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Psychotherapy and cognitive and neuronal plasticity. The social brain and post-traumatic stress disorder. Notes on *The interpreter* by M. Gazzaniga

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ABSTRACT. – Starting from a reflection of *The interpreter* by Gazzaniga, the mechanism of cognitive and neuronal plasticity underlying the effectiveness of rehabilitation therapies of the 'social brain' is addressed and described, particularly from a post-traumatic stress disorder point of view. Descriptive research seeks to highlight how drug therapy can be risky in terms of masking and concealing the symptom. This is an open reflection, which, in turn, wants to open some reflections on psychotherapy in light of recent discoveries in the neuroscientific field. The dynamic interaction between the amygdala on the one hand, and the more informed reactions of the prefrontal cortex on the other offers a neuroanatomical model to explain how psychotherapy reshapes deep-rooted emotional models that are now devoid of adaptive value.

Key words: psychotherapy, neuronal plasticity, social brain, post-traumatic stress disorder, psychology.

Michael Gazzaniga's publications, and in general his speeches, are enlightening and, at the same time, rigorous on a medical-scientific level. For this very reason, I consider it useful to discuss a passage from a popular book published in Italy entitled *L'interprete* (*The interpreter*), from which I quote the following passage:

> "It is easy to understand why my approach rejects therapeutic modalities such as psychoanalysis or dynamic psychiatry, as they are confronted with posthumous rationalizations of the cause of the disorder, rationalizations that are artificially

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> created by the left brain hemisphere. On the contrary, I believe that the use of medications that can detect and treat symptoms, modify emotion and disturbed mood, intervene directly on the right hemisphere before the interpreter even starts working on it, dig back into childhood or adult relationships, and end up sinking into a sort of black hole is of greater value and gives the best results." (Gazzaniga, 2007, pp. 34-35)

Firstly, I would like to make it clear that I, too, agree that pharmacological intervention has significant value, especially in cases where the symptomatic component is a source of pain and discomfort for the individual.

That is, in all cases where alleviating the symptom does not render the pathological source (and therefore the source of the symptom) inaccessible at the same time and keeps the patient, his/her self, and his/her person accessible, and especially when he/she does expect to end the pathological situation by simply eliminating the symptoms, which could find other far more dramatic ways of expression.

Nor do I agree with self-help approaches (except to the extent that they may be *good advice from a friendly neighbour*) nor with broadly understood new-age therapeutic approaches.

It is precisely by agreeing with the Gazzaniga interpreter proposal, and with the functional dialog between the right and left hemispheres, that I consider the approach – which is both psychological and dynamic psychiatric – to be useful and relevant in a context of widespread plasticity, both neuronal and systemic, that has now been established.

I would like to go into more detail about what I mean, starting from the idea of the social brain model.

In this model, the social brain consists of a set of wide-ranging neural networks that cooperate during our contacts with others.[1] The spread of such a network is further reinforced by the discovery of mirror neurons, which are not only located in Broca's area. The social brain operates at the level of systems in which neural networks are coordinated toward a unified purpose.[2]

One of the first proposals for this network identifies certain structures of the prefrontal area, in particular in the orbitofrontal and anterior cingulate neocortex in connection with areas in the subcortex, involving mainly the amygdala.

More recent studies show that this is a good foundation on which further details need to be added.

Considering the vastness of the social brain circuits, the characteristics of

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[1] Leslie Brothers. The Social Brain: A Project for Integrating Primate Behavior and Neurophysiology in a New Domain. 1990 in Concepts in Neuroscience – 2002 in Foundations in Social Neuroscience, chap. 26.

[2] Preston, S. D., & de Waal, F. B. M. (2002). Empathy: Its ultimate and proximate bases. *Behavioral and Brain Sciences, 25*, 1–20.

the neural networks involved depend largely on the type of interaction we are engaged in.

Mirror neurons in the prefrontal cortex and parietal areas handle the shared representations, the mental images that arise in the mind when we talk to someone about a topic familiar to both of us.[3] Other mirror neurons involved in movement activate when we simply observe the actions of other people.[4]

The cells of the right parietal lobe encode kinaesthetic and sensory signals working as we organize movements in response to the person with whom we are conversing.

When it comes to interpreting and responding to emotional messages in the tone of another's voice, the circuit connecting the insula and the premotor cortex, with the limbic system, and above all, the amygdala again, is put into operation. As the conversation progresses, direct connections from the amygdala to the brain stem control autonomous responses by increasing the heartbeat (as well as other emotional responses mostly handled by the sympathetic system) in a conscious, or not fully conscious, response to the social situation.

Neurons in the fusiform area of the temporal lobe are dedicated both to the recognition and interpretation of emotions on faces and to controlling where the interlocutor's gaze is directed. Somatosensory areas come into play when we perceive another person's state of mind, as well as when we notice and consider our own reaction. As we send out the emotional messages of response, brain stem projections and facial nerves shape the appropriate expression. As we tune in with the other, the brain experiences two types of empathy: a rapid flow of the 'lower route' through the connections between the sensory cortex, the thalamus, and the amygdala, which causes the immediate reaction, and a slower flow of the so-called 'higher route', which runs from the thalamus to the neocortex and then to the amygdala, resulting in a slower, mediated reaction.

The connection of the limbic circuit to the orbitofrontal cortex and to the anterior cingulate cortex comes into play here.[5]

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[3] Iacoboni, M., & Lenzi, G. (2002). Mirror neurons, the insula, and empathy. *Behavioral and Brain Sciences, 25*, 39-40.

[4] Iacoboni, M. (2003). Understanding intentions through imitation. *Taking action: Cognitive neuroscience perspective on intentional acts*. 107-138.

[5] See the following:

- Kasai, K., Yamasue, H., Gilbertson, M. W., Shenton, M. E., Rauch, S. L., & Pitman, R. K. (2008). Evidence for acquired pregenual anterior cingulate gray matter loss from a twin study of combat-related posttraumatic stress disorder. *Biological Psychiatry*, *63*(6), 550–556.
- Shin, L. M., Wright, C. I., Cannistraro, P. A., Wedig, M. M., McMullin, K., Martis, B., Macklin, M. L., Lasko, N. B., Cavanagh, S. R., Krangel, T. S., Orr, S. P., Pitman, R. K., Whalen, P. J., & Rauch, S. L. (2005). A functional magnetic resonance imaging study of amygdala and medial prefrontal cortex responses to overtly presented fearful faces in posttraumatic stress disorder. *Archives of General Psychiatry*, *62*(3), 273–281.

These areas are active in perceiving other's emotions and regulating the emotional reaction. The prefrontal cortex, in general, has the task of modulating emotions in appropriate and effective ways. Furthermore, behind all this activity, the cerebellum keeps its focus on one goal, allowing us to control the other person and gather the subtle signals of rapid and uncontrollable facial expressions and micro expressions.[6]

Non-verbal and unconscious synchrony offers us a continuous flow of social instructions. This, in turn, depends on the ancient structures of the brain stem, particularly the cerebellum and basal ganglia.

In this general context, their role in less demanding interactions assigns these areas of the so-called 'inferior brain' an auxiliary role in the circuits of the social brain.

All these areas come together in the structuring of social interactions (even imaginary ones): if any of these are damaged, our ability to tune in is weakened. The more complicated the interaction, the more complex the networks of interconnected neurons that are activated.[7]

Social neuroscience describes how multiple paths of knowledge and action work in interactions with others. Moreover, as anthropological studies have now demonstrated,[8] these multiple paths of knowledge and action in interactions are highly adaptive aspects of the social and human repertoire

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- Shin, L. M., Orr, S. P., Carson, M. A., Rauch, S. L., Macklin, M. L., Lasko, N. B., Peters, P. M., Metzger, L. J., Dougherty, D. D., Cannistraro, P. A., Alpert, N. M., Fischman, A. J., & Pitman, R. K. (2004). Regional cerebral blood flow in the amygdala and medial prefrontal cortex during traumatic imagery in male and female Vietnam veterans with PTSD. *Archives of General Psychiatry*, *61*(2), 168–176.

6 The first scientific publication in the field of psychoanalysis was by Haggard, E. A., & Isaacs, K. S. (1966). Micro-momentary facial expressions as indicators of ego mechanisms in psychotherapy. In L. A. Gottschalk & A. H. Auerbach (EDS.), Methods of Research in Psychotherapy (pp. 154-165). New York: Appleton-Century-Crofts studied during analysis of non-verbal behaviour in psychotherapy. The study was further elaborated by Paul Ekman, who theorized its universality based on his studies of Charles Darwin in Darwin, C. 1998. The Expression of the Emotions in Man and Animals, 3rd ed. Introduction, afterwords, and commentaries by Paul Ekman. Harper Collins. London (US ed.: Oxford University Press, New York City).

7 On interconnected and independent circuits, see Blair, J. R., & Perschardt, K. S. (2002). Empathy: A unitary circuit or a set of dissociable neuro-cognitive systems? *Behavioral and brain Sciences*, *25*(1), 27–28.

8 Premack, D., & Woodruff, G. (1978). Does the chimpanzee have a theory of mind? *Behavioral and Brain Sciences, 1*(4), 515-526. From this literally revolutionary study emerged what is still discussed today as Theory of Mind. On the related brain mechanisms, see the studies of Rebecca Saxe at MIT and those of the team of Rizzolatti Gallese and Fadiga in Parma. For literature on the wider cognitive, systemic, and psychological implications, see:

- Dennett, Daniel C. (1983). Intentional systems in cognitive ethology: The 'panglossian paradigm' defended. Behavioral and brain Sciences 6(3), 343–390.

and have a survival function, as well as non-cognitive skills such as primary empathy, synchrony and solicitude.

The old concept of purely cognitive social intelligence assumes that social intelligence can differ from general intelligence. But focusing solely on the mental capabilities of social intelligence does not allow us to grasp the irreplaceable role that the 'lower route' has.

It is undeniable that the ways of functioning of the paths of knowledge and action in interactions with others include skills of the higher route as well as of social cognition. But social intelligence has called into question functions of the lower route, such as synchrony, tuning in, social intuition, and empathic solicitude, and probably the impulse for compassion.

In this context, what it demonstrates is that these are intuitive non-verbal capabilities that manifest themselves in a matter of microseconds, much faster than the mind takes to formulate a thought.[9]

The prerogatives of the lower route constitute the very basis of a good social life as they are non-verbal qualities: they exclude what can be observed in a written text, for example, those used and administered for the quantitative assessment of IQ.

One example we can examine in concrete terms is post-traumatic stress disorder.

As Spencer Eth[10] summarized, "At the heart of the problem is the intrusive and harassing memory of violent action […] memories are intense perceptual experiences" consisting of sight, sound, the smell of traumatic experiences such as a gunshot, the silence of the victim, bloodshed, police sirens, explosions and very loud and sudden noises.

These intense and terrifying moments become memories built into the emotional brain circuits.

The symptoms of post-traumatic stress disorder reveal an overactivity of the amygdala that harnesses these intense memories of trauma forcing them to enter the space of consciousness.

The memory of the trauma, as such, becomes a very sensitive triggering mechanism, almost a sort of neural trigger, ready to set off an alarm at the slightest indication of the imminent recurrence of the traumatizing event.

- Simon Baron-Cohen, precursors to a theory of mind: Understanding attention in others, in Andrew Whiten (ed.), Natural theories of mind: evolution, development, and simulation of everyday mindreading, Oxford, UK Cambridge, Massachusetts, USA, B. Blackwell, 1991, pp. 233–251.

9 On moral judgments and empathy, see Eslinger, P. J., Moll, J., & de Oliveira-Souza, R. (2002). Emotional and cognitive processing in empathy and moral behavior. *Behavioral and Brain Sciences*, *25*(1), 34–35.

10 Eth, S. (2018) Progress in PTSD. *The American Journal of Psychiatry*, *175*(10), p. 1023-1023.

More generally, this phenomenon is characteristic of all emotional trauma, including those resulting from repeated physical and sometimes psychological abuse during childhood.

Every traumatizing event can impress these triggering memories onto the amygdala. In detail, violent acts are more dangerous than natural disasters because, unlike the victims of a natural disaster, where chance and fate predominate, or at any rate non-human factors of choice, victims of violence feel that they have been chosen as the target of an intentional violent act. This feeling shatters the assumptions of confidence in people and the security of interpersonal relationships, assumptions which, on the other hand, natural disasters leave intact and sometimes even strengthen. In an instant, the world, understood as a social place, becomes dangerous, populated by people who represent potential threats to their safety and survival. In these cases, the neural trigger may even become more powerful because it is generalized to a set of acts or events not immediately related to the traumatic event.

Post-traumatic stress disorder leads to a dangerous lowering of the neural threshold that triggers the alarm: the individual then reacts to normal life events as if they were emergencies.

The neural basis of these memories seems to lie in extensive alteration of brain chemistry triggered by a single experience of unbearable terror.[11]

As Dennis Charney[12] put it, "It doesn't matter whether the trauma was the relentless terror of combat, torture, or ill-treatment suffered during childhood […] all uncontrollable stresses have the same biological impact". A key element here is the adjective uncontrollable.

If, in a catastrophic situation, an individual thinks he/she can do something that he/she can exercise some control over, no matter if even in a limited form, this individual is psychologically better off than those who feel completely powerless.

It is the impotence at the end of the day that makes us feel subjectively overwhelmed by a particular event. It is precisely the individual who feels powerless that is most vulnerable to post-traumatic stress disorder: the brain begins to alter at the very moment when it feels that life is in danger and the individual is aware that there is nothing he/she can do to save himself/herself.

The main symptoms of this learned fear, including the most intense ones, are explained by considering the alterations that take place in the circuits of the limbic system, especially those concentrated in the amygdala.[13]

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[11] LeDoux, J. E., Romanski, L., & Xagoraris, A. (1989) Indelibility of Subcortical Emotional Memories. *Journal of Cognitive Neuroscience,* 238–243.

[12] An account of PTSD's underlying brain modifications and the role of the amygdala in these is found in Charney, D. S., Deutch, A. Y., Krystal, J. H., Southwick, S. M., & Davis, M. (1993).

[13] Experiments with animal pairs were described by John Krystal, and were repeated in

Some of the most important changes take place in the locus coeruleus, a structure that regulates the secretion in the brain of catecholamines (including adrenaline and noradrenaline) that mobilize the body and prepare it for emergency; these substances also cause recollections to imprint in the memory with particular intensity.

In patients with post-traumatic stress disorder this system becomes overactive, secreting exceptionally high doses of catecholamines in response to situations that actually involve reduced threats but that somehow resemble the original trauma.[14]

The locus coeruleus and the amygdala are closely linked to each other and to other structures of the limbic system, such as the hippocampus and hypothalamus, while catecholaminergic circuits extend to the cortex.

It is believed that some alterations of these circuits are the basis of the symptoms of post-traumatic stress disorder. Other modifications take place in the circuit that connects the limbic system to the pituitary gland that regulates the release of corticotropin-releasing factor CRF (also known as corticoliberin), the main stress hormone responsible for triggering the fight-escape response. Changes in this gland lead to hyper secretion of this hormone, which puts the body in a state of alert triggered by a non-existent emergency.[15] A third set of alterations occurs in the opioid system, *id est*, the structures responsible for secreting endorphins to dampen the sensation of pain, a system which, as a whole, also becomes hyperactive. The amygdala also participates in this neural circuit, this time together with a region of the cerebral cortex.

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several laboratories. The most important studies were conducted by Jay Weiss, at Duke University.

[14] Charney, D. S., Deutch, A. Y., Krystal, J. H., Southwick, S. M., & Davis, M. (1993). Psychobiologic mechanisms of posttraumatic stress disorder. *Archives of General Psychiatry*.

[15] In short, the brain, in trying to lower the rate of CRF secretion, compensates by decreasing the number of its receptors. An eloquent sign that this is what happens in individuals with post-traumatic stress disorder comes from a study in which eight patients received injections of CRF. Usually, an injection of CRF triggers a wave of ACTH [adrenocorticotropic hormone], the hormone that spreads through the body to trigger catecholamines. But in patients with this disorder – unlike in a control group of normal subjects – no appreciable change in ACTH levels was observed, indicating that their brains had reduced CRF receptors because they were already overloaded with stress hormone. The research is described by Charles Nemeroff, Duke University psychiatrist. In particular, the following reference works are mentioned:

- Sherin, J. E., & Nemeroff, C. B. (2011). Post-traumatic stress disorder: the neurobiological impact of psychological trauma. *Dialogues in clinical neuroscience*, *13*(3), 263–278.
- Heim, C., & Nemeroff, C. B. (2009). Neurobiology of posttraumatic stress disorder. *CNS spectrums*, *14* (Suppl. 1), 13–24.
- Sanders, J., & Nemeroff, C. (2016). The CRF System as a therapeutic Target for Neuropsychiatric disorders. *Trends in pharmacological sciences*, *37*(12), 1045–1054.

When the individual has a high level of opiates, they have increased tolerance for pain, and changes in endorphin levels add new elements to the neural cocktail triggered by re-exposure to trauma, further obfuscating a whole spectrum of feelings.

This seems to explain several negative psychological symptoms such as anhedonia, general emotional torpor, the feeling of being cut off from life, and not feeling interested in others' feelings.

Structural neuronal alterations immersed in this extraordinarily altered chemical cocktail also seem to make the individual more susceptible to further trauma.[16]

These considerations could also explain why the development of post-traumatic stress disorder is selective, even though several different individuals are exposed to the same traumatic event: the amygdala is programmed to detect the danger, and when it is exposed to risk again in real life, its alarm sounds louder.

It is therefore likely that a subject who is more exposed to even non-excessive trauma but quantitatively frequent is more susceptible to the development of this disorder in case of a traumatic event compared to others, and this is precisely because of the basic state of hyper-activation of the amygdala circuit (think, for example, of the experiences of war or the devastating episodes of school shootings: not all subjects exposed to the same trauma develop post-traumatic disorder, and individually they may develop it with different intensities).

All of these neural alterations offer short-term benefits for dealing with the dreadful and ruthless emergencies that caused them.

In difficult situations, being highly vigilant, activated, pain-resistant, with a body ready for prolonged physical exertion, has, and is, an obvious adaptive and added survival advantage. That is why this complex set of physical-physiological-psychological responses and, ultimately, electrochemical responses at the level of neuronal networks originates in the oldest part of the brain organs and exploits the more archaic mechanisms of the often unconscious physiological responses. However, these immediate benefits can become

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[16] For further reading of cross-sectional reference studies, see the following:

- Pitman, R. K., Rasmusson, A. M., Koenen, K. C., Shin, L. M., Orr, S. p., Gilbertson, M. W., Milad, M. R., & Liberzon, I. (2012). Biological studies of post-traumatic stress disorder. Nature reviews. *Neuroscience*, *13*(11), 769–787.
- Pineles, S. L., Suvak, M. K., Liverant, G. I., Gregor, K., Wisco, B. E., Pitman, R. K., & Orr, S. P. (2013). Psychophysiologic reactivity, subjective distress, and their associations with PTSD diagnosis. *Journal of abnormal psychology*, *122*(3), 635–644. Erratum in: J Abnorm Psychol. 2015;124:287.
- Glover, H. (1992). Emotional numbing: A possible endorphin-mediated phenomenon associated with post-traumatic stress disorders and other allied psychopathologic states. *Journal of Traumatic Stress, 5*, 643-675.

problematic in the long run if the brain changes to the point where they become predispositions.

When the amygdala and the brain regions connected to it are recalibrated during an intensely traumatic event, this alteration in excitability means that all of life is always on the brink of an emergency, and even an objectively neutral event can trigger an emotional explosion of terror, panic, and fear (in some cases accompanied by a violent response). These traumatic memories interfere with subsequent learning.

When fear is acquired, the mechanisms of memory learning become blocked: the amygdala, among all the brain regions involved, plays a key role, but the neocortex is essential to overcoming the acquired fear. In the brain, the key structure that learns, stores, and implements these fear responses is the circuit that connects the thalamus, the amygdala, and the prefrontal lobe.[17]

Usually, when you learn to fear something through conditioning, fear fades with time. This phenomenon appears to be due to natural re-learning.

In post-traumatic stress disorder, this spontaneous re-learning does not take place. This is probably due to the fact that the brain alterations typical of post-traumatic stress disorder are so severe that the amygdala can trigger 'a sequestering' whenever something even vaguely similar to the original trauma occurs. The extinction of fear, therefore, seems to involve an active learning process, which in individuals suffering from this disorder is compromised. This re-learning is cortical in nature.

The original fear that has taken root in the amygdala never completely disappears; rather, the prefrontal cortex actively suppresses the signals that the amygdala sends to the rest of the brain to trigger the fear response.

In children, one of the ways that can lead to a spontaneous psychological recovery is through play.

One example is the Purdy case, a game that was used to treat children who had experienced the tragedy of the Cleveland Elementary School massacre.[18]

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[17] The brain data reviewed – albeit concisely and schematically – are based on Charney, D. S., Deutch, A. Y., Krystal, J. H., Southwick, S. M., & Davis, M. (1993). Psychobiologic mechanisms of posttraumatic stress disorder. *Archives of General Psychiatry*, *50*. Cited article. However, see also for specific details:

- Thiel, F., Berman, Z., Dishy, G. A., Chan, S. J., Seth, H., Tokala, M., Pitman, R. K., & Dekel, S. (2021). Traumatic memories of childbirth relate to maternal postpartum posttraumatic stress disorder. *Journal of anxiety disorders*, *77*, 102342.
- Wood, N. E., Rosasco, M. L., Suris, A. M., Spring, J. D., Marin, M. F., Lasko, N. B., Goetz, J. M., Fischer, A. M., Orr, S. p., & Pitman, R. K. (2015). Pharmacological blockade of memory reconsolidation in posttraumatic stress disorder: three negative psychophysiological studies. *Psychiatry Research*, *225*(1-2), 31–39.

[18] Lenore Terr, 1990, Too Scared to Cry: Psychic Trauma in Childhood - Harper Collins NY. For an analytical review: Cohen, P. M. (1991). Review of *Too scared to cry: Psychic trauma in childhood* [Review of the book *Too scared to cry: Psychic trauma in childhood,* by L. Terr]. *Psychotherapy: Theory, Research, Practice, Training, 28*(1), 192–193.

The game-system opens two paths to recovery. On the one hand, the memory is repeated in a context of low level of anxiety, leading to desensitization and allowing association with non-traumatized responses. Another way to heal is one that allows children, at least in their minds, to give tragedy another ending.

While adults who have gone through tremendous trauma may experience a psychic numbness in which they exclude memories or feelings related to the catastrophe they have experienced, the psyche of children often manages the trauma differently. Because they use the fantasy of play and daydreams to recall and mentally retrace their torments, children become (or *may become*) less sensitive to trauma than adults. This deliberate re-enacting of the trauma seems to block the need to contain it in the form of vivid memories, which later can explode as a flash back. One way to get to this scene frozen in time within the amygdala is through art, which itself is a means of the unconscious.

The emotional brain is in tune with the symbolic meanings and the mode that Freud called 'primary process':[19] in other words, with the messages of the metaphor of the history of myth and art.

In the artistic production of traumatized children, there is almost never a lack of these hidden references to the traumatic scene. Such overbearing memories, which haunt the child, creep into his/her artistic production just as they appear in his/her thoughts. The very act of drawing is therapeutic in that it initiates the process in which the child comes to dominate the trauma.

According to Judith Herman,[20] a Harvard psychiatrist, there are three stages in which it is possible to recognize the main steps of recovery from trauma: the achievement of a sense of security, the remembrance of the details of the trauma and the pain for the loss it entailed, the restoration of a normal life.

In the sequence of these stages, there is a biological logic: it seems to reflect the way the emotional brain learns that life should not be regarded as an impending emergency.

There is a sort of reprogramming of the state in which the memory is fixed in the amygdala, which in turn affects the brain chemical response that determines the physiological-symptomatic response. The first step, which translates into a sense of security, is presumably to find ways to calm the generally overactive, fear-prone neural circuits, to the point where they can relearn more normal reactions.

To achieve this result, you often begin by helping patients understand that

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[19] Freud, S. (1900). The interpretation of dreams. Stollati Boringhieri, 1st Edition, 1973, p. 532-50.

[20] Lewis Herman, J. (1992). Trauma and Recovery. New York: Basic Books.

their excitability and nightmares and their hypervigilance and panic are part of the symptoms.

Another initial step is to help patients regain a certain sense of control over life's events, and this is essential for overcoming the lesson of impotence that the trauma has taught them.

It is true that, at this stage, drug therapy can be of help and support. There are certain drugs (only some) that antagonize some of the changes we have mentioned, in particular, antidepressants, which act on the serotonin system, or beta-blockers that block the activation of the sympathetic nervous system.

Achieving a state of physiological calm allows traumatized emotional circuits to rediscover that life is not a threat and allows patients to regain some of the confidence they had before the trauma.

Nevertheless, and not only in cases of post-traumatic stress disorder that we are taking as an example here, the use of drug treatment must necessarily and strictly be ascribed to a phase in which the symptoms are so strong, and generate such a degree of pain, that they do not allow a participatory life for the patient. Pharmacological intervention can never be a substitute for reprogramming the amygdala circuit and the limbic system in general, nor can it be so strong as to completely interrupt the life and physiology of the symptom, which remains an extraordinary indicator of the underlying disease and the trauma to be overcome.

In healing, the next step involves re-telling and reconstructing the trauma story under cover of the newly acquired security, allowing the neural circuits that process emotions to understand the memory of the trauma and to react to it and to the factors that trigger it in a new and more realistic way.[21]

Memory then begins to transform both in psychological significance and in its effects on the emotional brain.

Far from being a progressive linear path, the path of rewriting trauma can be seen as a fluctuation that seems to allow spontaneous reconsideration of the trauma and re-learning for trial and error of the emotional reaction.

Emotional re-learning is carried out, to a large extent, by reliving events and emotions related to them, but this time in a safe environment, in the company of a trusted therapist, in which two simultaneous communication mechanisms intervene. This is a very important point.

As Beau Lotto[22] clearly stated:

> "Perception is not something that takes place in the isolation of our brain, but is part of an incessant process within an *ecology*, by which I mean the relationship of everything with the things that surround it, and their mutual inferences [...] understanding the condition of being human involves understanding the interac-

[21] Horowitz, M. (1986). Stress Response Syndromes. Northvale, NJ: Aronson.

[22] Lotto, B. (2016). Deviate: The Science of Seeing Differently. London: Weidenfeld & Nicolson.

> tions between our body and our brain and between other bodies and brains, as well as all of the world around us. That is why, I believe that life is an ecology, not just an environment. Life, and what we perceive, lies in what I call the 'the space between'."

On the one hand, there is the rewriting of the narrative history of the trauma of one's own experience and of the acquisition of one's response to the traumatic stimulus, a passage then of rational metabolization of a traumatic event that left a strong trace at the limbic system level, generating a traumatic experience and the acquisition of an out-of-scale stimulus-response. On the other hand, the communication mechanism comes into play, which is linked to emotional intelligence that unconsciously, through the exchange between two human beings, restores the levels of social trust and environmental security that have been lost.

In this double passage, neither of the two moments can be separated from the other, and the therapeutic intervention takes place on two levels that do not have the same chronology at all, nor do they have overlapping chronologies.

We cannot know how hard and how long the so-called lower route needs to establish a level of harmony such that the unconscious part can restore plastic and electrochemical levels, which are the foundation and solid basis for a purely rational path of redefinition of historical experience, which in itself constitutes the traumatic and traumatizing element on the one hand and the structuring of the automatic and physiological response on the other.

These two levels of not only rational reprogramming (in a dialogue with the interpreter) but also of the lived basis with the outside world of the traumatic element and the traces it left in terms of injury in the amygdala and the limbic system as a whole, acting at levels much broader than a single organ or range of neurotransmitters, in no way and form can be delegated or entrusted exclusively to a chemical-pharmacological intervention. Indeed, as already mentioned, unnecessary pharmacological intervention, intervening both on the external symptom and on the chemistry of brain structures, can constitute an obstacle to the two processes we have identified.

The pain that follows the narrative of painful events has a crucial function: it marks the ability to let out the trauma. It means that instead of being constantly prisoners of that tragic moment in the past, people can start looking ahead, even hoping and rebuilding a new life free from the trauma and emotional injuries that affect their psychophysical responses. The same circuits that fix traumatic memories so markedly are supposedly at work, even in the quietest moments of life.

The most common suffering in childhood may never reach the level of trauma, but it certainly leaves its mark on the emotional brain by creating distortions in the individual's subsequent intimate relationships.

In general, emotional intelligence comes into play at the very moment when you must learn how to skilfully manage these psychologically charged reactions.

The dynamic interaction between the amygdala on the one hand and the more informed reactions of the prefrontal cortex on the other offers us a neuroanatomical model to explain how psychotherapy reshapes deep-rooted emotional models that are now devoid of adaptive value.

As Joseph LeDoux states:

> "Once your emotional system learns something it seems you never forget it. What therapy can teach you is how to control it: it teaches the neocortex how to inhibit the amygdala. The inclination to act is thus suppressed while the fundamental emotion remains in attenuated form."

Given the brain architecture that underlies emotional re-learning, what seems to remain, even after successful psychotherapy, is a vestigial reaction, a remnant of the original sensitivity or fear that underlies a relatively emotional problem.

The prefrontal cortex may refine the impulses from the amygdala or restrain them, but it cannot prevent them from reacting.

During therapy, overall, what seems to change are the individual's responses once an emotional reaction has been triggered, but the tendency to have an immediate reaction does not disappear completely.[23]

In brain terms, we can assume that the limbic system continues to send alarm signals in response to the warnings of a feared event, while the prefrontal cortex and the areas connected to it have learned a new and healthier response.

In short, the emotional reactions learned, even more radically because they have been acquired during childhood, can be reshaped. This kind of learning lasts a lifetime and can be considered an essential part of neural plasticity.[24]

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[23] Therapy research has been described in detail in Lester Luborsky and Paul Crits-Christoph, Understanding Transference: The Cort Method. New York: Basic Books, 1990.

[24] Finally, and we can say conclusively, Marian Diamond has found definitive proof that the brain is shaped not only during the developmental age but throughout its life, generating major changes in perception. The ability to conform to the environment and adapt to stimuli enriches the physical structure of the brain through the release of growth factors – in particular those identified by Rita Levi Montalcini – that lead to the development of nerve cells and their connections, and the connections between them. See Diamond M.C. (2001). Response of the brain to enrichment. *Anais da Academia Brasileira de Ciencias*, 73(2). But brain development also has its limitations. A study of neurodevelopment of children affected by institutionalization in deprivation settings found that some cerebro-behavioral circuits could over time reach normalized levels. But the memory visualization and inhibition control were not recovered. See Pollak, S. D., Nelson, C. A., Schlaak, M. F., Roeber, B. J., Wewerka, S. S., Wiik, K. L., Frenn, K. A., Loman, M. M., & Gunnar, M. R. (2010). Neurodevelopmental effects of early deprivation in postinstitutionalized children. *Child development*, *81*(1), 224–236.

Appendix Notes

The reference bibliography of the topics covered, as well as the indications of further specific sources of information, have been included in the notes to the text.

Special thanks go out to the scientific community for creating a free public database that is indexed and shared, such as PubMed, which, since 1996, allows for the consultation of millions of scientific articles published all over the world. While 'Dr. Google', on which many seek *prêt-à-porter* diagnoses, has never gone to school (but is overrun with sponsored pseudoscience), authors indexed on databases such as PubMed (and others) are all graduates and specialists, and their articles are reviewed and verified 'by peers' prior to publication. Our world is increasingly complex, and in this globalized complexity, we should orient ourselves by considering a model of shared intelligence and knowledge.[25]

The *circuitry model* that I use in this article to describe the neuro-physiological substrate process of post-traumatic disorder derives from a broader and different work on autism spectrum disorder, in which the model of '*empathy circuit*' is central and described admirably by Simon Baron-Cohen.[26] Finally, by abandoning a locationist idea of the brain, we are moving toward a systemic and correlated view of cooperating and integrated brain circuits and systems. In this revolution of our vision of the mind (which goes far beyond the brain), we can only be astonished by its extraordinary complexity. We are like astronomers who, for the first time, see beyond the solar system. This new direction is a debt that we all owe to people with autism and an extra motivation to study it. For everything that these extraordinary people can enable us to understand about our minds and our world by entering their minds and their worlds with delicacy and humility, we are grateful.

These endnotes and empirical reference follow-up notes would not have been possible without the valuable advice during the review process. If the text has improved, it is the merit of the reviewers. If there are gaps or things that have not been completely clarified, it is my responsibility.

As I said, the world we live in today is increasingly complex, and in this globalized complexity we should orient ourselves by considering a model of intelligence and shared knowledge, and the way forward must be travelled together.

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[25] On the subject, see the recent and very useful book by Steven Sloman and Philip Fernbach. (2017). The Knowledge Illusion. Why we never think alone (ed. it.: Raffaello Cortina Editore, 2018).

[26] See also the following for specifics:

- Baron-Cohen S. The Science of Evil (Ed. it.: Raffaello Cortina Editore, 2012).
- Baron-Cohen S. The pattern seekers. Basic Books, 2020.

In this common process, divergent opinions are an essential asset, and I, therefore, hope that this contribution will not be exhaustive, that it will raise questions, concerns, and doubts that will lead to new analyses and critical comments, that it will stimulate the refutation of the arguments put forward and extend the topics and objects discussed.

If so, this short contribution will have achieved its purpose in the intentions of its author.

To use the words of Lotto:

> "The brain does not always want toys […] it needs to learn that it can get back up after being knocked down, and in doing so becomes more resilient […] but it is not just [people] that have to take risks, our culture has to do it too. The past of our brains also includes the ecology of our culture. After all, culture itself is simply another product of the brain, a collective manifestation of thought and behaviour, so it also grows and adapts to meet the challenges [...] it is not just the human brain that is changing: the collective cultural brain changes too. Both constantly redefine the concept of 'normal' by creating a new normal with every passing second."

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