Short Communication

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A Brief Neurological Explanation of Suicide

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The human neocortex can be viewed as a huge memory systems interacting with one another. There are several exquisitely evolved memory filing systems. Most autobiographical memories are linked to a particular emotion. The neurochemistry of the emotion is an integral part of the memory, such that the emotion must be triggered for the memory to become accessible. However this is a two-way system, where the memory can trigger the emotion. Another filing memory method is by context. The context can be spatial, social, emotional or a combination of such contexts. Part of the context can trigger the memory, such that when similar contexts are encountered, memories associated with the context become accessible.

I propose an elegant neural circuit, comprised of a few small structures buried deep in the middle of the brain (primarily, the habenula and the medial and lateral septal nuclei, including the diagonal band of Broca), receives its major inputs from the thalamus and the hippocampus and is continuously adjusting the internal emotional state to the external, ever-changing environment, attempting to produce, at all times, the adequate emotional response. This system, through the signals it sends to the reticular formation, is an emotional arousal and, balancing and tuning system. It summarizes the activity of the entire brain, taking into account emotions, cognitive signals translated into contexts and meanings, and accordingly, fine-tunes the signals of all neurotransmitter systems in the brain stem.

This arousal system, in a slow gradation of excitability, from less to more, associated with negative and positive emotions, using asymmetries in the brain, detects and generates very fine emotional distinctions covering a whole range: from happy, changing slightly to blissful, playful, grateful, safe, relaxed, undecided, uneasy, frustrated, alert, tense, cautious, anxious, afraid, all the way to panicky; or conversely, starting from lonely, changing to bored, humble, bashful, ashamed, envious, determined, optimistic, joyous, all the way to loved.

The primary function of this circuit is to try to balance activity in both hemispheres, continuously attempting to restore neutral calmness, reflecting its evolutionary path when lateralization was less prevalent in more primitive brains. This habenula-septal circuit is important enough to deserve it own name, the isorropic circuit (from the Greek, isorropia=balance).

The cholinergic (acetylcholine) projections of the isorropic circuit act through muscarinic receptors. The muscarinic receptors are slow-acting, consequently their effects occur with a time lag. This time lag, in chaos-theory terms, allows the isorropic circuit to wander about a strange attractor, which under normal conditions represents the point of neutral calmness. Try to visualize a small, variable circular trajectory moving about a point, but never settling on the point.

Under most conditions, this strange attractor (mathematically represented by a point) is the state towards where the isorropic circuit's actions tend to modulate the cortex's activities. Let's call this attractor the isorropic attractor. The subtle wandering of the isorropic attractor around neutral calm permits the isorropic circuit to generate and regulate a wide spectrum of emotions. Potentially, this wandering permits the generation of myriad slightly differing emotions as a response to a vast array of changing internal and external stimuli.

When the isorropic attractor is close to neutral calmness a wider spectrum of potential emotional responses is available, positive and negative, and by activating memories associated with the emotions, allows for activation of a greater pool of memories. The speed at which small emotional changes can be generated also allows the search for the greatest potential number of contexts. Thus the brain achieves a state that can generate the maximum variety of behaviors in the quickest time possible as response to the ever-changing environment. This places the organism in a state of maximum adaptability. When the brain detects this healthy state of maximum adaptability it is felt as a sense of self.

Under certain conditions the isorropic attractor locks into a state different than neutral calmness, in which case the brain produces a mood or a long lasting emotional state.

The wider the spectrum of emotional responses that can be triggered, the more intense the sense of self is felt. Conversely, the narrower the spectrum, the less intense the sense of self is perceived.

When the isorropic circuit is close to neutral calmness, autobiographical memories can be more easily activated because of the continuous wandering of the isorropic attractor; as a result, most all emotions associated (through handshakes) with all the memories can be quickly triggered.

When the isorropic attractor is far from neutral calm, short term, working and explicit memories can be affected, primarily because of the intense emotion(s) present and because thalamic control of the attentional systems can be disrupted.

In contrast, semantic, source and procedural memory, because of a low or inexistent relation to emotions can still be activated even when the isorropic attractor is far from neutral calmness. The hippocampus can detect a particular context, independent of an emotion, and send the appropriate signals and activate these types of memories because they are independent of the emotional state.

If environmental or internal stimuli push the isorropic attractor outside the chemical ranges that permit homeostatic balance, the sense of self becomes distorted; the spectrum of emotions that can be potentially generated, thus the memories that can be activated, are greatly diminished and the behavioral responses limited. The limited choice of behaviors, imposed by stressful conditions, under a wide array of circumstances, can still generate a correct response. But the longer the stressful conditions prevail, the smaller the number of responses that can be activated and the less the probability that the correct response will be generated.

In extreme cases, when the isorropic attractor is pushed far enough from the range of homeostatic balance, the brain will no longer be able to perceive a sense of self; the organism can no longer adapt.

There are many paths that can be part (or not) of the journey that can culminate in suicide, such as anxiety, fear, loss, hopelessness, loneliness, anhedonia, aggression, alcoholism, masochism, preoccupation with death, a wish to kill or to be killed, revenge, spite, escape, rebirth, or reunion with loved ones (the list could easily be expanded), depression, schizophrenia, personality disorders, panic attacks, and so on. However, walking on many of these paths at different times in life or even simultaneously, does not remotely guarantee suicide will be the destination. On the other hand, these paths do point clearly to roads that can lead to depression. Depression, as we have seen, is a state that can be extremely painful and confusing. And depression, in its numerous incarnations--unipolar depression, bipolar (I and II) depression, depression in schizophrenia with positive symptoms, and depression in borderline personality disorder—is the highway to suicide. And still, suicide does not happen among most depression sufferers (more than 80% don't kill themselves). About 90-95% of suicides are related to depression (in one of its incarnations) with another 3-5% being borderline personality disorder cases. And still, for suicide to occur several other elements have to converge [1,2].

First, and probably foremost, a person must experience a loss of the sense of self. This happens when the activity of the isorropic circuit is pushed beyond the ranges of homeostatic balance towards the negative side, because of concomitant negative emotions triggered by depression or in addition to an extremely intense negative emotion. This condition is often, but not always, produced during depression and the depressed phase of manic-depression (especially when anxiety or panic attacks are concomitant with this condition), occasionally in schizophrenia and schizophreniform disorder concomitant with depression (with positive symptoms), or some of these conditions concomitant with extreme, intense emotional responses (anger, jealousy, panic, bereavement), and occasionally when personality disorders are present. The condition can also present itself in the face on one single intense negative emotion. As I have mentioned, when slowed or disorganized thinking is present, negative emotions are triggered and brought to the surface. The isorropic attractor is pushed to the negative side in proportion to the quantity and mix of the negative emotions. Under these conditions the isorropic circuit's balancing functions (represented by the isorropic attractor) are locked into a negative emotional state and contribute to perpetuate the depressed state.

Because I believe it is so important to understand this mental state, I have given it its own special name, idiozimia (from the Greek, idios=self and zimia=loss). And, I have named a partial reduction or diminution of a sense of self, archidiozimia (from the Greek archi=beginning).

Archidiozimia is almost always present in depression, and depending on the degree of emotional mix in depression this state will drift towards idiozimia, the total loss of self. Idiozimia or archidiozimia are also possible in various intense emotional states, such as anger, mania, fear, sadness, and even hatred or jealousy. At the same time, because of the effect of the negative emotions on the brain, certain modes of thinking, primarily all the memories associated with positive emotions, are partially or totally inaccessible. A mix of negative emotions produces confusion in the form of inaccessibility to the related memories of each individual emotion. The emotion (and its neurochemistry) is an integral part of the memories associated to it. But, when several emotions are triggered simultaneously none of the respective memories can be activated because of the mixed neurochemistry. The fact that these latter memories are inaccessible produces a distortion of the sense of self, archidiozimia sets in, and ultimately idiozimia, when there is a complete loss of self. Under these conditions, the isorropic circuit is unable to restore balance. Eventually, the range of choices becomes limited to only one or perhaps two.

Idiozimia produces a constriction of cognition, affect and intellect. As the sense of self is distorted by the intensity and mix of negative emotions, insight and understanding diminish as more memories become inaccessible. Idiozimia can be perceived, when depressed, as a menagerie of simultaneous negative feelings, which produce in the individual who suffers from it, a torment greater than the sum of each of its parts. The particular combination of negative feelings is individual to each person, and this makes each individual's experience different. Just as each person has unique memories, so each person will experience depression uniquely. Each individual's thoughts and experiences are unique, and when they are activated by the negative emotions, will be expressed in special ways. An individual in the idiozimic

state will present a serious deficit in autobiographical memories, not only because of the confusion of negative emotions and their associated memories, but more importantly, because there is no accessibility to the memories associated with positive emotions. Depressed individuals suffer a negative memory bias where positive experiences from their past are inaccessible.

An important affective aspect of idiozimia, because memories of loved ones are nonexistent, is the perception of an eternal feeling of aloneness. It is a state that seems always to have been and that will never end. Some have described it as an experience of utter isolation, accompanied by a sense of unreality, with a quality of emptiness, horror, devastation and denigration. The idiozimic person does not take others into consideration, for the simple reason that most memories of others are inaccessible. Social isolation normally exacerbates needs for belonging, respect, caring, and attention.

Because of this, when idiozimia is present, social isolation is another contributing factor to suicide, but the cause is the idiozimic state, not the isolation per see.

Idiozimia, in a similar fashion to the fight-flight response, is accompanied by a lack of sensitivity or fear of physical pain. Both of these conditions are conducive to possible self-harm, or at minimum, conducive to disregard potential injurious consequences of one's actions. During idiozimia people can break away from the natural fear of death or self-harm.

Another mental condition needs to be present and exacerbates the idiozimic state. Second element: damage to the hippocampus. Because of prolonged and intense stress, secretions of glucocorticoids damage the hippocampus. The hippocampus normally stores and retrieves the memories of all related objects to the event, in this way creating a context. When the hippocampus is damaged, the emotional, social, and spatial context will also be perceived wrongly. In essence, the context, which is an integral part of a memory, will be disrupted and the respective memories will be inaccessible; this further facilitates idiozimia. Moreover, in the face of depression and/or idiozimia, the negative emotions, put into the wrong context, will exacerbate the effect of the next three elements. The wrong context will tend to exaggerate the perception of negative events, thus leading to increased self-pity and occasionally triggering rage and/or paranoia. The wrong context will lead inevitably to faulty reasoning.

Suicide is associated with two to five years of suffering depression with comorbidity of panic attacks or anxiety, expressed suicidal ideation or intent, and prior suicide attempts, and is highest in schizophrenic patients with positive symptoms and depression This might be a manifestation of the cumulative stress effects of mental health disorders on the hippocampus. The lack of control, such as associated with panic or anxiety attacks, mood disorders such as depression (unipolar and bipolar), and posttraumatic stress disorder or personality disorders, exacerbates the stress factor over time. With a faulty context—social, emotional, or spatial—faulty reasoning becomes much more probable and thus suicide more likely.

A third element, are suicidal thoughts. These thoughts, in an idiozimic state and seen in the wrong context, become part of logical actions. In this sense suicide is a rational act.

To this we add a fourth element: the slow vanishing of inner strength. The intense suffering that accompanies depression and some combination of symptoms in schizophrenia and schizophreniform disorder can be so acute that ending one's life becomes a real alternative. The effort to stay alive in the face of extreme torment is emotionally exhausting and seems insurmountable.

Naturally, all these mental states lead to a fifth element: hopelessness and helplessness. A sixth possible element: the aftoktonic switch. Within the context of the last four elements, or because of their repetitiveness in past experience (suicidal ideation, hopelessness-helplessness, vanishing strength, and a damaged hippocampus), the echoes streaming from the cortex reinforce and increase the chance that the thalamus will match incoming signals with external sensory signals and erroneously, but automatically, trigger the respective emotions linked to hopelessness and vanishing strength, which in turn activate the related suicidal thoughts. Once this happens, the amygdala will signal the nucleus accumbens and amplify these, and turn the suicidal thoughts into a belief—the belief that death is desirable, that death is a goal. Simultaneously, the hippocampus signals the nucleus accumbens and increases the urge signaled from the ventral tegmental area. With a stressed, damaged hippocampus, the hippocampus and amygdala trigger, respectively, an urge to end life within the context of hopelessness, and a belief that death is welcome. This binary combination becomes an unbearable urge to commit suicide by whatever means available. This is what I call the aftoktonic switch. This is a specific case, among many, of the archipraxi (from the Greek=begin action) switch sending a signal to initiate action. Suicide is not a random, purposeless act. To the sufferer, under these conditions, it becomes the only available solution to his or her problems.

There are many pointless deaths, but suicide is never a needless act. More often than not, during the act of suicide, it is perceived as the best and only possible response to alleviate the excruciating situation.

Sevent element: this is so important that I have given it a special name, the phobothymic switch (from Greek, phobos=fear and thymos=wrath or anger). This element might be independent of the second, third, fourth and fifth elements just described. It is common that depression or schizophrenia is accompanied by anxiety or panic attacks. Depression and schizophrenia are states that clearly indicate that something is wrong, and the fear and anxiety associated with these states could be normal, healthy responses to a dangerous mood disorder. In order for the amygdala to build up a fear response the prefrontal region must be shut down, otherwise the prefrontal lobes signal the amygdala and fear slowly dissipates when danger is not present. However, when the amygdala is completely unchecked by the prefrontal lobes, the fear escalates and a panic attack ensues. An unbearable inner terror builds up.

The first step of the fight-flight response is freezing, a strategy that helps fool predators into thinking that you are dead, or makes it

harder for them to detect you. At this moment the phobothymic switch can go either way, fear or aggression is equally possible. Freezing also provides a small amount of time to evaluate the threat and determine to flee, attack, or simply return to a resting state: false alarm. This state feels strangely both like anger and fear, a sort of physical prickling sensation down the spine.

When the anxiety attack or the fear escalates into a full-blown panic attack, there is a moment when suddenly the phobothymic switch is activated: the flight mechanism switches to a fight response. The uncontrolled fear turns into uncontrolled aggression. In this aggressive mode, the perceived threat, in this case an irrational panic, which is translated to a feeling of imminent death, is immediately confronted with an anger attack. A confrontation with the biggest known fear, imminent death, becomes not only logical, but seems the only solution, the only way to win, the only way to survive. When the tiger has chased us into a dead-end canyon and there is nowhere to flee, we inevitably turn to face it and fight it to the death. There is no other choice. In a normal, healthy situation, at some point the threat is evaluated (not rationally, there is no time) by the cortex and determined that escape is impossible, or conversely, that the threat can be removed by quick action: the phobothymic switch turns from flight to aggression.

Uncontrolled aggression is channeled against the source of the threat (the panic attack), which is being generated by one's own brain. Suddenly, extinguishing the root of this threat becomes absolutely imperative. An attack against oneself, suicide, is the focus of the aggressive behavior. It is an experience of relentless scorn against oneself. It is easy to try to see this as a form of self-hate, but aggression against a threat (even an internal threat) is more in line with this behavior.

At some point the amygdala receives a signal from the prefrontal cortex, and automatically the response changes from flight to fight mode; from fear to anger; from a panic attack to an anger attack. It is two sides of the same coin. The phobothymic switch is an elegant engineering solution to automatically and quickly change the strategy for survival; at some point, or under certain conditions, the organism is better off fighting than fleeing.
In a nut shell, this is how suicide happens.

References

1 R.W. Maris, Assessment and Prediction of Suicide. 1992.

2 Douglas Jacobs, The Harvard Medical School Guide to Suicide Assessment and Intervention.