

# Psychoanalysis and Neurophysiology

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## A New Domain

Research regarding the neurophysiology of emotions is moving ahead by leaps and bounds. Due to technical advances in Neuroimaging (Positron Emission Tomography and functional Magnetic Resonance), brain areas that become metabolically more active are observed, registered and correlated with clinical states of depression, compulsive obsessive disorder, agoraphobia, etc. Comparison studies between psychotherapy and pharmaceutical therapy have already shown that these two approaches affect different neuronal pathways when patients improve. (Roffman,2004)

This article will explore the implications of recent neurophysiological research for the psychoanalyst. Although the psychoanalytic process has not yet been studied in a direct way by Neuroimaging studies, the cortical and sub-cortical neuronal maps that are emerging from recent research can already begin to "talk to" the psychoanalyst. Thus, our epistemological basis is not "support by direct observational evidence," but more hypothetically, "support by correlation." (Liss, 1986) This means that the clinical-research correlations presented in this article are merely suggestive, calling for modification, correction, enlargement and higher precision.

A potential obstacle for the psychoanalyst is to dismiss the Neurosciences as "reductive." This view comes from a misunderstanding. The multiple levels of brain dynamics – 1. activated brain regions and their interactions, 2. neuronal pathways, 3. chemical processes (hormones, neuromodulators and neuropeptides) and 4. receptor-molecular interactions – all taken together give a vast neurophysiological map for helping us understand psychophysiological events.

But this map does not claim to explain consciousness. And consciousness cannot be reduced to this map. Rather, the goal is to delineate specific phenomena in the two fields – clinical experience and research-based knowledge – and highlight those events that seem to have similarities and show correlations.

## The Sub-Cortical Amygdala Regulates Our Emotions

We will begin with the work of Joseph LeDoux.(1996) LeDoux has investigated the amygdala, and proposes that this area represents the central regulator of the emotions of anger and fear, correlating to the well known polarity, "fight or flight." The amygdala is a sub-cortical regions shaped like an almond. It receives inputs from at least 12 neuronal areas and offers outputs to 12 neuronal areas. Therefore, it is a true "hub," integrating neuronal processes with a high degree of complexity. The clinical implication is that emotional phenomena are equally complex; the amygdala receives (as input) and influences (as output) a multiplicity of brain regions. In fact, looking at LeDoux' map, we can better understand how amygdala-controlled emotions integrate inputs coming from the **higher cortical regions** and that are connected to **conscious** psychological experiences: thought, memory and perception. At the same time the cortex receives an "input" from **lower sub-cortical regions**: the hypothalamus, below this the central gray matter, and from still lower centers, the visceral nuclei that reside in the brain stem. Outputs from the amygdala then return to these same cortical and sub-cortical areas, creating mechanisms of feedback or, as Gerald Edelman defines it, "re-entry mechanisms." (Edelman, 1989)

What does this neuronal map show? We now have a picture of how amygdala-based emotions are influenced by thoughts (from the cortex) and visceral processes (from the lower

visceral nuclei). At the same time, amygdala-based emotions will influence these very same cortical thought processes, by sending messages "upwards," and visceral processes involving the heart, lungs and gastro-intestinal system, by sending messages "downwards."

What does this map offer the clinician? We now have a picture of how **psychosomatic processes** work, that is, from the cortical-based conscious psyche to unconscious visceral regulation, and how **somato-psychic processes** occur, from body processes to the visceral nuclei, and from there, through "small climbing chains of neurons" (Nauta, 1972) to amygdala regulation. This map can help overcome the either-or polarity that comes from unilateral thinking. **Emotions are at the center of both psychosomatic and somato-psychic processes.** This means that we have **vertical mechanisms of interaction.** (Edelman, 1989, first suggested the concept of "vertical brain mechanisms"). Since messages are constantly running up and down the brain, to posit the psyche as primary or, alternatively, the soma as prima, is to return to an outmoded concept of unilateral causality. "Re-entry" means that we always have interactive, reciprocal and circular causality.

For example, an age-old argument between psychotherapists who start with the "psyche," and "organically-oriented" medical doctors who prescribe psycho-pharmacies, cannot have an either-or resolution. In this line of thinking, Massimo Biondi, (2005) from the University of Rome, has shown the synergy between pharmaceutical and psychotherapeutic treatment. His research is summed up by the title of his important paper: "Increased Maintenance of Obsessive-Compulsive Disorder Remission after Integrated Serotonergic Treatment and Cognitive Psychotherapy Compared with Medication Alone."

### **Rational and Emotional Causes of Action**

One additional output of the amygdala is to the basal ganglia. The basal ganglia is the sub-cortical region that regulates behavioural initiation and behavioural habits at the unconscious level. This sub-cortical region has important connections with the cortical motor areas that are correlated with conscious, intentional actions. (Cortical motor areas send messages directly to the basal ganglia. The basal ganglia returns messages, first to the thalamus, and from there back to the motor cortex as well as other cortical areas.) (Edelman, 1989) This explains how **emotions can influence our action.**

Objection: But sometimes our emotions do **not** influence our actions, and thankfully so, for example, when we are driving a car under stressful circumstances. How can this be? Once again, a more extensive brain map helps us understand this fact: 1. Messages from the frontal lobe, which are rationally "computing" our action strategy, go directly to the cortical motor areas. 2. The basal ganglia also receives input's from the lateral parts of the frontal lobes, which integrates environmental perception (posterior brain areas) with space/time **calculations** (parietal lobes). (Rolls, 1999) Thus we have neuronal pathways demonstrating both "rational" roots and "emotional" roots when the job is to regulate the cortical-subcortical action strategy.

### **A Paradox: "The Conscious Emotion Does Not Regulate Our Emotional Life"**

Another consequence of LeDoux' research and, in fact, of all research involving sub-cortical dynamics (not just the of amygdala, but regarding all sub-cortical areas), brings us to a much more radical point of view: **The emotions that we are conscious of, and which are verbalized in psychoanalysis, are NOT the emotions that regulate our thoughts, behaviour and visceral reactions.** This creates a paradox in awareness, almost like saying, "The Tao is not the Tao." Or: "The Tao that we call the Tao is not the Tao." The emotion that we are aware of and that we verbalize - "I feel panicky," "My anguish is overwhelming," "I feel

terribly sad" – is connected to **cortical** processes. But this does not represent the central dynamic of the emotion, Rather, the **regulation** of the emotion, and its power to influence thoughts, perceptions, behaviours, hormonal and visceral processes, is **sub-cortical**. **Since only cortical processes can correlate with consciousness, while sub-cortical processes can influence consciousness but not directly correlate with the conscious event, the physiologically unconscious sub-cortical process cannot be accessed directly by consciousness. The sub-cortical process sends messages "upwards," and the result is a conscious message.** This means that the psychoanalyst is hearing the cortically-accessed conscious emotional dynamic, but the main center of regulation – the amygdala -- which the psychoanalyst would like to influence, cannot be directly accessed by consciousness. The clinical consequence? The psychoanalyst hopes that by working with the conscious and, therefore, **cortical** substrate of the emotion, the sub-cortical amygdala mechanisms of the physiological, unconscious emotion can be modified. How can the psychoanalyst intuit that a conscious emotion is in connection with the unconscious dynamic of the amygdala? One way is by noting whether there are verbal and non-verbal signs, within the patient's verbal discourse, and pauses, that suggest visceral reactions: breath, tremors, voice sounds, etc.

A neurophysiological clarification: It is universally accepted that only the cerebral cortex has an neuronal architecture sufficiently complex to directly support consciousness. The more primitive sub-cortical architecture can **influence** consciousness, but it cannot create the neuronal events that sustain consciousness directly. Attention! We must avoid a confusion. The "Freudian unconscious," which refers to experiences that are repressed, but which can re-enter consciousness, is a **cortical** process. In contrast, the "neurophysiological unconscious" is another matter. The "neurophysiological unconscious" refers to processes that are outside of consciousness awareness, like the "Freudian unconscious," and influences consciousness. Nevertheless, the "neurophysiological unconscious" refers to a process that can never be directly accessed by consciousness, because it is a **sub-cortical** process.

Antonio Damasio (2003) and Alan Schore (1996) suggest that the neuronal underpinnings of conscious emotion are probably most closely correlated to processes in the orbito-frontal lobe. Given that the orbito-frontal lobe receives input from brainstem visceral nuclei, the hypothalamus, the amygdala and the anterior cingulate gyrus – areas of the "limbic system" that are highly inter-connected, there is logical support for this hypothesis. But the total emotional experience is undoubtedly a highly complex phenomena that involves, at any one moment, many parts of the cerebral cortex: posterior areas (perception), temporal areas (memory), parietal areas (space/time calculations, generalizations, verbal production). (Please note: The above correlation between "region and function," is an over-simplification. Each of these regions do not "contain" entire function, but each region has a central role for each function, while the total function involves a vast neuronal network. Edelman's concept of "distributed processes," meaning a neuronal phenomena that involves almost **all** parts of the brain, helps us understand this point.) (Edelman, 2004)

In psychotherapy training groups, we have found that this paradoxical concept, namely, that the emotion that is in consciousness and that is verbally expressed is **not** the central dynamic of emotional regulation, is not easy to grasp. Therefore, psychoanalysts, who have only recently been exposed to this new notion of the sub-cortical dynamics of emotions, may also have a certain difficulty in grasping this conceptual paradox.

### **Neurophysiological Consequences of "Lying Down on the Couch"**

I have focused on the sub-cortical mechanism of the amygdala in order to raise a number of issues regarding emotional regulation and change. But the neuronal map that comes from today's research involves many other areas that influence, directly and indirectly, emotional processes as well as their impact on thought, behaviour and visceral feelings.

More details regarding cortical and sub-cortical neuronal mechanisms can be found in several other articles. (Liss) Here is a summary of the mechanisms presented:

Ernst Gellhorn (1967) shows how contradictions between sympathetic and parasympathetic discharge (within the autonomic nervous system) could create psychological symptoms such as depression, apathy or mania as well as physiological symptoms such as nausea, diarrhea, vomiting and headaches. Emotional exploration can reduce these sympathetic-parasympathetic "knots" and thus help return the autonomic nervous system to normal functioning. Alan Schore's work expands the anatomical map of parasympathetic-sympathetic interactions and shows that several brain levels are involved: frontal lobe, hypothalamus, tegmentum and the brain stem. Henri Laborit (1969) proposes that prolonged inhibition of action, or "paralysis when faced with stress," involving the limbic system, could also create psychological and psychosomatic symptoms. For example, the resulting increase of corticosteroids interferes with the immune system. Gerald Edelman (1989) shows how the motor cortex sends collaterals to the basal ganglia. From the basal ganglia are sent "upward" messages to the thalamus, therefore influencing attention patterns and, perhaps, reducing the "overwhelming" emotions that arise during "helplessness," that is, when there is no action pattern to offset the emotional pattern. At the same time "downward" discharge from the action-coordinating basal ganglia to lower brain centers (locus ceruleus, raphe nucleus, pendulopontine tegmentum) then result in the production of activating neurotransmitters (dopamine, serotonin, catetcholamines) that are sent "upwards" and "wake up" the entire brain. Chronic depression, therefore, may result from the lack of this brain activation that requires basal ganglia action patterns. Finally, Bessel Van Kolk's research (1996) suggests that "indelible memory traces" involving the amygdala create the traumatic imprint. This problem is associated with diminished functioning of Broca's Area, suggesting that there is a reduction of language "containment" during traumatic experience. Thus the neurophysiological mechanism favours the continuous re-emergence of traumatic memories and flashbacks, symptoms characteristic of Post Traumatic Stress Disorder (PTSD). Based on these neural mechanisms, what can the psychoanalyst find useful?

When the patient lies down on the couch, he is favouring **parasympathetic "receptive" processes** as against the **sympathetic "active" processes** that characterize the normal daily routine. While sympathetic action processes favour attention toward perception of the outer environment, parasympathetic receptive processes favour attention toward the inner world. Is there neurophysiological evidence to support this distinction between sympathetic outer world attention and parasympathetic inner world introspection? First, it has been observed that corticosteroids, a stress hormone that is secreted in moments of stress and bodily activation, have the effect of pushing the thought sequences toward rapidity, fragmentation, and a compulsive attention toward external reality that neglects inner and subtle visceral experiences. In fact, this "thought pressure" might even reaches a manic state, as sometimes seen in Cushing's Disease, a physiological abnormality characterized by an abnormally high corticosteroid level. (Victor, 2001)

A second support comes from Gerald Edelman's concept of the "core dynamic," that is, a process of thalamic-cortical interaction that organizes attention.(Edelman, 2004) Edelman shows how basal ganglia action can orient attention to the immediate environment and also influence thinking. Thus, action-oriented attention decreases the limbic system's input (that is, input coming from the hypothalamus, amygdala and anterior cingulate gyrus), which usually reaches the orbito-frontal lobe and produces "visceral experience." This represents another mechanism that supports the psychoanalytic process of introspection. In summary, the Freudian principle that posits "acting out" as an escape from the therapeutic process finds a neurophysiological justification: The "acting out" obscures attention to subtle, internal visceral processes and the work of therapeutic exploration becomes blocked.

## **"Free Association" Changing Neural Connections**

An interesting study by Quarti and Renaud (1967) suggests another neurophysiological mechanism that explains the positive impact of psychoanalysis. Their EEG studies show that when a person is under a high level of stress, messages from the amygdala (the emotional hub) reach the thalamus (the major sub-cortical gate for ascending sensorial, motoric and emotional messages that reach the cortex). The thalamus, receiving this "high stress" input from the amygdala, shunts its activation from the thalamic lateral "discrete" nuclei, that are sending differentiated messages to the cortex, to thalamic midline "diffuse" nuclei that send less differentiated messages to the cortex. Two additional "emergency operations" occur at the very same time: the interaction among cortical associations is diminished, and feedback from cortical areas to the thalamus are also diminished. This creates a state of "high vigilance," but low thought complexity (lack of cortical interaction) and low flexibility (diminished feedback from the cortex to the thalamus.) Evidently, this favours rapid and incisive action for the emergency situation.

But when the amygdala's high stress" input to the thalamus becomes prolonged, as in chronic stress patterns and personality disorder, this creates a psychological disturbance: constant vigilance, repetitive and simplified thinking (obsessions), and rigidity. At the time of Quarti and Renaud's study, (1967) Edelman's concept of "distributed processes" was not yet known. But the researchers, aware of the psychoanalytic method of "free association," suggested a therapeutic approach based on systems theory: Changing one part of a system can potentially change other parts. Specifically, by means of the free association process, interaction among cortical areas is re-stimulated. This can affect the entire "high stress" amygdala-thalamus-cortical circuit, returning the thalamus to an activation of its "discrete" lateral nuclei, reinstating cortical feedback to the thalamus and diminishing the amygdala's "high stress" state. Thus the free association method of psychoanalysis has another mechanism of neurophysiological justification. (Other potential mechanisms will be cited below.)

### **From "Helplessness" and Total Action Inhibition to Psychoanalytic "Elaboration"**

Henri Laborit (1969) suggested that being in a state of "**paralysis**" or "total action inhibition," when confronted by stress, creates the preconditions for psychological disturbance as well as for psychosomatic illness. He described the limbic system nuclei (amygdala, septum and hippocampus) that regulate this state of action inhibition.

It is now understood that the amygdala sends both activating messages and inhibitory messages to the basal ganglia. The basal ganglia, also receiving collaterals from the cortical motor areas, represents the major sub-cortical regulator of automatic actions. (Edelman, 1989) This means that whenever an action is inhibited for emotional reasons, there will be found a barrier that prevents the activating emotional messages coming from the amygdala from reaching the action-producing basal ganglia.

The great pain and discomfort that comes from an inhibited action, "Stop that!", is not yet understood. One mechanism that can partially account for this specific emotional pain comes from studies involving the Nucleus Accumbens. (Kelley, 2004) This Nucleus represents the "gateway" to the basal ganglia, receiving inputs from sub-cortical centers (hypothalamus, amygdala and anterior cingulate gyrus) and also from cortical regions (motor areas, frontal lobes, parietal lobes and posterior sensory areas). The Nucleus Accumbens is dependent upon dopamine, which it receives from the substantia nigra, the lowest part of the basal ganglia, and also from the ventral tegmentum, one of several brainstem sources of "neuromodulators." (More about that in a later section.)

How does the Nucleus Accumbens affect our mood? When it receives a sufficient quantity of dopamine, the basal ganglia action system can be activated and the person's mood will be generally positive. This explains the effect of cocaine. The ingestion of cocaine results in this drug replacing the essential dopamine necessary for normal Nucleus Accumbens stimulation of the basal ganglia. In fact, the drug momentarily overstimulates the Nucleus Accumbens. The

result? The person's basal ganglia action system becomes highly aroused, creating a feeling of "power," "potency" and huge self-confidence. But the effect is short-lived. Once the drug is metabolized, the Nucleus Accumbens activation, suffering from dopamine depletion, is totally blocked. Furthermore, the return to normal levels of dopamine may take one or two months, even after one dose of cocaine. Thus "cocaine withdrawal," which goes with a feeling "impotence" (no power) and emotional pain, is explained by dopamine depletion of the Nucleus Accumbens. This brings to light one neurophysiological mechanism, among many, that can account for the emotional pain and depression stemming from chronic action inhibition. (An interesting study by G. Mac Donald, (2005) suggests that the very same neuronal networks that underlie physical pain overlap with the areas that produce emotional pain.)

How does psychoanalytic work diminish this state of action inhibition and paralysis? By means of verbal elaboration of the emotional pain. **To "talk about inner experience" is a very significant action!** In addition, "word sequences" may be highly connected to "action sequences," given that the architecture of motor areas that regulate behaviour then evolved to create the specific language regions, Wernicke's area and Broca's area. (Lieberman, 2000) The therapeutic significance is that psychoanalytic elaboration, especially during states of emotional desperation and psychological impotence, interrupts the pathological state of total inhibition. We can imagine how the Nucleus Accumbens begins to renew its dopamine reserves by means of the "talking cure"; in conclusion, the patient, over time, can reach a renewed state of brain equilibrium.

### **Mechanisms of Emotional Containmentment**

Clinical experience shows that emotions that are described, elaborated, verbally defined and analyzed become less intrusive and dysfunctional. The emotion becomes "contained." One way to picture this therapeutic phenomena is to imagine a conscious emotion becoming excessively intense, and then, by means of therapeutic elaboration, a different conscious force composed of verbal and cognitive elements is called forth to define, surround and limit the emotion, thereby giving it "frontiers of containmentment." This point of view involves an integration of two **cortical** processes.

But recent neurophysiological research supports another type of mechanism that involves sub-cortical processes: The excessive emotion, according to this model, is understood to stem from non-conscious sub-cortical processes, especially involving the amygdala. The cognitive-verbal elaboration, stemming from cortical regions (frontal, parietal and temporal lobes) sends inhibitory messages "downwards" (cortex to sub-cortex), and this can account for the emotional containmentment.

How does the amygdala undergo pathological changes that result in excessive increases of both neuronal processes and manifest emotions? Since the amygdala receives input from a multiplicity of cortical and sub-cortical regions, we can suggest different mechanisms which can produce this problem of "run-away" or "getting out of control."

### **The Frontal Lobes**

There is a great deal of evidence that cognitive conscious frontal lobe processes have an overall **inhibitory effect** on the unconscious amygdala emotions. Frontal lobe studies by Damasio (2003) and Rolls (1999) support this mechanism. Roffman's review of the neurophysiological correlates of psychotherapy (2004) shows that in the successful treatment of various disorders, Neuroimaging shows an increase in frontal lobe "cognitive" processes and a decrease in amygdala "emotional" processes.

### **The Anterior Cingulate Gyrus: "A Major Self-Other Modulator"**

Recent research by G. Bush (2000) and Kevin Ochsner (2001, 2004), among others, has added a new element to our cortical/sub-cortical brain map by revealing various psychological functions of the anterior cingulate gyrus: registering disappointed expectations, revealing novelty, indicating mistaken actions, expressing separation distress, etc. These researchers have shown that when cortical input to the anterior cingulate gyrus increases, the emotional process within this region begins to diminish. (We can expect that this important region will reveal yet other functions of great psychological importance – including the body scheme and self-other dynamics -- given its intermediate position between the orbitofrontal lobe, which probably registers the conscious component of emotions, and the amygdala, which regulates the unconscious component of emotions.)

## Verbal Containment of Emotions

What does this mean for the psychoanalyst? We can imagine that the cortically-based “free association” process results in an increased number of message being sent “downwards”, first to the anterior cingulate gyrus and then to the amygdala. Thus the psychological elaboration of free association diminishes the unconscious emotional system. This is especially useful when the amygdala emotions are “getting out of hand” or “going wild,” that is, dysfunctionally excessive.

Bessel Van Kolk, in his important book, **Traumatic Stress, (1996)** suggests that the amygdala becomes **over-active** when there is a traumatic experience. He notes that in post-traumatic stress disorder (PTSD), not only is the amygdala over-active, but Broca’s area (language regulation) is **under-active**. This is believed to correspond to the clinical experience, “There are no words available to transmit the traumatic experience.” Thus, to simply “find the words” that correspond to the experience can have a certain benefit. We can imagine how the rich multiplicity of Broca area linguistic patterns, a cortical phenomena, result in new verbal messages being sent “downwards” to the sub-cortical amygdala, and thus, the emotional “run-away” of traumatic experience begins to be “contained” by the verbal elaboration and sharing.

The therapeutic strategy of Pat Ogden, (2000) a psychotherapist who collaborates with Van Kolk, can have important implications for psychoanalysis. Ogden points out that it is essential for the patient to experience a “tolerable dosage” of the traumatic experience in order to obtain therapeutic benefits. Being “overwhelmed” by the trauma can have negative consequences. But also – and this is the point for psychoanalytic exploration – to not feel at all a part of the traumatic emotion is like “escaping” from the experience. The patient may verbalize many things, even “about” the experience, but the emotion itself is not accessed or modified.

This therapeutic principle can apply to many psychotherapeutic approaches: A certain dosage of the difficult emotion – the pain, the depression, the anguish, the rage, the rancour – must be actually felt and re-lived during the therapeutic association. **It is possible that the psychoanalytic work involving the analysis of the transfer relationship can become the vehicle for the re-living of painful emotions at a tolerable dosage.** The point is that a part of the emotional charge must be actually felt during the psychoanalytic session for the various brain levels – cortical and sub-cortical – to be actively engaged and modified. Only then can the new mental associations, fantasies, images, cognitive contextualization, and so on, produce therapeutic benefit. Without coming into contact with the underlying emotion, the patient might conclude, “I talked and talked about my problem, but nothing has changed.”

In a sense, D.O. Hebb, in the 1950’s, had predicted this necessity of linking the emotion to the mental elaboration: **“Neurons that fire together, wire together,”** was his oft quoted dictum. This means that **both** neuronal circuits that correspond to the emotion must become **activated**: the orbitofrontal lobe for the conscious component and, at the same time, the sub-cortical amygdala for the unconscious component. Only then can the new neuronal circuits

generated by psychoanalytic "elaboration" have a significant emotional impact. Without this neural connection between the disturbing emotion and the cortical-based elaboration, the two neural circuits remain separated. The words are empty and the emotion stays the same.

Objection: Can the psychoanalytic work of metaphor, fantasy and dream analysis substitute for the "felt emotion", given that their neural circuits can still connect to the primary trauma? Response: A neurophysiological map permits the answer, theoretically, to be "yes," based on the concept of "distributed processes." (Edelman, 2004) Nevertheless, the connection remains hypothetical until empirical evidence gives support or opposition.

### **Cell Death in the Over-Active Hippocampus**

Another mechanism for amygdala "run away" is related to the **hippocampus**. This sub-cortical region, very closely connected to the amygdala, regulates several functions, including remembering the spacial dimension of events, the focus of "attention" (upwards discharge to the cortex), and "contextualization" of the amygdala's emotional processes. The overall impact of hippocampus discharge upon the amygdala is inhibitory. (Rolls, 1999)

An interesting finding is that **long-term depression is connected with a reduction of neurons in the hippocampus**, and therefore, a reduction of amygdala containment. How does the hippocampal undergo cell destruction? The hypothesis is that in the "immediate stress phrase" of an emotion – loss or attack – there is an increase of corticosteroids (Liss, Miriam, 2005). (Mechanism: From amygdala to hypothalamus to adrenal cortex, for corticosteroid production, and then corticosteroid feedback to the amygdala and hippocampus for brain regulation.) The hippocampus is especially sensitive to corticosteroid levels; when its plasma concentration rises, cell metabolism within the hippocampus becomes immediately increased. And what happens when the heightened corticosteroid level becomes prolonged over time, as in chronic emotional stress? The hippocampal cells become exhausted, losing dendritic substance, until undergoing cell atrophy and death. (Rolls, 1999)

Two consequences of this disturbed hippocampal-amygdala mechanism: First, it explains why chronic depression may sometimes be refractory to psychotherapy. The hippocampal-amygdala "containment" mechanism is no longer "structurally" possible. The amygdala run-away becomes embedded, and therapeutic efforts fail. Second, it explains why it is essential to treat a depression in the earliest period. Waiting too long can permit the **chronicity** of neuronal patterns. The risk of chronicity is that the dysfunctional neural mechanism then becomes "structured" within the brain circuitry. (As we learn more about NMDA receptor mechanisms and their influence on Long Term Potential (LTP) circuitry changes, we will enhance our multi-level knowledge of neuronal events.) (Rolls, 1999)

### **The Locus Coeruleus-Noradrenalin Run-Away**

The impact of brain neuromodulators is a new frontier of psycho-neurophysiologic research. For example, it has been shown that the amygdala discharges downwards to the locus coeruleus, which then increases its noradrenalin production and secretion. Noradrenalin exerts an important function in activating the organism in order to face stress: This neuromodulator increases attention to stimuli that are "salient" to the stressful event, thus increasing mental focus.

The noradrenalin secreted by the locus coeruleus creates a positive feedback effect upon the amygdala while, at the same time, exerting a negative feedback effect upon the anterior cingulate gyrus (ACG). If these feedback mechanisms become excessively intense and prolonged, the brain enters a dysfunctional state: The amygdala process increases because it receives positive feedback from the noradrenalin, while the ACG, which normally inhibits and reduces amygdala emotions, becomes itself inhibited by the noradrenalin and, thereby, can no longer effectively "contain" the amygdala. (Berridge, 2003)

## Multiple Causality in All Brain Events

In the last section we have presented various sub-cortical-cortical mechanisms which can explain how a dysfunctional amygdala "run-away" can create excessive emotionality.

If we now ask, "**Which one** of these brain mechanisms explains the dysfunctional amygdala "run-away?", we are posing a question which is no longer appropriate. To search for a **single** brain mechanism is a part of A to B "unilateral thinking." The complexity of the brain with its multiple levels and circularity of interactions is best understood by means of "Systems Thinking." (DeRosnay, 1975)

Systems Thinking creates an epistemological orientation based on the assumption that every psychological disturbance will be based on multiple mechanisms. Edelman's concept of "distributed processes" (2004) offers a general neuronal picture that emphasizes the extensiveness of neuronal networks. More specifically, a recent study Anne Kelly (2004) of the University of Wisconsin shows that "**distributed processes**" can involve repeated molecular mechanisms functioning in different brain areas at the very same time. (see Addendum, "The Repetition of Molecular Mechanisms")

Evidence for "repeated brain mechanisms" comes from another source. Both Antonio Damasio (2003) and Edmund Rolls (1999), who are bringing to light various neuronal mechanisms involving the frontal lobes, have proposed that **frontal lobe connections partially reproduce amygdala connections**. For example, the frontal lobes show important connections with the temporal lobe, the basal ganglia, the hypothalamus and visceral nuclei. The more archaic amygdala shows the same connections. Both authors suggest that the frontal lobes, by means of evolution, offer a more complex architecture that reproduces and enlarges amygdala functions. Both regions are activated at the same time. This means that "distributed processes" creates a repetition of neural processes, based on multiple levels of cerebral architecture, that have developed in various moments of species evolution.

And the psychoanalyst? Understanding that multiple levels of the brain, and especially, the "invisible" sub-cortical levels, must be involved in emotional change, justifies several aspects of the psychoanalytic protocol: **intensity**, to reach deeper brain areas); **complexity**, to integrate the various distributed brain processes pertinent to a single moment, and **repetition over time**, to respect the fact that long term memories that are almost "indelible" require therapeutic methods based on repetition over time in order to become modified.

## Lower Brain Neuromodulator Activators

Let us return to the example of neuroadrenalin secreted by the locus coeruleus. This is but one example of a new field of brain research: the lower brainstem **neuromodulators**. Here is a larger picture of the situation.

In the brain stem (pons and medulla) we have four centers of neuromodulator production and secretion. These centers produce (at least) four different neuromodulators. Once a neuromodulator is produced, it is secreted in a global way "upwards" toward higher centers reaching both the sub-cortical and cortical levels. Thus they have a panoramic effect upon brain functioning.

Gerald Edelman (2004) calls these centers the "valuational system", based on their effect of orienting a brain state toward a positive or negative valence. For the moment I will call them the neuromodulator "activating system" in order to emphasize their diverse ways for activating the brain, whether the overall impact is toward a negative or positive valence. (In fact, many brain states, such as when getting prepared for a new challenge, mulling over past actions, evaluating family relationships, etc. represent mixtures of negative and positive feelings.

What is more certain than the presence of a strictly positive or negative emotional valence is the fact that the brain is more "active", thus, the term, "brain stem activating system".)

The following is a list of brain centers, neuromodulators and functions (always remembering that such categorization is over-simplified and will be modified with future research):

<b>Lower Brain Region</b>	<b>Neuromodulator</b>	<b>Functions</b>
Tegmentum	Dopamine	Action for Reward
Raphe Nuclei	Serotonin	Bonding, Visceral Feeling
Locus Coeruleus	Noradrenalin	Vigorous action, vigilance
Pendulopontine Nuclei	Acetylcholine	Learning, memory

Pharmacological treatment is often based on these neuromodulators. More specifically, the tricyclic compounds and monoamineoxidase inhibiting anti-depressants increase noradrenalin at the synaptic level while Prozac increases serotonin. These treatment approaches favour a positive mood and decreased negative emotions. This supports the notion that neuromodulator production and secretion, originating from brainstem centers, have enormous effect on overall mood. But while the use of pharmacological products represents one form of "organic" treatment, mind-body connections permit us to consider all forms of psychotherapy as partially "organic" in their effects, that is, as affecting cortical and sub-cortical neuronal processes as well as the body's somatic processes. (At the very beginning, for example, we pointed out how the lying down position of classical psychoanalysis shunts activating sympathetic processes toward receptive parasympathetic processes. This is an example of "organic" change elicited by a psychotherapeutic method.)

The neural pathways stimulating these neuromodulator activator centers are only now beginning to be worked out. We can imagine that each center will be shown, in the future, to be influenced by several higher centers. For the moment several connections have been revealed: The substantial nigra of the basal ganglia stimulates dopamine production in the tegmentum. The amygdala stimulates noradrenalin production in the locus coeruleus. In both cases, the neuromodulator reinforces functioning of the higher center. That means that the dopamine prolongs the action activation of the basal ganglia while the noradrenalin increases the emotional activation of the amygdala. We can expect that social bonding will increase serotonin production while novel situations requiring new learning will activate acetylcholine production.

And the consequence for the psychoanalyst? Once again, the overall message is simple: The psychotherapeutic elaboration, with intensity, complexity and repetition over time, will not only modify the cortical neural circuits connected to thoughts and consciousness, but will also modify the numerous sub-cortical circuits that represent the subterranean dynamics of our emotions.

### **Conclusion:**

Brain studies are revealing the specific dynamic of how emotions are regulated by both cortical and sub-cortical processes. The field is new and we can expect greater precision and enlargement of the map in the coming years. The psychoanalyst, working with free association, dream analysis, the transfer-countertransfer relationship, etc., can find support and new stimulation as the neurophysiological maps of emotional dynamics become more extensive and precise.

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**Addendum, "The Repetition of Molecular Mechanisms in Different Brain Areas"** (from page 12)

Anne Kelley (2004) shows that sub-cortical molecule-receptor mechanisms can involve both Dopamine<sup>1</sup> and NMDA receptors which, when acting together, increase dramatically neuronal activation and long-term memory. This molecular mechanism comes into action when there is stimulation of the Nucleus Accumbens. Kelley describes the "core" of the Nucleus Accumbens Nucleus as a feedforward mechanism that influences basal ganglia action patterns. At the same time, the "shell" of the Nucleus Accumbens sends feedback messages that return to limbic system regions, in fact, to those very same regions from which the Nucleus Accumbens received its input: the amygdala, the hypothalamus and the orbitofrontal lobe. These multiple connections not only create a functional unit that combines limbic emotions to N. Accumbens action preparation, but **distributed regions are all activated simultaneously by the same dopamine and NMDA-receptor mechanisms**. Kelley suggests that this principle of "repeated mechanisms" will be generalized to other **molecule-receptor interactions**. We can enlarge this prediction. When "distributed processes" underlying brain states will be more thoroughly studied, we will probably discover a repetition of **neuronal circuits** that are similar to one another and that will become activated during any specific function.

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