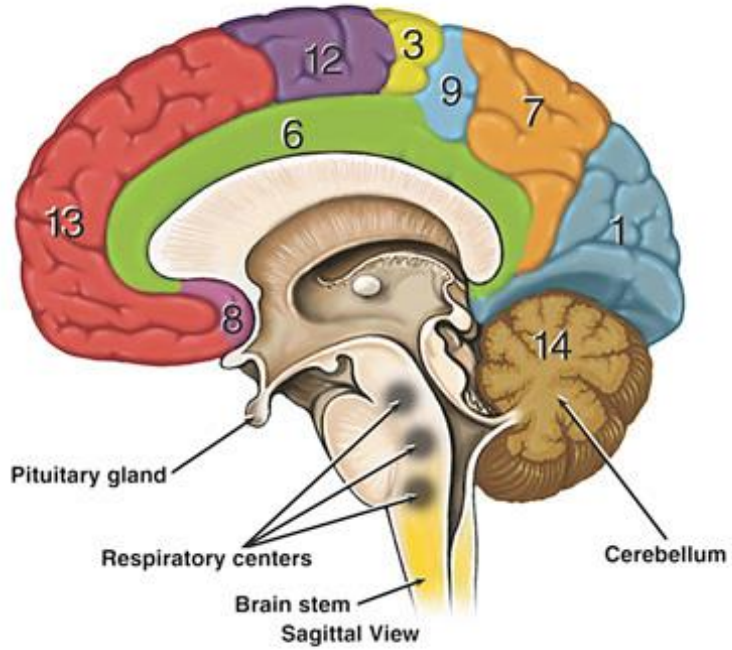
1. **Visual Area:**
   Sight
   Image recognition
   Image perception
2. **Association Area**
   Short-term memory
   Equilibrium
   Emotion
3. **Motor Function Area**
   Initiation of voluntary muscles
4. **Broca's Area**
   Muscles of speech
5. **Auditory Area**
   Hearing
6. **Emotional Area**
   Pain
   Hunger
   "Fight or flight" response
7. **Sensory Association Area**
8. **Olfactory Area**
   Smelling
9. **Sensory Area**
   Sensation from muscles and skin
10. **Somatosensory Association Area**
    Evaluation of weight, texture, temperature, etc. for object recognition
11. **Wernicke's Area**
    Written and spoken language comprehension
12. **Motor Function Area**
    Eye movement and orientation
13. **Higher Mental Functions**
    Concentration
    Planning
    Judgment
    Emotional expression
    Creativity
    Inhibition

Functional Areas of the Cerebellum

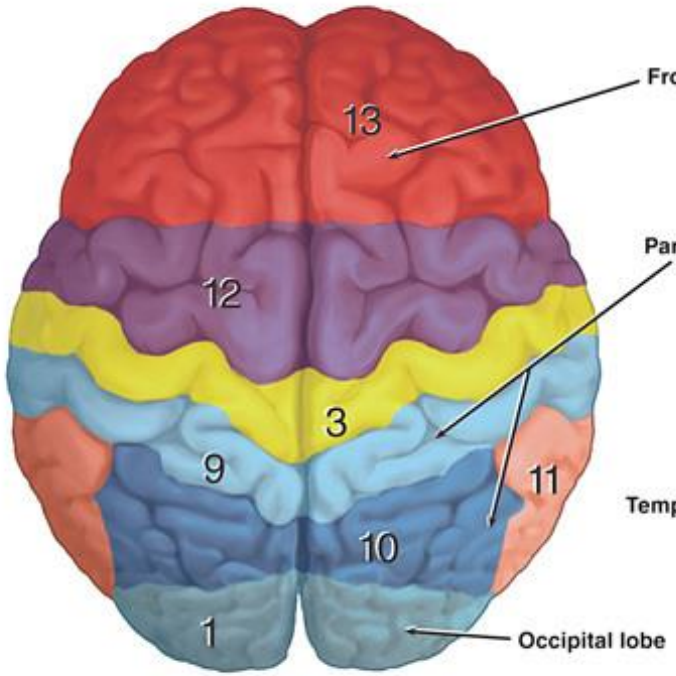
14. **Motor Functions**
    Coordination of movement
    Balance and equilibrium
    Posture



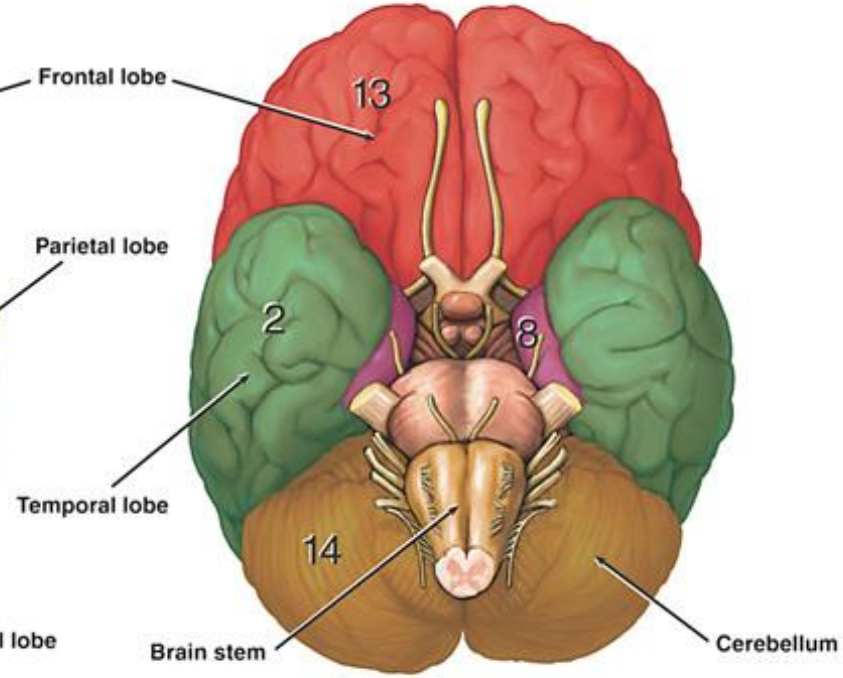
Lateral View



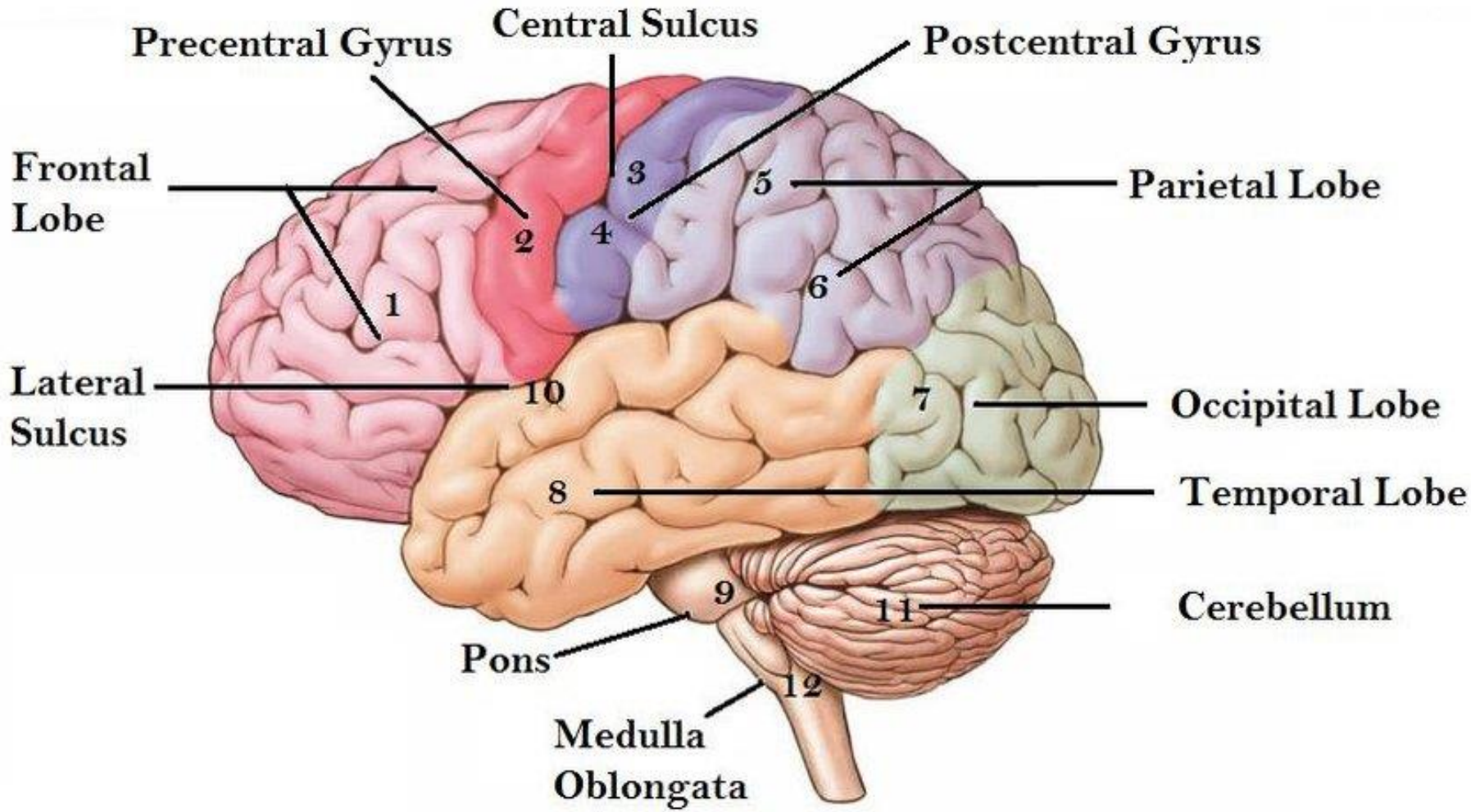
Sagittal View



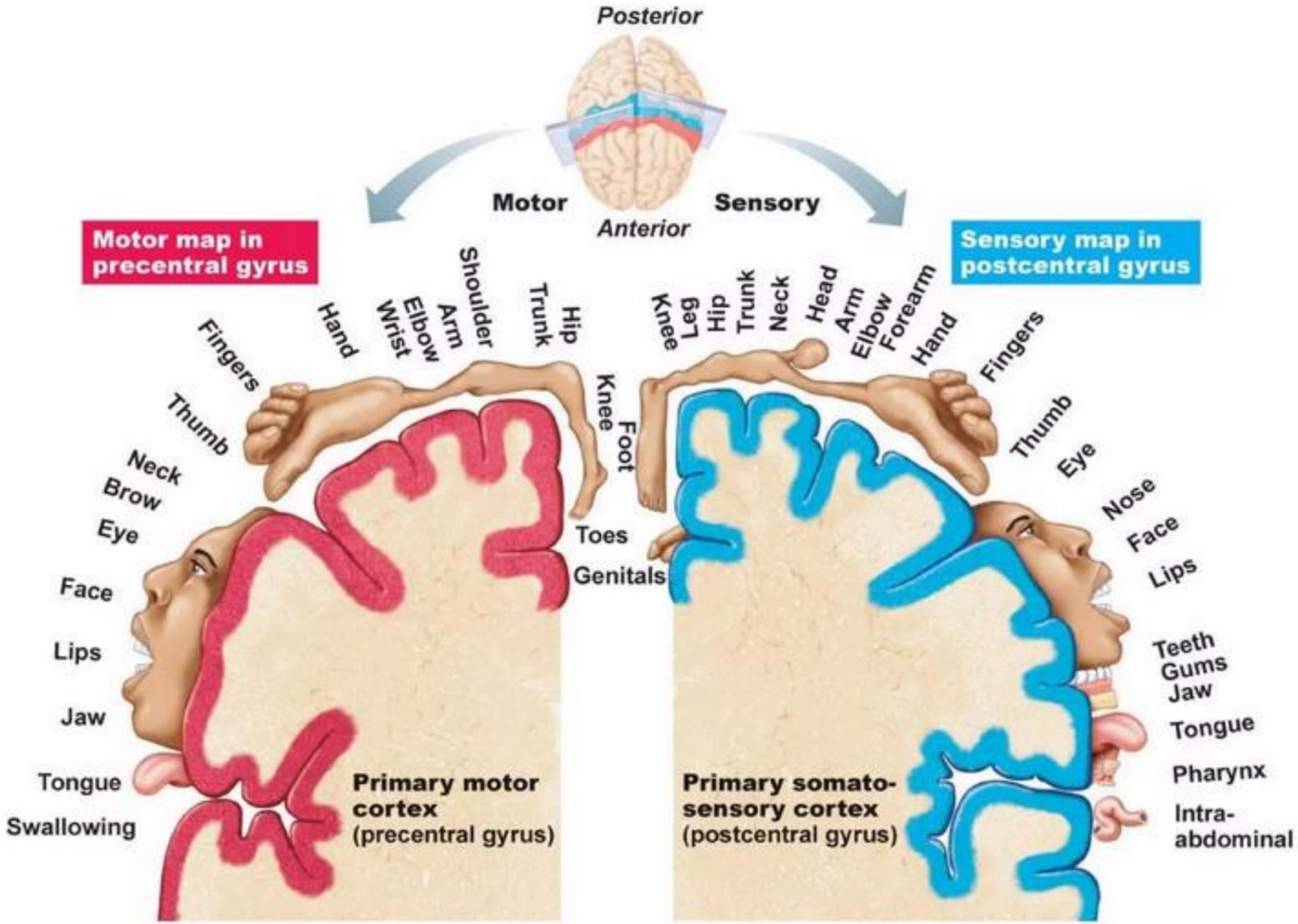
Superior View



Inferior View

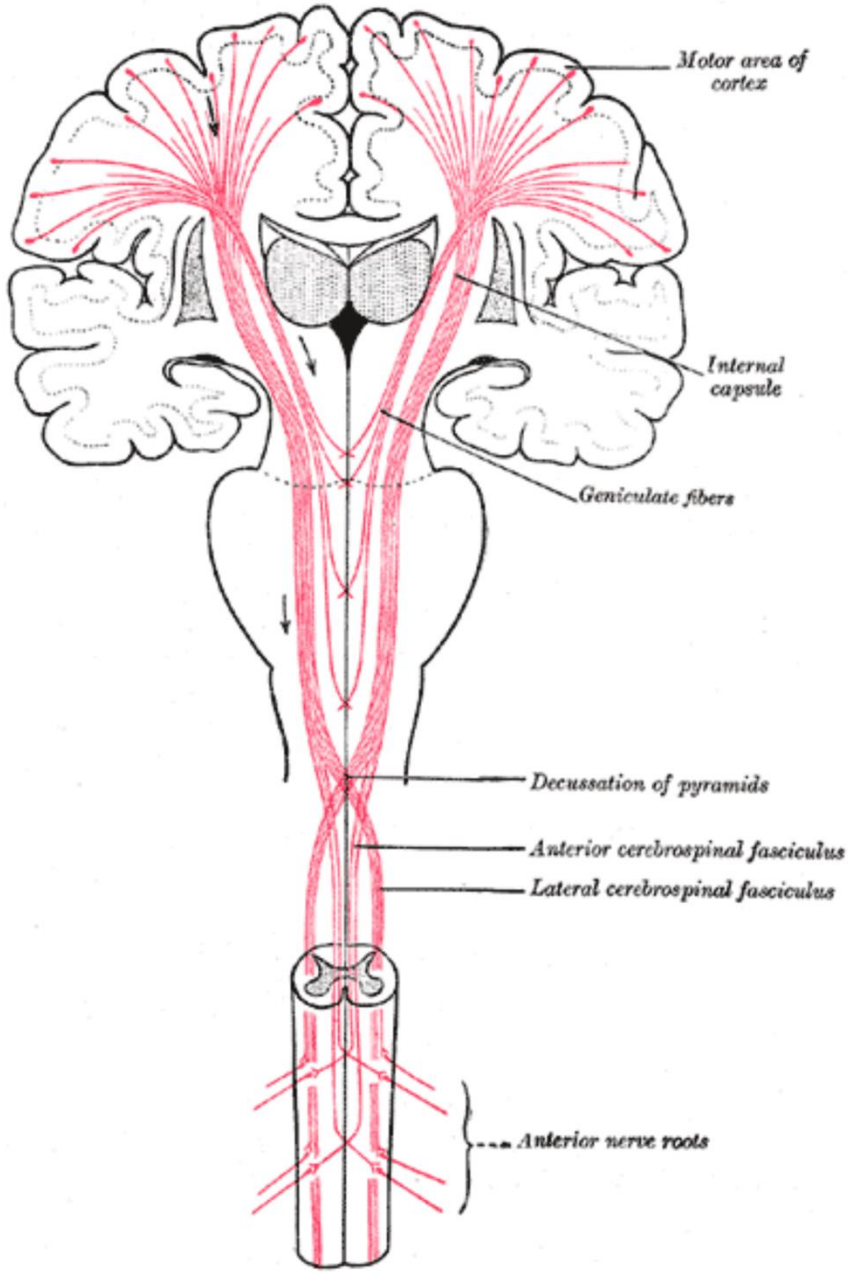

Precentral Gyrus
Central Sulcus
Postcentral Gyrus
Frontal Lobe
Parietal Lobe
Lateral Sulcus
Occipital Lobe
Temporal Lobe
Cerebellum
Pons
Medulla Oblongata
1
2
3
4
5
6
7
8
9
10
11
12

Pre and Post Central Gyrus



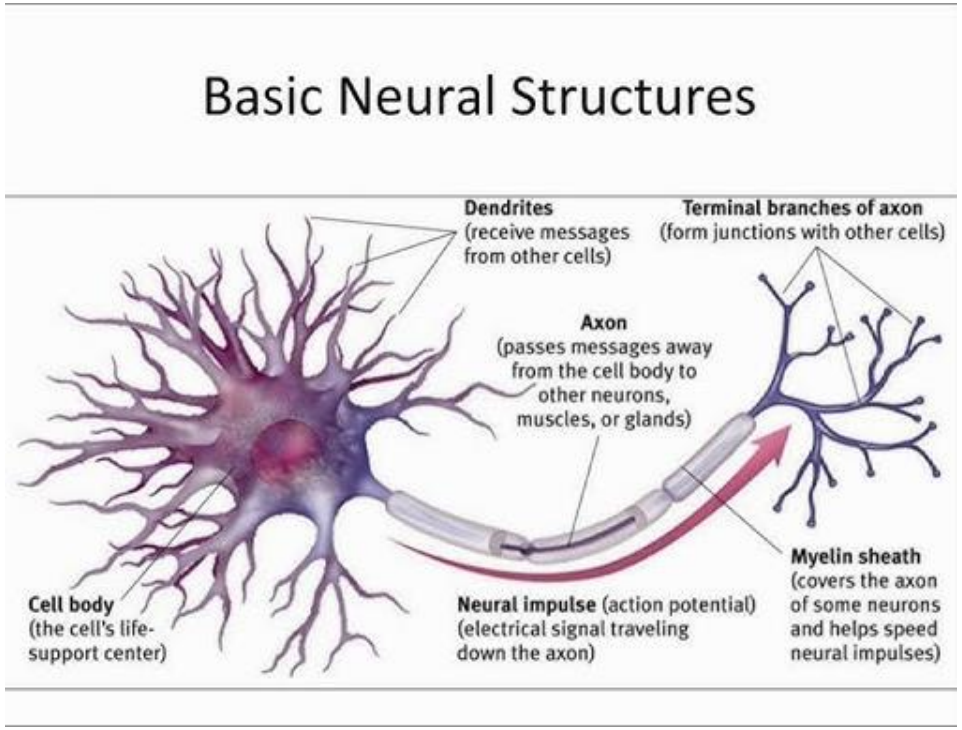
Pre and post Central Gyrus

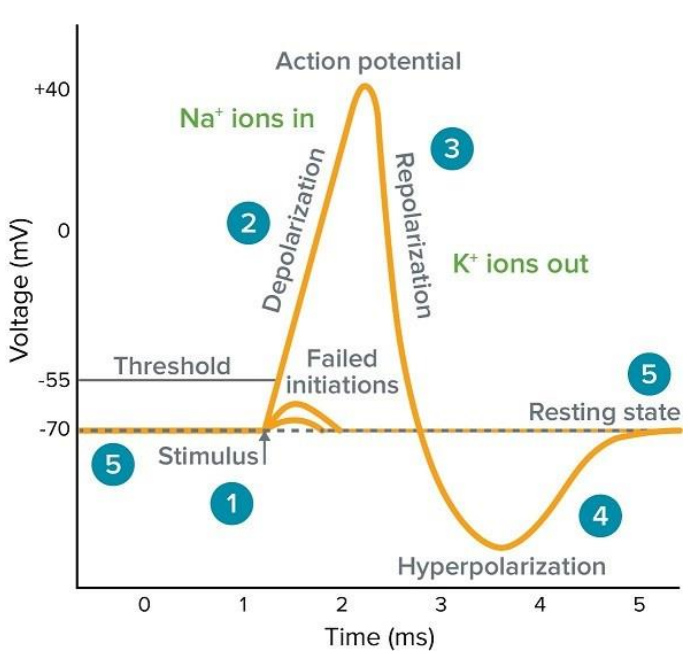
- Provides motor control over muscles and senses (outputs and inputs)
- Most (but not all) neurons cross-over to the opposite side of the body

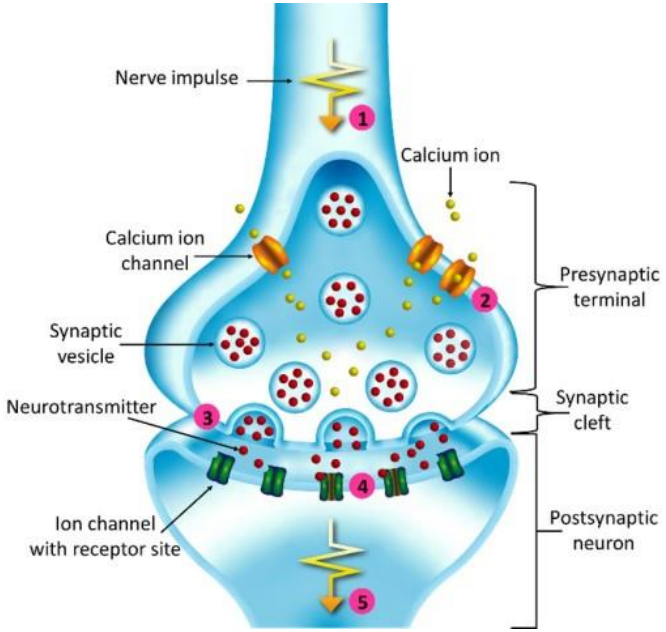


How Neurons ‘fire’

- Electric ‘pulses’ run thru the neurons like wires
- Junctions use chemicals called neurotransmitters to bridge the gap between neurons







What happens when a stroke occurs?

- During a stroke the normal highly regulated electrical activity of neurons is severely disrupted due to a lack of oxygen and glucose. This disruption leads to an initial period of hyperexcitability followed by a prolonged state of hypoexcitability (reduced firing), and eventually, widespread cell death.
- Acute Phase: Excitotoxicity and Hyperexcitability
- The primary event in an ischemic stroke (the most common type) is the blockage of a cerebral artery, leading to a rapid depletion of ATP (energy) in the brain cells. This energy failure causes a cascade of events:
- Ion Pump Failure: The critical sodium ion (Na+) pumps that maintain the neuron's resting membrane potential fail. This leads to an accumulation of sodium inside the cell and the collapse of ion gradients.

What happens when a stroke occurs?

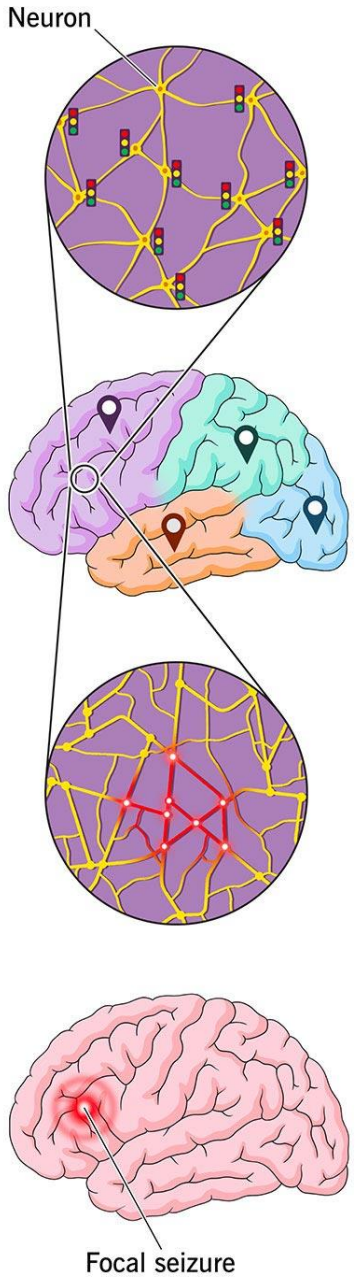
- Massive Glutamate Release: The change in membrane potential and influx of calcium ions cause neurons and astrocytes to release excessive amounts of the excitatory neurotransmitter, glutamate, into the synaptic cleft and extracellular space.
- Overactivation of Receptors: This excess glutamate overstimulates glutamate receptors (especially NMDA and AMPA receptors) on adjacent neurons, causing a massive influx of calcium ions into the postsynaptic cells.
- Cell Death Cascade: This calcium overload triggers a variety of cell death pathways, including necrosis (rapid, uncontrolled cell bursting) in the stroke core and apoptosis (programmed cell death) in the surrounding area (the penumbra).
- Spreading Depolarizations: This initial high excitability manifests as "spreading depolarizations" (or peri-infarct depolarizations) which radiate from the stroke core like a fire, consuming energy and pushing penumbral neurons toward cell death.

What happens when a stroke occurs?

- Subacute and Chronic Phases: Hypoexcitability and Reorganization
- In the hours to days following the initial injury, the surviving neurons in the peri-infarct region (tissue bordering the dead core) enter a period of hypoexcitability.
- Increased Inhibition: A key mechanism for this reduced firing is an increase in tonic (persistent) GABA signaling, the brain's main inhibitory neurotransmitter system.
- Reduced Plasticity: This increased inhibition, while initially potentially neuroprotective, later hinders the brain's ability to form new connections (neuroplasticity) which are essential for recovery.
- Network Disruption: The loss of direct connections to the stroke core also causes damage signals to be relayed throughout connected brain networks, leading to a general disruption in normal synchronized neuronal activity and function.

Seizure

Seizures happen in two main ways, generalized and focal. This example shows a focal seizure.

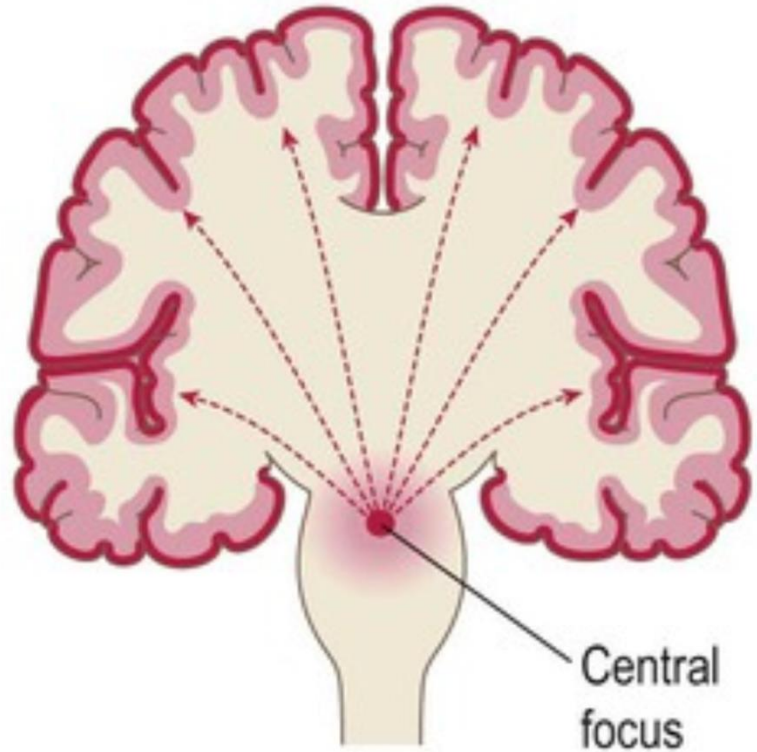


Your brain has billions of cells called neurons that all connect to create electrical networks. This network would be similar to roads and traffic lights.

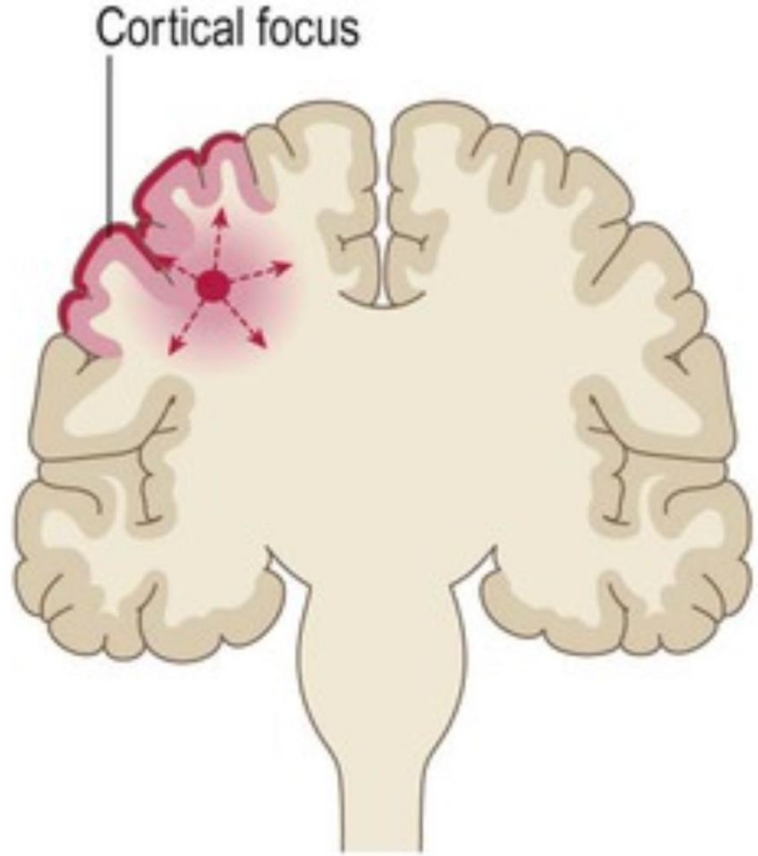
Those networks make up and connect different parts of your brain, like different towns.

Seizures happen when the neurons malfunction and electrical signals fire uncontrollably. Think of traffic lights short-circuiting.

The more neurons (traffic lights) that go haywire, the more intense or widespread the seizure and bodily effects.



Primary generalized



Partial (focal)

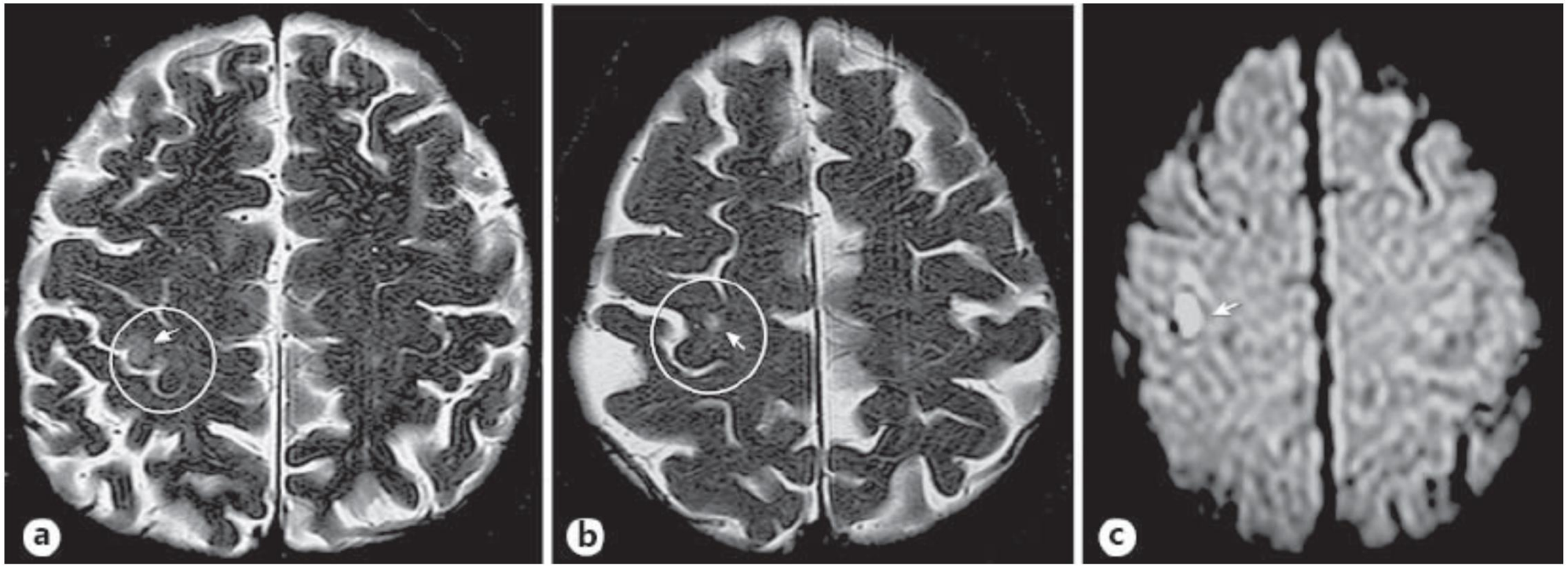
Seizures and related neuron hyper-excitability after having a stroke

- The stroke itself, by causing a critically decreased regional blood flow and oxygen supply, makes the brain more prone to seizures
- Provoked focal seizures are caused by abnormal, disorganized electrical activity in the damaged brain area, often triggered by the scar tissue that forms after a stroke which disrupts normal electrical signals. A buildup of excitatory neurotransmitters like glutamate can also lead to increased brain excitability.
- Electrical disruption: Scar tissue can send out abnormal electrical signals, disrupting the brain's normal communication pathways.
- Excitatory neurotransmitters: In the acute phase, a stroke can cause an imbalance of neurotransmitters. The concentration of excitatory transmitters like glutamate increases while inhibitory ones like GABA decrease, which makes the brain more excitable and prone to seizures.
- Inflammation following a stroke can damage the blood-brain barrier, increasing the brain's susceptibility to seizures.

Managing hyper-excitability and recovering …

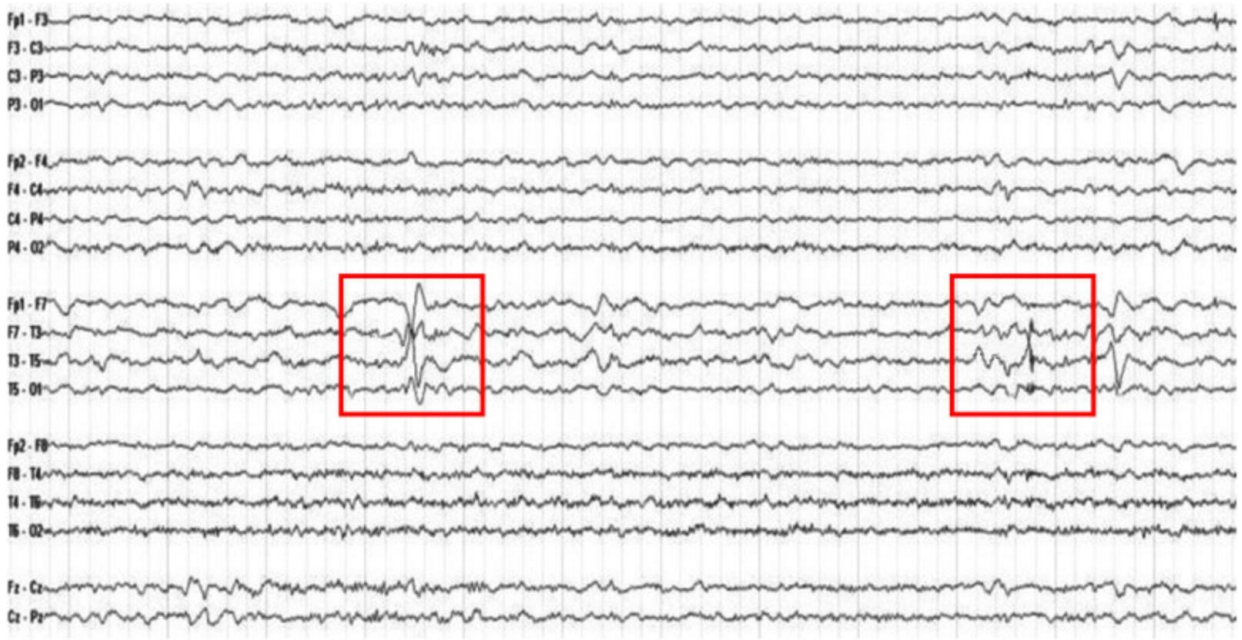
- At 5 weeks+ I continue to experience a range of hyper excitability issues: tingling in the finger tips which can spread up my arm to my face; ‘pins and needles’ on the soles of my feet, legs and arms, muscle tightness, sudden brief headaches, leg, arm and face spasms
- Seizure meds called ‘anticonvulsants’ reduce the excitability of neurons, limiting their ability to start a chain reaction of neuronal firing
- An initial loading dose of Keppra was administered to me at VGH emergency and then 750mg x2 daily. It takes a few weeks to fully stabilize but I experienced a calmer brain the first day
- Relaxing at night often brings on my symptoms as my brain down-regulates
- Loss of driver’s license until free of these issues 6+ month out
- Avoiding over stimulation and stress is important for healing to proceed
- I am carrying on with life, albeit at a slower pace (for now)

MRI imaging to determine stroke location



a T 2 -weighted MRI showing a small infarction in the lateral part of the epsilon-shaped knob of the right precentral gyrus. b T 2 -weighted MRI showing a small infarction in the inverted omega-shaped knob of the right precentral gyrus. c DWI MRI showing a small infarction in the right lateral part of the hand knob on

EEG Monitoring to spot hyper-excitability



- Focal seizures and related hyper-excitability can be seen by EEG monitoring which can identify the location within the brain