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FRONTOTEMPORAL AND DOPAMINERGIC CONTROL OF IDEA GENERATION AND CREATIVE DRIVE

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Abstract

This paper presents a three-factor anatomical model of human idea generation and creative drive, focusing on interactions between the temporal lobes, frontal lobes, and limbic system. Evidence is drawn from functional imaging, drug studies, and lesion analysis. Temporal lobe changes, as in hypergraphia, often increase idea generation, sometimes at the expense of quality. Frontal lobe deficits may decrease idea generation, in part because of rigid judgments about an idea's worth. These phenomena are clearest in verbal creativity, and roughly parallel the pressured communication of temporal lobe epilepsy, mania, and Wernicke's aphasia--compared to the sparse speech and cognitive inflexibility of depression, Broca's aphasia, and other frontal lobe lesions. The phenomena also shape non-linguistic creativity, as in that of frontotemporal dementia. The appropriate balance between frontal and temporal activity is mediated by mutually inhibitory corticocortical interactions.

Mesolimbic dopamine influences novelty seeking and creative drive. Dopamine agonists and antagonists have opposite effects on goal-directed behavior and hallucinations. Creative drive is not identical to skill—the latter depends more on neocortical association areas. However, drive correlates better with successful creative output than skill does. Traditional neuroscientific models of creativity, such as the left brain – right brain hemispheric model, emphasize skills primarily, and stress art and musical skill at the expense of language and mathematics. The three-factor model proposed here predicts findings in a broad range of normal and pathological states, and can be tested in many experimental paradigms.

Keywords

Frontal lobe; temporal lobe; hypergraphia; bipolar disorder; dopamine; motivation

Introduction

Creativity has been essential to the development of human civilization, and thus of neuroscience. Neuroscientists have nonetheless been hesitant to study creativity, distrusting its animal models and perceiving it as difficult to quantify even in humans. Anatomical and physiological understanding of the phenomenon is growing, however. For the purposes of this paper, a creative idea will be defined simply as one that is both novel and useful (or influential) in a particular social setting (Perkins, 1988; Csikszentmihalyi, 1999). The definition captures the cultural relativity of creativity (using a lever to move a rock might be judged novel in a

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Cro-Magnon civilization, but not in a modern one), and it also captures the distinction between the creative and the merely eccentric or mentally ill (novelty without utility).

Based on a psychometric approach to novel idea generation (Guilford, 1950), there now exist behavioral tests of creativity with both inter-rater reliability and predictability for future performance (Cropley, 2000; Carson et al, 2005). Psychometric studies complement developmental approaches (Sawyer et al., 2003) and historiometric analyses of the lives of eminent creators (Gardner, 1993). They have provided new support for the age-old theory that creativity is associated with psychopathology, most often hypomanic or mildly psychotic personality traits without full-blown illness (Andreasen, 1987; Jamison, 1989). However, the neurobiology linking these personality traits and creativity is unclear.

The last influential neuroscientific theory of creativity was the hemispheric lateralization model that grew in the 1970's from studies of patients who had had corpus callosotomies for epilepsy (Bogen and Bogen, 1988; Hoppe, 1988). Researchers postulated that the nondominant hemisphere is specialized for creative activity such as holistic pattern recognition, art, and music. The lateralization model applies poorly to language-based innovation. This is a significant defect, since symbolic verbal communication underlies most creative thought and its cultural transmission—and may have driven the evolutionary increase in the size of the human brain (Diamond, 1995). The lateralization model's exaggerated emphasis on the nondominant hemisphere was tempered by later evidence that maximizing the function of both hemispheres is probably more important than selectively activating the right hemisphere (Martindale, 1999). Moreover, right or left hemisphere lesions rarely affect creativity selectively; their effect is always clouded by their profound impairment of more general skills.

The model of creative idea generation presented here differs in four ways from the hemispheric model. First, although the model does not turn the lateralization theory completely on its head, it turns the axis by 90 degrees, arguing that connections between the frontal lobes and temporal lobes are more important than those between left and right hemispheres. Second, it incorporates the role of the limbic system in generating this drive. Most neuroscientific studies of language have focused on skill, but the limbic drive to communicate is neurally independent of the skill to do so, and probably more important for creativity. Third, the model focuses on aspects of creativity that are relatively independent of domain. In particular, it applies to idea generation using language and mathematics, as well as artistic and musical composition. Fourth, it predicts a wider range of findings in normal subjects, drug-treated ones, and in patients with a variety of lesions--rather than solely in surgically treated epileptics. Although not all aspects of this model have yet been tested, they are testable. Thus they make it clearer that creativity can and should be a phenomenon worthy of scientific study.

The Temporal Lobes and Idea Generation

The neurological phenomenon of hypergraphia, a compulsive drive to write, helps anatomically localize creative drive. It was first characterized in some temporal lobe epileptics (Waxman and Geschwind, 1974). Hypergraphia is generally proposed to reflect interictal decreases of temporal lobe activity. It is most common when the lesion is in the right hemisphere, perhaps because the left, language-dominant side is then disinhibited (Yamadori et al, 1986).

Temporal lobe epilepsy is not the only brain condition that produces hypergraphia; indeed, most patients with hypergraphia have mania and related states of agitation (Kraepelin, 1921). Manic patients show a resting state increase in right anterior temporal SPECT signal and decrease in lower left temporal quantitative EEG (Gyulai et al, 1997; Small et al, 1998). Behavioral activation paradigms have produced more complicated, task-dependent activation patterns (Haldane and Frangou, 2004). Temporal lobe lesions, especially on the right, are the most likely lobar lesions to generate mania (Braun et al, 1999). (When frontal lesions

occasionally produce mania or pseudomanic disinhibition, PET scans also show altered temporal lobe activity (Starkstein et al, 1990). Manic hypergraphia and pressured speech reflect underlying idea pressure and loose, cross-modal associations that are, when the manic state is mild enough not to be disabling, significantly associated not only with novel ideas but with socioeconomic success (Coryell et al, 1989).

While the correlation between manic states and creativity is strongest for language-based fields, temporal lobe changes can also produce the equivalent of hypergraphia in other creative fields. Frontotemporal dementia is the best-known example. A subset of these patients has neurodegeneration that selectively affects the temporal lobe. Up to 10% of that subset develops compulsive artistic or musical interests, even when they had no pre-existing artistic tendencies (Miller et al, 1998).

Other conditions that both affect the temporal lobe and increase creative drive are listed in Table 1. Notably, left temporal lobe lesions in or near Wernicke's area can increase speech output, even though they produce deficits in understanding speech. Such pressured speech in part reflects the fact that the patient's speech is not inhibited by comprehension of his errors. The model proposed here allows rephrasing the mechanism of increased speech output in anatomical terms. Because temporal lobe activity inhibits the frontal lobe (Menzel et al, 1998), speech output increases when the temporal lobe speech comprehension area no longer inhibits the frontal lobe speech generation area. Right hemisphere lesions, which do not impair comprehension in left-dominant patients, and can disinhibit left hemisphere language function, are the most likely of all brain regions to trigger pressured speech (Braun et al, 2004).

Is the temporal lobe, then, the seat of creativity? The data above suggest that it might equally be described as the seat of creativity suppression, since most of the temporal lobe conditions known to trigger creative drive seem to disrupt temporal lobe function. The relative contributions of lateral temporal cortical areas and the medial, amygdalohippocampal system may not be equal, however. Wernicke's speech area needs to function for successful linguistic creativity. Bipolar disorder, the disorder best correlated with successful creativity, is associated with left or bilateral amygdala enlargement (Haldane and Frangou, 2004). The relative absence of hippocampal atrophy may explain the cognitive sparing in manic depression compared with schizophrenia, the latter disorder being much less correlated with creativity. Alterations in amygdalar function, in assigning emotional meaning or affective valence to events or ideas, may underlie the idiosyncratic passionate interests of manic patients. Although in most cases their pursuits are misguided or overly risky, in mild bipolar disorder they can be turned to creative use.

The Limbic System, Dopamine, and the Relation between Drive and Skill

Hypergraphia reflects a drive to write, not always writing skill. Although a few great writers such as Dostoevsky were thought to have epileptic hypergraphia, many hypergraphic writers merely write interminable office memos, or make lists of their favorite songs. Nonetheless, creative drive can secondarily improve creative skill. One way is a practice effect: the more subjects write or paint, the better they get at doing so. A second way, proposed as as the Darwinian theory of creativity, depends on a Gaussian distribution of the idea quality generated by a subject (Simonton, 1999). When high motivation increases the number of ideas produced, the number of novel and useful ideas increases proportionately. The common strategy of brainstorming attempts to take advantage of this phenomenon. The Darwinian model predicts that the subjects generating the best ideas will on average also be the most driven and productive, and that innate skill will be less relevant. Indeed, there is substantial evidence that creativity is essentially independent of I.Q. above about 115 (Sternberg and O'Hara, 1999).

Above this threshold, the model predicts that brain motivational systems will be more relevant than networks primarily subserving skill.

Creative subjects have higher baseline levels of arousal and greater response to sensory stimulation (Martindale, 1999). Dopamine decreases latent inhibition, a behavioral index of the ability to habituate to sensations (Ellenbroek et al, 1996; Swerdlow et al, 2003). Low latent inhibition can flood an organism with stimuli, and is seen in psychosis (Swerdlow et al, 1996). But low latent inhibition is also characteristic of creative individuals with high intelligence (Carson et al, 2003). It may be that highly intelligent subjects can find patterns in what would otherwise be a disorienting barrage of sense data.

Dopamine does not merely raise baseline arousal. The focused aspect of creative drive, its high goal-directedness, may be driven by mesolimbic dopaminergic activity (see Fig. 1). Dopamine mediates reward-seeking activity ranging from gambling and cocaine addiction to the appreciation of beautiful faces and music (Aharon et al, 2001;Breiter et al, 2001). It can trigger the drive to communicate (Wintink and Brudzynski, 2001), although too much dopaminergic activity may cause stuttering or coprolalia. High doses of levodopa can cause excessively focused, highly complex motor stereotypies, such as repeatedly disassembling and reassembling one's motorcycle engine (Fernandez and Friedman, 1999). Whereas dopamine agonists can induce hypomania and hallucinations (Peet and Peters, 1995), the dopamine antagonists, generally used as antipsychotics, are notorious for their ability to suppress not only hallucinations and stereotypies but also creativity.

One possible mechanism for dopamine's role in focused reward-seeking behavior is a center-surround inhibition model (Mink, 1996). On this scheme, dopamine facilitates voluntary, goal-directed activity and inhibits competing behaviors. In the motor rather than cognitive system, the balance between these two poles may explain motor phenomena such as dystonia and tics (Mink, 2003). Dopamine clearly drives center-surround inhibition in sensory systems such as the retina (Bodis-Wollner and Tzelepi, 1998). Dopamine may also play a role in creative discovery through its effect on novelty-seeking. An allele of the D4 receptor has been postulated, somewhat controversially, to be a novelty-seeking gene (Keltikangas-Jarvinen et al, 2003; Savitz and Ramesar, 2004).

The temporal lobe plays a role in this interaction between dopaminergic drugs, novel creative thought, and novel thought that is merely psychotic. Functional MRI shows that schizophrenic auditory hallucinations—which can resemble the experience of having a creative idea dictated by the muse—selectively activate the temporal lobe (Shergill et al, 2001). Metaphoric, cross-modal thought, is selectively impaired by temporal lobe lesions (Jakobson and Halle, 1972). Although metaphors, when vivid enough, can be a step along the psychotic spectrum towards delusion (“I suffer like Jesus” becomes “I am Jesus”), metaphoric thought is nonetheless vital for creativity because metaphor depends on detecting analogies between phenomena previously though unrelated. This is as true for non-literary creativity as for writers. Even scientific models are metaphors, ones tight enough to allow predictive power (Martindale, 1999).

The Frontal Lobe and Creative Block

A much more common complaint than excessive creative drive is its lack. Is creative block caused by an increase in temporal lobe activity, and creative drive caused by its decrease? Probably not. There seems to be a better correlation between frontal lobe malfunction and creative block (See Fig. 2). Evidence comes from several conditions associated both with frontal lobe dysfunction and with creative block (see Table 1.) Since the frontal and temporal lobes are to a first approximation mutually inhibitory, creative block and pressured output do

not usually occur together. They can, however—as in the highly repetitive hypergraphia of some epileptics.

The first condition linking frontal dysfunction and block is depression. Many techniques, including functional brain imaging and lesion analysis, have demonstrated frontal deficits in depression. During depression, motivation and cognitive flexibility decrease, as do goal-directed activities such as eating and sex. Although creative subjects paradoxically more often have a history of depression than the average, their creative work is not done during their depressions, but in rebound periods of increased energy between depressions (Jamison, 1989; Flaherty, 2004).

When depression is treated, frontal lobe function normalizes on functional imaging (Goldapple et al, 2004). Creative block usually improves as normal levels of motivation return—with the caveat that side effects such as mood flattening or agitation from antidepressants can be counterproductive. Stimulants can help depression, as well as creativity as described above. Non-pharmacologic treatments of depression such as exercise and phototherapy may help creativity and productivity even in blocked subjects with no signs of depression (Norden and Avery, 1993; Steinberg et al, 1997).

The second frontal lobe condition similar to creative block—especially writer's block—is a lesion in Broca's area. Broca's lesions produce a selective deficit in speech production, in contrast to the speech comprehension problems of Wernicke's aphasia. Although writer's block is not an aphasia or agraphia, it shares with Broca's aphasia such features as painfully retained awareness of speech errors, as well as decreased linguistic output. This awareness causes frustration and depressed mood that further inhibit speech. Table 2 shows the parallels between Broca's aphasia and block, and Wernicke's aphasia and hypergraphia.

A third group of conditions, frontal lesions outside Broca's area, can cause depression and decreased speech independent of aphasia. Frontal lobe lesions can cause cognitive deficits such as perseveration that are similar to the dogged and unproductive efforts of blocked writers. Both appear to respond to sudden environmental changes—blocked subjects can escape their mental ruts when distracted by a break from the problem.

Anxiety is a fourth condition that, like depression, shows frontal lobe changes in a number of paradigms (Cannistraro and Rauch, 2003), and anxiety is highly associated with creative block. Block of this form, which resembles performance anxiety, should respond to antidepressants just as anxiety does. Arousal levels are higher in anxiety than depression, however, and stimulants can be counterproductive. This reflects the Yerkes-Dodson law that task performance is an inverted U-shaped function of arousal level. Anxiety states exceed the ideal arousal for task performance. Task performance has a similar inverted U-shaped relationship to the beneficial cognitive effects of dopamine agonists (Kimberg et al, 2001).

Fifth, features of writer's cramp—a focal dystonia that shows changes in sensorimotor and premotor cortex activity (Lehericy et al, 2003)—suggest that it may be a more motor, posterior frontal analogue of writer's block. Writer's cramp, like block, appears to be induced by highly attended, repetitive, stressed practice of the task. Continued attempts to write often only worsen the cramp, whereas enforced inactivity—like the breaks that help perseveration and block—can sometimes be therapeutic (Byl et al, 1996).

Sixth, electromagnetic studies demonstrate that the functioning frontal lobe stimulates creativity. When subjects with high and low creativity are compared, the former have both higher baseline frontal lobe activity and greater frontal increase while performing creative tasks (Carlsson et al, 2000). There is preliminary evidence that transcranial magnetic stimulation over frontal lobes can increase creativity in normal subjects during both drawing and writing

tasks (Snyder et al, 2004). There are case reports of patients whose creativity increased after receiving subcortical deep brain stimulating electrodes near the nucleus accumbens (Gabriels et al, 2003; Flaherty et al, In press). The accumbens' connections to the frontal and temporal lobes, and its role in limbic generation of drives, may help explain this effect.

Relative contributions to creativity from the temporal and frontal lobes may in part reflect the distinction variously described as divergent versus convergent thought, primary versus secondary process thought, or the writing versus editing stages (Flaherty, 2004). What about relative contributions to idea generation from different frontal subsystems? Lesions of medial prefrontal cortex can produce amotivational, abulic states of decreased creative drive. Dorsolateral prefrontal cortex's importance for working memory and flexible problem solving suggests a greater role in creative skill than in drive. Motor and premotor cortex are probably more necessary for performance than for conception of a creative plan. While lesions of all of these systems would be detrimental to idea generation, orbitofrontal lesions may have a partly opposing effect, as they can produce disinhibition syndromes that at least superficially resemble mania.

Summary

Overall, creative drive has the advantage of being a simpler, more tractable phenomenon than creativity itself. Creative drive's links to better understood systems, such as the drive to communicate, provide both direct and indirect evidence for a three-factor anatomical model of creative drive coordinating frontal, temporal, and limbic systems. The frontotemporal interactions are probably mediated by mutually inhibitory corticocortical projections, whereas the limbic contribution is likely to be primarily dopaminergic.

These hypotheses are oversimplified. Because activation in the frontal and temporal lobes is mutually inhibitory only to a first approximation, a more accurate model than that presented in Figure 2 would require a three-dimensional plot in which frontal and temporal activation can vary independently. Much needs to be clarified about differential contributions from lateral and medial temporal subsystems, and from orbitofrontal, dorsolateral, and medial frontal regions. Nonetheless, testing the predictions of the simple model proposed here may help characterize the role of these frontal and temporal subsystems, as well as permit direct comparison with older theories such as the hemispheric model of creativity. Doing so may prepare the way for the rigorous neuroscientific studies of creativity that are greatly needed.

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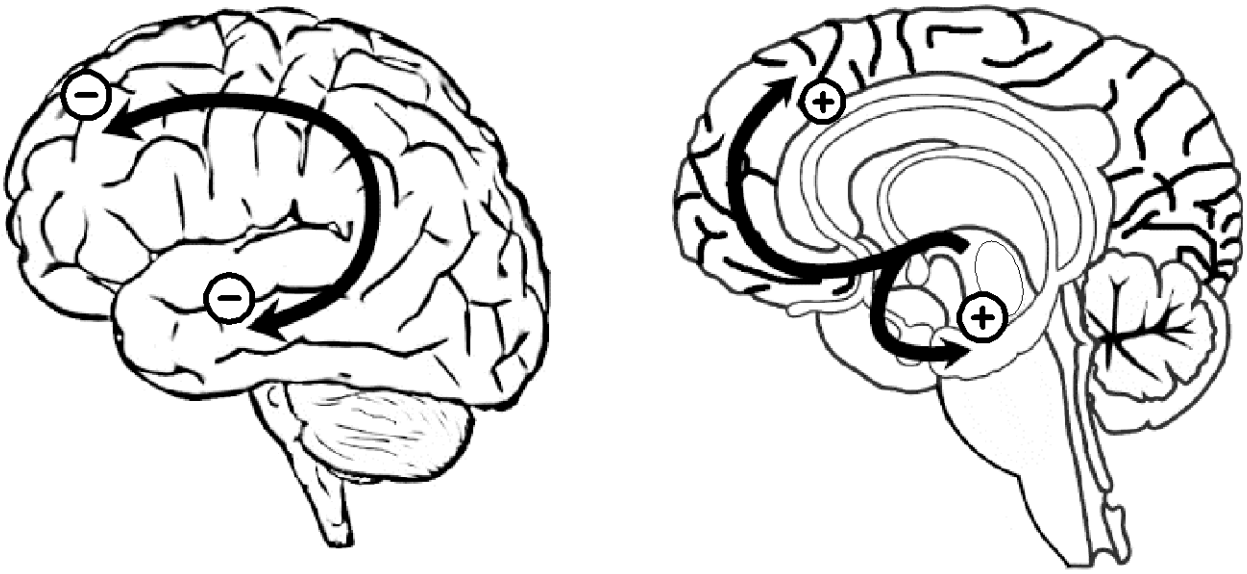

Figure 1.

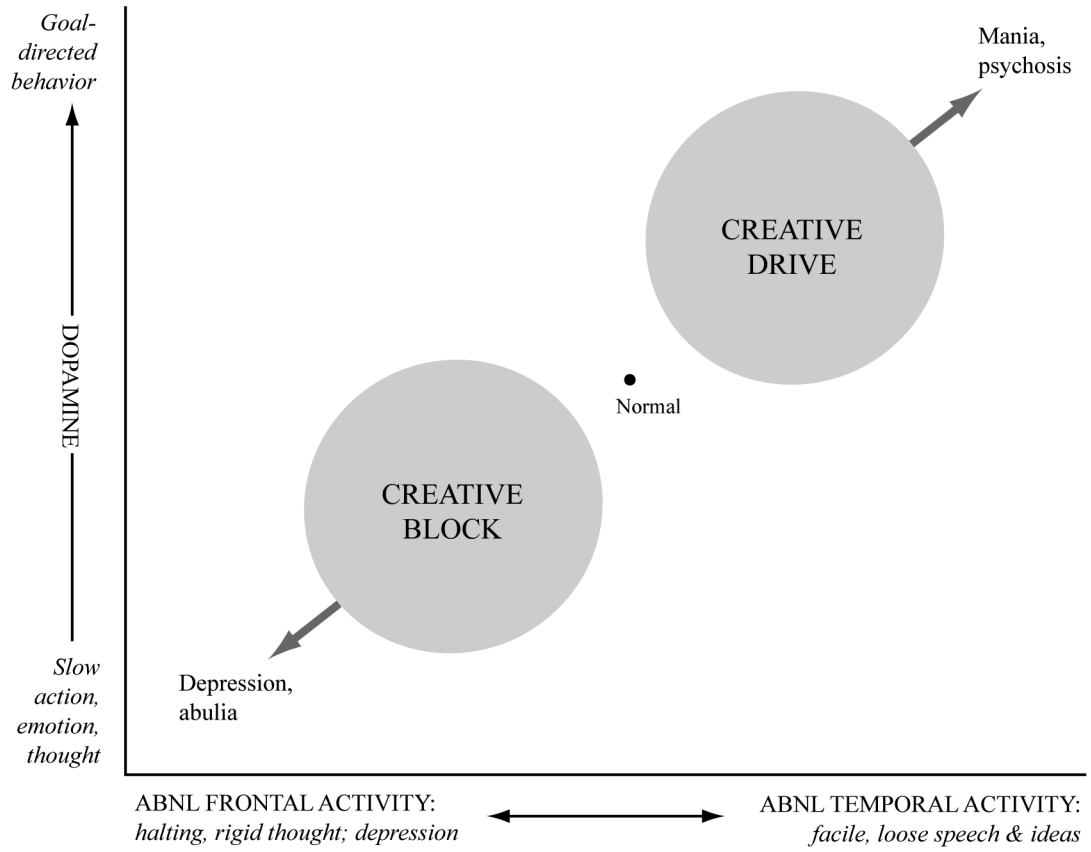

Figure 2.

Table 1

Conditions in which altered creative outputs are associated with temporal, limbic, and frontal changes. Abbreviations: DA = dopamine, EEG = electroencephalogram, fMRI = functional MRI, PET = positron emission tomography, SPECT = single photon emission computed tomography, TMS = transcranial magnetic stimulation

Condition	Behavioral Evidence	Physiological Evidence
Temporal Lobe		
Temporal lobe epilepsy	Hypergraphia, viscosity	EEG, MRI, PET, lesion studies
Hypo/mania, mixed states	Pressured speech & writing; muse experiences	fMRI, EEG, PET
Wernicke's aphasia	Pressured speech	Lesion studies
Other temporal lobe lesions	Hypomania and mania	Lesion studies
Frontotemporal dementia	Pressured artistic and musical expression	SPECT, PET, MRI, fMRI
Command hallucinations	Auditory hallucinations/muse experiences	fMRI, TMS
Metaphorical thinking	Tendency to analogies, associative thinking	Lesion studies
Limbic/Dopaminergic		
Levodopa, DA agonists	High productivity/drive, lower latent inhibition	Drug studies, PET receptor binding
DA antagonists	Decreased drives and creativity	Patient reports, receptor binding studies
Psychosis	Hallucinations/muse experiences	DA agonists cause, antagonists treat
Psychostimulant use	High productivity/punding, lower latent inhibition	Drug studies, PET receptor binding
Nonspecific arousal	Exercise, phototherapy, Mozart effect raise creativity	
Frontal Lobe		
Depression	Self-critical, low energy and output	fMRI, PET, lesion studies
Anxiety	Self-critical, “stage fright” inhibits output	fMRI, PET, lesion studies
Broca's aphasia	Frustrated awareness of speech production deficit	Lesion analysis
Other frontal lobe lesions	Abulic mutism, perseveration, solution fix	Lesion analysis
Normal/creative subjects	Tests of idea fluency	f MRI
Writers/musicians cramp	Focal dystonia from stressed practice	fMRI, PET
Metonymical thinking	Organization by spatial/temporal sequence	Poor in frontal lesions, depression, block
Antidepressants	Improve depression and secondary block	fMRI, PET
Electromagnetic stimulation	Increased productivity, creativity	DBS, TMS

Table 2

Relation between states of creative drive, aphasias, mood states, and lesion locations.

	Wernicke's aphasia	**Hypergraphia**	**Broca's aphasia**	**Writer's block**
Linguistic output	↑	↑	↓	↓
Awareness of deficit	↓	~↓	↑	↑
Depression & anxiety			↑	↑
Mania, irritability	↑	↑		
Location of lesion	Temporal lobe	Temporal lobe	Frontal lobe	Frontal Lobe?