

The Neurophysiology of the Emotions and of Consciousness: Recent Research

Edelman, Schore, Van Kolk, Gellhorn, Laborit, Quarto & Renaud, Shapiro

By Jerome Liss, M.D.
j.liss@fastwebnet.it

Why Study the Neurophysiology of Emotions and Consciousness?

In daily life, we do not have to know the structure of our brain in order to know what we think and feel. Humanity has done very well without this knowledge.

And for the clinical psychologist, we have had more than 100 years of progress, without the knowledge of brain mechanisms and structures. It is only in the past 30 years that research regarding “brain mechanisms” has made substantial progress. **Why ask the clinical researcher to bone up on his neurophysiology?** In fact, there is a large gap between the knowledge gleaned from formal research in the field of neurophysiology – research that is usually conducted on animals or that regards human beings suffering from neurological diseases like cardiovascular strokes and tumors – and the experience of the person with emotional difficulties. But despite this large gap, we propose that connecting these two diverse fields – neurophysiology and clinical psychology – can bring fruitful results. How? Through their **correlations**.

The goal of this article is to present a number of such **correlations** between neurophysiological research and clinical experience. Correlations do not create empirical proof, but only generate suggestions based on indirect evidence¹. Nevertheless, as we shall show in the following pages, these suggestions based on research correlation help the clinician focus on certain experiences and behaviours of the patient, and even of himself. **Hypotheses can emerge that will often support clinical ideas already formulated on an intuitive basis.** This is a help for the clinical researcher. With so many clinical models of psychology available, those that are correlated with neurophysiological mechanisms can merit greater attention. In this way the clinical researcher will be encouraged to develop further certain pathways of theory and method while giving less attention to other possibilities.

Even this idea, namely, that certain clinical models can become reinforced and developed further because of a matching with neurophysiological models, can itself find a neurophysiological parallel: Gerald Edelman suggests that neuronal groups present a huge diversity of patterns². **How does the brain “choose” those neuronal groups that are most adaptive? By a mechanism of “selection based on use.”** Edelman points out that this is the same as Charles Darwin’s Theory of Natural Selection applied, however, not to a species, but to particular neuronal groups within the brain. Thus we are proposing that **to use correlations between neurophysiology**

and clinical experience can help determine the “choice” regarding those psychological models that seem to be the most promising for theoretical-methodological progress. In summa, to determine such correlations can help us develop “a natural selection” regarding “the most useful” psychological models.

In this article we will use a series of designs. The purpose is to give a clear picture of the neurophysiological mechanism. Designs also help understand complex phenomena in which there are simultaneous reciprocal interactions³.

Finally, the neurophysiological research material is taken from the works of Alan Schore, Bessel Van Kolk and Gerald Edelman. (See bibliography for references.)

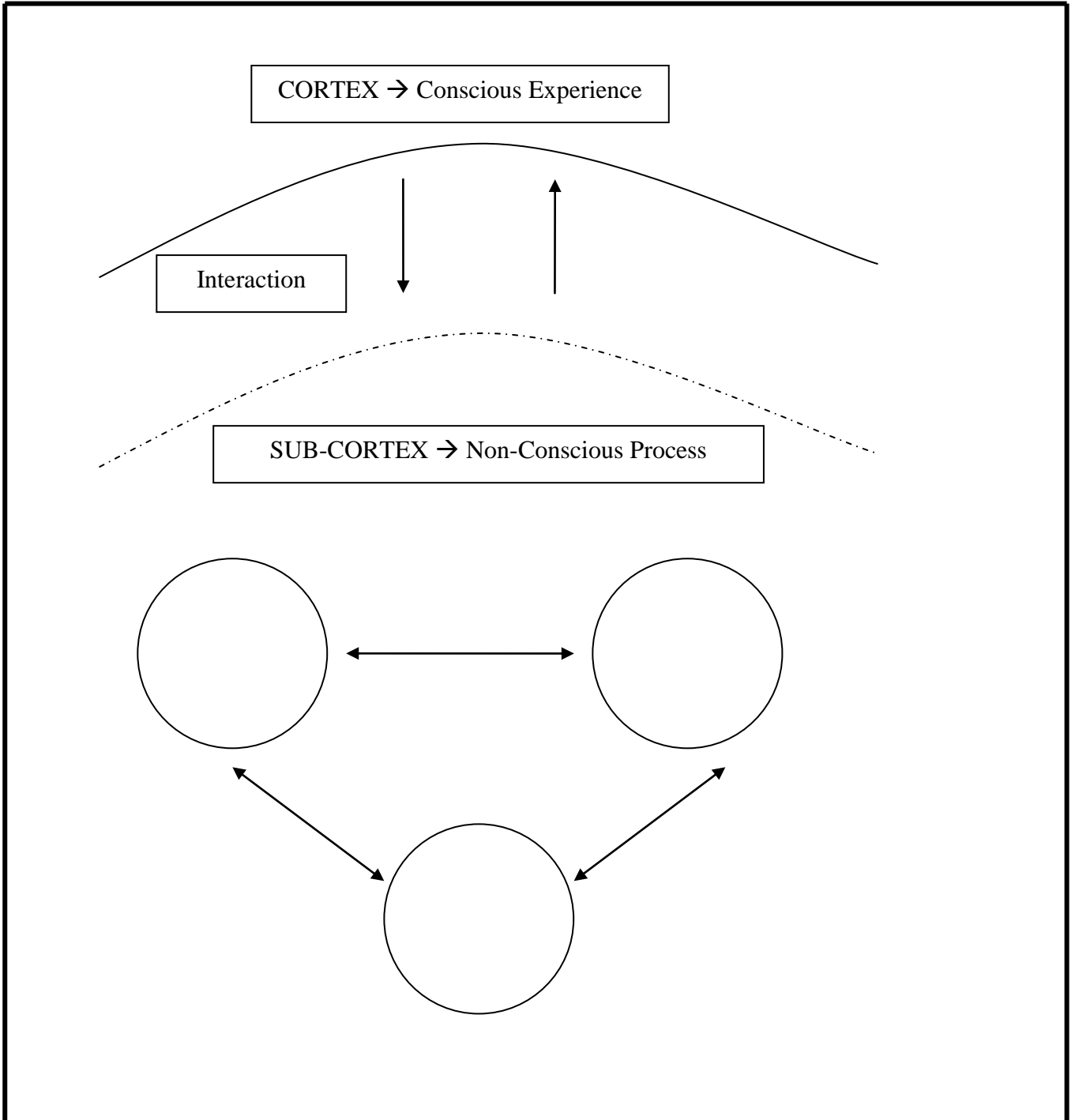
Cortical Conscious Experiences and Sub-cortical Non-Conscious Processes

The brain consists of numerous levels with neuronal centers connected horizontally and vertically.

The highest level, the **cerebral cortex**, contains many areas connected with one another: the visual cortex, the auditory cortex, areas for proprioceptive input, areas for symbolic thought, for memory, for language, for emotions, for planning and for action. **Conscious processes** are correlated with neuronal firing in most of these areas.

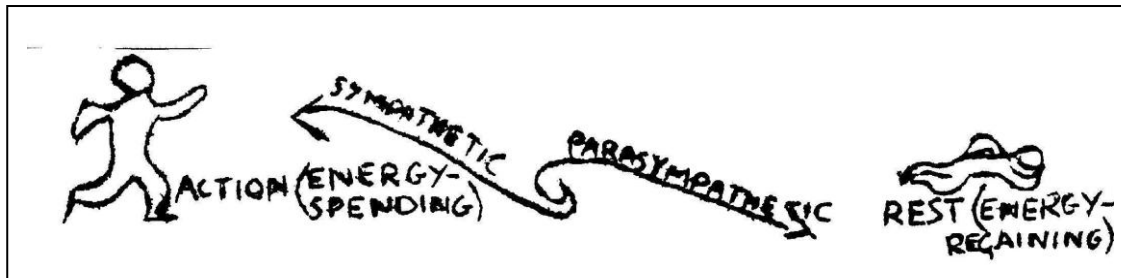
Non-conscious processes are correlated with neuronal firing in lower areas of the brain, which we will call **sub-cortical areas**. There is a constant interaction between conscious cortical processes and non-conscious sub-cortical processes.

The Brain: Cortical Conscious Experiences and Subcortical Non-Conscious Process



Sympathetic and Parasympathetic Connections Between Cortical and Sub-Cortical Areas

The autonomic nervous system controls “autonomic functions” of the body, that is, functions in which there is no direct conscious control. This means a control of blood distribution, heart rate, digestive processes, sweating, etc. **The autonomic nervous system is divided into an “activator,” the sympathetic component which uses up energy, and a “relaxer,” the parasympathetic component that renews energy.**



Sympathetic and Parasympathetic Functions

The sympathetic and parasympathetic help the body, on the one hand, sustain vigorous activity (by means of the **energy-spending** sympathetic activator) and then help the body **to renew the energy** that was used up (by means of the parasympathetic relaxing or “receptive” component). What part of the brain modifies the “active” sympathetic and the “receptive” parasympathetic? According to Ernst Gellhorn⁴, it is **the hypothalamus** that regulates these two functions. (We will later present Alan Schore’s research which enlarges Gellhorn’s map regarding the neuronal pathways that regulate sympathetic and parasympathetic functions.) And how do the sympathetic and parasympathetic functions interact? **Gellhorn proposes that the sympathetic and parasympathetic should work in alternance!** One component **intensifies** (the sympathetic during vigorous activity, thereby using up energy) while the other component, the parasympathetic, **diminishes**. And vice versa: The parasympathetic function increases during rest, in order to renew the energy used up, while the activating sympathetic diminishes.

How can this important mechanism which regulates our total body physiology, even down to the cellular level, become disturbed? This mechanism becomes disturbed when both the sympathetic and parasympathetic **become activated at the very same time**. This is called the pathological “additive” mechanism of sympathetic-parasympathetic firing. **This “additive” mechanism may be compared to putting one’s foot on the accelerator of an automobile and at the same time pressing down the brake with the other foot**. We have “Go!” and “Stop!” at the very same time. And the outcome? The screech of the motor of the automobile is like the body manifesting signs of disturbance due to these contradictory messages.

Another disturbance can come from “dissociation” between the two components: In this case the action of one does not influence the action of the other. The two components are totally “out of synchronisation” with each other.

“Sympathetic” Anger and “Parasympathetic” Vulnerability

An important clinical implication is that **emotions** are also associated with these two energy systems: **Active emotions** like “jumping joy” are associated with the **sympathetic**. Active emotions can also be “disturbing” or “negative,” like rage, anger, frustration and protest. On the other hand, **“receptive” emotions** are associated with the **parasympathetic**: Tenderness, melting joy, soft love. “Receptive” emotions can also be difficult or disturbing: Emotional hurt, sadness, disappointment, shame and loss⁵. These emotions may be painful, but they are useful. They give us important information: “Change your behaviour!” “Change your attitude!” “Change your understanding!”

“Sympathetic” Anger and “Parasympathetic” Vulnerability

SYMPATHETIC

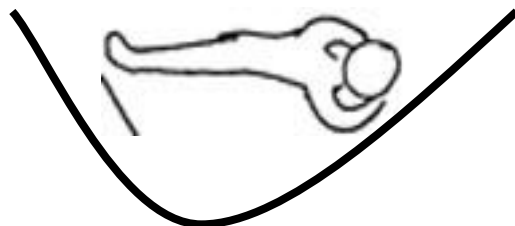


**RAGE
ANGER
PROTEST
FRUSTRATION**



JUMPING JOY

PARASYMPATHETIC



**HURT
SADNESS
SHAKING FEAR
SHAME**



MELTING JOY

From Chronic Emotions (Sympathetic-Parasympathetic “Knot”) to Dynamic Emotions (Sympathetic-Parasympathetic Alternance)

On the other hand, our emotions can become altered and dysfunctional when the sympathetic and parasympathetic have lost their normal alternance; they become *stagnant*. In summary, each emotion can have two versions: A **dynamic** version in which there is sympathetic-parasympathetic alternance, and a **chronic or stagnant** version in which the sympathetic-parasympathetic alternance has been lost. How can we differentiate the dynamic from the stagnant forms of an emotion? In the dynamic form, we see a wave-like “intensification-followed-by-reduction” sequence. That means that the emotion is intimately connected to wave-like sympathetic or parasympathetic processes. In the stagnant or chronic form, in contrast, the emotion is prolonged without this intensification-reduction sequence. There is no crying with sadness. There is no raising of the voice or sudden, vigorous limb movements with the anger. The emotion is “talked about” in a flattened way. Perhaps the person seems calm or perhaps up tight. In either case the talk does not invigorate the emotion or make it dynamic.

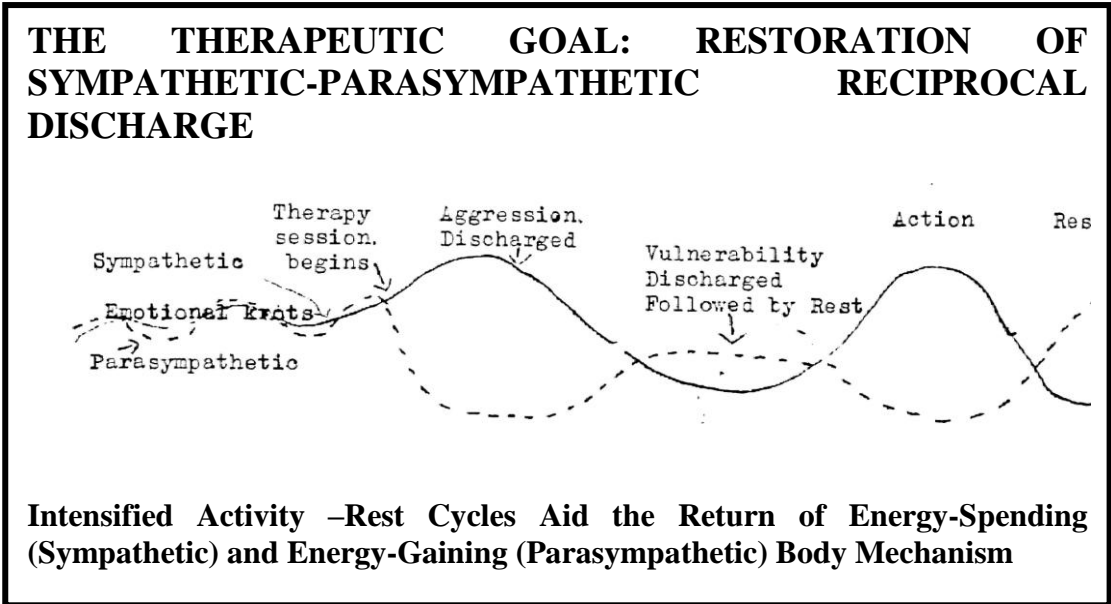
What do we see in the clinical session? On the one hand, we can see “**dynamic anger**,” with a normal rising-and-diminishing of the emotion. In the rising phase, the voice may be louder, a bit trembling, with the sound of the phrases indicating, “This is what I’m telling you!” At the same time a brief moment of lively and rhythmic gestures may accompany the verbal expression. This will usually be followed by “relief,” at least partially, and there will be a sense of tension reduction, with the breathing open, and perhaps even accompanied by a smile. In contrast, when we see “**stagnant anger**” (**resentment, bitterness, hostility**), the rise and fall of the sympathetic dynamic has been lost: The person seems “controlled,” the voice in a monotone and there is a lack of “vitalising” gestures.

Another clinical example: We may have “**dynamic sadness**,” which is stimulated by a situation of loss, with crying or near-to-tears expression: eyes down, words slowed down, voice sounds muffled, perhaps sounds and facial expressions of pain breaking through, the torso curved forward. When we hear “**sobbing**,” we can feel “the dynamic” breaking through. We have an intuitive feeling of the person “**hitting the bottom**” or, more often, **approaching “inner depths.”** With time, the acute hurt-sadness feeling subsides, usually with a sense of relief or, at least, partial relief. The person may remain in a state of “inner reflection and sensitivity,” (parasympathetic dominance), but there is a certain degree of calm as if a hurdle has been overcome. In contrast, we will often see, at the **beginning** of therapeutic work, people showing signs of “**chronic sadness**”: Depression, emptiness, apathy, hopelessness, withdrawal. The connection with the vitalizing and wave-like parasympathetic process has been lost, and the person may feel imprisoned in this psychological strait-jacket for months and years.

From “Emotional Discharge” to “Construction”: Two Steps in the Process of Healing

What happens to the sympathetic and parasympathetic components of the autonomic nervous system when we express anger in a moment of frustration, or when we allow ourselves to cry in a moment of sadness and loss? Here is our hypothesis: To deepen and discharge our emotions can create a vital connection between the emotion (limbic system) and autonomic the nervous system (hypothalamus). **The vitalising emotion can stimulate the sympathetic and parasympathetic components of the autonomic nervous system.** The outcome is to replace an **abnormal interaction** between the sympathetic and the parasympathetic – perhaps one which is **“additive”, or** perhaps one which is **“dissociated”** -- by a **healthy and dynamic interaction that is expressed by “synchronised alternance”** of the two components. We might also call this healthy alternance between the sympathetic and the parasympathetic **“reciprocity” or “complementarity.”**

In other words, we are proposing that to experience the “acute” emotion can transform a chronic emotional state by stimulating the sympathetic and parasympathetic and inducing their return to a normal, healthy alternance. This means that “emotional discharge,” which has sometimes been criticised, as a therapeutic tool, because its effects are not long-lasting, could have a useful role in the psychotherapeutic process. In the Biosystemic School, emotional discharge is permitted and sometimes encouraged because of its “vitalizing” and “relieving” effects. But the emotional work is not concluded at this point.

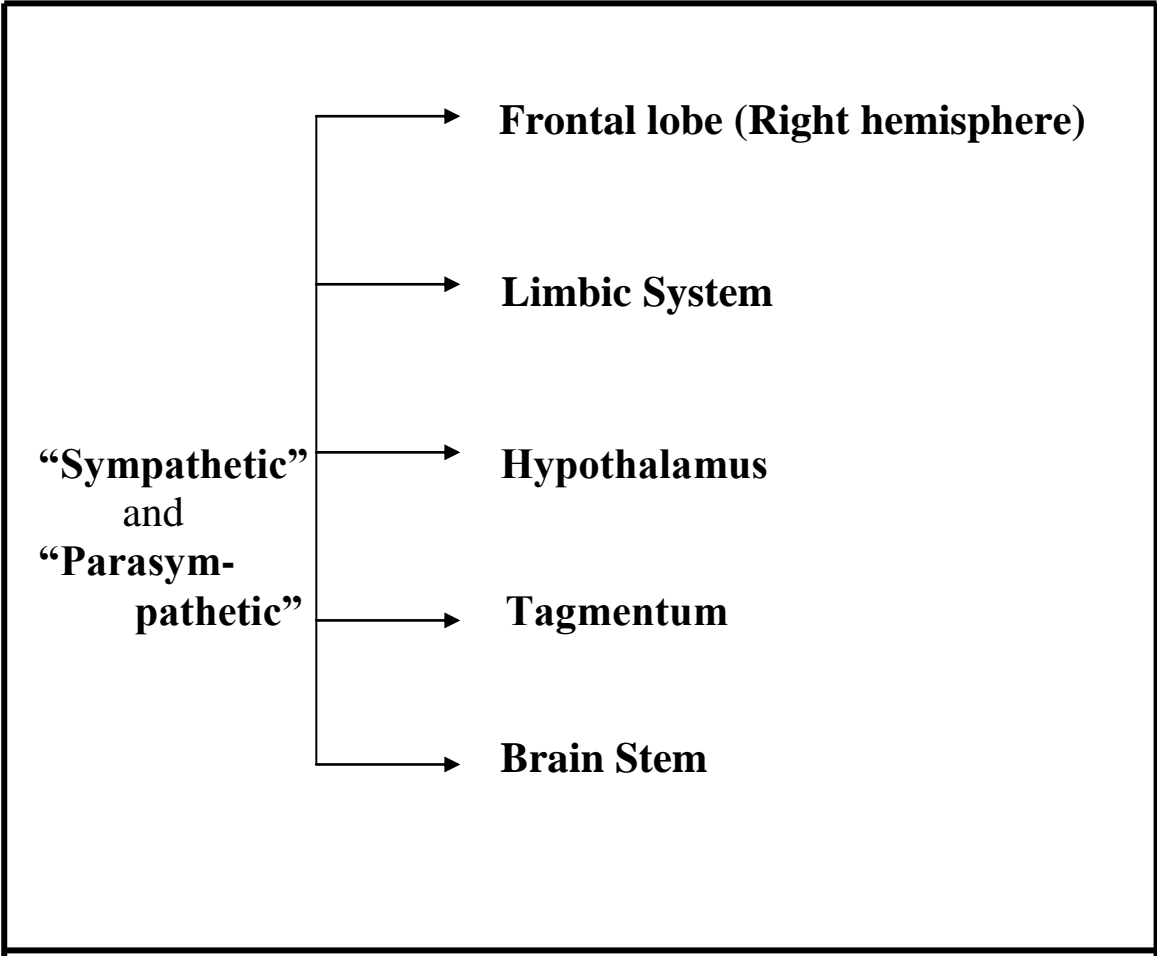


A second therapeutic step – “construction work” – is initiated, so that the benefits of vitalization and relief are canalized into the task of developing thoughts, attitudes and behaviours that help solve the problem⁶. It is our hypothesis that those who have refused “emotional discharge” as a therapeutic step have not profited from the temporary psychophysical benefits of emotional discharge as a prelude for “construction work.

The Interaction Between Conscious and Non-Conscious Processes

Prof. Alan Shore **extends** Gellhorn’s model concerning sympathetic and parasympathetic states⁷. The neural pathways involve additional brain levels running up and down the brain like a double chain: the sympathetic and parasympathetic. Shore’s model looks like this:

Sympathetic-Parasympathetic Pathways Throughout the Whole Brain



Each level can both receive inputs and send out outputs when interacting with the other levels. In addition, each level can also intensify or reduce its output so that it can influence the sympathetic-parasympathetic process. We can say that each center is “semi-autonomous.” That means that there is an **internal dynamic**, intensification or reduction, that is **partially independent** and, at the same time, an **interactive dynamic**, stimulating or inhibiting, that creates a **partial dependence** among levels⁸.

Schore’s extension of the Gellhorn model regarding sympathetic and parasympathetic processes has several clinical implications:

1. The **emotions** that correlate with sympathetic dominance (anger, frustration, protest) or with parasympathetic dominance (sadness, hurt, emotional pain) involve neural pathways that **cover all brain levels**.
2. **Conscious cortical processes – thoughts, memories, perceptions -- can influence an emotional state:** The **frontal lobe** receives messages from diverse parts of the cortex and sends messages **downwards** to the limbic system and below. The messages may be anger-inducing (sympathetic) or vulnerability-inducing (parasympathetic).

At the same time, **non-conscious sub-cortical states that originate in diverse brain levels can send sympathetic or parasympathetic messages “upwards” to the frontal lobe and thereby influence consciousness.** In this way our inner physiological processes can influence our conscious emotional states. In summary, “psychosomatic medicine” emphasizes how our “psyche” influences our “somatic” condition. (Top to bottom) “Somatopsychic medicine” brings to light the reverse situation of our body influencing our psychological state. (Bottom to top) **Systems Theory predicts this interactive causality.** Alan Schore’s research presents the neural pathways that demonstrate how both systems of causality are valid.

Why is this up-down and down-up double dynamic of great importance? It shows that the traditional opposition between “psychological causes” and “organic causes” must be superseded by a mind-body integrated approach.

Clinical Examples:

I. From Cortex to Sub-Cortex: (Top to bottom)

- a. A **conscious perception** – a photo, a phrase, a place – can “stir up” old memories and feelings that were buried for years and almost entirely forgotten. An inner thought – an idea, a flash image, a wish – or a

visceral perception (“the feeling in the guts”) can do the same thing. Thus, the conscious cortical process -- the perception, thought, memory, wish, feeling -- sends down messages to the amygdala-hippocampus complex of the limbic system (centers that integrate unconscious emotions and memories). Messages go yet further down to the levels of the hypothalamus, tegmentum and brain stem. All these lower brain levels then send messages back up to the cortex. These ascending messages then evoke the enlarged cortical pattern of the emotional memory stemming from childhood.

- b. An **emotional arousal** can change our breathing, make our heart beat rapidly or create a weight in the belly. These are the effects of the **downwards** sympathetic-parasympathetic emotional messages that reach the brainstem. Brainstem outputs include control of breathing, heart-rate, circulation and gastro-intestinal activities including peristalsis and secretions for digestion.

- c. Frontal lobe and limbic system-regulated emotions can send messages **downwards** to the hypothalamus and influence its various functions. What functions? According to Ernest Rossi⁹, we can observe at least six hypothalamic functions:
 1. Sympathetic-parasympathetic regulation
 2. Hormonal output (corticosteroids, adrenalin, noradrenalin, thyroxin, sexual hormones)
 3. Motor patterns
 4. Immunological processes
 5. Sexual desire
 6. Appetite

Thus, all of these functions can be disturbed or altered by our emotional states.

Here are several specific patterns: Chronic emotions associated with intense **sympathetic predominance** might produce **hypertension** (excessive noradrenalin), reduced appetite and chronic abdominal tension (blocked peristalsis), agitated and impulsive movements (uncoordinated motor patterns), **vulnerability to infection and tumors** (deranged immunological processes associated, as Laborit points out, with excessive corticosteroid secretion that blocks protein-building and thereby reduces anti-body production), **headaches** (increased muscular tone with arterial constriction), **chronic back pain** (increased muscular tone with reduced flexibility of the articulations), etc.

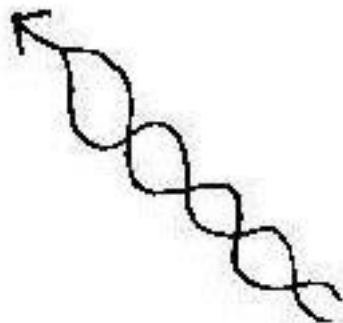
In contrast, here are several results of chronic emotions associated with **parasympathetic predominance**: **Chronic passivity and depression** (lowered

muscle tone, loss of activation for motor programs), **obesity** (increased appetite and loss of motor activity), **apathy** (abnormal decrease of “tonifying” stress hormones, reduced activation of cortical “interest systems”), **social isolation** (loss of “charge” or “drive” regarding social interests and expressive vitality), **lowered self-esteem** (loss of motor competence, repetitive “self-abasing” thoughts without cortical “contextual mediation”), etc.

AUTONOMIC NERVOUS SYSTEM “KNOTS” AND RESULTING SYMPTOMS:

SYMPATHETIC DOMINANT SYMPTOMS

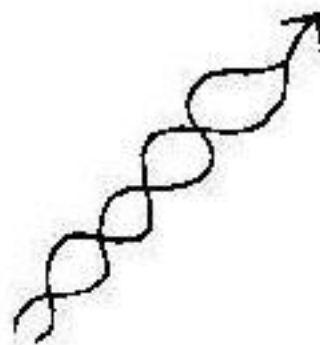
agitated movements
hypertension
vulnerability to infection and tumors
headaches
chronic back pain



SYMPATHETIC

PARASYMPATHETIC DOMINANT SYMPTOMS

chronic passivity and depression
obesity
apathy
social isolation
lowered self-esteem



PARASYMPATHETIC

However, we might well remember, as indicated in a previous section, that it is **not** the **dynamic emotion** that creates these physiological and psychological symptoms but, rather, the **stagnant or chronic emotion that fails to undergo the wave-like “intensification-reduction”, in other words, a loss of sympathetic-parasympathetic reciprocity.** The therapeutic work of re-vitalizing emotional reactions to re-establish this sympathetic-parasympathetic alternance should then **diminish** these pathological processes: If the emotional knot has not yet afflicted the person for too long, that is, if the emotional problem is not yet “somatically structured,” then the therapeutic approach, “to open our emotions,” may act as an effective means of prevention and support the maintenance of good health.

II. From Sub-Cortex to Cortex: (Bottom to top)

“Priming” or Emotionally-Base Sensory Orientation

Schore describes a neuronal pathway in which unconscious emotions (limbic system) send up messages to the frontal lobe. These messages are then sent to the posterior part of the brain involved in sensory input and perception. **The posterior sensory areas are “primed” or “activated” by this emotional input and will tend to favour stimuli pattern that correspond to the emotion.** Thus, “fear of mother’s disapproval” (the emotion) can activate posterior sensory areas in which “mother’s face” or “mother’s tone of voice” becomes especially attended to. The baby is seeking out “confirmation”, that is, a “matching” of the fearful emotion to an (even slightly) facial disapproval or brusque tone of voice that may be perceived in the mother’s expression. Thus, the emotion can lead to a perceptual orientation that favours, by feedback circuits, a reinforcement of the original emotion. This resembles the circularity of “self-fulfilling prophesies.” (Disegno)

Insufficient Activation

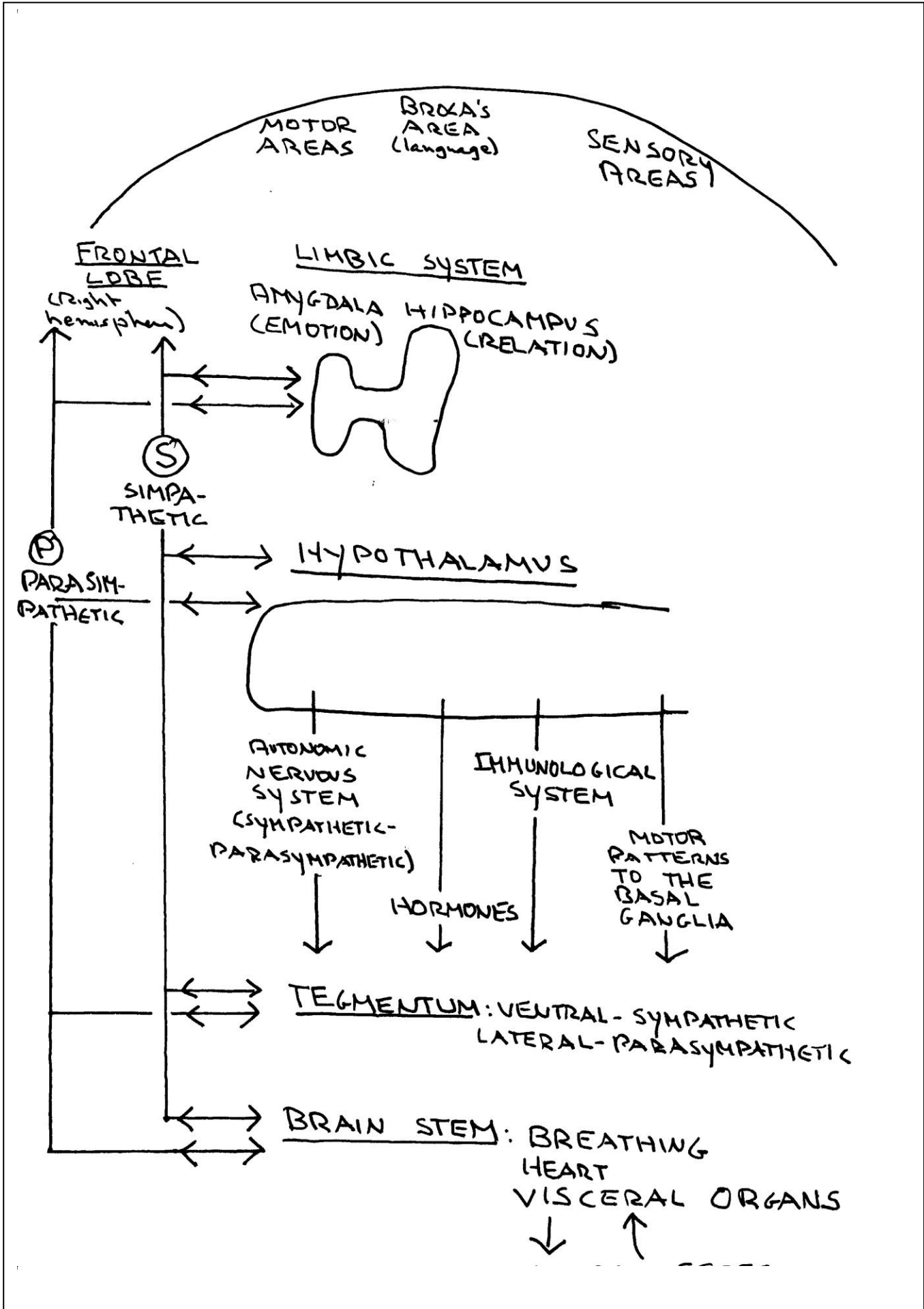
Certain patterns of depression and apathy might be due to bottom-to-top processes. We can imagine how **insufficient activation** of lower brain processes (which might be due, as we will later discuss when presenting Gerald Edelman’s research, to an **inhibition or loss of motor patterns**) can then result in an **insufficiency of activating messages sent upwards.** What clinical phenomena might appear? Slowing down of thought, dispersed and unstructured thought patterns, slowed down and inhibited action patterns, lack of desire or interest, etc. These “internal patterns” might then create an “external pattern” of “low-energy”: voice subdued, eyes looking in the distant, gestures and movements hardly seen. All this can create disinterest or even aggression when the “low energy person” meets others. The person, as a result, feels refused, self-esteem collapses and social isolation is reinforced, **thus creating a**

vicious circle between low vitality and social isolation. This is called “the depressive syndrome.”

Other abnormal situations within the sub-cortex – toxic states, infections, metabolic abnormalities, cardio-vascular obstruction, drug withdrawal states, tumours, prolonged sensory isolation, hallucinogenic and other drug effects, etc. – can also create **upwards** consequences and a great range of disturbances: deep anguish, unpredictable action, mental confusion, hallucinations, paranoia, mental absence, etc.

In summary, Schore’s model of sympathetic and parasympathetic neuronal centers interacting at multiple levels of the brain offers a neuronal map for understanding complex and varied mind-body phenomena.

Sympathetic-Parasympathetic Interactions Throughout the Whole Brain



The Motor System Organizes Our Attention

The research of Gerald Edelman opens up another frontier in this new domain of “maps of the brain.” Edelman’s work highlights **the importance of the motor system for organizing consciousness and attention**¹⁰. The design shows how motor neurons that descend from the cortex in order to create expression and action (cranial nerves for the head, spinal column nerves for the trunk) send “collaterals” (side-routes) to the basal ganglia. From this nuclear complex, messages are sent to the thalamus.

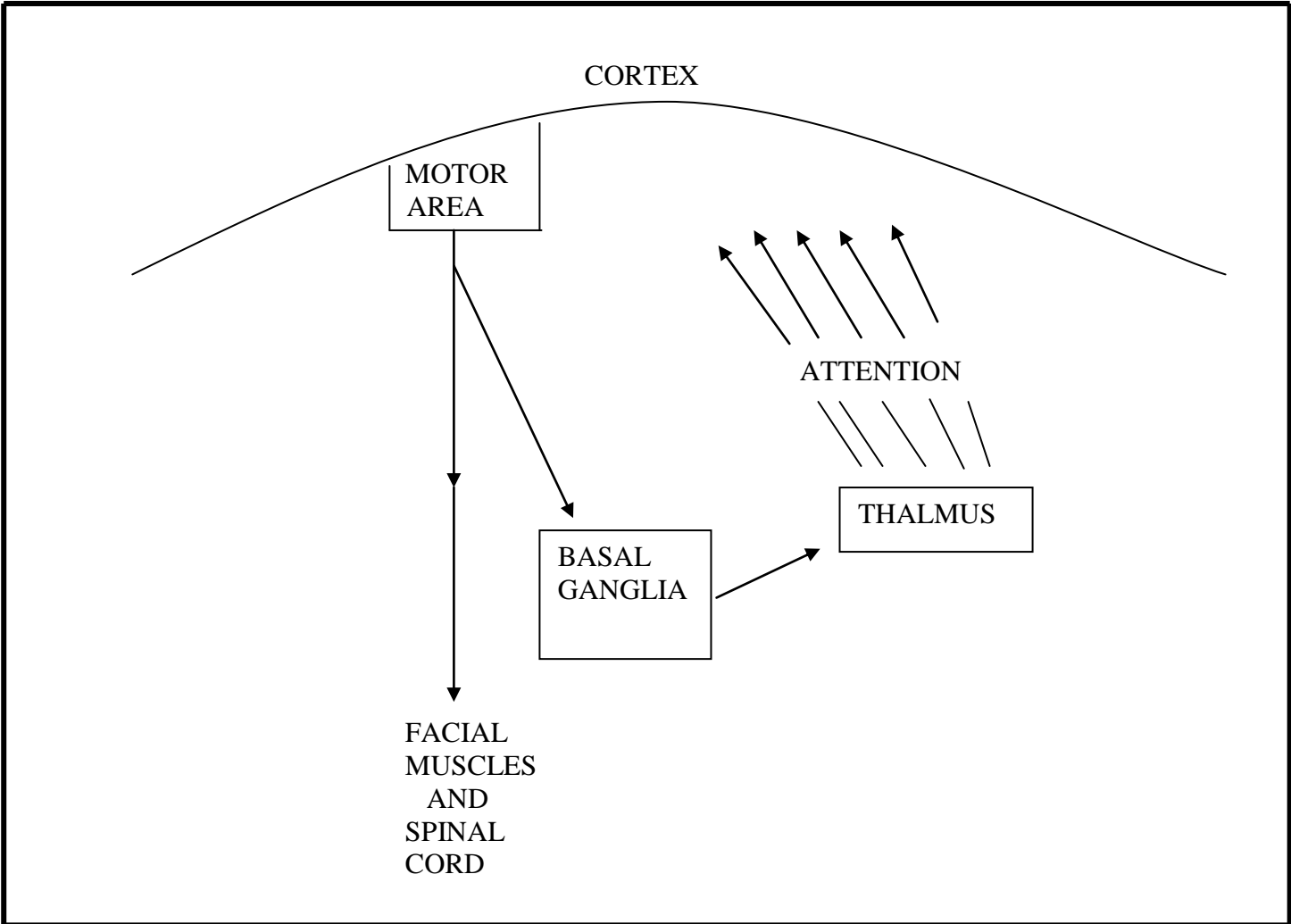
What is the function of the thalamus? For decades, the thalamus had been understood to act as the main “first station” in the brain for sensory input. Sensory messages of diverse types – proprioceptive, tactile, heat, pressure, pain, etc. – climb up through the lower brain by means of the Reticular Ascending System (RAS) until reaching this “first station” thalamus. From there, patterns are sent up to the sensory cortex, where they transform from “sensations” (primary sensory cortex) to “perceptions” (associative sensory cortex). Thus, **the thalamus has this special role of organizing our sensory experience, directing our conscious attention to one dimension or another within the range of sensory input.**

But thalamic output is not limited to cortical **sensory** areas. **All** of the cortex – parietal, temporal, frontal areas, cingulate gyrus, entorhinal cortex, etc. – receive activating and organizing messages from the thalamus.

Thus, while Schore has emphasized that the presence of **anterior** sub-cortical (non-conscious) pathways lead to cortical (conscious) areas, more specifically, from the limbic system and lower brain sympathetic-parasympathetic organisers to the frontal lobe, Edelman’s work highlights a more **posterior** set of pathways: From the RAS (sensory input) to the thalamus and from there a spread of axons to reach different areas of the cortex. What is more, it seems that the thalamus has a greater potential to influence consciousness and attention, given its complexity and the large range of upward climbing axons, than the limbic system – frontal lobe connection.

The important contribution of Gerald Edelman’s research, for psychotherapists, is that **the thalamus receives an important input from the basal ganglia!** To repeat the neuronal sequence: motor neuron – basal ganglia – thalamus – return to cortex. In summary, **the motor system influences attention!** When we take a walk, bring a glass of water to our lips, design a figure, drive our car, etc., we need to direct our attention to the part of the environment that we are acting upon. Otherwise, major errors and even catastrophe! (I’m always amazed how people are so accurate while speeding on the highway. “Hmm,” I think, “our emotional system doesn’t work with the same precision!”)

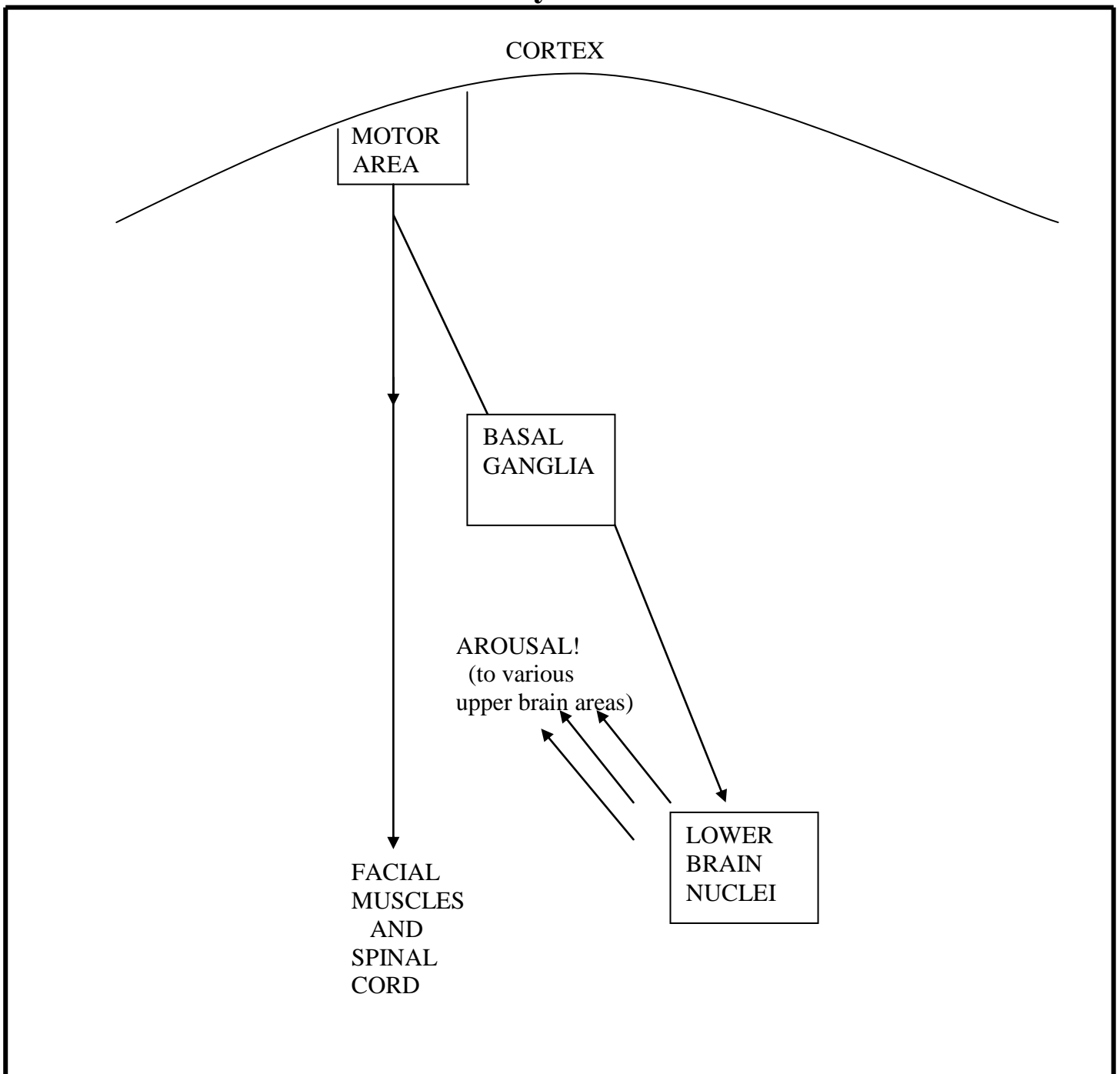
Motor Neuron Pathways to Organize Our Attention



The Motor System “Wakes Up” the Brain

In fact, the motor system – basal ganglia connection not only works through the thalamus, but produces another output that affects the entire brain: From the basal ganglia, we have descending axons that reach the substantia nigra and, from there, that descend still further into the lower brain in order to activate “lower brain nuclei”: locus ceruleus, raphe nucleus, pedunculopontine tegmentum, etc. **These nuclei send up neurotransmitters – cholinergic, catecholnergic, dopaminergic, etc. -- to sub-cortical and cortical regions, thus “waking up” the entire brain¹¹.**

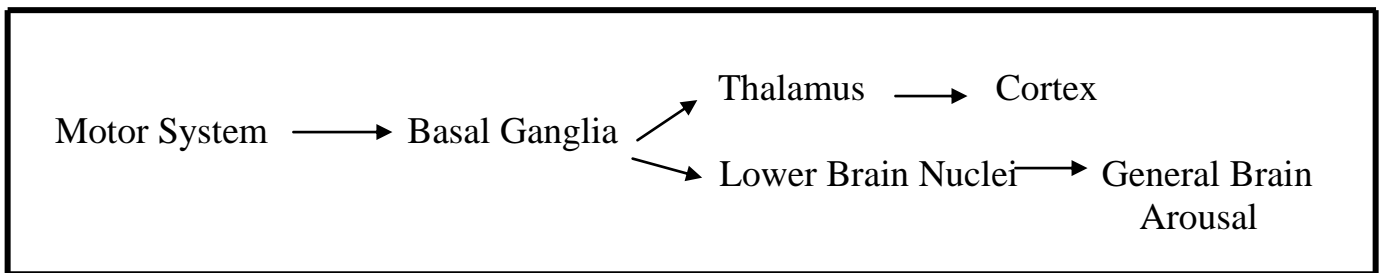
The Activation of the Motor System Creates “Arousal”



So we have a map that can explain still further an important psycho-physiological principle that Henri Laborit declared thirty years ago. (Even the title of his book, **Action Inhibition**, emphasizes this fundamental principle.) **Paralysis in front of stress is the pre-condition for emotional suffering**¹². But while Laborit focused on the internal dynamic of the limbic system, in particular, the amygdala, hippocampus and septum, and their descending connection to the hypothalamus, the more recent research by Gerald Edelman enlarges considerably the neuro-anatomical picture.

Let us repeat the fundamental sequence:

Action Arouses the Brain and Organizes Attention



Emotions Are “Contained” When We Have a Plan of Action

Let us see what happens when we attempt to integrate Schore’s “Sympathetic-Parasympathetic Model” and Edelman’s “From-Action-to-Attention Model.” The direct implication is clear: **When an emotional stimulus becomes connected to a plan of action, the field of attention integrates awareness of the emotion (primarily, a frontal lobe process) with attention-oriented-by-action (primarily, a thalamic process).** The person can feel stressed by the emotional stimulus, but will not feel overwhelmed, intensely distraught or acutely anguished. **The motor system plan acts to contain the emotion, maintaining a part of consciousness on “the job to be done.”**

But what happens when an emotional provocation occurs for which there seems to be “no answer, no solution, no means of effective action”? At that point Henri Laborit’s principle, **“paralysis when confronting a situation of stress,”** comes into play. The person feels intense suffering, anguish, even despair and hopelessness and, if this persists, symptoms of grave depression can appear, or else, somatic symptoms like dermatitis, gastric ulcer, asthma, colitis or high blood pressure can emerge, thereby producing a psychosomatic illness.

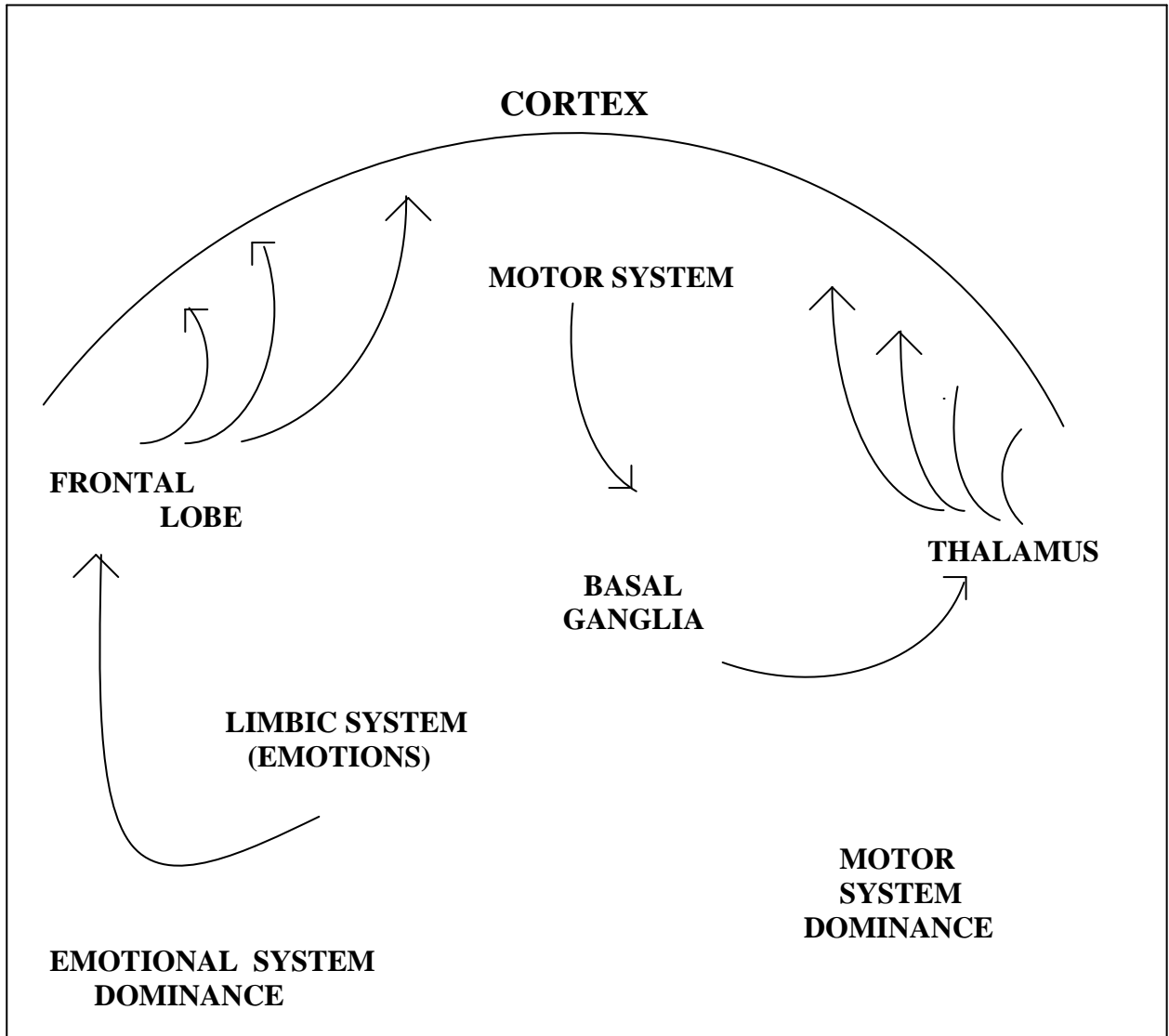
Let us examine the very different outcome between “emotional stress **without** an action plan” and “emotional stress **with** an action plan.” If there is no action plan,

“no way out,” “no way to respond to the problem,” the person feels overwhelmed by the situation: confused, anxious, panicky, “losing the ground,” with “a pressure in the chest” or else “a rock in the stomach.” Our interpretation: **We have an intense parasympathetic reaction that especially involves the gastro-intestinal tract** -- perhaps with the accumulation of blood in the gastro-intestinal system while peristalsis becomes blocked, secretions accumulate and voluntary muscle tone is lost -- all leading to a feeling of “collapse”: *“It’s too much!” “I’m overwhelmed!” “The worst has happened!”*

Schore shows that this type of parasympathetic reaction may be due to intense and/or prolonged emotional stress that takes place in early childhood. This is called “trauma”. Why is this childhood experience so profound? so lasting in its effects? Schore cites research literature that demonstrates that the child’s parasympathetic system is developing during the period of 16 to 22 months¹³. This has a great impact upon the child’s emotional and behavioural growth. If the child undergoes frequent and intense emotional traumas, such as constant refusal or punishment by the mother, or by whomever represents the primary caretaker, there will be seen, in later stages of life, **a special vulnerability to emotional stress** and the tendency toward helplessness and collapse.

By integrating Schore’s psychological and neuro-anatomical analysis with that of Edelman, we can formulate a more complete scenario. **What happens when the traumatic emotional stimulus involving parasympathetic pathways has no basal ganglia-mediated action program? At that point messages from the limbic system to the frontal lobe then spread to the rest of the cortex, without a pattern of thalamic regulation, based on an action plan that can organise attention.**

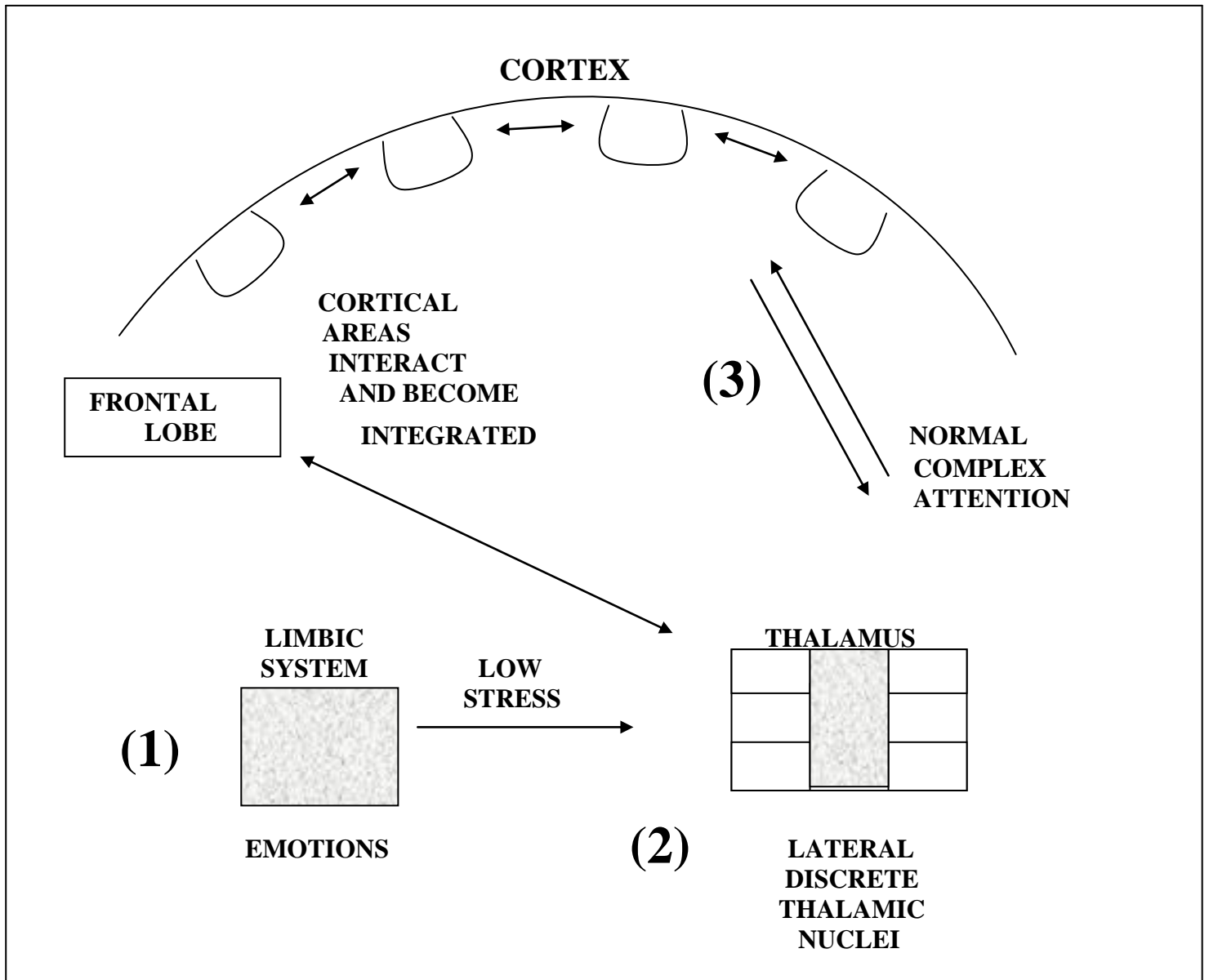
**ATTENTION OVERWHELMED
BY EMOTION OR “CONTAINED” BY ACTION**



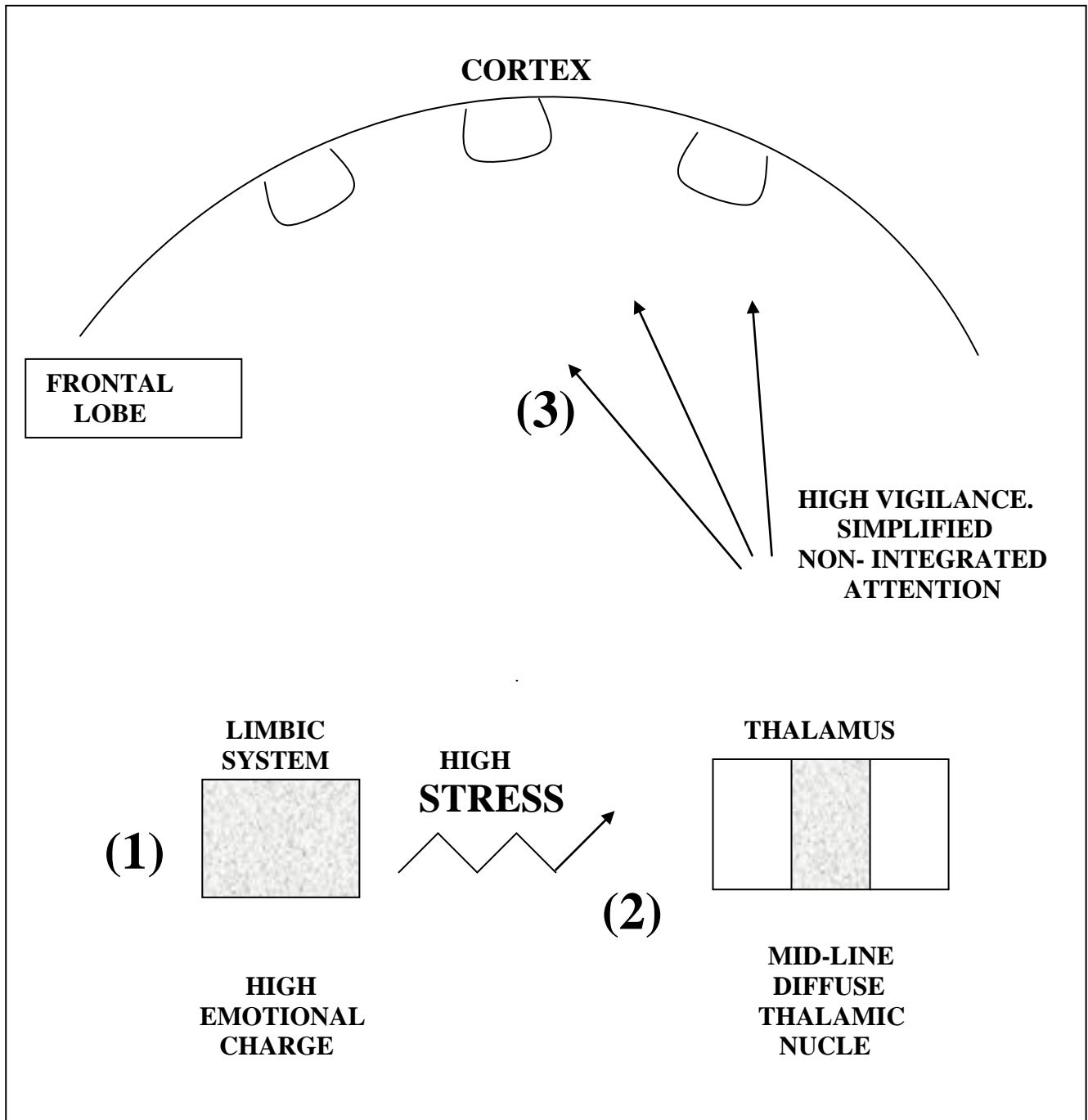
The Emotional Emergency Regulates the Thalamic Attention Pattern

An additional mechanism is also possible for explaining “attention disrupted by intense emotion.” Because the limbic system also has neural connections with the basal ganglia and the thalamus, **it is possible that the “overwhelming emotion” also directly disrupts normal thalamus-to-cortex connections.** This mechanism in which emotions directly disrupt thalamic organisation of attention is suggested by research studies conducted by Quarti and Reynaud¹⁴. Their EEG studies suggest the following: **Thalamic functions are usually dominated by the large, lateral, discrete thalamic nuclei. These nuclei integrate the RAS (Reticular Activating System) sensory input coming from below and send up well-patterned messages to the cortex.** In addition, the functioning of this “precise” thalamic system is associated with feedback from the cortex and an integrated play of diverse cortical areas. Thus, the entire system is fine-tuned and adapted to complexity. **But** when the emotionally based limbic system sends high stress messages of emergency to the thalamus, we see **a shift** in thalamic functions. **Instead of the large lateral discrete nuclei being stimulated, the mid-line “diffuse” nuclei become activated. These nuclei work very differently: They send “diffuse” messages to the cortex. Therefore the cortical organisation of attention also becomes “diffuse.”** Beyond that, when the diffuse mid-line nuclei of the thalamus become activated, they block feedback from the cortex. This stops feedback regulation. And, to make matters worse, these nuclei also block the interaction among diverse cortical areas, thus pushing our thoughts toward primitive simplicity and preventing a complex evaluation of the provocative situation. **In summary, the emotional emergency creates a state of high vigilance, but with loss of complexity and fine-tuning.** We can understand the usefulness of this emergency system for brief, rapid adaptations, but if the emotional emergency becomes prolonged, the result will be a restriction of conscious attention and flexible behaviour.

LOW EMOTIONAL STRESS: COMPLEX ATTENTION



HIGH EMOTIONAL STRESS: SIMPLIFIED ATTENTION



Thus we have proposed **two mechanisms** by which the limbic system based “overwhelming emotion” can block normal, thalamic, action-based attention: First, the lack of an action strategy prevents the basal ganglia to give organizing messages to the thalamus and, in compensation, permits frontal lobe “spread” of emotions. Second, the limbic system emotional stress **directly** disrupts thalamic functioning. Whether one or both mechanisms are in play, the resulting disturbance is clear: **When we are confronted with an intense emotional stress and, at the same time, “We don’t know how to handle it,” that is, when the action-plan control of attention is absent, the cortex can become bombarded by emotional messages without the “containment” or “boundaries” that an action plan normally produces.** Traumatic experience and other high emotional stress states function in this way.

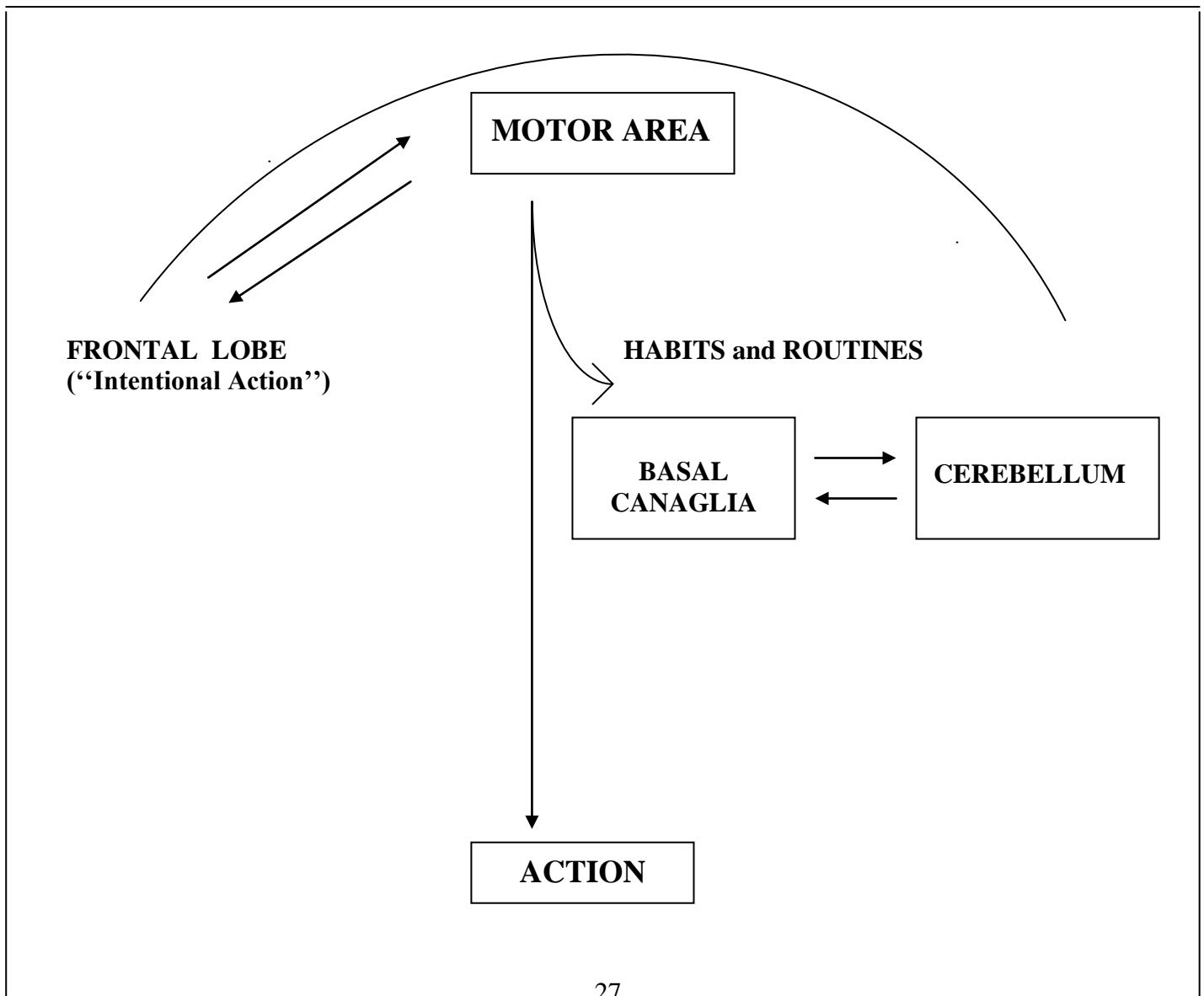
Let us now return to Edelman in order to enrich still further our understanding of **how “action inhibition” alters brain functioning.** We mentioned previously Edelman’s finding that the basal ganglia also send **descending messages** to the lower brain nuclei: substantia nigra, locus ceruleus, raphe nuclei, peduncopontine and tegmentum. These nuclei, normally, activate the entire brain by sending **upwards** various neuro-transmitters: cholinergic, catecholamergic, dopaminergic, etc. However, if a person experiences paralysis when faced with stress,” (Laborit’s “action inhibition” hypothesis), this normal mechanism of brain activation is also repressed. And the result? **The brain capacity for integrating complex interactions and for thinking about the stress situation in sequential terms and with precision becomes significantly diminished.** The person can feel, in this circumstance of “stress without solution,” a state of urgency, but thought becomes paralyzed as well as action: *“I couldn’t think straight!” “My mind went blank!” “I felt frozen with fear.” “I felt lost in the chaos!”*

Intentional Action (Frontal Lobe) Vs. Habitual Action (Basal Ganglia and Cerebellum)

Edelman’s work shows how the motor neurons from the motor cortex create the collaterals to the basal ganglia and thus influence thalamic attention patterns and lower brain nuclei activation processes. **Does this mean that only the action itself, when executed, can create this motor neuron – basal ganglia connection? Or might it be possible that the psychological awareness, “I know what to do, I have a plan of action,” can itself exert an impact on the basal ganglia?**

Research on “new actions” versus “habitual actions” has shown an interesting brain pattern. A “new action” is connected to frontal lobe processes. We know that the frontal lobe regulates both emotional functions and action strategies. What seems to happen is that the new action plan is conceptualized in the frontal lobe and the strategy is then sent posteriorly to motor areas in order that the action be carried out. Nevertheless, **the new action is carried out with hesitation, effort, a certain discomfort and a certain degree of uncertainty.** After the action is carried out a number of times, it becomes more rapid, graceful and precise. In a word, it becomes “a habit.” And what happens in the brain? **The frontal lobe shows less functioning while the basal ganglia shows increased functioning. That means that the passage from a first time intentional action to a twentieth time “habitual action” involves a shift from frontal lobe to basal ganglia functioning.** (An article, “The Cerebellum,” published in Quaderni di Scienza, also suggests that basal ganglia functioning is associated with cerebellar processes. Thus, when we talk about “habits,” we are talking about basal ganglia – cerebellar “routines.”)¹⁵

FROM “INTENTIONAL ACTION” TO “HABIT”



Therefore, when the action that resolves the emotional stress is a habitual action rather than a “first time” action, there is greater involvement of the basal ganglia, and therefore, the mechanism described above of **basal ganglia “containment” of the emotion is more effective**. A clinical consequence: To practice a new action during a therapeutic session, such as “role-playing” several times a new initiative or a new type of communication, can make the patient feel “more sure of himself” when anticipating the circumstances that create stress. (Examples: Practicing a new type of communication with one’s boss, with a colleague, with parents, with the spouse, etc.)

Let us take this one step further: The basal ganglia are not only involved when we actually carry out a habitual action, but even **thinking of the action** can stimulate the basal ganglia – cerebellum “routines.” This idea is supported by Jacobsen’s classical experiment in which “imagined actions” are accompanied by contractions of the muscles involved in those actions¹⁶. That means that the brain mechanisms that accompany an imagined action are similar to the brain mechanisms called into play at the moment that the action is actually carried out.

This means that the imagining of a **habitual action**, as an effective response to a stressful situation, will create more emotional containment than the imagining of a new action plan. The clinical consequence is the almost the same: Practice the new action until it feels habitual. And if actual practice is not possible, **imagine the new action** a number of times until the imagined sequence feels “as easy as pie,” that is, habitual.

Let us summarize the major argument based on an integration of the brain models offered by Schore and Edelman: **When our emotion is associated with an action program, the emotion will be contained. We will feel the stress, but we will not feel overwhelmed.** Our self-esteem will be maintained as well, since we can feel security in our competence. On the other hand, when we are faced with an intensely stressful situation and, at the same time, don’t know in what way to react, our emotions can overwhelm us. We can feel *anguish, helplessness, panic* and so on, and if there is no resolution, we can enter into a state of *chronic anxiety or depression*.

Obsessive-Compulsive Disorder

An interesting hypothesis based on Edelman’s neurological map concerns the mechanism of obsessive-compulsive disorders¹⁷. If we remain within the limits of Schore’s model, we can hypothesize an obsessive-compulsive disorder as due to repetitive processes involving specific neuronal tracks connecting the limbic system to the frontal lobe and, from there, other repetitive tracks connecting the frontal lobe

to posterior areas: 1. to the motor system for compulsive disorders and 2. to the parietal lobe for conceptual disorders.

Edelman's neurological map offers another possibility: That the neuronal tracks for repetitive thoughts involve basal ganglia and cerebellar "routines." The emotional "input" might still come from the frontal lobe (the area of "emotional consciousness") with neuronal tracks then stimulating the motor cortex, and from there descending tracks that reach the basal ganglia. Another "emotional input" could involve **a direct connection** from the sub-cortical limbic system to the basal ganglia, given that the basal ganglia does receive neurons originating in the limbic system.

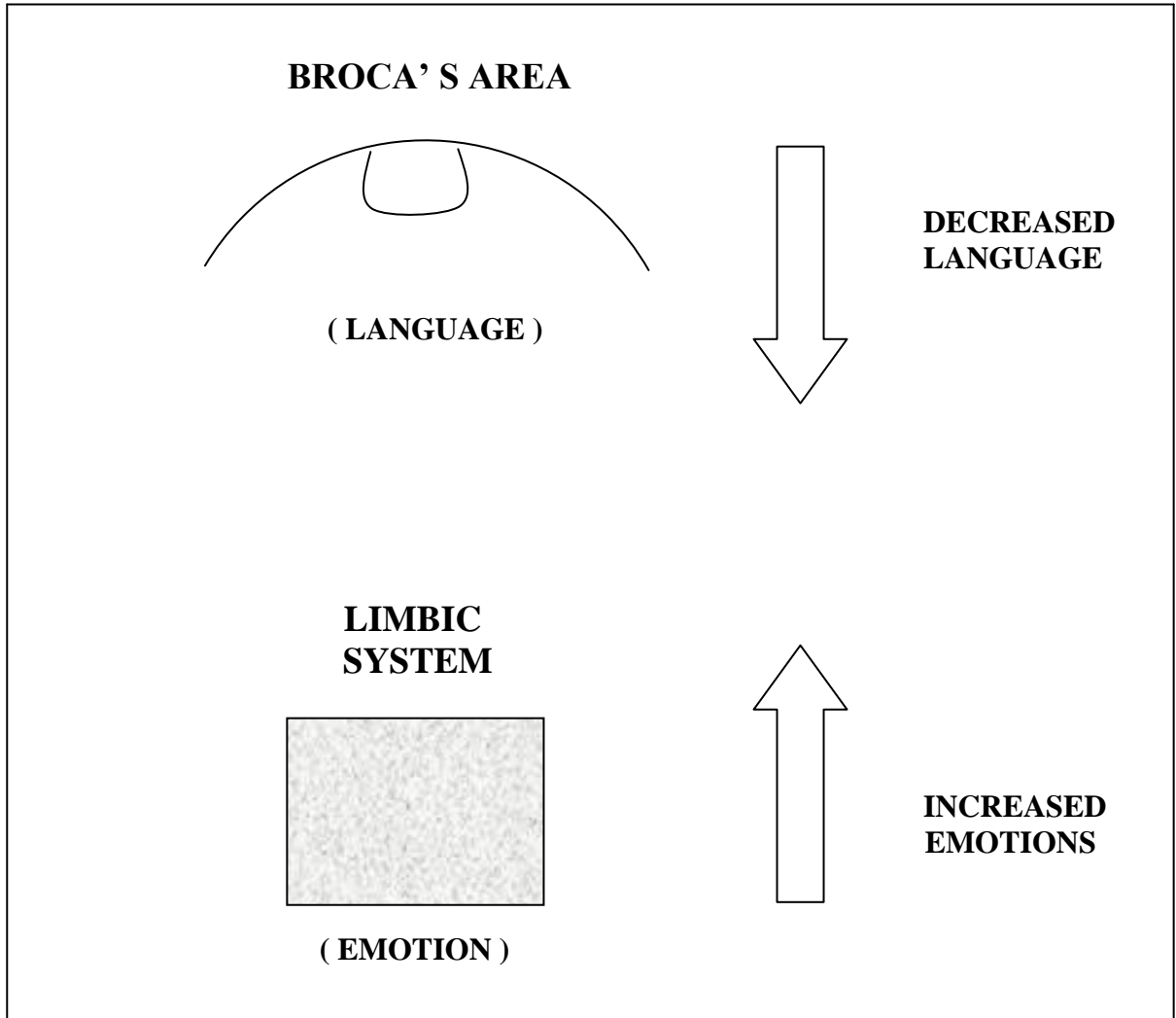
Why is the basal ganglia/cerebellum connection an interesting hypothesis? Because these brain areas show an architecture in which neuronal tracks run **in parallel**, while other brain areas, and especially the cortex, show **a network** form of architecture. All this makes functional sense. **The basal ganglia/cerebellum architecture must support "action routines" that are specific and sequential**¹⁸. A "network" architecture would result in confused actions with overlapping and error-filled strategies. Thus, the specific and repeatable "action routines" organized by the basal ganglia/cerebellum areas can account for the inexorable repetition of compulsive actions and obsessive thoughts. The idea that the obsessive-compulsive disorder may be based on sub-cortical routines rather than cortical repetitions can help us understand the inaccessibility of this problem to rational (cortical) change.

Traumatic Experience and the Loss of Language

A specific research finding cited by Bessel Van Kolk offers an interesting perspective regarding the problem of emotional trauma. Van Kolk discovered that **when a patient recalls a traumatic episode, the limbic system (especially the amygdala, which is noted for maintaining "indelible memory traces") and the frontal lobe show increased functioning.** (This is shown by increased blood flow to these regions.) **At the same time, the region of language functions, that is, Broca's area, shows diminished blood flow**¹⁹. Interpretation: The occurrence of traumatic experience as well as subsequent memory of the trauma take place with a diminished access to language. **Thus the traumatic experience is "overwhelming," not only because of its intensity, but because language functions, which can delineate and contain emotions, are momentarily "out of order."**

The therapeutic implication is clear-cut: The psychotherapeutic process of "putting the emotional experience into words" is a method to re-awaken the repressed language functions and help contain the overwhelming trauma with verbal language: "If I put it into words, I can get a handle on it."

TRAUMATIC EXPERIENCE



Rhythmic Movements to Shunt the *Parasympathetic* to the *Sympathetic*

Francine Shapiro proposes another method for “containing” traumatic emotions. In her book, **Eye Movement Desensitisation and Reprocessing (E.M.D.R.)**²⁰, Dr. Shapiro presents a specific method for overcoming traumatic “flashbacks” and other sequelae of the “post-traumatic reaction syndrome.” Dr. Shapiro asks her patients, first, to permit a re-living of the traumatic episode at a tolerable level. At the moment that this painful experience is “accessed,” Dr. Shapiro asks her patients to make a series of “up and down movements” with the eyes. These movements are regular in terms of rhythm and intensity. **Dr. Shapiro proposes that these eye movements “disconnect” the image of the trauma from the painful and overwhelming emotional discharge.**

In the second part of the book, Dr. Shapiro suggests that **other types of body gestures and movements might also have similar beneficial effects.** In the Biosystemic school, we have found that gestural movements that correspond to the patient’s verbal output – especially if these movements are **regular in rhythm and intensity** – seem to “contain” the overwhelming traumatic experience and, therefore, create beneficial effects²¹.

Can our neurophysiological map explain this therapeutic hypothesis? If we return to Schore’s hypothesis, we can understand that the traumatic experience involves a strong parasympathetic reaction²². The person feels “lost” in the visceral emotion of fright, panic and bombarding images. What does the rhythmic movement accomplish? First, a basal ganglia action pattern is developed that influences the thalamic organisation of attention. We have already suggested how this can offset the limbic system /frontal lobe “unbounded” emotional process. Second, muscular action and sympathetic tone go hand in hand. (Gellhorn had proposed this functional interplay between the autonomic nervous system and the voluntary muscle system.) Therefore, the regular rhythm and intensity of the expressive gestures can “shunt” the autonomic process from the parasympathetic to the sympathetic. **The “unbounded” parasympathetic process becomes “contained,” at least partially, by the structuring sympathetic process.**

Dr. Shapiro makes an important point that integrates neurophysiological dynamics with clinical principles: To achieve a therapeutic effect, the neuronal pattern of the trauma must be **changed!** The **helplessness** of the traumatic experience (a limbic system / frontal lobe neuronal pattern) must become **connected** to the **action plan** (a basal ganglia / cerebellum “routine”). To create this **connection**, the traumatic experience must be accessed and, **at the very same time**, the “sympathetic/muscular” action plan must be brought into play. The therapeutic art involves helping the patient live out these two very dimensions – hopelessness and helplessness / action

and competence – at the very same moment. If the two events are experienced in two different and separated moments, the connection will not be made.

This therapeutic strategy was also suggested by creators of Neurolinguistic Programming (NLP)²³. In addition, in the article, “The Key Word,” (see J.Liss, 1996) the same neuronal model and therapeutic strategy was presented, namely, to access the emotional disturbance and an effective action plan in close temporal order²⁴.

Albert Pessó’s Approach: “The Corrective Emotional Experience”

But is an action strategy the only way to change a traumatic imprint? An alternative approach can be seen in the clinical work of Albert Pessó (who works in association with Prof. Bessel Van Kolk in Boston, Massachusetts). Dr. Pessó helps the person re-live the traumatic episode at a tolerable dosage. (We also see this therapeutic principle of re-living traumatic episodes in “tolerable dosage” in films presented by Prof. Van Kolk. The therapist is Dr. Pat Ogden.) **Dr. Pessó’s original contribution is to ask the person to create “an ideal supportive figure”. This may be an “ideal mother” or “ideal father”²⁵.** This ideal figure, role-played by a group member, will then enter the scene while the protagonist is re-living the traumatic experience. Dr. Pessó proposes, in very clear terms, “Let this symbolic figure, this ideal parent, enter into your world at the very moment that you are re-living the trauma. Our goal is **to change the memory!**”

Franz Alexander proposed a similar therapeutic strategy in his classic work, **Psychosomatic Medicine**. He called this approach “the emotional corrective experience”²⁶.

In summary, this therapeutic method is aimed at helping people who are devastated by traumatic memories is, painful memories that keep returning in the present, **to re-connect the neuronal patterns of these memories to positive experiences**. Thus, an effective therapeutic approach can interrupt the pathological and debilitating effects of traumatic experiences, even those originating in childhood. As one patient said, “I have a new lease on life!”

Summary

This article presents diverse neurophysiological models that come from research studies conducted over the past three decades. To repeat our epistemological premise: Such models are not regarded as “true,” since they will be modified yet further in the coming years. More important, the “clinical applications” are neither “right” nor “wrong.” The clinical methods begin as therapeutic “intuitions.” The research studies that “correspond” to these intuitions are cited, encouraging the

clinician to explore yet further his methodological intuition. **Thus we have a positive interplay between therapeutic method and neurophysiological research.**

Another argument, **not** developed in this article, is as follows: Is it possible for the **scientific researcher**, when becoming acquainted with the clinical literature, to find new hypotheses based on clinical experience that can guide and enrich research studies for the future? In that way we can develop “positive reciprocity” between the scientist and the clinician.

¹ Liss, Jerome, *"The Philosophy of Science and the Clinical Researcher: A Proposal for a New Scientific Psychology,"* published in Italian: "Filosofia della Scienza e la Ricerca Clinica: Una Proposta Per una Psicologia Scientifica Nuova," in **Psicologia Clinica** (ed. Prof. Mario Bertini, Università di Roma "La Sapienza"), Vol. 2, No. 2, May-August, 1983, pp. 143-163, and in **La Psicoterapia del Corpo**, (by Liss, Jerome and Boadella, David), Rome, Ed. Astrolabio, 1986, Chapter XIV.

² Edelman, Gerald M., **The Remembered Present**, A Biological Theory of Consciousness, BasicBooks, 1989.

³ Hyerle, David, **Visual Tools for Costructing Knowledge**, Alexandria, 1996.

⁴ Gellhorn, Ernst, **Principles of Autonomic-Somatic Integration: Physiological Basis and Psychological and Clinical Implication**, Minneapolis, University of Minnesota Free Press, 1967.

⁵ Liss, J., **Il sistema nervoso autonomo nelle turbe emotive e in condizioni di emotività normale**, in Boadella D. e Liss J., **La psicoterapia del corpo**, Roma, Astrolabio, 1986, pp.80-91.

⁶ Liss, Jerome, "From Problem to Solution: Guiding Emotional Work with Deepening Followed by Construction," in **Energy and Character**, (editor: David Boadella) Vol. 29, No. 11, June, 1998, pp. 40-46.

⁷ Schore, Allan N., **Affect Regulation and the Origin of the Self** (The Neurobiology of Emotional Development), Lawrence Erlbaum Associates, Publishers, 1994.

⁸ Liss, Jerome, "**The Systems Model Applied to Bioenergetic Therapy, Psychology and Psychosomatic Medicine**," *Energy and Character*, Vol. 13, No. 2, August, 1982, pp. 12-28, and Vol. 14, No. 1, April, 1983, pp. 18-36; Liss, Jerome,

"**Psychothérapie et Psychanalyse: La Complexità en Question**," Le Journal des Psychologues, No.19, Juillet-Aout, 1994, pp. 45-48.

⁹ Rossi, Ernest, **La Psicobiologia della Guarigione Psicofisica**, Rome, Ed. Astrolabio, 1987.

¹⁰ Edelman, op. cit. pp. 202-204

¹¹ Edelman, Gerald M., and Tononi, Giulio, **A Universe of Consciousness**, (traduzione italiana, **Un Universo della Coscienza**, ed. Einaudi, 2000), New York, Basic Books, 2000.

¹² Laborit, Henri, **L'Inibizione dell'Azione**, (traduzione di A. Meluzzi) Milano, Il Saggiatore, 1986.

¹³ Schore, op. cit., pp.16-22.

¹⁴ Quarti, C. and Renaud, J., **Neurophysiologie de la Douleur**, Paris, Hermann, 1972.

¹⁵ Joaquin M. Fuster, **La Localizzazione della memoria**, Le scienze edizione italiana di Scientific American n°14 inverno 2002 pp 12/20

¹⁶ Jacobsen, E., **Biology of Emotions**, Springfield, Charles C. Thomas Pub., 1967.

¹⁷ Edelman, op. cit. p. 217

¹⁸ Fuster, op. cit. p. 18

¹⁹ Van der Kolk, Bessel A., **Traumatic Stress**, The Guilford Press, 1996.

²⁰ Shapiro, Francine, **Eye Movement Destination and Reprocessing**, New York, The Guilford Press, 1995.

²¹ Shapiro, op. cit., p.314.

²² Bandler, R. e Grinder, J., **Frogs Into Princes, Neurolinguistic Programming**, Moab, Utah, Real People Press, 1979.

²³ Bandler, R. e Grinder, J., op. cit.

²⁴ Liss, Jerome, "Key Words for Unlocking Our Unconscious," in **Energy and Character**, (editor: David Boadella) Vol. 29, No. 2, Dec., 1998, pp. 79-93.

²⁵ Pessó, Albert, "*The Effects of Pre- and Peri-Natal Trauma*," **Energy and Character**, Vol. 22, No. 1, April, 1991.

²⁶ Alexander, Franz, **Psychosomatic Medicine**, New York, W.W. Norton Pub., 1950)

Jerome Liss, M.D.

P.zza S.M. Liberatrice, 18

00153 Rome

Italy

Tel. 00.39.06.5744.903

e-mail: j.liss@fastwebnet.it