

Vertical Brain Mechanisms for Understanding the Psychotherapeutic Process

How Neurophysiological Maps Justify the Different Schools of Psychotherapy

(references to be added)

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Why is Neurophysiological Research a New Scientific Frontier?

Science advances with changes in technology. The use of Neuroimaging techniques, and especially of Positron Emission Tomography and Functional Magnetic Resonance, permit the visualisation of certain brain processes. (Goldin,2005) More particularly, when the subject is exposed to specific stimuli – photos of faces, problem-solving tasks, etc. -- to measure the Subject's brain processes can show their correlation with the stimulus. In this way conscious experiences are becoming correlated with physiological brain processes. This is certainly a great advance for scientific inquiry. As Gerald Edelman says, scientific study means going back and forth between physiological mechanisms and phenomenal issues. (Edelman,2004)

The P.E.T. does not measure the neural firing pattern directly. Rather, the increased heat is due to increased metabolism. This means that a specific brain area is being activated by specific stimuli or particular experiences. Although this is not a direct measure of the neural firing pattern, the observation of increased metabolism is sufficient to show pertinent correlations.

Our goal in this article is to present the main models that emerge from these laboratory studies, followed by the hypotheses one can make for understanding clinical phenomena that cannot be observed in the laboratory. The emphasis will be upon the work of Gerald Edelman (Nobel Prize Winner), Edmund Rolls and Joseph LeDoux. Other researchers will also be mentioned.

A New View of Psychology

Before presenting the brain models, let us first suggest some of the important consequences derived from this type of study.

1. The model of emotions presented by Freud can be very misleading and must be entirely overhauled.
2. Our approach toward introspection can change. Previously, the goal of introspection was to bring into explicit (clear, verbalized) consciousness emotions and experiences that have been repressed and, therefore, put outside of our explicit conscious awareness. (The Freudian strategy) The new goal is to permit intuitive (implicit) experiences and unconscious (or non-conscious) physiological knowledge to influence conscious experience. (Stern,2004) These non-explicit mind-body processes need not be brought into consciousness in order to change their pattern. In fact, the traditional psychoanalytic goal, "to make the unconscious conscious," at certain neurophysiological levels, is impossible! (We are referring to sub-cortical levels of the brain which cannot

be directly correlated with conscious experience, but which can **influence** the conscious experiences that are occurring at the cortical brain level. Many neurophysiologists agree that the complex architecture of the cortex can support consciousness, while the less complex architecture of the sub-cortex cannot create conscious experience.)

3. **The problem of "pathology" is not due to the negative emotions.** Negative emotions and other disturbances of the personality are the outcome of a **disequilibrium** among many brain processes. These processes include specific sub-cortical areas and functions: the **basal ganglia** for action, the **amygdala** for emotions, the **thalamus** for attention and optimal stimulation levels, the **hippocampus** for spacial representation, various **lower brain centers** for the production of specific activating neuromodulators, and the **solitary nucleus** and dorsal motor nucleus of the vagus for visceral regulation and feedback.

4. Therefore, if we wish to help the patient diminish his anxiety, overcome a phobia, resolve a depression, raise self-esteem, find hope, the therapeutic approach is **not** to alter a single factor but, rather, to create changes that influence all brain levels and permit a return to equilibrium.

These consequences as well as others will be developed in the text.

Multiple Inputs and Outputs of the Sub-Cortical Amygdala

The amygdala gives a sub-cortical basis for rage and fear reactions. This was understood in research conducted in the 1930's. Ablation of the amygdala and/or its connections with the temporal lobe produced an apathetic non-reactive animal. This is called the Kluver-Bucy syndrome. (Emery,2000)

Joseph LeDoux has now made extensive studies of the amygdala. This sub-cortical structure receives at least ten incoming neural pathways from different brain areas, and at least ten outgoing neural pathways to activate a diversity of functions. This makes the amygdala a central coordinator for numerous brain processes. (LeDoux,1999) In other words, our emotional life is due to the integration of many parts of the brain and, in turn, "emotional reactions" constitute a great complex of neural-somatic processes. These neural processes produce "emotions" as a complex psychophysical process. The emotional process includes present experience, past memory, behaviour, autonomic nervous system processes, hormonal effects, arousal level, and so on.

The amygdala is also composed of at least twelve different centers, and these too in systemic interaction. For example, many of the inputs first reach the amygdala at the level of the Lateral Nucleus, while other inputs enter at the level of the Nucleus Basalis. What happens to these inputs? The Central Nucleus of the amygdala receives information from both of these nuclei, as well as from others, and serves as the major **output** center. (Rolls,1999) Thus, given this high complexity of **input, output and internal integration**, the neurophysiological map of the amygdala justifies the conclusion that our emotional life has a central role in our existence.

Before turning to the clinical hypotheses that can be derived from this particular neurophysiological map, let us present another recent dimension of brain research, that of Gerald Edelman.

From Motor Sequence to Verbal Sequence

Gerald Edelman's research (1989,2004) focuses on the interaction between cortical and sub-cortical levels of the brain. More specifically, Edelman shows how the interaction between two non-conscious sub-cortical centers – the basal ganglia and the thalamus – affect conscious cortical processes. It was already known that that basal ganglia – thalamus interplay influences motor acts by means of the thalamus sending patterned messages to the motor areas of the cortex. Edelman adds that the sub-cortical dynamic also influences the cortical functions of perception and thinking. For example, the basal ganglia (in coordinated activity with the cerebellum) organizes our **sequences of motor movement**, absolutely necessary for goal-directed action. But Edelman adds another function of the basal ganglia – thalamic circuit: to influence our **sequences of thought**. For example, we can make verbal phrases because we have the capacity of creating **sequences of words**.

The architecture of the motor system permits **sequences** of neural firing. The cortical areas for verbal thought production were evolved from these motor structures. It is as if evolution used the motor sequence architecture as a template for the development of new brain areas (Brocca's and Wernicke's areas), and this permitted the creation of verbal sequences.

Vertical Brain Mechanisms that Produce "Helplessness" and "Hopelessness"

The thalamus, in classical neurophysiological studies, is known to receive inputs from the environment through the Ascending Reticular System (ARS). Edelman adds to this scheme the influence of the non-conscious muscular coordination upon the attention-organizing thalamus. Edelman presents this specific circuit as influencing the "dynamic core" that controls consciousness. In summary, the sub-cortical basal ganglia connect to the thalamus, and from there messages are sent up diverse cortical areas.

Edelman's model becomes yet more complex when he adds the influence of lower brain centers. These areas within the pons and the medulla send neuromodulators in an upwards direction to be distributed throughout the entire brain, that is, to both cortical and non-cortical areas. (These neuromodulators and the brainstem areas that secrete them, are presented in the Table, "Brain Stem Neuromodulators.") Such neuromodulators create arousal, influence mood, enhance memory and learning, and orient our awareness toward positive or negative experiences. It is interesting that many psychopharmaceutical medicines increase the presence of these neuromodulators, supporting our hypothesis that mood, emotion, thought and behaviour are dependent on these complex **vertical brain mechanisms**. (Note: Edelman calls these brainstem neuromodulators the "value system", because of their capacity for orienting the person toward positive or negative experience. I prefer the term, "activating system," in order to emphasize their impact upon attention, learning and motor activation.)

Going backwards in the causal chain, what stimulates and intensifies this lower brain "activation system"? Here is where Edelman's model has important consequences for the clinical psychotherapist. **The sub-cortical basal ganglia action system has an especially important role in stimulating our lower brain "activating system."** We now have one link in the chain (and later, we will examine others) that explains how "helplessness" (no action possible) creates "hopelessness" (a negative state with reduced brain activation) and, if prolonged in time, can create depression.

We can now enlarge our mind-body model: The **sub-cortical** basal ganglia receive an important input from the **cortical** frontal lobe. What is the function? The frontal lobe (which creates conscious intentions) sends patterned messages **downwards** to the basal ganglia in order to prepare and realize actions according to our conscious goals. And from the basal ganglia messages are sent **further down** into the brain stem (Edelman's work) in order to stimulate the lower brain activators (dopamine, etc.) And these lower brain activators then send messages **upwards** to stimulate the entire brain. In synthesis, just our intention to act, as well as the action itself, can stimulate the lower brain activating system, that then sends messages upwards for general brain activation. And if we have no plan or hope for action, when faced with stress, we can feel "lost," "defeated," "broken down," and enter into a state of depression.

This is the state of "prolonged emotional paralysis" that Henri Laborit (1969) describes as the pre-conditions for emotional and psychosomatic disturbance.

A life style of passivity can also favour this state of "action inhibition": sitting all day in the classroom, in the office, in front of the computer, at the telephone, and then returning home – "too tired" to do aerobic exercise of sport or running – and so plumping oneself down in front of the television for the entire evening. (Becoming a "couch potato.") What is missing? Vigorous action that can return the sub-cortical brain centers to a state of reciprocal activation and healthy equilibrium.

Sharing Emotions is a Type of Action

This neurophysiological map also helps us understand why "activating" psychotherapeutic approaches can be effective. As the person remembers scenes of emotional provocation – conflicts, separation, criticism, misunderstanding – there is often a primary reaction of, "I don't know what to do." The amygdala fight-or-flight mechanism is activated, but the connection to the basal ganglia action system is blocked. "I feel like strangling him!" may be the primary impulse of the amygdala aggression system. Or, "I wanted to run and hide" may come from the amygdala fear system. But since these intentions cannot be carried out by the basal ganglia – they are "taboo," "dangerous" or "humiliating" – the person remains stuck.

Traumatic experiences are extreme examples of "nothing could be done." The memory of physical abuse, sexual abuse, war-time bombardments, humiliation in the classroom, loss of a parent, or other "overwhelming events" – "It was too much!" – is maintained from childhood as an "indelible memory trace" (Bessel Van Kolk's term, 1996). In fact, every time an element of the experience returns to mind, the memory traces are re-activated and reinforced. We can imagine how negative experiences return to mind at night while the person toss and turns in his bed, unable to sleep. The memory traces of the unhappy episodes are re-activated and reinforced, usually as a single image or visceral sensation, rather than as a more comprehensive sequence, while the sensation of "Nothing to do!" is also reinforced. Thus we have neuroses and emotional knots.

When the psychotherapist asks, "What do you remember of that unhappy episode?", just recounting the event attaches a certain degree basal ganglia action to the amygdala episode. This is important because many negative experiences are retained as visceral and visual memories that have no language component. Giving words to experience is both a symbolic

and motor activity. Even when the words are not said out loud, the internal language includes a motor component: "internal talk." (Lieberman,2000)

As the patient describes the event, several action components become attached to the negative event: the spoken language, as we have just said, and also other actions that are inherent to communication: gestural language, activated facial expression and increased breathing. Some therapists, for example, focus on the breathing as an essential element. They ask their client, "Can you help your breath as a you recount that very difficult moment?" The blocked breathing is a classical part of prolonged action inhibition. (Boadella, 1986)

Also recounting the negative event blow by blow, "What first happened?....And then?....", changes the conscious cortical component from an isolated instance to a sequence of instances. Pat Ogden, a psychotherapist who collaborates with Bessel Van Kolk in the study and treatment of traumatic experience, emphasizes to her patients, "Let's get the sequence." (Ogden, 2000) Neuroimaging studies in the future may reveal the beneficial changes of conscious-cortical sequencing of traumatic memories. Not only are cortical associations opened up, but we can hypothesize that this will feed back to alter sub-cortical basal ganglia routines, thalamic attentional processes and amygdala emotions.

Another interesting mechanism involves the anterior cingulate gyrus. This area has shown a decrease of emotional processes when there is an increase of cognitive processes, giving a neurophysiological basis – and there are undoubtedly others --for the control of emotionality by means of cognition. (Bush,2005)

Understanding "Catharsis"

One of the mysteries of the psychotherapeutic process, and of all emotional change, is how **catharsis** works. Catharsis means that active expression of an emotion leads to a two-step process: intensification of the emotions, like "tears of sadness" or "discharge of rage," will be followed by a certain relief. It is like an arrow shot in the air; the emotion gets more intense and then comes down. It becomes subdued, at least, for the moment.

We can easily imagine some of the sub-cortical correlations: the amygdala increases its connection with basal ganglia behaviour and with hippocampal spacial relations. We can call these connections, more or less, **horizontal** connections. Also, we can expect to see a momentary intensification in amygdala **downward** discharge to the hypothalamus, central gray matter, lower brain stem activator centers and the nucleus solitarius, (activating the autonomic nervous system and influencing hormonal output). At the same time, the amygdala creates an **upward** discharge to the cingulate gyrus, orbital frontal lobe, temporal lobe and parietal lobe (influencing thoughts, attitudes, and the consciousness of the emotion). This period of emotional intensification and discharge, that is, this period of "catharsis," is followed by a feeling of emotional relief. We can expect, furthermore, that some of the brain regions noted above, but not necessarily all, will show a certain degree of de-activation, when the subjects expresses emotional relief.

Let us try to imagine the clinical events that this intensification/de-intensification of **catharsis** might correspond to. A very strict scientist might criticise this hypothesis-making as pre-mature. On the other hand, given that Neuroimaging researchers are still limited to hypotheses that are mainly pertinent to the laboratory setting, but not very relevant to full blown life situations, such hypothesis-making may stimulate researchers to undertake studies

that are closer to reality. (Ochsner,2001) The philosopher of scientist, Carl Hempel (1952) pointed out the importance of science evolving, over time, to the study "natural systems."

Hypotheses Regarding Catharsis

What Might Happen When the Amygdala Momentarily Intensifies

Brain Area	Clinical Correlation
Horizontal Processes	
Basal Ganglia	Behavioral expression
Hippocampus	Spacial memory
Downward Processes	
Hypothalamus	Sympathetic-parasympathetic activation (Increased sympathetic in anger, increased parasympathetic in crying)
Central Gray Matter	Increased contact with the "emotional pain"
Brain Stem Activating Nuclei	Increased intensity of experience, especially of attention and recall
Solitary Nucleus, Dorsal Motor	Changes of peristalsis, gastrointestinal secretion, gastrointestinal blood flow and
Nucleus of the Vagus	other gastrointestinal phenomena
Upward Processes	
Cingulate Gyrus	More intense feeling of body presence
Orbito-frontal Lobe	Increased "emotional feeling"
Parietal Lobe	More intense feeling of "reality"
Temporal Lobe	More vivid memory

Let us avoid misunderstanding: This scheme is immensely oversimplified. Its purpose is only to create a frame of reference for reflecting upon clinical phenomena and for imagining a neurophysiological map that presents a certain degree of correlation. Here are several of its limitations:

Limitations of Mapping Neurophysiological-Clinical Correlations

1. Each brain state involves “distributed processes” that involve activation of all (or, at least, numerous) areas of the brain. No function is found “in” a single area, but involves a vast repertory of areas. The areas noted in the correlation signify that these areas have an important and, sometimes, essential role in the overall function.
2. This model of brain interaction might, at first, look like a model based on “linear causality”: A causes B causes C, etc. In the above example, it might appear that the emotional processes connected with the amygdala represent the starting point. However, in the brain, there is no single starting point. The concept of “**re-entry**” (Edelman) means that at every moment there are **reciprocal interactions** among many brain area, in this case, involving the amygdala. Thus, when there is an emotional disturbance, **many** of these subcortical brain interactions are in disequilibrium.
3. There are many brain areas that have not yet been included in this scheme.
4. The “downward processes” in the above map are limited to regions of the brain. But this is not the whole picture. There are other “downward processes” based on neurons that descend still further, beyond the brain stem, into the somatic body, in order to regulate **body functions**. (Simultaneously, there are downward processes that start out from the higher cortical motor area and descend to the body’s muscle system.) Beyond that, the somatic body is sending its own innumerable messages **upwards** to the brain. Some therapeutic methods, such as massage, Shiatsu, acupuncture, hydrotherapy, reflex therapy, pressure point therapy, as well as Bioenergetic exercises, are based on altering body processes directly as well as upon changing the body’s input to the brain.

The Neurophysiological Basis of Different Schools of Psychotherapy

We have emphasized the cathartic process of psychotherapy because it is also relevant to daily life. Any time we are sharing our feelings, living out emotional outbursts, or even experiencing emotions vicariously while watching a dramatic film, there is some degree of catharsis going on.

We can employ our neurophysiological map to hypothesize the way various psychotherapeutic approaches can influence our emotional life. Each approach represents another type of input to the amygdala.

Brain Area	Psychotherapeutic Approach
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Frontal-Parietal-Temporal	Psychoanalytic Insight
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Lobe Integration

Frontal-Parietal-Posterior Lobe Integration	Gestalt Therapy (emphasizing here-and-now experience)
Posterior-Temporal Lobe with Motor Cortex – Basal Ganglia – Thalamus integration	Body-Oriented Psychotherapy
Frontal-Parietal-Temporal- Posterior Lobes integrated with the Basal Ganglia and Motor Cortex	Psychodrama
Frontal-Parietal-Temporal Lobe Integration	Cognitive Therapy
Basal Ganglia-Thalamus- Motor Cortex Integration	Behavioural Therapy
Frontal-Parietal Lobe Integration	Transactional Analysis (categorizing and guiding emotional impulses according to parent-adult- categories)
Posterior Lobe	Positive Visualization
Posterior Lobe – Basal Ganglia – Thalamus	Guided Daydream (visualization combined with imagined movement)

Once again, we are creating a model based upon vast over-simplification. Each psychotherapeutic approach can justifiably claim that its methods are multiple and that the brain-psychological correlations involved in its psychotherapeutic strategy involve functions more complex than those indicated in the above Table. The purpose, nevertheless, is to indicate that even at this early point of neurophysiological research, we can begin to hypothesize brain mechanisms for different psychotherapeutic processes.

Furthermore, each method is valid! The fact of “distributed processes” means that the entire brain is involved in every psychotherapeutic intervention. Therefore, if the intervention emphasizes one or several particular brain functions (“insight” in psychoanalysis, “here-and-now experience” in Gestalt Therapy, and so on), the long-term impact of the method will be “distributed” and affect all brain regions, that is, all levels of the personality.

Clinical-Neurophysiological Correlations Are Useful For Clinicians and Researchers

In summary, recent research in the neurophysiology of emotions allows us to make hypotheses about the neural mechanisms of emotion. The **vertical mechanisms** of the brain show how the physiological unconscious is in reciprocal interplay with the psychological state of consciousness. For example, the disconnection between the emotional amygdala and action-producing basal ganglia can explain why "hopelessness" (amygdala) and "helplessness" (basal ganglia) reinforce one another. In contrast, to merely share emotions with another person can partly overcome this disconnection by means of activating the basal ganglia.

In conclusion, the diversity of approaches represented by the different Schools of Psychotherapy becomes justified. The neurophysiological principle of "distributed processes" means that a new input, in any area, can potentially change all other areas involved in a particular circuit. The neurophysiological map helps us picture the functional relations that correlate with these neuronal pathways. Given that the map will become more detailed and complex over time, the functions that are correlated will become correspondingly detailed and complex.

For the researcher in the field of Neurosciences, these correlations can encourage research protocols, using Neuroimaging, that involve complex emotional experiences rather than simple perceptual and cognitive tests. Thus the job of correlating brain functions with psychotherapeutic processes can be of benefit for both clinicians and researchers.

Bibliografia

Boadella, David, e Liss, Jerome, **La Psicoterapia del Corpo**, Roma, Ed. Astrolabio, 1986.

Bush, George e al, "**Cognitive and Emotional Influence in anterior cingulate Cortex**", Elsevier Science Ltd, 2000, pp. 215-217.

Edelman, Gerald M., **The Remembered Present**, A Biological Theory of Consciousness, BasicBooks, 1989.

Edelman, Gerald M., and Tononi, Giulio, **A Universe of Consciousness**, (traduzione italiana, **Un Universo della Coscienza**, ed. Einaudi, 2000), New York, Basic Books, 2000.

Edelman, Gerard M., **The Remembered Present**, BasicBooks, 1989.

Edelman, Gerard M., **Wider Than The Sky**, Yale University Press, 2004.

Emery, Nathan and Amaral, David, "The Role of the Amygdala in Primate Social Cognition", (Ch. 8), in Lane, Richard D. & Nadel, Lynn, **Cognitive Neuroscience of Emotion**, Oxford University Press, 2002.

Goldin P.R. et al, "The Neural Bases of Amusement and Sadness", **Neuroimage**, 2005 Aug. 1; 27(1): 26-36

Hempel, Carl, (1952) **Fundamentals of Concept Formations in Empirical Science**, International Encyclopedia of Unified Science, Vol II, No. 7, Chicago, University Press.

Laborit, Henri, **L'Inibizione dell'Azione**, (traduzione di A. Meluzzi) Milano, Il Saggiatore, 1986.

LeDoux, Joseph, **The Emotional Brain**, Phoenix, 2003.

Lieberman, Philip, **Human Language and Our Reptilian Brain**, Cambridge, Mass., Harvard University Press, 2000

Ochsner, Kevin and Barrett, Lisa, "A Multiprocess Perspective on the Neuroscience of Emotion", (Ch.2) in Mayne, Tracy J. & Bonanno, George A., **Emotions. Current Issues and Future Directions**, New York, The Guilford Press, 2001.

Ogden, Past & Minton, Kekuni, "One Method for Processing Traumatic memory", **Traumatology**, Volume VI, Issue 3, Oct. 2000.

Rolls, Edmund T., "The Rules of the Olfactory Representations Found in the Orbitofrontal Cortex Olfactory Areas in Primates", **Symposium AchemS XXII**, 2004.

Rolls, Edmund T., **The Brain and The Emotion**, Oxford University Press, 1999.