

How Does Our Brain Constitute Defense Mechanisms? First-Person Neuroscience and Psychoanalysis

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Key Words

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Abstract

Current progress in the cognitive and affective neurosciences is constantly influencing the development of psychoanalytic theory and practice. However, despite the emerging dialogue between neuroscience and psychoanalysis, the neuronal processes underlying psychoanalytic constructs such as defense mechanisms remain unclear. One of the main problems in investigating the psychodynamic-neuronal relationship consists in systematically linking the individual contents of first-person subjective experience to third-person observation of neuronal states. We therefore introduced an appropriate methodological strategy, ‘first-person neuroscience’, which aims at developing methods for systematically linking first- and third-person data. The utility of first-person neuroscience can be demonstrated by the example of the defense mechanism of sensorimotor regression as paradigmatically observed in catatonia. Combined psychodynamic and imaging studies suggest that sensorimotor regression might be associated with dysfunction

in the neural network including the orbitofrontal, the medial prefrontal and the premotor cortices. In general sensorimotor regression and other defense mechanisms are psychoanalytic constructs that are hypothesized to be complex emotional-cognitive constellations. In this paper we suggest that specific functional mechanisms which integrate neuronal activity across several brain regions (i.e. neuronal integration) are the physiological substrates of defense mechanisms. We conclude that first-person neuroscience could be an appropriate methodological strategy for opening the door to a better understanding of the neuronal processes of defense mechanisms and their modulation in psychoanalytic psychotherapy.

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Introduction

The term ‘defense mechanisms’ was coined over 100 years ago to describe a construct of psychological mechanisms for coping with intrapsychic conflicts [1] (table 1). Defense mechanisms and conflict are two hypothetical constructs that have remained at the core of psychodynamic approaches to understanding and treating clinical

Table 1. Defense mechanisms: hierarchy of mature/cognitively oriented and immature/emotionally driven mechanisms of defense according to psychoanalytic theory

<i>Mature/cognitively oriented mechanisms of defense</i>		<i>Immature/emotionally driven mechanisms of defense</i>	
Intellectualization	Dealing with emotional stressors by excessive use of abstract thinking or complex explanations to control or minimize disturbing feelings.	Somatization	Dealing with emotional stressors by physical symptoms involving parts of the body innervated by the sympathetic and parasympathetic systems.
Rationalization	Dealing with emotional stressors by inventing a socially acceptable or logical reason to justify an already taken unconscious emotional action.	<i>Derivatives of self/nonself loss of boundaries</i>	
Repression	Moving thoughts unacceptable to the ego into the unconscious, where they cannot be easily accessed.	Dissociation	Temporary and drastic modification of one's self-image to avoid emotional distress. Disconnection from full awareness of self, time and/or external circumstances. Often connected with childhood trauma and posttraumatic stress disorder.
Displacement	Dealing with emotional stressors by redirecting emotion from a 'dangerous' object to a 'safe' object.	Projective identification	Repeated cycle of projection and introjection: hateful impulses are projected onto the significant other who becomes the bad object. Some of the bad impulses are still retained in the self; they are reinforced by taking into one's self, introjecting, what has originally been projected onto the object.
Isolation	Dealing with emotional stressors by splitting off the emotional components from a difficult thought. The mechanism of isolation is commonly overutilized by people with obsessive-compulsive personalities.	Psychotic introjection	Psychotic internalization of the object to overcome overwhelming anxieties of loss.
Reaction formation	Dealing with emotional stressors by converting an uncomfortable feeling into its opposite.	Psychotic projection	Hallucinatory and paranoid externalization of inaccessible thoughts and their connected affects.
Identification	Occurs in various stages of development, in particular in its role as an intrinsic part of object relationships. Serves the function of structure building and makes it possible to deal with separations from loved objects. Plays a role in some types of conversion.	Splitting	Splitting off and rejecting parts of the object image and/or of one's own body.
Identification (with the aggressor)	By becoming an aggressor towards others, one avoids becoming a victim of aggression.	Fragmentation	Reflects a primitive stage in psychic development, preceding the formation of part self and part object images. Breaking up of the self or the object image into components which may operate independently.
Idealization	Dealing with emotional stressors by overestimating the desirable qualities and underestimating the limitations of a desired object.	Denial	Dealing with emotional stressors by failing to recognize obvious implications or consequences of a thought, act or situation.
Introjection	Dealing with emotional stressors by internalizing the values or characteristics of another person; usually someone who is significant to the individual in some way.	Catatonia	Psychomotor syndrome showing a specific constellation of affective, behavioral and motor symptoms. Sensorimotor regression reflecting an immature mechanism against the uncontrollable overflow of anxieties.
Projection	The opposite of introjection. Attributing one's own emotions or desires to an external object or person.	Autism	Extreme withdrawal and avoidance of contact and interpersonal relationships to overcome overwhelming anxieties of losing one's own self when near the object.

psychopathology. From a psychoanalytical perspective, defense mechanisms mediate between an individual's wishes, needs and affects on the one hand, and both internalized object relations and external reality on the

other. Through specific constellations of affective and cognitive function, defense mechanisms help resolve conflicts, whether triggered by internal or external stressors. One could therefore hypothesize that defense mech-

anisms are complex emotional-cognitive constellations. As such, the recent progress in affective and cognitive neuroscience [2–5] raises the question of their underlying neuronal processes, which, in turn, could also contribute to a more refined and detailed definition of the construct of defense mechanisms. However, the dialogue between psychoanalysis and neuroscience has only recently emerged [6–21].

The reception of neuroscience by psychoanalysts ranges from strong skepticism to equally strong enthusiasm. Skeptics [22–24] doubt that there can be common conceptual grounds for linking the hypothesis about unconscious aspects of the mind (as advanced by psychoanalysis) and knowledge about the brain (as framed by neuroscience). They claim that the complexity and richness of subjective human experience can become lost in empirical neuroscientific investigation. Another argument, as put forward by Gruenbaum [25], is that possible psychodynamic processes cannot be attributed causal relevance in the same way as other scientifically investigable processes. This makes it rather difficult to empirically test and validate psychodynamic processes such as defense mechanisms. Moreover, skeptics have recently argued that it remains impossible to associate the complex subjective experience with neuronal activity in specific brain regions; they thus argue against neuronal localizationism of psychodynamic processes [22]. Our answer to these challenges is the following.

Those endorsing the integration of psychoanalysis and neuroscience usually refer to Freud's 1895 project, in which he sought a unitary conception of mind and brain [26]. Kandel [10] recently articulated the hope 'that biology might reinvigorate the psychoanalytic exploration of the mind'. He emphasized the need for combined research approaches in psychopathology (implicit and explicit memory, development, etc.) and psychotherapy [17, 27–29]. This led some authors to investigate the neuronal mechanisms of psychodynamic processes. For example, emphasizing the developmental features of the right orbitofrontal cortex, Schore et al. [7, 8] propose neurobiological mechanisms of unconscious processes such as projective identification. Based on psychoanalytic treatment in patients with orbitofrontal cortical lesions, Solms [6] and Solms et al. [30] make inferences about the localization of early, immature and somatic defense mechanisms. Furthermore, Northoff et al. [20] were able to link sensorimotor regression, as observed in catatonic patients, to a complex neural network including the orbitofrontal, medial prefrontal and premotor cortexes (see below for details). The orbitofrontal cortex also plays a role

in emotional-cognitive interaction, which Westen and Gabbard [13, 14] consider to be crucial in conflict and compromise. However, despite these studies and the crucial role of defense mechanisms in psychoanalytic theory and practice, their underlying neuronal mechanisms remain yet to be explored.

One of the main methodological challenges in investigating the neuronal processes underlying defense mechanisms is to link first- and third-person data. Being based upon subjective experience, psychoanalysis relies on first-person data. This contrasts with neuroscience, which requires third-person observation of neuronal states. Due to the neglect of subjective first-person experience, neuronal states as third-person data can be quantified and objectified. This, in contrast, remains impossible in the case of first-person data (see below for discussion of potential criticisms of the concept of first-person data), which are rather qualitative and subjective. If, however, the neuronal processes of defense mechanisms are to be investigated, subjective experience and neuronal states (i.e. first- and third-person data) have to be linked to each other in a systematic way. For this purpose we created an appropriate methodological strategy, 'first-person neuroscience', which aims at systematically linking first- and third-person data (see below for a discussion of the distinctive features of first-person neuroscience when compared to neuroscience and psychology as usually practiced). After describing first-person neuroscience, the utility of this methodological approach will be paradigmatically demonstrated by means of the example of sensorimotor regression as observed in patients with catatonia.

Considering sensorimotor regression and other defense mechanisms, it becomes clear that first-person neuroscience requires a shift from neuronal localization to neuronal integration: instead of localizing particular defense mechanisms in specific brain regions, we hypothesize that defense mechanisms might rather correspond to specific functional mechanisms of integrating neuronal activity across several brain regions. We conclude that the methodological approach of first-person neuroscience in association with neuronal integration might eventually open the door to greater knowledge about the neuronal mechanisms of the various defense mechanisms and their modulation in psychoanalytic psychotherapy.

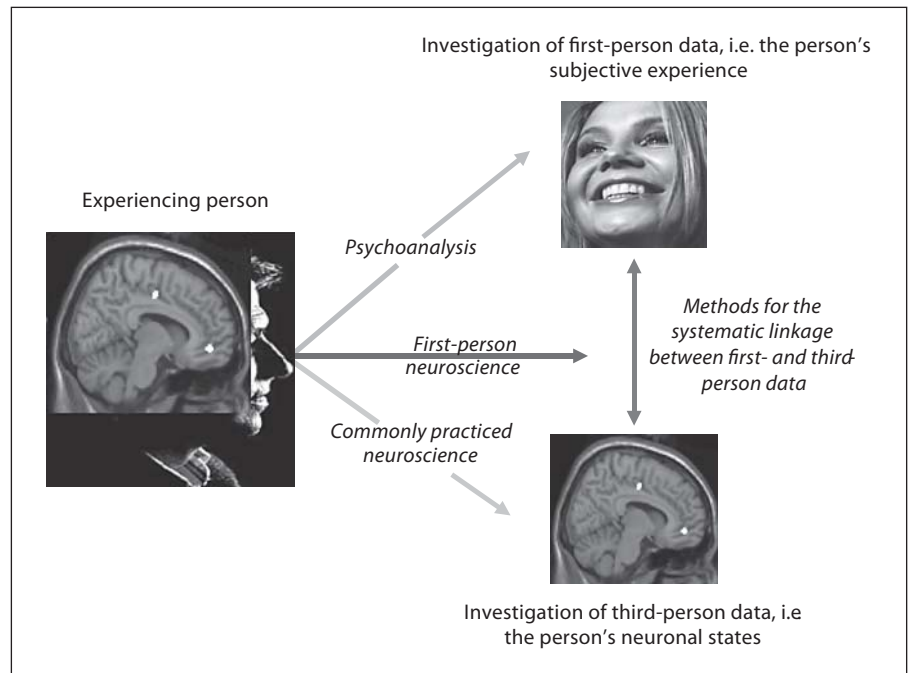


Fig. 1. Psychoanalysis and neuroscience: linkage between subjective experience and neuronal states in first-person neuroscience.

What Is First-Person Neuroscience?

Definition of First-Person Neuroscience

We define ‘first-person neuroscience’ as a methodological strategy to systematically link subjective first-person experience¹ to third-person observation of neuronal states [3, 32–34]. The development of such methods distinguishes first-person neuroscience from neuroscience as it is commonly practiced, which most often relies on third-person observation of neuronal states more or less independently of subjective experience (fig. 1). First-person neuroscience should also be distinguished from psychology. Though psychology considers data from the first-person perspective as well, for example in the case of emotional feelings, as in the case of third-person neuroscience, it too relies on third-person observation of psychological states. This means that the individual contents of psychological states are lost. Such generalization independent of individual contents presupposes what has been called the ‘nomothetic approach’. In contrast, first-

person neuroscience, as we understand it here, aims at preserving the individual contents of the psychological states, as obtained from the first-person perspective – this has been called the ‘ideographic approach’. The difference between first-person neuroscience on the one hand and third-person neuroscience and psychology on the other thus consists in the difference between the ideographic and nomothetic approaches, i.e. in whether the individual contents are preserved or not.

The main challenge in establishing first-person neuroscience, however, does not only consist in pursuing an ideographic approach to account for individual contents as subjectively experienced in the first-person perspective. An even more demanding challenge is to link the individual contents of subjective experience to neuronal states. How can we link subjective experience to neuronal states? The linkage between subjective experience and neuronal states requires two steps: (1) subjective experience needs to be evaluated systematically, including objectification and quantification of subjective data – such ‘science of experience’ is a necessary precondition for any linkage between subjective experience and neuronal states; (2) the systematically objectified and quantified subjective data then enable the linkage to analogous data about neuronal states. For this, special methodological strategies need to be developed – this is the core of what we call ‘first-person neuroscience’: ‘It would be futile to

¹ It should be noted that this also includes introspection of the individual’s own subjective experience. Introspection of subjective experience has been linked to the second-person perspective, as distinguished from subjective experience itself in the first-person perspective [31–33]. However, for pragmatic reasons we refer to introspection and second-person perspective under the terms ‘subjective experience’ and ‘first-person perspective’, using both in a broader sense.

stay with first-person descriptions in isolation. We need to harmonize and constrain them by building the appropriate links with third-person studies. ... To make this possible we seek methodologies that can provide an open link to objective, empirically based description' [35, 36]² (fig. 1).

Science of Psychodynamic Processes

The systematic examination and evaluation of subjective experience must preserve its richness and complexity on the one hand and objectively quantify its main characteristics on the other³. The objectification and quantification of subjective first-person data allows for scientific investigation and consequently for establishing what can be called a 'science of experience' [40]⁴. Based on a science of experience, a 'science of psychodynamic processes' needs to be developed. The science of psychodynamic processes should place great emphasis on patients' mental life or inner experience in order to preserve the richness and complexity of subjective experience and clinical description [12, 41]. At the same time, these subjective features must be objectified to provide reliable and quantifiable data. This can be achieved by asking the subjects to complete rating scales. For example, visual analog scales [33, 34, 42] with regard to personal identity or idiographic instruments like the Repertory Grid test (see below for further details) might be applied to let the subjects themselves evaluate their experiences. One might also apply structured interviews with valid and reliable instruments for the evaluation of the subjects' relevant psychodynamic features by an experienced investigator. General instruments include, for example, the Karolinska scale that assesses different psychodynamically relevant dimensions of a person's structure [43, 44]. Another

instrument is the Operationalized Psychodynamic Diagnostic (OPD; Arbeitskreis OPD 2004), which, based on ICD and DSM, develops five psychodynamically relevant axes (illness experience and expectations, relationship, conflict, structure, and psychic and psychosomatic disturbances). More specific instruments are scales for quantifying defense mechanisms, e.g. the Defense Mechanism Rating Scale [45, 46] or the Defensive Style Questionnaire 40 [47, 48], and psychodynamic psychopathology in personality disorders (Shedler-Westen Assessment Procedure 200 [49, 50]). The functional status of different psychopathological phenomena as mechanisms of defense and compensation has been highlighted by several former studies (e.g. [51–55]).

A potential point of criticism of such science of psychodynamic processes, relying on ideographic approaches, is that the individual contents from the first-person perspective cannot be considered 'data' in the way that this term is understood in science. For example, Metzinger [56] argues that the concept of data implies their generalizability to groups, their possible validation in an intersubjective process, their extraction by technical measuring and their linkage to public procedure. He subsequently considers first-person data, the individual contents as subjectively experienced from a first-person perspective, as a fusion of 'semantic elements with a concept borrowed from the theory of science' [56]. However, we do not consider this argument as applicable to the science of psychodynamic processes for the following reasons: individual contents which are central in psychodynamic processes can indeed be generalized to groups by comparing the similarities and differences of individual contents between different individual subjects. One of the main features of psychodynamic theory is that it was able to reveal the same individual contents and structures of psychological processes across different individuals. As the above examples of operationalization demonstrate, it is quite possible to validate and objectify individual contents in psychological processes. Though not requiring technical measures, it can be considered a public procedure. Thus, in view of these arguments, individual contents of psychological processes can be understood as data in the scientific sense. This enables us to refer to first-person data characterizing the subjective experience of individual contents. The purpose of our present paper is not to create new methodological approaches but to combine existing ones in such a way that it is possible to link first-person data, characterizing individual contents of subjective experience, and third-person data, accounting for neuronal states. We believe that this could not

² If the 'science of experience' is not related to the 'science of observation' and thus neuroscience, the former degenerates into 'first-person mentoscience', which is occupied with mental states exclusively, without any relation to neuronal states [32, 33, 37].

³ An interesting development in this respect is the emergence of what has been called 'clinimetrics', as distinguished from psychometrics. Clinimetrics arises from clinical observation, clinical methods and clinicians in its attempt to link clinical phenomena and subjective experience [38, 39]. In contrast psychometrics incorporates methods where all variables have the same weight.

⁴ Varela and Shear [35, 36] also refer to 'first-person methodologies' in this context. The difference between first-person methodologies and our concept of first-person neuroscience is that only the latter aims at developing methods to systematically link first- and third-person data, whereas the former concerns only the quantification and objectification of subjective experience, i.e. of first-person data, thus aiming at the development of what we called 'science of experience', as distinguished from neuroscience as 'science of observation'.

only provide insight into the underlying neuronal states but also reveal, diagnose and possibly define psychodynamic processes in relation to specific individual contents more clearly and in more detail. This, however, remains a scenario of the future.

To avoid misunderstandings, it has to be pointed out that we do not intend to handle defense mechanisms as if they were facts in the same way as, for example, assumptions about neuronal states. We consider defensive reactions as constructs⁵ which need to be investigated empirically, both psychologically, as for example in operationalization scales, and neurally, as for example by first-person neuroscience. Defense mechanisms – as part of the psychoanalytic theory – cannot be considered common psychological functions (as can be, for instance, working memory, attentional shift, etc.) because, unlike these functions, they are inherently associated with individual contents that are subjectively experienced. Instead, defense mechanisms are supposed to reflect structures and processes according to which individual contents of subjective experience are organized across different individual subjects. For example, different individual contents of subjective experience might correspond to different defense mechanisms, for instance to more mature and cognitively oriented ones or to rather immature and emotionally driven ones (table 1). As such, defense mechanisms can only be accounted for by what we called first-person data (see above) as distinguished from third-person data. Bearing in mind the methodological shortcomings and problems in operationalizing defense mechanisms and defining the concept of first-person neuroscience, we consider our paper to be a preliminary and speculative attempt to explain defense mechanisms in psychological and neural terms.

In this paper we aim to seriously confront the criticism of empirical validation of psychodynamic processes. One main point of criticism is that defense mechanisms cannot be validated in the same way as other scientific data (see for example Gruenbaum [25] in the 'Introduction' section). Our answer to this is the development of first-person methods such as the operationalization of first-person data. Another point of criticism is that defense mechanisms cannot be localized in particular brain regions. In reply to this we would like to state that instead

of neuronal localization we pursue the concept of neuronal integration. Accordingly we have a somewhat critical view concerning the nature of defense mechanisms. We therefore prefer to consider them as constructs (see above) awaiting further psychological and neuronal validation.

How Can We Investigate the Neuronal Processes of Defense Mechanisms? Sensorimotor Regression in Catatonia

Defense Mechanisms in Disorders: Catatonia and Sensorimotor Regression

Catatonia is a psychomotor syndrome showing a unique constellation of affective, behavioral and motor symptoms [58–63]. Acute catatonic patients are totally immobilized, adopting a bizarre posture and becoming totally mute. Psychodynamically, bizarre motor behavior has been interpreted as sensorimotor regression reflecting an immature and emotionally guided defense mechanism against the uncontrollable overflow of anxieties, i.e. 'immobilization by anxieties' [61, 62, 64]. At the same time more mature and cognitively guided defense mechanisms like internalization and externalization seem to break down in the patients' handling of their emotional conflicts [65, 66]. According to psychodynamic theory, unlike schizophrenic patients, catatonic patients are no longer able to develop paranoid ideas with respect to their environment and thus to externalize their emotional conflicts. Unlike affective patients, catatonic patients remain unable to attribute emotional conflicts to themselves and express them, for example, in ideas of guilt and sin and thus to internalize them [66]. These cognitive mechanisms of internalization or externalization are apparently no longer available for catatonic patients, whose sensorimotor regression aims at compensating for their overwhelming anxieties [65–67]. In the following, our own findings in catatonia are presented. They will be discussed in the various sections concerning neuronal integration.

Methods: Psychodynamic and Biological Measurements of First-Person Psychological Processes

We applied the Role Construct Repertory Grid in catatonic and noncatatonic psychiatric patients, as well as in healthy subjects (see [20] for details). The Role Construct Repertory Grid is an idiographic instrument accounting for psychological characteristics from a subjective point of view [68–71]. It is based on a semistandardized, operationalized and validated methodology, combining intra-

⁵ Whether defense mechanisms, though being constructs, can be considered universal of human nature, as is claimed in the case of language, for example [57], is a question to be answered by future investigation. Although psychoanalysis would certainly claim so, it has yet to be confirmed.

subjective self-appreciation with intersubjective categorical evaluation [68–71] and thus enabling the nomothetical use of idiographic data by means of different mathematical-statistical procedures. This methodology was developed in the context of the psychology of personal constructs [72, 73]. The psychology of personal constructs [69] follows the assumption that subjective experiences of the self and other persons are actively created or constructed and that therefore certain related psychological characteristics are manifest, which, in turn, determine personal constructs. These psychological characteristics include various emotional and cognitive functions whose particular ways of interacting might correspond to different defense mechanisms. Personal constructs might subsequently be well suited to give insight into the first-person experience of psychological mechanisms associated with defense mechanisms.

In addition to the Repertory Grid, we used functional magnetic resonance imaging (fMRI) to investigate catatonic patients, noncatatonic psychiatric control patients (bipolar depressive, unipolar depressive, unipolar manic and schizoaffective) and healthy subjects during emotional stimulation with a motor response (see [32, 33] for details). In this paradigm the subjects viewed emotional pictures and had to give an immediate response to the picture by the arbitrary pressing of buttons. The focus of our analysis was on those regions presumed to be involved in emotional (orbitofrontal and medial prefrontal cortex) and motor (premotor and motor cortex) processing. Moreover we performed an analysis of functional connectivity between these regions [32, 33]. Finally we correlated signal changes in the relevant regions with catatonic symptoms and results from the Repertory Grid test to determine the relationship between psychological characteristics and regional neuronal activity in catatonic patients. One should be aware that other paradigms might be used to elicit implicit nonconscious emotional processing, as for example in masking, and to distinguish it clearly from explicit and conscious processing. The paradigm in our investigation might be considered a mixture of both, since the duration of emotional picture presentation was about 4 s, without any request to give an emotional judgment which would have induced the explicit and conscious component. Finally neurochemical challenge studies during emotional stimulation, for example with lorazepam that relieves the acute catatonic state [61, 62], might also be of interest. This is especially so given the fact that lorazepam in healthy subjects modulates neural activity in those regions (the orbitofrontal cortex) [74] that are altered in catatonia.

Results: Grid and fMRI

The Grid test, which was applied after significant recovery from the acute symptoms, revealed that catatonic patients characterized themselves by the terms ‘low emotional arousal’, ‘low self-esteem’ and ‘lack of social contact’ in both the acute and postacute states. The imaging results showed an altered pattern of signal changes in the medial orbitofrontal cortex [MOFC; Brodman areas (BA) 11 and 12] and lateral orbitofrontal cortex (LOFC; BA 11, 47) in catatonic patients compared to noncatatonic psychiatric and healthy controls. Specifically, we observed reduced signal changes in the MOFC (BA 11, 12) and enhanced signal changes in the LOFC (BA 11, 47) during negative stimulus presentation (see [32, 33] for details). Analysis of functional connectivity revealed abnormal connections from the orbitofrontal to the medial prefrontal cortex (BA 8, 10) and to the premotor (BA 6) and motor cortex (BA 4) in catatonic patients when compared to noncatatonic psychiatric and healthy controls.

Correlation analysis between Grid and fMRI results revealed the following specific findings in catatonic patients: emotional arousal and self-esteem correlated significantly with signal changes in the MOFC (BA 11, 12). In contrast the dimension of social contact correlated significantly with motor symptoms as well as with signal changes in the orbitofrontal and medial prefrontal cortex (BA 8, 10) and their connectivity to the premotor cortex (BA 6).

What Is Neuronal Integration?

Neuronal Integration: Organization of Neuronal Activity across Different Regions

Neuronal integration describes the coordination, organization and adjustment of neuronal activity across different regions. Neuronal organization and modulation is mirrored in specific functional mechanisms of integrating neuronal activity [32, 33]. Even though the exact functional mechanisms of neuronal organization underlying emotional-cognitive interaction are largely unknown, we would like to discuss two possible examples derived from recent imaging studies on emotional-cognitive interaction in relation to specific defense mechanisms. We should, however, emphasize the hypothetical nature of our reflections, since neither the exact mechanisms of neuronal integration nor the defense mechanisms have been completely and satisfactorily validated at this point.

Reciprocal Modulation: Cognitively and Emotionally Guided Defense Mechanisms

Recent studies [32, 33, 75, 76] demonstrated a pattern of opposite signal changes in the medial and lateral prefrontal cortex during emotional-cognitive interaction. These results are compatible with the assumption of functional mechanisms of reciprocal modulation and reciprocal attenuation during emotional-cognitive interaction. Reciprocal modulation can be defined by signal changes in opposite directions (i.e. signal increases and decreases) in different regions. While emotional processing is known to lead to signal increases in the medial prefrontal cortical regions (BA 8, 10, 11) and concurrent signal decreases in the lateral prefrontal cortex (BA 9, 46, 47), cognitive tasks might induce the reverse pattern with signal increases in the lateral prefrontal cortex (BA 9, 46, 47) and signal decreases in the medial prefrontal cortex (BA 8, 10, 11). Emotional-cognitive interaction is then associated with the functional mechanism of reciprocal attenuation: the inclusion of an emotional component in a cognitive task leads to smaller signal decreases in the medial prefrontal cortical regions and, at the same time, smaller signal increases in the lateral prefrontal cortical regions, which shall be called attenuation. Since attenuation concerned both the medial and lateral prefrontal cortical regions in opposite directions (i.e. smaller signal decreases/increases, respectively), one can call it reciprocal attenuation.

Catatonic patients showed an altered pattern of reciprocal signal increases and decreases in MOFC and LOFC (see above and [32–34] for further details). There were diminished signal increases in MOFC (BA 11, 12) and more signal decreases in LOFC (BA 11, 47) during emotional stimulation. This indicates that the reciprocal modulation (and possibly reciprocal attenuation) of emotional-cognitive interaction might be altered in these patients. We assume that altered reciprocal modulation could possibly be related to their inability to use cognitive defense mechanisms and thus to cognitively defend the emotional overflow. The exact relationship between altered reciprocal modulation and the inability to use cognitive defense mechanisms, however, remains unclear. Either the altered reciprocal modulation causes changes in defense mechanisms, or a different use of defense mechanisms leads to changes in neural patterns with altered reciprocal modulation. Alternatively, changes in both reciprocal modulation and cognitive defense mechanisms could possibly be traced back to a third factor underlying and thus causing both, possibly resulting in a bidirectional relationship.

Based on these observations, we assume that the orbitofrontal cortex plays a crucial role in constituting more mature and cognitively guided defense (like intellectualization, rationalization, isolation or reaction formation). Dysfunction in this region and consecutive dysbalance in reciprocal modulation with lateral regions might make the constitution of cognitively guided defense mechanisms impossible. This, in turn, might induce regressive processes with the consecutive predominance of rather immature and emotionally guided defense mechanisms like splitting, projective identification, denial and psychotic introjection/projection. For example, one would suspect dysfunction in the orbitofrontal cortex in patients with a borderline personality, where projective identification predominates. Imaging studies indeed show severe alterations in the orbitofrontal cortex and connected subcortical regions (amygdala, hippocampus) during emotional stimulation in these patients (see [7, 8, 77–79] for a detailed neurobiological account of projective identification). However, altered reciprocal modulation between the medial and lateral prefrontal regions remains to be demonstrated in these patients.

Functional Unit: Sensorimotor Regression and Hysterical Conversion

Another example of a possible functional mechanism of emotional-cognitive interaction might be the constitution of functional units among several brain regions over a certain time period. Such transient functional units might be identified based upon the psychophysiological characteristics or the functional connectivity of the respective regions [80–86]. For example, in contrast to other regions (like the lateral prefrontal cortex), so-called cortical midline structures (CMS, [32]) show a continuously high level of neural activity during resting conditions, such as passively focusing on a certain point (cross) [87–91]. Moreover regions in the CMS seem to be characterized by close anatomical connections. Finally Greicius et al. [92] investigated the functional connectivity among CMS regions in both resting and activation states. They observed increased functional connectivity between anterior and posterior CMS regions in the resting state, whereas it was decreased during active cognitive tasks.

These data provide compelling evidence for the existence of CMS as a functional unit, which seems to be particularly active and cohesive in the resting state [92–95]. Interestingly analogous observations of the CMS as a functional unit have been made during the processing of self-referential stimuli as distinguished from non-self-referential stimuli [32, 88, 96]. Since the resting state can

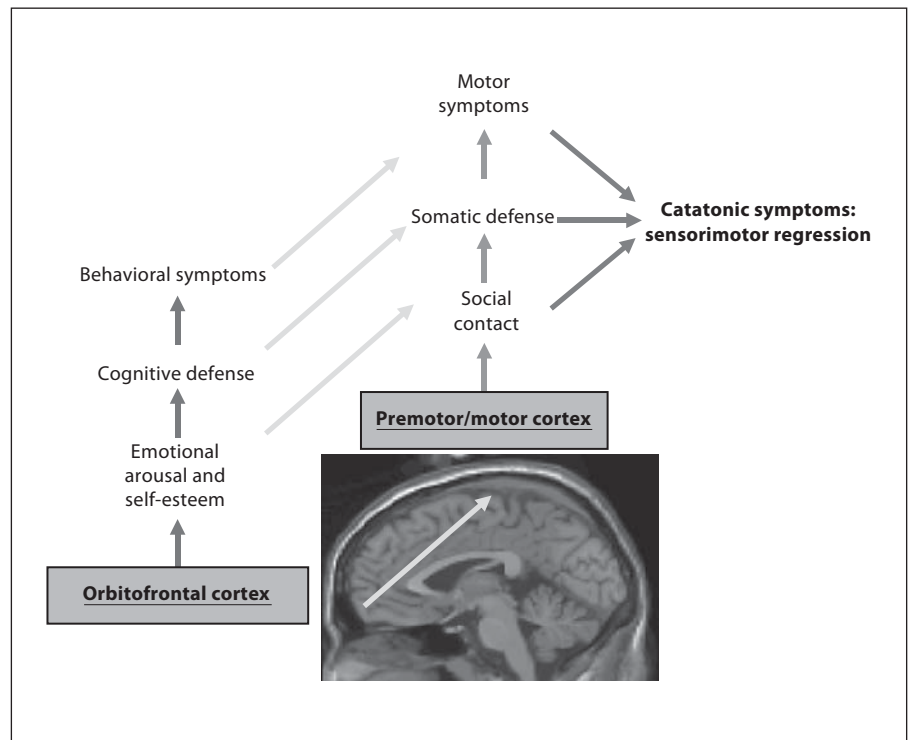


Fig. 2. Sensorimotor regression and orbitofrontal-premotor/motor cortical function in catatonia.

be characterized by strong self-directed and thus internal activity, it might also show a high degree of self-referential stimulus processing. This might explain analogous CMS results during both rest and self-referential processing.

Catatonic patients showed alterations in anterior CMS with decreased functional connectivity among the MOFC, the LOFC and the premotor/motor cortex (see above). These changes significantly correlated with the patients' alterations in their self-esteem, which reflects changes in self-referential processing (see above). This indicates that decreased functional connectivity in the CMS as a functional unit is somehow related to changes in self-referential processing, as manifest in low self-esteem and abnormal social contact. Decreased functional connectivity from the orbitofrontal to the medial prefrontal and to the premotor cortex might not only disrupt self-referential processing but also concurrent behavior. We speculate that such concurrent disruption of self-referential processing and behavior might ultimately result in the manifestation of predominantly somatic defense mechanisms with catatonic motor symptoms as a form of sensorimotor regression (fig. 2).

The relationship between an abnormal functional unit of anterior CMS and somatic defense mechanisms is fur-

ther supported by studies in patients with conversion symptoms. Conversion with hysterical paralysis might be regarded as a form of somatic defense in relation to an emotional-cognitive conflict which can no longer be solved by cognitive defense exclusively. Imaging studies in acute paralytic patients revealed deficits in various regions of the anterior CMS, including the orbitofrontal and the premotor/motor cortex [97–100]. Why, however, is there a symptomatic difference in somatic defense between hysterical and catatonic patients, the former showing conversion and the latter sensorimotor regression? It should first be noted that hysterical patients can show a catatonic-like picture and that, conversely, catatonic patients can appear strongly hysterical [61, 62, 101–103]. Such a symptomatic overlap suggests that both catatonia and hysteria overlap in the neuronal mechanisms underlying somatic defense. They might share the abnormal functional unit of anterior CMS resulting in abnormal motor behavior. However, reciprocal modulation (and reciprocal attenuation) of neuronal activity across the MOFC and the LOFC might be altered to different degrees in both disorders, being stronger in catatonia and less pronounced in hysteria, possibly corresponding to emotional and behavioral differences and thus to different forms of somatic defense. Finally, additional regions

might be implicated in hysterical conversion. This is indicated by a recent study by Lanius et al. [104]. They investigated patients with posttraumatic stress disorder in whom flashbacks or dissociative responses to script-driven imagery were observed in fMRI. Patients with dissociative responses could be characterized by an increased connectivity between the anterior cingulate cortex and the ventrolateral prefrontal cortex. This suggests that (i) different additional regions like the ventrolateral prefrontal cortex might be involved in hysterical conversion and (ii) functional connectivity might also increase, which might possibly compensate for the decreased connectivity among other regions. However, at present we remain unable to fully explore the similarities and differences between hysterical and catatonic conversion. Finally one might use different forms of stimulation, for example emotional pictures [33, 34] or trauma-oriented script-driven imagery [104], in both hysterical and catatonic conversions. We speculate that both types of patients might eventually show differential response patterns to both types of stimulation⁶.

Conclusion

We introduced an appropriate methodological strategy, first-person neuroscience, to overcome the methodological problem of linking first- and third-person data. Psychodynamic processes such as defense mechanisms must be considered first-person data, since they reflect the individual contents as they are subjectively experienced in the first-person perspective, whereas neuronal states as observed in the third-person perspective are typically regarded as third-person data. As such, first-person neuroscience allows for psychodynamic processes associated with defense mechanisms to be related to neuronal activity in our brain. Concerning neuronal activity, first-person neuroscience requires a shift from neuronal localization within one or more brain regions to neuronal in-

tegration across multiple brain regions. We hypothesize that the various defense mechanisms – constructs as hypothesized in psychoanalytic theory – may correspond to specific functional mechanisms by means of which neuronal activity is coordinated and thus integrated across different brain regions. As our knowledge of the functional mechanisms of neuronal integration grows, the future holds the promise of a deeper understanding of the different neuronal processes associated with the various defense mechanisms. A better understanding of these neuronal processes will open the door to an appreciation of the neurophysiology underlying the transition from immature defense mechanisms to more mature ones in psychotherapy.

Since the emotional interaction between patient and therapist is crucial for inducing changes in the pattern of defense mechanisms, investigating the underlying neuronal changes in the brains of both might be of future interest. First-person neuroscience – in contrast to third-person neuroscience – subsequently considers that our brain is embedded in social interaction. One could consequently use the terms embedded first-person neuroscience and embedded neuropsychanalysis [32, 33, 37, 106]. First-person neuroscience in this sense will provide insight into the neuronal processes of defense mechanisms and emotional interaction. This, in turn, might help lay the foundation for the development of a neurophysiologically informed psychoanalytic psychotherapy.

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⁶ Another issue is the question of lateralization. A large body of literature (Lanius et al. [104] and especially for a very good overview Schore 2003 [7, 8]) suggests the right hemisphere to be involved in primitive defenses. Though in this paper we have focused on cortical midline structures, this is still compatible with catatonia. In other studies right hemispheric dysfunction in the lateral parietal cortex was observed to correlate with spatial deficits [105]. The exact relationship between the midline and the right lateral parietal cortical changes, however, remains unclear [74]. What is ultimately needed are studies that link the different subjective experiences and their different individual contents in catatonic and hysterical conversion to different though probably overlapping neural networks with specific patterns of increased and decreased functional connectivity.

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