

## **THE SPECULATE BRAIN**

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### **Summary**

This work deals with the theory according to which the clinical expressions manifested in the schizophrenia could be considered like the "symptomatologic reflex" of the wrong operation of the subcortical zones , clinical expressions that we from years attribute to the prefrontal cortex . The Author hypothesizes that the cortical areas are above all associative and that their hard function "release" the subcortical zones that would be the greater responsible of the schizophrenic symptoms . On the base of this theory the author describes the possible neurological streets implicated in the genesis of the deliriums and of the hallucinations. The purpose of the study is to point out any incongruities in the sketch of the circuits so far described and expresses perplexity around the methods of study so far envoys in action. Such methodic they have assembled their attention above all on any cerebral zones (prefrontal cortex , tegmento-ventral area, nucleuses of the base, head of the caudate nucleus ecc) neglecting of examine the amygdale (from little time in prominence) the hypothalamus , the entorhinal cortex , the insula, the motor and sensitive streets , the red nucleus and so much other cerebral districts stayed so far in the shade. This study wants to be a stimulus to widen the strumental examinations on all the subcortical zones to can have a "vision of together" of the operation of the cerebral zones in the schizophrenia.

### **Introduction**

The concept of brain speculate originate from the considerations expressed from many neurologists and psychiatrists around the functioning of the brain and the psychic expressions that characterize the mental pathologies . In last century a skilful scientist Paul Maclean (1) hypothesized that the human brain had developed itself in three following phases, in the course of which three zones could be identified: the first very ancient definite reptilian , correspondent approximately to the encephalic trunk , the second chronologically following developed to the of above of the trunk, identifiable with part of the diencephalon and finally the latest zone correspondent to the cortex, born at the end of the cerebral development of the man. It being understood that it is my conviction that our brain will continue to become larger and that the zones cortical will be in the next millennia "overlapping" from other zones new-developed , from this consideration we could draw aside fascinating hypothesis. The first zone (reptilian) is without conscience, in the sense that to each stimulus corresponds an instinctive answer, compulsory, not reasoned.

Probably the living beings that possess this cerebral structure doesn't try sensations or feelings, but only antiseptic instinctive answers. The living beings that reached the second stadium of evolution probably possess a "emotional coloration" correlated to the stimuli and to the answers: they try fear, anger, desire of escape etc. This emotional coloration is also she primordial, in the sense that there is little of conscious, the "reasoned consciousness" of the perception of a stimulus and of the adequate answer, aren't present. In these living beings the part that would act from "simil-cortex" could reside in the convolution of the cingulum and in the part medial of the temporal lobes (areas of Heschl, cortex of the insula, limbic cortex) (2) where they have the role of coordinators of the function "stimulus-answer adequate" in the beings that have this cerebral organization. The limited functionality of this pseudo-cortex is also testified from his structure; if we analyze microscopically the neocortex, we find that it is fully grown from six layer of cells, respect at three that are in the underlying zones (gyrus of the cingulum), that presupposes, in the neocortex, a better organization of the interactions between the structures and a more complex cerebral functionality (3).

To this point bears spontaneous a question: why never the human being would have needed the cortex and because does it have developed? After all the underlying zones were able to work adequately to allow the life of relationship, within some limits. It's evident that the answer is sought in the improvement of the analyses of the stimuli and of the answers, but above all in the language. The human being in the course of the evolution began to associate behaviors to the consequences of the behaviors, they began to associate the acquired experiences creating logical connections between experiences, memoirs, behaviors and consequences to them connected. To this we are been adding a sketch of language that put the premises to that they develop zones superior cortical. To do this the man has slowly developed a series of structures cortical that has the function to put in connection all the centers interested for the elaboration of "logical constructions" that spring out of the experiences. From these considerations starts the idea that the zones cortical is exclusively "associative," that they serve to put the informations together, to correlate them with the centers of the memory and subcortical, to perform a "reasoned answer." Departing from this point of view we could understand like the underlying zones are "little associative" as regards those of the cortex, and that the neuronal stations of the diencephalon are, under any appearances, more specialized. This means that to a lesion subcortical can easily correspond a symptomatology that derives, in maximum part, from the function lost in the specific one zone. For example the lesion of the zones 32 and 24 of the cingulum gives birth to an excessive and unnatural loquacity, the specific stimulus of the nucleuses cortical-medial of the amygdala can cause behavioural expressions of anger, the lesions of the areas of Heschl carry to auditory symptoms, while the lesions of the hippocampus and of the libic system of left they cause alterations of the language and motor (4). These is only any of the examples that could be facts on the zones subcortical. This means that a specific lesion is able to give symptoms that are prerogative of the particular one zone of the diencephalon. The same doesn't happen for the cortical zones: this is motivated from the fact that the cortex has superior functions, less specific, more from "supervision."

It's fitting the example of a square of soccer : if a player plays badly, will hear again only the zone and the role in which the player has appointed to play ( subcortical zones ); if to mistake is the trainer (the supervisor) , for wrong strategies of play, the whole square will stay some damaged. This means that the underlying areas to the cortex are, under some appearances, "pseudoautonomous," in the sense that they could express symptoms both under the influence of the cortex , and if they are release from her. But , to this point, it is necessary to do an ulterior consideration: the brain and his functions don't ever stop ; this means that exists a "functionality of base" always present, an equilibrium of functions, stimuli, answers that are "basally " always present. A kind of "functional tension" of base, that doesn't ever cancel out, not even during the sleep. This presupposition, easily shareable, bring to the logical conclusion according to which:

- The brain possesses varied stations that work in equilibrium of strengths between them, in the sense that perception, elaboration of a stimulus, feeling and response, are the resultant of this equilibrium.

But the cortex , that for his role of supervisor is above all associative, has the purpose of integrate the information between them and express the more adequate answers. From that is deduced that a lesion of a specific zone cortical trouble this mechanism of the equilibrium, for which the nervous stations subtended and directly connected to her, they come in a certain sense " released" from the influence of the cortex . And are above all the zones that have intense connections with that portion of cortex ,that from it are mostly influence. These zones released, because they are underlying to the cortex, they are free to can express themselves , to can give symptoms, feelings, functions, that don't come ,in this case, filter from the supervisor. On the base of this hypothesis , the consequent deduction could be that:

- The symptom that is expressed to consequence of a lesion cortical, it is not given from the stricken cortical zone, but from the zones subcortical that from them depends and in better measure those zones subcortical that has intense connections with the damaged cortex.

In simple terms this means that the concepts developed in the years according to which to a lesion of a zone cortical corresponds a determined symptom are partially wrong: the symptoms don't correspond the cortical zone damaged , but to her been missing supervision on the underlying zones and/ or on other associative near areas, that are "free" to express themself without control. It's like if we have analyzed so far the brain to the mirror, a brain in which the symptoms are only the "reflex" of other cerebral districts: in the schizophrenia , for example , we have identified the cortex prefrontal like responsible of the positive and negative symptoms, symptoms that would result be, to a large extent, expression of the underlying zones.

On the base of this theory it's therefore supposable that come seen again the routes of the schizophrenia, setting particular prominence to the zones subcortical. If, for instance, we take the delirium in consideration we will think that the responsible routes of the symptoms are above all those subcortical .

The delirium is a distortion of the interpretation of the reality. It departs from a "psychic pain" not metabolized, not sublimed, from an external visual input, acoustic and/ or visceral. For this reason the routes of the delirium depart necessarily from nucleuses ponto-mesencephalic (nc of the raphe, interpeduncular, visual streets, acoustic and reticular formation ) (2) that spending for the ventral tegmentum project themselves, through the longitudinal fascicle mediate , to the thalamus, that is best relay of the brain for what concern the afferent sensorialities . The thalamus, in its turn, projects to the amygdala stimulated it, and to the hippocampus. The amygdala through the nucleuses basal-lateral projects the cortex prefrontal, that checks the same amygdala with inhibitory action. The symptoms of the delirium schizophrenic derives not so much from a lesion of the zone prefrontal *in primis* , in the sense that is not the zone prefrontal to express the delirious symptomatology, but the mental rigidity, the poverty of introspection, the deficit of planning, judgment and criticism, (5) derives from a been missing associative action of the prefrontal zone on the underlying stations to her mostly connected, like hippocampus, amygdala, and thalamus. These stations try to could be revealed all one series of symptoms like anger, fear, escape (amygdala) alteration of the perception of the emotions and of the memory (hippocampus) and alterations of the analysis of the sensory perceptions deriving from the outside (thalamus). This circuit, not more checked from the cortex prefrontal, intensifies the symptoms only just described : but the anger, the fear, the unorganized memoirs and not filtrates from the prefrontal cortex, carry the brain of the patient to elaborate false convincements, false memoirs, in short deliriums for face the input that emerges from the subcortical stations not regulated. The triad "thalamus-amygdala-hippocampus" would go to constitute a kind of "circuit obsessive delirious ," a circuit autoreflexion that in a state of obsessive overexcitement (typical of the first phases of the delirium) would oppose the function of control that the prefrontal cortex has on these stations, in the sense that the input would be such and so much, continuous in the time that the overloaded cortex would not be able to develop his assignment of "supervisor." And ,in this context, the amygdala develops a role of first plain in as station of meeting between cortex, thalamus, hippocampus, parahippocampalis cortex and nucleuses of the base (5).

This principle of the symptomatologic expressiveness derived not from the direct interest of the zone of cortical malfunction , carries us to duty see again the interpretations of the symptoms and of the derivations of them in the schizophrenia. The mental rigidity of the schizophrenic, the absence of introspection of the behavior and of the analysis of the stimuli, of the lack of criticism, derive only partly from the prefrontal cortex , because the mailing of signals altered from part of the underlying zones, released few working from the cortex, translate themselves in a "imbalance " of the relationship cortex-undercortex, caused to cognitive alterations, own of the schizophrenia.

The theory of the "brain speculate" could be applied also to the hallucinations in the schizophrenic patient . In this case the routes of beginning is always to departure from the nucleuses of the trunk, that spending for the ventral tegmentum, arrive at to amygdala and thalamus. It is correct to specify that, to my notice, the zone tegmentum-ventral is , probably, only one zone of transit in as in the ventral tegmentum is present only small nucleuses like the nc paranigric, nc pigmented parabrachyal, nc interfasciculus, the black substance and the A10 area , that are not the routes of

beginning like we thought but probably are only of intermediary stations; the stimulus part, probably, from much more in low: from the raphe, from the nc interpeduncle, from the locus coeruleus, from the streets visual and acoustic ponto-mesencephalic. The difference as regards the deliriums is that the way common ending of the cortex mostly interested is, probably, the temporal one. Also in this case the "overloaded of impulses" on the temporal zone lets frees outlet to those underlying zones that are to them connected like the areas 37, 17, 18-19 (visual streets), 22, 52, 41-42 (auditory streets). These stations not more regulate from the temporal cortex , send stimuli and released input, leaving that amygdala and thalamus can " overload" tha subcortical-temporal cortex circuit , caused auditory hallucinations and/ or visual. This mechanism of best afferent of fibers would also explain how come are more frequent the auditory hallucinations, as regards the visual: that depends on the fact, probably, that the temporal cortex is strongly connected with the auditory zones (that they are a part of the same temporal lobe) and less with those visual, that derives from the calcarinus cortex, that has set to better distance.

It's clear like this discourse is only a theory, based however on comparisons brought again in literature and his secluded meaning is that of stimulate to a more accurate reconstruction of the routes of the schizophrenia , holding even present, that the express symptoms could represent a "vision speculate," an indirect reflex of the malfunction of the responsible zones. It would be therefore desirable that the studies performed with RM, SPECT, functional RM, are more and more turned to the zones subcortical, what real districts of origin of the psychotics symptoms in general. And if that had shown also the new pharmacological aids should go to interest the stations subcortical mostly, modulating the compromised diencephalic functionality.

### References

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