Auto-Activation Deficit Explained by a High-Level Functional Model of the Brain

Frank Heile Ph.D. (Physics, Stanford)

Poster submitted to the Stanford Neurosciences Institute for the Oct 11, 2018 Symposium

Introduction: Auto-Activation Deficit (AAD) is a very rare neurological syndrome characterized by extreme passivity, apathy, blunted affect, and a profound generalized loss of self-motivation and conscious thought. AAD patients do not initiate behaviors, but when prompted by another person, they are able to accurately perform quite complicated behaviors. Without external prompts, they can sit or lie motionlessly for a long time, and if asked, "What are you thinking?" most of them report "Nothing."

The explanation of these observed symptoms is provided by a proposed high-level functional model of the human brain. An agent, such as a human being, is an entity that can sense the world and can act on the world, often in the pursuit of goals. Decomposing a complex agent into multiple sub-agents is one strategy for gaining insight into underlying mechanisms. The high-level functional model proposed here decomposes the brain into three interconnected sub-agents: The Thinker, Doer, and Experiencer.

Auto-Activation Deficit: The neurology literature has several different names for AAD, such as: Loss of Psychic Self-Activation, Athymhormia, Pure Psychic Akinesia or Reversible Inertia. AAD is often associated with bilateral lesions to several specific sub-structures of the basal ganglia. The major symptoms of this disorder include:

- **Psychic Akinesis**: An AAD patient "spent 45 minutes with his hands on a lawn mower, totally unable to initiate the act of mowing. This 'kinetic blockade' disappeared instantaneously when his son told him to move." [1]
- **Blunted Affect**: An AAD patient "described her reaction to the death of her nephew this way: 'It's quite tragic. Before, I would have been totally devastated. But now, it's really not such a big deal.' "[2]
- **No Thought**: An AAD patient sat unmoving for a while, when asked what he was thinking, he said, "No, I'm just thinking of nothing, no idea, no question, no thought at all." [3]

Thus, these AAD patients report no thoughts, have decreased (but not absent) emotions, and cannot initiate a behavior. However, when they are externally prompted they can perform quite complicated behaviors—for example, this could include a behavior as complicated as playing bridge. [4]

The Three Agent Model: The functionality provided by the three proposed agents are:

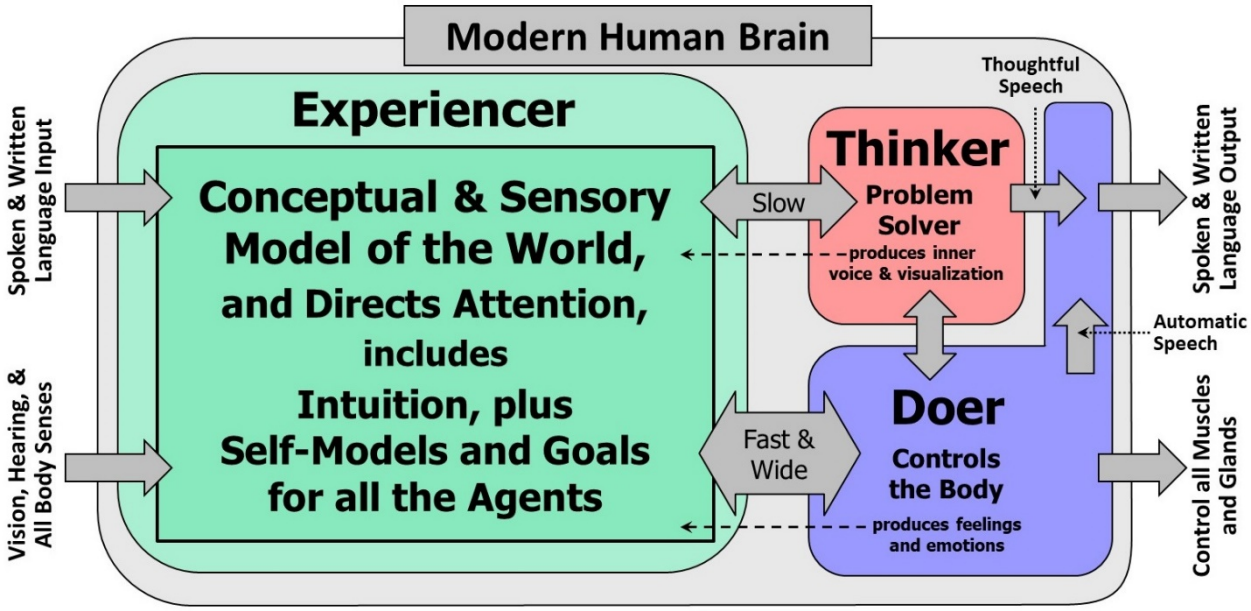
Functionality Provided by the Agents	
Thinker	**Generalized problem solving:** plans non-automatic thoughtful behaviors, thoughtful speech and produces inner voice and inner visualizations.
Doer	**Controls the body directly using automatic behaviors and automatic speech:** produces emotions & feelings, and implements the Thinker's thoughtful behaviors, and the Thinker's thoughtful speech.
Experiencer	**Constructs the world model** (sensory and conceptual) and **directs attention**

The proposed Thinker and Doer agents are **consistent** with experimentally derived models of cognition in both **psychology** (Dual Process Theory [5], **DPT**) and **neuroscience** (Yin and Knowlton [6], **YK**):

Consistency with Other Models	
Thinker	**DPT:** System 2: slow, deliberative, explicit, sequential, controlled & conscious **YK:** Associative Network / Action-Outcome contingency system
Doer	**DPT:** System 1: fast, intuitive, implicit, parallel, automatic & subconscious **YK:** Sensorimotor Network / Stimulus-Response habit system

The Good Regulator Theorem[7] suggests that an agent needs to contain a model of the world in order to exert control over the world. Given both the Thinker and Doer, the Experiencer is suggested by the Good Regulator Theorem since these two agents need to access a single shared world model. A human agent can be viewed as maintaining two different world models, one sensory, and the other conceptual, therefore the Experiencer would construct both of these world models.

The **interconnections** and **interfaces** between the agents are:



Auto-Activation Deficit Explained: The hypothesis is that the AAD patient does not contain a working Thinker. The bilateral basal ganglia lesions that cause AAD apparently disables the Thinker while leaving the Doer and Experiencer intact. More general damage to the basal ganglia or to the frontal lobes would usually damage both the Thinker and Doer and those patients will be much more disabled than an AAD patient.

The default problem the Thinker solves is: "What do I do next?" With no functioning Thinker, this problem is never addressed and thus the Doer never gets direction about what to do next—leading to a stationary AAD patient. When the patient is externally prompted, the Doer treats the external prompt as if it came from the Thinker and the Doer is then able to perform the requested behavior.

During the performance of an automatic behavior, when the Doer gets distracted or interrupted, there will be a pause in the behavior. With an intact Thinker, the Doer is reminded by the Thinker to continue the behavior. Without a Thinker, additional external prompting will often be required to enable these patients to complete the behavior.

This disabled Thinker hypothesis explains the externally reversable psychic akinesis and the lack of thought (since the Thinker is the source of conscious thoughts), but how does it explain the blunted affect? The hypothesis is that the Thinker generally amplifies affect. In the example given[2], the patient's Doer and Experiencer experiences the nephew's death as tragic (i.e. sad). If the Thinker were intact, it would try to

"solve" the problem of the nephew's death. Since the Thinker cannot solve that problem, the negative affect would have been amplified by the frustrated Thinker's inability to solve the problem—this normally would result in "total devastation." In the AAD patient the non-functional Thinker cannot amplify negative emotions, hence their affect is blunted compared to normal humans.

A Testable Prediction: Some of the evidence for dual process theory comes from multiple choice tests where the intuitive (wrong) answer is often chosen (by the Doer) whereas the correct answer, which takes some effort by the Thinker, is not chosen as often. The prediction is that AAD patients would **never** chose the correct Thinker answer since they only have a Doer and Experiencer—this would always result in the choice of the wrong (intuitive) answer.

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[1] Laplane & Dubois (2001) Auto-Activation Deficit: A Basal Ganglia Related Syndrome; Movement Disorders; 16, 5

[2] Verstichel & Larrouy, (2005) The Drowning of Mr. M.; Scientific American Mind; 16, 1

[3] Habib & Michel (2004) Athymhormia and Disorders of Motivation in Basal Ganglia Disease; J. Neuropsychiatry & Clinical Neurosciences; 16, 4

[4] Laplane & Baulac (1984) Pure psychic akinesia with bilateral lesions of basal ganglia; J. Neurology, Neurosurgery, and Psychiatry; 47

[5] Nobel Laureate Daniel Kahneman popularized Dual Process Theory in his 2011 book, "Thinking, Fast and Slow."

[6] Yin & Knowlton (2006) The role of the basal ganglia in habit formation; Nat. rev. Neuroscience. 7. 464-476

[7] Conant & Ashby (1970) Every Good Regulator of a System Must Be a Model of That System; Int. J. Systems Sci.; 1, 2,

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The following page shows what the actual presented poster looked like:

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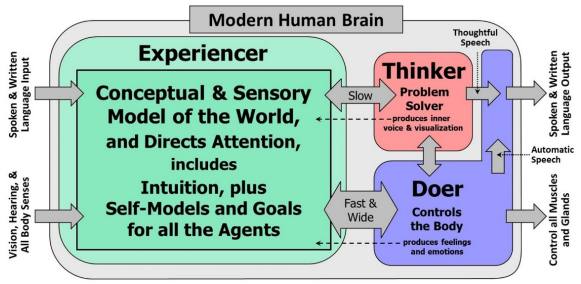

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