# The Implosion of Reality. Schizophrenia, the Anterior Cingulate Cortex and Anticipation

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**Abstract**
In contemporary neuroscientific and psychiatric research into schizophrenia, we can observe a shift in focus from the clinical dysfunctions (positive and negative symptoms) towards a mapping of the cognitive function. In this paper we look at a specific cognitive problem area in schizophrenic brain functioning, the Anterior Cingulate Cortex (ACC). We show what the ACC is, what it probably does and how this is relevant in research concerning certain psychiatric disorders. Then we explain the role of the ACC in choice anticipation. In this we underline the possible link between choice anticipation and the lack of 'Error Related Negativity' (ERN) in this specific area. Lastly we incorporate this approach to the problem of schizophrenic anticipation within the neuropsychoanalytical framework and the role it might play in the formation of hallucinations and delusion.
**Keywords:** Schizophrenia, ACC, (Neuro)psychoanalysis, Anticipation, Delusion.

## 1 The unapparent connection is more powerful than the apparent one.[1]

In this publication I would like to present you with material which links the idea behind anticipation with the research concerning schizophrenia from a complex neuropsychoanalytical viewpoint. For this choice, I give three reasons.

The first one is that the ideas underlying an complex anticipatory approach enrich any problem you put your mind to. It makes the model more natural, dynamic and from my own point of view more accessible to thought experiments and clinical applications. Although the model itself may become more difficult to *translate* into experimental research directly and you need an open mindedness not commonly found among scientists and philosophers nowadays, it will be worth it in the long run.

Second of all, I am very interested in the neurology behind schizophrenia. As opposed to other mental 'illnesses' such as hysteria, neurasthenia, depression,

[1] Herakleitos, quoted in Hippolytus, Refutations
(http://community.middlebury.edu/~harris/Philosophy/heraclitus.pdf)

personality disorders and the like, the brain has always seemed as *the* site where schizophrenia is supposed to be located. “In 1837, Dr. W.A.F. Browne, the best-known English psychiatrist of his generation, wrote: "Insanity, then, is inordinate or irregular, or impaired action of the mind, of the instincts, sentiments, intellectual, or perceptive powers, depending upon and produced by an organic change in the brain." In that same year, Dr. Amariah Brigham, one of the founders of American psychiatry, also wrote that insanity "is now considered a physical disorder, a disease of the brain."”[2]

Only a handful of researchers have the courage to look beyond the actual locus of the problem in the brain and look at the complex connections between different cognitive or psychological phenomena and different brain states. Schizophrenia is a complex problem, so to do it justice we can only hope to find a very complex answer to this question, no matter what level of the schizophrenic question we want to address (be it the social, psychological or neurological level).

Thirdly, as a psychotherapist, I work with schizophrenic patients on a daily basis. In this work, it is very remarkable to witness two very distinct phenomena. On the one hand, they seem to fail miserably when it comes to making realistic expectations about the future and their own place in it. On the other hand, they do not seem to stop anticipating events, which from a logical point of view can’t and won’t ever happen. These wrongly anticipated events are commonly known as delusions.

Especially in the case of paranoid schizophrenia, these two quirks in the anticipatory process really stand out. Paranoid patients don’t seem to bother too much with the anticipation of real dangers and other possible stressful events (such as losing a job, a partner, a friend or even making certain specific errors in everyday life situations and the implications these might entail; things like crossing the road even). This makes them from a ‘normal’ point of view seem reckless, aloof or even outright demeaning in social situations. But when it comes down to their paranoid delusions themselves, they are very much aware of how the danger they fret is going to come and knock on their door. They know what the specific portents are of this imminent doom and what they will have to do to counter or accelerate this process. In short, from a clinical perspective paranoid patients lack a realistic anticipatory attitude and they make up for this through a paranoid anticipatory approach.

In paranoid schizophrenia the process of anticipation has gone haywire, and no one understands how this has come about. One of the major advances that a psychoanalytical approach to this problem might have, is an insight in the logic behind (paranoid) delusions and hallucinations. They are not mere whim nor folly, there is a certain anticipatory logic behind it.

To help a schizophrenic patient in a state of anticipatory anxiety is to understand what is going on in his mind, so as to anticipate what possible interventions might be able to alleviate the burden of this anguishing anticipatory process and the social turmoil that follows in its wake.

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[2] http://www.schizophrenia.com/family/disease.htm

So, to keep things simple, in this paper we link the neurological approach to the problem of anticipation, haphazardly located for now in the anterior cingulate cortex, and look at where this avenue of thought leads us to.

First we look at schizophrenia from a psychiatric point of view and comment upon the view of (ab)normality. Next, we discuss the Anterior Cingulate Cortex, followed by a look at the involvement of this brain area in anticipation. We then incorporate the ACC anticipation cortex within a psychoanalytical framework regarding psychosis to come to our conclusion about the ACC-anticipation-psychosis link.

## 2 Schizophrenia, the 'global brain disease' in sickness and in health?

From the first moment the term schizophrenia was first coined by Eugene Bleuler in 1911, this group of mental disorders was seen as one of the most severe and pervasive, with a very pessimistic prognosis. Even Freud it is said deemed them almost untreatable at times (1911 [1910]). The severity of this illness is only matched by Alzheimers'dementia, which normally only manifests itself later in life. But, schizophrenia has its onset for most patients between the age of 18 and 25 years, where a subject should normally have his or her entire life before him. Before Bleuler in 1887, Emile Kraepelin named this group of patients the dementia praecox, those who irreversibly become demented too young because of their illness.

Even in popular culture the term schizophrenia equals dangerously mad, people in need of being locked away for life. And for a great spell, this was actually the case, schizophrenic patients where locked away in asylums for the rest of their days. This view on schizophrenia continues up to this very day, where people with schizophrenia are seen as disordered and malfunctioning individuals.

This approach is countered from a Lacanian psychoanalytical point of view by stating that schizophrenia is actually just a form of psychosis and that psychosis is a different biopsychosocial structure, another existential position alongside the 'normal' neurotic structure (Maleval, 2000b). Schizophrenia has its disadvantages and its benefits, but it should be measured or appraised in its own right.

The question of developmental 'normality' does not come into play as such from a Lacanian point of view. The crux of psychoanalytical treatment for any patient is the specific subjective suffering and the questions surrounding it, which bring them into therapy. The normative diagnostics behind the reasons for this suffering come second, although they are important as avenues to approach the specific suffering, to understand it and to help alleviate it. In short, from a psychoanalytical point of view the cause of the subjective suffering is the main issue, not the question of (ab)normal thought or behaviour.

So, from a psychiatric point of view now, what is schizophrenia? Schizophrenia is a group of psychotic disorders characterized by disturbances in thought, perception, affect, behaviour, and communication that last longer than 6 months. In the most frequently used psychiatric guide to mental illness, the DSM IV, a patient is deemed schizophrenic if the following criteria are met.

DSM-IV diagnostic criteria for schizophrenia[3]

*Characteristic symptoms:* Two or more of the following, each present for a significant portion of time during a one-month period: Delusions, hallucinations, disorganised speech (eg, frequent derailment or incoherence), grossly disorganised or catatonic behaviour, negative symptoms (ie, affective flattening, alogia, or avolition). Only one of these symptoms is required if delusions are bizarre or hallucinations consist of a voice keeping up a running commentary on the person's behaviour or thoughts, or two or more voices conversing with each other. *Social/occupational dysfunction:* Since the onset of the disturbance, one or more major areas of functioning, such as work, interpersonal relations, or self-care, are markedly below the level previously achieved. *Duration:* Continuous signs of the disturbance persist for at least six months. This six-month period must include at least one month of symptoms (or less if successfully treated) that meet the characteristic symptoms.

The DSM IV further specifies *4 Subtypes*: Paranoid Type, Catatonic Type, Disorganized Type, Undifferentiated Type and the Residual Type.[4]

## 2.1 Positive and negative symptoms: the dopamine hypothesis and beyond?

For all forms of schizophrenia, psychiatrists since Bleuler usually divide the symptoms in positive[5] and negative[6]. These symptoms are then treated with medications, commonly known as neuroleptics. These are divided into two groups: typical[7] and the atypical[8]. The typical ones are all strict dopamine antagonists, meaning

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[3] http://www.mja.com.au/public/mentalhealth/articles/hustig/husbox1.html

[4] Also, schizophrenia should be differentiated from psychotic disorder due to a general medical condition, delirium, or dementia; substance-induced psychotic disorder; substance-induced delirium; substance-induced persisting dementia; substance-related disorders; mood disorder with psychotic features; schizoaffective disorder; depressive disorder not otherwise specified; bipolar disorder not otherwise specified; mood disorder with catatonic features; schizophreniform disorder; brief psychotic disorder; delusional disorder; psychotic disorder not otherwise specified; pervasive developmental disorders (e.g., autistic disorder); childhood presentations combining disorganized speech (from a communication disorder) and disorganized behavior (from Attention-Deficit/ Hyperactivity Disorder); schizotypal personality disorder; schizoid personality disorder; paranoid personality disorder. (see, http://www.mentalhealth.com/dis1/p21-ps01.html)

[5] delusions, hallucinations, disordered thought and disorganized speech.

[6] flat affect, anhedonia, social withdrawal, emotional detachment, cognitive deficits and poverty of speech

[7] Typical neuroleptics are generally $D_2$ antagonists. $D_2$ antagonists are particularly effective in treating the positive symptoms of schizophrenia. Adverse side-effects are common with dopamine antagonists due to the blockade of dopamine receptors within the neostriatum. Major side-effects include dystonias (acute spasms), Parkinsonism (bradykinesia, shuffling gait, muscular rigidity, and tremor), and akathisia (motor restlessness). Tardive dyskinesia is the term used to characterize a neurological disorder caused by long-term treatments with classical neuroleptics. Tardive dyskinesia is particularly associated with abnormal movements of the hands and face.

[8] Since the 1960's, several new compounds have been developed with unique properties. Collectively, these compounds are referred to as atypical neuroleptics, that have an effect not only of $D_2$ but also on the serotinin receptors in the brain for example. Atypical neuroleptics are generally (more) effective in treating both the positive and negative symptoms of schizophrenia and with fewer side effects or so it is believed. The lack of extrapyramidal side effects may be due in part to its potent anticholinergic effects, in addition to a high affinity for 5-$HT_2$ (serotonin) receptors. Atypical neuroleptics generally have high

that they block dopamine in certain parts of the brain.[9] Four major dopamine pathways are distinguished: nigrostriatal (involved in movement and Parkinson's disease), mesocortical (alterations in this are supposed to give rise to the negative symptoms), mesolimbic ( these are the main psychiatric focus point as they are seen to give rise to the positive symptoms) tuberoinfundibular pathway (this tract controls the prolactin secretion from the anterior pituitary gland)

The success of these medicines, combined with an insight into their function in the brain has lead to a theory known as the dopamine hypothesis: schizophrenia as a whole is to be understood as a problem in the dopamine circuitry only, $D_1$ and $D_2$ receptors, because blocking these (especially $D_2$) greatly reduce the positive symptoms. This seems to be at odds with recent findings in the working mechanisms of atypical neuroleptics and further brain research (Dolan et al, 1995; Coyle, 2006; Abi Dargham, 2004).

Other neurotransmitters and their receptors have also been shown to play a crucial role in schizophrenia: acetylcholine, glutamate (NMDA), GABA, cannabinoid, nicotinic and opioid receptors, for example (Coyle, 2006; Benes et al, 2001).

All this leads to the conclusion that a limited look at the working mechanisms behind neuroleptics as an explanatory framework for schizophrenia is flawed at best. We need a broader perspective to place the relevance of the inner workings of dopamine and its link to positive symptoms in a meta-theory of schizophrenia. For this enterprise we need more building blocks and more possible ways to combine them. Crucial within this framework is that the positive symptoms themselves are looked at from another angle.

The positive symptoms may be very bizarre and hard to follow, but perhaps they are not the core of the schizophrenic problem. Yet they are usually the reason why a schizophrenic patient is sent into treatment, because they seem out of their mind. One very important focal point still for most psychiatrists is to eradicate these positive symptoms, which may not be necessary. It can't be or at least it shouldn't be the goal to 'normalize' schizophrenics, unless it is their own goal in life. Most patients are only hampered by their hallucinations and/or delusions if they are threatening or depressive in nature, or if these cause social strife in interpersonal interaction. To me, trying to annihilate them because they are the symptoms par excellence of a sick mind is missing the point at best, unethical at worst.

## 2.2 Cognitive deficits: the broader perspective, or is it?

In recent decades a lot of research has gone into the cognitive functioning of schizophrenic patients (Brazo et al, 2002). This has lead to the idea that the cognitive

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affinity for 5-$HT_2$, $D_2$, $M_1$, and/or $H_1$ receptors. With the discovery of multiple subtypes of dopamine receptors, recent efforts have focused on the development of $D_3$ and $D_4$ antagonists. $D_3$ receptors are found at higher levels in the limbic system than in the striatum, suggesting that $D_3$ antagonists might be useful neuroleptics with a reduced incidence of extrapyramidal side effects.

[9]Dopamine pathways project from the mesencephalon (ventral tegmental area) to the frontal cortex (mesocortical projection, $D_1$ group) and to the cingulate cortex, amygdala, lateral septum and the olfactory bulbs (mesolimbic projection, $D_2$ group). These pathways seem to control cognitive function and emotion and give rise to the common symptoms of schizophrenia.

deficits in schizophrenia are not immediately linked to the positive and negative symptoms, but that they are a category all by themselves.

The most common cited deficits are in working memory, attention/vigilance, verbal learning and memory, visual learning and memory, reasoning and problem solving, speed of processing and social cognition.[10] All of these seem severely damaged in schizophrenia. Furthermore, research in longitudinal studies and twin research has shown that the illness itself is detrimental to cognitive functioning (Bressler, 2003; Harvey et al, 1997). Schizophrenic patients perform worse -cognitively speaking- after the onset of the disease than before and an affected twin scores lower on cognitive tests than the unaffected twin (Goldberg, 1990). Overall, this has lead to the conclusion that schizophrenia is also a cognitive *disease*, that a psychotic breakdown is neurotoxic even and that schizophrenic subjects are handicapped because of their erroneous psychotic convictions and cognitive decline (Heinrichs et al, 1998). I personally believe this statement is rather blunt, verging on academic and clinical naivety. Two biases greatly influence this debate.

The first, which I would like to call the *normative bias*, is the view that schizophrenia is a deficit in and of itself. It is only a deficit when compared to 'normal' individuals, and who makes up these norms and for what reason? This approach starts off from the accepted idea that the brain is a normative thing, and that certain differences in cognitive brain processes are transparently linked to mental aberrations or social inadequacies. Anyone truly looking at brain functioning knows that there are a million reasons why a person could be dysfunctional in certain situations. From my own therapeutic experience with these patients, I must say that it seems completely biased, you need a better picture at the interactions between these specific deficits and their importance in certain social or psychological situations, not just in test situations based on some kind of normative benchmark.

The second bias I call the *psychiatric bias*, is that the people usually included in this research are patients in institutions, people who have come into trouble because of certain problems in their social and/or mental functioning. This is a very specific subgroup of what is probably a large population of people with a certain psychotic vulnerability. Only those caught up in the psychiatric web are seen as *the* schizophrenic population, where this is probably not true as is shown in numerous epidemiological studies (Stefanis et al, 2002; Johns et al, 2001). Moreover, what is commonly labeled as the schizophrenic disease is probably not a homogeneous group of patients (Weiser et al, 2005). Again, there are a lot of reasons (social, psychological, neurological) how someone could come to have the positive and/or negative symptoms of schizophrenia and maybe some schizophrenics do not even have these symptoms as such (Myin-Germeys et al, 2003).

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[10] as measured with the Repeatable Battery for the Assessment of Neuropsychological Status (RBANS), The Brief Assessment of Cognition in Schizophrenia (BACS), or as stated in the Measurement and Treatment Research to Improve Cognition in Schizophrenia (MATRICS) project: Speed of Processing, Attention/Vigilance, Nonverbal attention,Verbal Learning and Memory, Visual Learning and Memory, Reasoning and Problem Solving and Social Cognition (http://www.matrics.ucla.edu).

Concluding this lengthy introduction to schizophrenia, let's sum up our main pretences and focal points.

Right of the bat, schizophrenia is *not a disease*. It is a complex structure and anyone truly looking at this problem should feel humbled by the degree of its complexity.

Second, schizophrenia is *different* and should be understood in its own right, according to its own criteria. It is useless to impose some kind of normative approach which might lose track of the problem at hand, trying to understand schizophrenia and schizophrenic patients in particular.

Third and last, schizophrenia is *not* at all *a stable or fixed problem*. Symptoms change over time as people change and adapt over time. When dealing with schizophrenia, we should always remember that we are talking about people in interaction with their environment, trying to make the best of things. We need a viewpoint which takes change and chaos into account. Complex anticipatory models do just that, as we will try to show in part 4.

## 3 The anterior cingulate cortex: knowing when you're wrong?

Numerous scientific studies with schizophrenic and other patients have shown the functioning of the Anterior Cingulate Cortex is noteworthy. The ACC (BA 24-32) is a part of our neocortex, on the medial surface of the frontal lobes, right over the corpus callosum. At first it was thought that it was actually not a part of the neocortex at all, but an older subcortical part of our limbic system (Allman, 2002). More recent research has shown that this is not the case.

The ACC is richly interconnected with a lot of other functional areas.[11] The most important are the prefrontal cortex (PFC), notably the dorsolateral prefrontal cortex (DPFC)[12], the (pre)motor cortex (PMC), the Parietal Cortex (PPC) and subcortically with the thalamus, the amygdala, the hippocampus and the cerebellum (Wang et al, 2004; McDonald et al, 2000).

EEG and fMRI studies have shown that the ACC consists of two distinct functional parts, the Caudal ACC and the Rostral ACC, where the Caudal ACC is more involved in cognitive functions, whereas the Rostral ACC is more involved when it comes down to emotional appraisal as such (Laurens et al, 2003; van Veen et al, 2002, ). Further data suggest that these two parts are richly interconnected, which leads to the hypothesis that the ACC is partly responsible for linking cognition and affect (Fan et al, 2003).

Now we know a bit more as to where the ACC is located and what it is linked to, what is its function? In the early '90s both Falkenstein and Gehring, using ERP

[11] In the ACC of humans and the great apes spindle neurons were found. These distinct neurons are probably projection neurons, sending and receiving stimuli from a wide array of other brain areas. These spindle neurons are dopamine receptive in nature, are only visible after the first four months after birth and they seem to announce the development of the maturation enhancements of attention. It wouldn't surprise me if in these spindle neurons the clue could be found as to where the developmental differences in schizophrenia could be found (Nimchinsky et al, 1995).

[12] Space limitations force us to leave the link between the ACC and the DPFC in the dark, although I cannot begin to stress the relevance of this enterprise. Combined they provide us with an explanatory framework beyond measure (Mc Donald et al, 2000).

research, discovered that a certain negative brainwave pattern occurred in the EEG when a subject performs a mistake in an experimental task.[13]

This pattern has been dubbed the error negativity ($N_e$) or Error Related Negativity (ERN). It seems to stem from the frontocentral part of the brain, near the ACC. This has lead to the hypothesis that the ACC is involved in action monitoring, more notably when errors have been performed.

From this point on a whole corpus of literature has been established to further extrapolate on this point and to bring some of these rather conflicting data results (from ERN and fMRI results) within a logical framework (van Veen et al, 2002b; Mathalon et al, 2003). Some say that the ACC is an action monitor for errors, providing information to other areas; other see it as a top-down control area in itself (Liddle, 2001; Botvinick et al, 2004; Erickson et al, 2004; Carter et al, 1998; Paulus et al 2002; Luks et al, 2002; Fan et al, 2003). They might just both be right, as we will come to see later on.

From the viewpoint of schizophrenia research, the ERN and the ACC functioning have received a lot of attention too, because the ERN pattern does *not* occur when schizophrenic patients make an error in an experimental task, at least not in the same way as 'normal' subjects (Nordhall et al, 2001). This, taken together with the popularisation of the ERN-ACC link as the brain's 'Oops' function has lead to the psychiatric cognitive hypothesis that schizophrenics don't know that they make a mistake, that they don't feel it as such. In short, schizophrenics are social error prone misfits and the ERN-ACC dysfunction explains it. Speaking to any schizophrenic patient will show you that this caricature hypothesis is flawed at best. They don't react on the spot, but the onslaught of their delusional reactions comes later on (see part 5).

Any delusional story they tell you is a testimony to the fact that they react *differently*, but that they react nonetheless, albeit not in the way we 'normal' people usually do. At the core of any delusion lies a erroneous conflict within the subject or with its environment, a place where a certain solution was expected, even longed for, but not presented as such, so they invent their own solution.

What is wrong then with the ERN-ACC hypothesis towards schizophrenia? First, the *reductionist interpretational context* is flawed. When you look at the brain as a problem solver, you can never look at it as if it is a linear computing machine. Although the ERN in the ACC is a very salient feature of our brain functions and of the difference in brain processing in schizophrenia, it is and remains a complex issue that cannot be understood too simplistically. The ACC is a very complex part of our very complex brain. The ERN not occurring after making a mistake does not lead to the conclusion that schizophrenics are not able to see faults on the spot, the only allowed conclusion according to me is that their brain reacts in another way, as is shown in ERP and fMRI research. In recent years an entire wealth of other features of the ACC have been discovered, most notably its role in mediating between cognition and affect, its role in reward dependent tasks, its role in the inscription of data in long term memory, the distinction in self/not-self in relation to the mirror neurons et cetera.[14] (Arbib et al, 2005; Lane et al, 1997; Wang et

[13] http://www-personal.umich.edu/~wgehring/intro.html

[14] A very recent study by Fellows and Farah (2005) has even shown that the ACC may not be necessary for cognitive control at all in people with cerebral damage to the ACC itself. Their problems are more

al, 2004; van Veen et al, 2002, 2002b) The ERN (dys)function should take these other features and the interrelations between these new discoveries into account, especially when studying schizophrenia. The brain is not a computer and schizophrenics aren't robots, although some of them believe otherwise themselves.

Second, the *relativistic temporal context* interaction is biased. The ERN seems to occur only after an error has been made, but some data suggest that the ERN also occurs when mistakes are likely to occur, in anticipation (Luks et al, 2002). Furthermore, Error rate and outcome predictability affect neural activation in prefrontal cortex and anterior cingulate during decision-making, which point at the role the context plays for certain specific brain phenomena (Erickson, 2004). You could call this *the error prone environment interactions*. Furthermore, the N2 pattern –also frontocentral, probably also stemming from the ACC- occurs prior to a correct conflict resolution in a task, where the ERN is usually shown after an error has occurred (van Veen et al, 2002b). The interactions between both are not well understood, nor studied.

To me, it is fascinating that both functions –anticipating success and (anticipatory and posterior) reacting to error- are located in the same part of the brain as processing attention, memory, motivation and emotional appraisal. It makes more sense that our brain should look at upcoming events from a relevant point of view *for the system itself*, taking into account previous reactions and possible future consequences (see part 4). What are the consequences of my (re)actions for me as a subject, what are my feelings towards this?

We are not built to perform well in relation to irrelevant stimuli, we are built to survive in an error prone environment with dire consequences if we falter or fail. Recent research into pain shows that the expectation of pain (for ourselves or for others) causes massive innervation in the ACC, which goes to show (Buchel et al, 2002; Jackson et al, 2005).

## 4 The ACC anticipation hypothesis: doubting that you're right!

What is wrong with the model up until now? Almost everybody looking at the working of the ACC and the role of ERN in it takes a very matter of fact attitude towards the problem: if the ERN occurs, then we know that we have made an error. In reality, it is not as easy. We need a broader context and a mapping of the subject environment interactions.

*My first hypothesis*: The ACC and its function is actually that part of the brain which evaluates differences in the constant temporal flux which makes up our subjective awareness. It seems that our brain has the capacity to distinguish between correct and incorrect information and to evaluate its own role in it.

From a psychoanalytical point of view, this function is crucial. In 1925, Freud wrote a very peculiar short text on this subject, and Lacan throughout his work has always hammered on the importance of our capacity to negate information and our own role in

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linked to motivational problems and reward expectancy. How to explain that from a reductionist viewpoint of the ERN?

it (Freud, 1925h). Alongside the ideas of repression and the discovery of the unconscious complex phenomena, negation is seen as a crucial human capacity, both for subjective development as for our use of language (Ver Eecke, 2006 ).

*My second hypothesis:* I personally believe that the ACC is the locus in our brain where the distinction between (pre)conscious affirmation (bejahung) and conscious negation (verneinung) over time is made. According to me, it is all the ACC might be doing, it takes possible error into account, it tells us that we may be right, but we might be wrong. Some have compared the function of the ACC to an exclamation mark, the brain's 'Oops!' or 'Damn!' function. I on the other hand would compare it to a question mark, the 'Huh?' or 'what the…?' function.[15]

How does this relate to what we have discussed so far? In a recent article Quintana et al (2004) investigated the ACC dysfunction in choice anticipation in schizophrenia. They discovered that in 'normal' subjects the ACC activation was high in response anticipation, but not when these subject had to remember a certain percept. While in schizophrenic subjects it was the other way around, no significant ACC activation in anticipation, activation when remembering a percept.

They also discovered that there was a significant influence in this process if the anticipated or remembered percept was a colour dot or a face diagram. In anticipation with the normal subjects the colour dots yielded more activation than the faces. In remembering in schizophrenia the facial diagrams resulted in more activation than the dots, which leads to the speculation that the saliency and the relevance of the stimulus is also important (irrelevant colour dots or symbols of expressed emotion in the face diagrams). Because the results from this experiment were obtained with fMRI, it is impossible to tell if this activation was a $N_e$ or an N2 wave pattern. We can only say that there was a distinction in activation as such.[16]

Nevertheless, speculating on the information thus far, I would like to propose a third hypothesis concerning the ACC, negation and anticipation. In a previous paper, I made a distinction between three forms of anticipation: real, imaginary and symbolic (De Grave, 2004).

*Real anticipation* is to be seen as the drive, the libido or the state of bodily arousal in expectation. The body prepares itself for a future event. *"I feel A, so B must be coming on." Imaginary anticipation* is the expectation of a certain percept following another percept, based on a previously learned link between the two. *"If A was first seen and B followed, then you anticipate that after the presentation of A, B will follow."* Imaginary anticipation is usually the one people hint at when using the word anticipation. The hard one to grasp is *symbolic anticipation*, which is the ambiguous subjective interpretation of what happens now, and what possible future states this might lead to without the immediate certainty of what the future is going to be.

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[15] Since the ACC is also the mediator between affect and cognition, the role of distinguishing between right and wrong might have far-reaching consequences if we add these two functions up. Knowing what is right or wrong is also a bodily state, an emotional appraisal.

[16] Throughout almost all ACC investigation, we see a clash between results from fMRI and ERN. Few studies combine the two.

*"If A is the case, then …?"* Symbolic anticipation starts off from lack of certainty about future states and is based on conventions, beliefs and convictions, derived from social symbolic interactions with others. If A is the case, then we all agree upon the fact that B should follow.

Language itself is probably the best example of symbolic anticipation. No signifier or word means anything in and of itself, it only generates its meaning through the context of other signifiers and subjects uttering them. Symbolic anticipation is the anticipation of certainty of meaning over time, with a lack of certainty at this moment in time. To me, it is the true and exclusively human complex form of anticipation. It is symbolic anticipation which makes up our common sense view of reality, which is nothing short of believing that you know what to expect based on social interaction. (Lacan, 1945).

*Third hypothesis:* The ERN is the feeling (real) that something which a subject sees or does (imaginary) does not lead to the anticipated result (symbolic).

Combining these three forms of anticipation, I speculate that the ACC might be the anticipatory interface where the real, imaginary and symbolic are intertwined to create the whole of existential anticipatory awareness. It entails being a certain way at a specific point in time, in relation to our remembered past and anticipated future. Some have named the ACC the interface between emotion and cognition and I think that this is true if we add that it links emotion and cognition into a meaningful event *over time*.

Mostly, this is a process we are not aware of, since mostly our beliefs and expectancies are seemingly correct. We only notice its function when it goes wrong, and that is where the ERN in 'normal' subjects comes into play. Of course, this hypothesis is overly speculative, but I believe it could result in a few novel avenues of thought, experimental research and clinical applications.

## 5 The collapse of reality: schizophrenic anticipation and delusion

Experimental research has clearly shown that the brain wave pattern representing the ERN function in the ACC in schizophrenia is mostly blunted. According to me, this doesn't say anything in its own right besides the fact that in schizophrenia, the brain react differently when making a mistake than in 'normal' individuals. But it is important nonetheless.

From a Lacanian diagnostical point of view towards psychosis and schizophrenia, the diagnosis is formed on the basis of certain language disorders, certain peculiar social ties, a special way of experiencing the body and the bodily states (Maleval 2000a,b). All this is tied up in a theorem developed by Lacan named the forclusion of the primordial signifier (Name-of-the-Father) (Lacan, 1957; Maleval 2000a). Although it is a very difficult theorem to understand, for this paper it is only necessary that Lacan stated that for psychotic subjects language works in a different way than in neurosis, based on another existential position towards the Other (De Grave, 2006).

In other words, what is important here is that for psychotic individuals language is not imbedded in the common sense idea we all share that words represent what we believe them to represent. A psychotic subject doesn't accept language as the shared belief system, it is forcluded (Freud named this verwerfung, a function next to

verleugnung and verneinung (Maleval, 2000a; Lacan, 1957; Freud, 1911c, 1918b, 1924e, 1940e)).

This has far-reaching consequences, since everybody needs to be able to communicate in life to survive, be able to remember past errors to learn from them and to know what to expect from the future so as not to be fixed in a harrowing unpredictable universe.

From a psychoanalytical point of view delusions in schizophrenia function as these ersatz anchor points, ways to distil a certain logic out of the events that happen in and around a psychotic individual (Maleval, 2000b). They are delusional because they are not tightly caught up in the network of symbols we all know as our shared reality. Delusions are a highly personal logical framework where things don't fit in.

To me, schizophrenic delusion should be understood as a form of direct interpretation. You think something and it is true, you feel something and it is true. There is no middle ground for taking the possible erroneous character of your beliefs and convictions into account.

We all know that our common sense reality is a lie or a fantasy up to a certain degree, words don't mean anything as such for example. But it is not strictly speaking our own lie, we didn't invent the words, they are just borrowed. So we play along and pretend that it is true. It seems to work fine. But if your perception of reality is not based on this common sense lie, you have nothing to fall back on. This is exactly the problem in schizophrenia according to me.

If we take the functioning of the ACC and the lack or blunting of ERN into account in this matter, we must say that schizophrenics may not be aware of mistakes as we are, but that is actually very logical if we look at it from the psychoanalytical point of view on delusions. They live in another symbolic universe than we do, with other rules and laws. Although they try to cope with the stresses everyday life imposes on us all, they take a very different approach to this. A lot of the time, they just do what is asked of them, because it is asked. In this, motivation for acts, even in experiments, is almost entirely external. This does not make it impossible to communicate with each other if we accept that sometimes we cannot understand each other.

To me, plain and simple as a psychoanalyst, it is clear that in my everyday work with schizophrenic patients that they are not capable of anticipating a possible future as I am. They long for and they fret a different future which is not my own. They react differently. When they come unto our ward in a state of anxiety they are clearly different. Neuroleptics as dopamine antagonists do their work at stripping away their awkwardness (positive symptoms). I am not against medication, quite the contrary, but in giving medication that alters someone's built-in expectation schemas, we should be very careful, maybe we are taking away someone's own personal even intimate reference manual to understand the world and their own role in it. (Zirnheld et al, 2004) If you know that the ACC's primary neurotransmitter is dopamine, you should think twice when administering a high dose of dopamine antagonists to solve the problem (Suhara et al, 2002; Nordhal et al, 2001). You might just be taking away a person's capacity to anticipate any future, delusional or not.

Clearly, schizophrenics on the whole mostly do not know on a conscious level why they have performed the acts that have brought them into trouble in the first place, they often don't see the problem. But their delusional framework is a testimony to the fact that they do react very sensibly, even on the spot, but *differently*. They are busy trying to piece together the missing parts, the things other people tell them are out of order, unhinged, unbecoming in *that* place, at *that* time.

To the outside world it seems as if time and logic have collapsed into nonsense, to them all forms of understanding have imploded into the magnificent insight which makes up their delusion. In this imploded reality everything is linked with everything, nothing can be seen loose from another fact. In all patients I have had the pleasure to speak to in length, this element of implosion of reality is present as a strange attractor in their delusional framework (Bazan et al, 2003).

In such a collapsed universe it is difficult to see mistakes or error, because there is always a reason why the thought or the action *could* be right. Schizophrenics are caught up into an self-referential here and now with few possible ways of negating what is going on inside their mind (Ver Eecke, 2006). It is up to us as therapists to stretch this delusional form of all too direct interpretation out into an debatable temporal form of cognition, by standing next to them and placing the punctuation marks in the debate about their delusion. It is not that their delusions are wrong per se, the fact is that they *could* be wrong, just like any other thought anybody might have. In this we are all equal and in this we can find a middle ground between 'normality' and schizophrenia. The work with my patients heartens me that this is a possible endeavour.

## 6 Conclusion: delusions of grandeur

In this paper I have argued against a very strict interpretation of the ACC dysfunction in schizophrenia. The ACC clearly does something different in schizophrenia, as is shown in the blunted ERN, but that does not mean it doesn't work, or that its function is abnormal. It all depends on the reference background as to what you expect the ACC to be doing in a certain case.

With my speculations concerning delusional anticipation and the ACC, I may be dead wrong. It remains to be proven that there is actually a link between the two. All I am saying is that it is an interesting avenue of thought and possible experimentation. For this enterprise, you need to respect the delusions for what they might be, not just a symptom of a severely diseased brain, but an alternative reaction to a certain stimulus over time, another form of symbolic anticipation.

As a therapist it is my job to try and better understand my patients, so as to be able to try and help them better. A normative stance doesn't fit into this picture, condemning because of their frame of mind is wrong. In this paper I have tried to show that this mind set of respecting the formations of the mind for what they are, might prove beneficial in other fields of dealing with schizophrenia: psychiatry, neurology, linguistics,… The relatively young branch of science named neuropsychoanalysis should take it upon itself to bring the qualities of psychoanalysis to the fore within the field of neuroscience, as a form of critique, but also as a clinical guideline where

interesting fields of research are waiting to be discovered. I hope that I have conveyed some of the fervour in this project concerning the ACC and schizophrenic anticipation/delusion.

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