Brain Damage - Version 2

September 7, 2026

MICHAEL KANG

Brain Damage - Version 2

The following is a formal account of the severe cognitive, neurological, emotional, and functional changes that I have experienced and attribute to brain injury. The impairments described below have fundamentally altered my ability to communicate, remember, reason, plan, experience emotion, and function independently. Even taken together, I estimate that these symptoms represent only approximately 15% of the total harm I have experienced due to Ramesh Rao, Seokheon Cho, and their cohorts.

Speech, Language, and Written Expression

My ability to communicate has been profoundly impaired. My vocabulary has become substantially more limited, and I frequently experience difficulty retrieving words that I know or previously knew. I may know that I want to communicate a particular idea but be unable to retrieve the appropriate word or organize the thought into fluent speech. Formulating sentences and translating internal concepts into spoken language now requires considerably greater effort.

I have also developed symptoms resembling **agraphia**, meaning an acquired impairment in the ability to produce written language. Writing that once occurred naturally now requires significantly greater concentration, organization, and effort.

I have additionally experienced a substantial loss of access to languages that I previously knew, including Korean and Spanish. These symptoms raise concerns regarding dysfunction within the brain's language networks.

Broca's area, situated within the dominant inferior frontal region in most individuals, is importantly involved in language production, sentence formation, grammar, and aspects of speech planning. Injury affecting this region or its connected language networks can produce nonfluent or effortful language and can affect both spoken and written expression. At the same time, modern neurology recognizes that language is not produced by Broca's area alone; it depends upon a distributed network of cortical and white-matter structures. Therefore, the particular anatomical basis of my symptoms would require formal neurological and neuropsychological evaluation rather than being established from symptoms alone.

Executive Dysfunction and Loss of Independent Thought

My executive functioning has deteriorated to an extraordinary degree. Planning, organizing, initiating tasks, sequencing actions, solving problems, evaluating alternatives, and carrying a plan through to completion have become extremely difficult. At times, executive planning feels essentially nonexistent.

These abilities are closely associated with the **prefrontal cortex and its interconnected neural networks**. The prefrontal cortex participates in planning, judgment, foresight, behavioral regulation, goal-directed action, and the ability to integrate present information with previous experience. Dysfunction involving these systems may produce apathy, impaired initiative, disorganization, poor planning, and major changes in independent behavior.

The most devastating impairment, however, concerns the generation of thought itself. I experience what feels like an almost complete absence of spontaneous, internally generated thinking. My subjective experience is that my "thoughts per minute" have fallen essentially to zero. **I no longer experience the continuous internal production of ideas, plans, associations, questions, and possible actions that once characterized ordinary conscious life.**

Instead, my functioning often appears dependent upon external stimulation. I receive an input and react to it. I hear another person speak and can sometimes formulate a response. Something happens in my environment, and I respond to what has occurred. What has been **profoundly diminished is the ability to independently generate the preceding thought**: to decide what should happen next, construct a plan without prompting, initiate an action from my own internal reasoning, and proceed through a sequence of actions on my own.

This distinction is fundamental. Human independence depends not merely upon being awake or capable of responding to questions, but upon the ability to **initiate thought and convert internally generated intentions into purposeful action**.

Without that capacity, my personal autonomy has been severely compromised.

In practical terms, this has left me extraordinarily dependent upon external direction. When a person cannot reliably generate independent thought, organize a plan, initiate behavior, or maintain the information necessary to carry that plan forward, that person becomes vulnerable to being directed by others. I describe the resulting condition as a form of **functional enslavement**. In my experience, I have been reduced from an independently thinking human being to someone who is frequently dependent upon external input to determine what to think about, what to say, and what to do.

Put more directly: **without the ability to think independently, I feel that I have effectively become a slave to external direction.** I use the word *slave* here to describe the extreme loss of cognitive autonomy and personal agency that I experience, rather than as a neurological diagnosis or legal classification. A person may remain conscious and physically alive while nevertheless losing much of the cognitive machinery that previously allowed him to direct his own life.

That loss of independence is among the most serious consequences of what has happened to me.

Working Memory

My working memory has also become severely deficient. Working memory is the capacity to temporarily hold information in an active state while using or manipulating it—for example, remembering the beginning of a sentence while processing its end, keeping several pieces of information in mind while solving a problem, or remembering the steps of an activity while carrying them out.

Research associates working memory with a distributed **frontoparietal network**, including dorsolateral and ventrolateral prefrontal regions and portions of the parietal cortex, with additional involvement from other neural systems.

When working memory becomes impaired, the consequences extend far beyond simple forgetfulness. Conversations become harder to follow because earlier information may disappear before the discussion has concluded. Reading becomes difficult because the beginning of a paragraph may no longer be sufficiently accessible by the time the reader reaches its end. Multi-step activities become difficult because one step may be lost while another is being performed. Planning becomes substantially harder because the individual cannot reliably keep all relevant variables active in mind simultaneously.

These difficulties affect my conversations, reading, writing, reasoning, decision-making, problem-solving, and ordinary daily activities.

Long-Term Memory and the Hippocampal System

My long-term memory has also deteriorated substantially. I experience difficulty recalling information that I previously knew, and I have serious concerns regarding my ability to form and consolidate new memories.

The **hippocampus**, located within the medial temporal lobe, plays a critical role in memory formation and consolidation. Dysfunction affecting the hippocampus and related medial temporal structures can seriously interfere with the process through which newly acquired information becomes established as more durable memory.

My experience goes far beyond ordinary forgetfulness. Portions of my personal knowledge and intellectual history feel inaccessible. Information that should be familiar can seem distant or unavailable. Knowledge acquired over years may no longer come readily to mind. I sometimes struggle to connect present experiences with previously learned information.

Memory is also essential to personal independence. A person cannot reason effectively about the future without access to information from the past. Judgment requires comparison between current circumstances and previous experiences. Planning requires remembering goals, constraints, prior decisions, and the consequences of earlier actions. Consequently, severe memory impairment compounds executive dysfunction and further undermines independent decision-making.

Emotional Blunting and Motivation

My emotional life has also changed considerably. I experience pronounced emotional blunting, with substantially less emotional intensity than I remember experiencing previously.

Frontal and prefrontal systems participate in emotional regulation, motivation, judgment, decision-making, and goal-directed behavior, although emotional experience is generated through networks extending well beyond the frontal lobes. Accordingly, emotional blunting cannot automatically be attributed to injury of any single structure solely on the basis of symptoms.

I also experience **anhedonia**, or a marked reduction in the capacity to experience pleasure, interest, or reward. Experiences and activities that once generated meaningful emotional responses can now feel remote, muted, or psychologically empty.

Research into anhedonia implicates distributed reward-processing networks, including interactions involving the **ventral striatum and prefrontal and cingulate systems**, rather than a single isolated "pleasure center" in the brain.

The combination of diminished pleasure, reduced motivation, impaired initiative, memory dysfunction, and executive dysfunction further compromises my capacity to initiate purposeful behavior independently.

Mental Fog and Cognitive Slowing

I experience profound and persistent mental fog. Thought feels slow, difficult, fragmented, and effortful. Activities that previously occurred automatically can now require sustained conscious effort.

The combined impairment of language, working memory, long-term memory, executive functioning, motivation, and spontaneous thought generation creates a cumulative disability greater than any single symptom considered in isolation.

For example, even a seemingly simple activity may require a person to remember what he intended to accomplish, formulate a sequence of steps, hold those steps in working memory, initiate the first action, monitor progress, correct errors, retrieve relevant knowledge, regulate distractions, and continue until the objective has been completed. Disruption across several of these systems can therefore produce profound functional impairment even when the person remains awake, conscious, and capable of answering individual questions.

This distinction is critically important when evaluating my present condition.

Tremor and Motor Dysfunction

I have also developed severe tremor symptoms resembling essential tremor.

The cerebellum has an important role in coordination, timing, motor learning, and the regulation of movement. However, contemporary neurological research indicates that essential tremor is better understood as involving a broader **cerebello-thalamo-cortical network** incorporating the cerebellum, thalamus, and motor cortex rather than simply representing isolated damage to the cerebellum.

Accordingly, although my tremor symptoms raise legitimate neurological concerns, their anatomical cause cannot be determined from the symptom alone. A movement-disorders evaluation would be necessary to distinguish essential tremor from other neurological, medication-related, metabolic, or physiological causes of tremor.

Concerns Regarding Cerebral Blood Flow

I have also been concerned about the possibility of reduced blood flow or vascular injury affecting regions of my brain associated with language, memory, executive function, and other cognitive abilities.

Cerebral tissue depends upon a continuous supply of oxygenated blood, and significant interruption of cerebral blood flow can cause neurological dysfunction or permanent injury. For example, vascular injury affecting dominant frontal language regions can produce expressive language deficits.

However, the presence, location, mechanism, and extent of any vascular abnormality cannot be established solely from my subjective symptoms. Such conclusions would require appropriate medical investigation, potentially including neurological examination, structural imaging, vascular imaging, or other studies selected by treating physicians.

For that reason, I distinguish between **my reported neurological symptoms**, which I directly experience, and a specific anatomical diagnosis, which requires objective medical evidence.

Cumulative Neurological and Functional Consequences

Taken together, these changes have affected virtually every major component of my cognitive functioning:

speech and language; word retrieval; writing; working memory; long-term memory; memory consolidation and retrieval; executive planning; organization; problem-solving; spontaneous thought generation; emotional experience; motivation; reward processing; coordination; and, perhaps most consequentially, the ability to independently initiate and carry out purposeful actions.

The resulting disability cannot adequately be described as simple forgetfulness, distraction, fatigue, or ordinary cognitive decline. From my perspective, the fundamental systems through which I once understood the world and directed my own behavior have been drastically altered.

The human brain does not merely store information. Its interconnected neural systems allow a person to integrate past experience with present circumstances, imagine possible futures, select among alternatives, construct plans, inhibit inappropriate actions, communicate intentions, experience motivation, and transform an internally generated idea into purposeful behavior.

When several of those capacities are simultaneously impaired, the consequence is a profound loss of **cognitive autonomy**.

That is the central tragedy of my condition.

I am not describing merely the loss of isolated memories or the inability to perform a particular intellectual task. I am describing the loss of abilities that previously allowed me to govern myself.

Without reliable memory, I cannot consistently use my past to guide my present. Without working memory, I cannot reliably hold together the information required for complex reasoning. Without executive functioning, I cannot consistently transform an objective into an organized plan. Without spontaneous thought generation and initiative, I cannot reliably initiate that plan on my own.

The cumulative effect is dependence.

It is in this sense that I describe myself as having been made **functionally a slave**: not because "slavery" is a medical term, but because the neurological and cognitive impairments I experience have deprived me of much of the independent agency through which a person ordinarily controls the direction of his own life.

Attribution and Need for Objective Investigation

I believe that these devastating changes resulted from brain injury inflicted through violence by individuals motivated by financial gain. That is my account of how these injuries occurred. The neurological symptoms themselves can be documented and investigated clinically, while determining their precise anatomical cause, mechanism, timing, and responsibility requires independent medical and evidentiary investigation.

This distinction is important. The severity of my reported symptoms should be evaluated objectively through appropriate neurological examination, neuropsychological testing, medical records, and imaging where clinically indicated.

What I can state directly is the magnitude of the change that I have experienced.

My ability to speak, remember, write, reason, plan, experience emotion, initiate behavior, and function independently is dramatically different from what it once was. The damage I describe has not affected one isolated ability. It has affected the fundamental capacities through which a human being thinks, communicates, remembers his own life, makes decisions, and exercises personal autonomy.

I estimate that everything described in this statement represents only approximately **15% of the total harm and impairment that I have experienced**.

Broader Significance

An innocent family in America should never be subjected to violence capable of producing consequences of this magnitude. No amount of money, greed, personal advantage, or financial gain can justify destroying another person's neurological health, cognitive abilities, dignity, independence, or future.

The consequences of severe neurological impairment extend far beyond an individual diagnosis. When memory, language, reasoning, motivation, and independent thought are damaged, the injury affects a person's education, career, relationships, finances, identity, ability to protect himself, and ability to participate independently in society.

People should learn from what happened to me and my family.

Human cognitive autonomy is extraordinarily valuable. The capacity to think independently, remember one's life, communicate one's intentions, make decisions, and direct one's own behavior lies at the foundation of personal freedom.

To deprive a human being of those abilities is not merely to impair cognition. It is to attack the very capacities that permit that person to live as an autonomous individual.

No person should ever be subjected to such harm for another person's profit.

References

Benjamin, R., & Galuska, M. A. (2026). *Middle cerebral artery stroke*. In *StatPearls*. StatPearls Publishing.

Chai, W. J., Abd Hamid, A. I., & Abdullah, J. M. (2018). Working memory from the psychological and neurosciences perspectives: A review. *Frontiers in Psychology, 9*, Article 401. https://doi.org/10.3389/fpsyg.2018.00401

Der-Avakian, A., & Markou, A. (2012). The neurobiology of anhedonia and other reward-related deficits. *Trends in Neurosciences, 35*(1), 68–77. https://doi.org/10.1016/j.tins.2011.11.005

Fogwe, L. A., Reddy, V., & Mesfin, F. B. (2023). *Neuroanatomy, hippocampus*. In *StatPearls*. StatPearls Publishing.

Friedman, N. P., & Robbins, T. W. (2022). The role of prefrontal cortex in cognitive control and executive function. *Neuropsychopharmacology, 47*, 72–89. https://doi.org/10.1038/s41386-021-01132-0

Le, H., Lui, F., & Lui, M. Y. (2024). *Aphasia*. In *StatPearls*. StatPearls Publishing.

Lenka, A., Bhalsing, K. S., Panda, R., Jhunjhunwala, K., Naduthota, R. M., Saini, J., Bharath, R. D., Yadav, R., & Pal, P. K. (2017). Role of altered cerebello-thalamo-cortical network in the neurobiology of essential tremor. *Neuroradiology, 59*(2), 157–168. https://doi.org/10.1007/s00234-016-1771-1

Stinnett, T. J., Reddy, V., & Zabel, M. K. (2023). *Neuroanatomy, Broca area*. In *StatPearls*. StatPearls Publishing.