

Psychosis as a Disorder of Reduced Cathectic Capacity: Freud's Analysis of the Schreber Case Revisited

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Approximately 100 years ago, a prominent German public figure name Daniel Schreber wrote memoirs of his experiences in asylums. His case was diagnosed Dementia Praecox at times and Paranoia at others by his treaters. Freud analyzed Schreber's memoirs from the perspective of his "libido" theory of developmentally organized mental "cathexes" or ideational/emotional investments in self and others. Revisiting Freud's analysis of the Schreber case suggests that it may represent the first theoretical articulation that the pathophysiologic core of psychosis is one of deficit, i.e., of diminished (organic) cathectic capacity for normal mental and affective investments in life.

Key words: psychosis/dementia praecox/dementia paranoides/cathexis/libido

Introduction: Dementia Praecox

At the turn of the 20th century, Emil Kraepelin defined the forerunner of today's psychotic disorders, dementia praecox (DP), or the dementing process that erupts before old age. It was characterized by a panoply of positive psychotic symptoms and deficits or deterioration in functioning. Postmortem scrutiny of the brains of patients with DP revealed nothing that was obviously different from normal. Nevertheless, Kraepelin assumed, largely by the disorder's deteriorating course, that organic processes were involved.

At about the same time, Sigmund Freud, operating from his own theoretical realm of psychodynamic forces and investments, came to the same conclusions about DP. He did this by analyzing the published memoirs of a patient suffering from psychotic paranoia, Daniel Paul Schreber. Based on his analysis, he concluded that the disorder could not be treated psychoanalytically because of the disorder's inherent deficits in the capacity

for object relatedness, including the therapeutically necessary development of a transference to the treating person.

This communication will revisit the notorious "Schreber case" and Freud's ad hoc analysis of it from the psychoanalytic perspective. The question to be addressed is whether he saw the case as a disorder of conflicting aims (wish and defense) or of cathectic capacity (cathexis according to *Webster's Dictionary* meaning the investment of mental or emotional energy in a person, object, or idea). In short, did the essence of psychosis to Freud reside in the positive or the deficit symptom domains of psychopathology?

The Case of Daniel Paul Schreber

Freud's 1911 monograph is about 70 pages long. Considerations of space require a severely condensed version here, but for the Freud scholar the now classic original is highly recommended.¹

Schreber was a public figure, a judge who developed a paranoid psychosis for which he was institutionalized on 3 separate occasions. He wrote memoirs of his illness experience that were published in 1903 and received considerable public attention. They attracted Freud's attention in 1910 and led to the writing and publication 1 year later of his *Psychoanalytic Notes on an Autobiographical Account of a Case of Paranoia (Dementia Paranoides)*.¹ Freud saw cases of paranoia and DP as a practicing neurologist but never as patients in psychoanalysis. He therefore found in Schreber's memoirs a unique opportunity for applying his newly formed libido theory of the psychoneuroses to the psychotic disorders.

Schreber was born in Leipzig in 1842. He married in 1878 at the age of 36 years. In 1884, at the age of 42 years, while a judge and a candidate for the Reichstag, he had his first illness, entered an asylum for about 6 months, and returned to public office in 1886. Seven years later, in 1893 at the age of 51 years and a presiding judge, Schreber was readmitted to 2 asylums for 8 years. In the last 2 of these years, he wrote his memoirs and took legal action against his commitment to asylum. He won the case and was discharged in 1902, 1 year before his memoirs were published. In 1907,

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however, in the context of his mother's death and his wife's stroke, Schreber fell ill again and was admitted to asylum where he spent the remaining 4 years of his life.

Schreber's first illness and hospitalization in 1884 was described by his physician Flechsig as severe hypochondria. Little more than this is recorded, and he left Flechsig's clinic in 1885 "completely recovered."

The second illness began in October 1893 with insomnia, forcing Schreber to return to Flechsig's clinic. There his condition worsened rapidly with hypochondriacal ideas that he had softening of the brain and the plague, was dead and decomposing, and was being persecuted and that his body was being handled, manipulated, and changed on behalf of a "holy purpose." He was often inaccessible, sitting motionless for hours in an apparent "hallucinatory stupor." He felt tortured, longed for death, asked to be given cyanide, and tried drowning himself in his bath several times. His delusions were religious, ie, he was in direct communication with God, and they were persecutory, ie, he was being injured, especially by Flechsig.

This state of total psychosis lasted for several years and more than one asylum. By 1899, however, the clinical picture began to "evolve" or "crystallize out" more clearly into a paranoid psychosis with a psychotic delusional core and a reconstructed personality "capable of meeting the demands of everyday life." Were it not for the delusion, Schreber functioned as if in full remission, and, in fact, he applied successfully to regain his civil liberties and to leave the asylum. However, his delusional ideas remained present, active, and "more or less fixed," and they were "inaccessible to correction by means of any objective appreciation and judgment of the external facts." The core of Schreber's delusion was that he had a mission to redeem the world and to restore mankind to their lost state of bliss. In order for this to happen, he had to be transformed bodily into a woman so that, as God's concubine, he could give birth to a new race of humanity. In his application to the courts for release from asylum, Schreber never disavowed these delusions nor did he hide his intentions to publish his experiences as memoirs.

Freud's Discussion of the Case

Freud tried to understand Schreber's most prominent symptoms, his paranoia of Flechsig, his end-of-world experiences, and his ultimate mission to redeem mankind as a woman. Here, he turned to his theory of psychosexual developmental levels of libidinal "cathexis." Depending upon context, libidinal cathexes may be expressed as attachments, investments, interests, commitments, motivations, salience, etc. The objects or targets of one's cathexes are multiple and can include significant others, oneself, and one's ideas, talents, possessions, etc.

The force and foci of an individual's libidinal cathexes change and evolve with development. In infancy, ca-

thexes cluster around experiences of nursing and personal comfort, in childhood around nuclear family and a growing body, in adolescence around sexual maturity and object choice, and in adulthood around vocation and procreation. Most developmentally earlier cathexes are superseded in time by later, adult interests. Nevertheless, earlier investments remain active, though largely subdued and unconscious, in the individual's mnemonic warehouse of behavioral habits and repertoires. These traces Freud termed fixations, and certain fixations came to be central to his theories of psychotic and psychoneurotic pathophysiology.

Libidinal cathectic fixations were central to Freud's explanation of the relation of paranoia to DP in the Schreber case. Freud postulated that Schreber's paranoid delusion about Flechsig represented an earlier fixation of homosexual attraction that underwent denial and reversal ("I do not love him; I hate him because he persecutes me"). Schreber's elaboration of this into his later grandiose delusion of saving the world as God's concubine represented the addition of cathectic fixations around homosexuality and omnipotence. These ideas, although psychotic, were rendered less disorganizing by becoming "compartmentalized." As the case notes record, Dr Schreber's thinking and behavior surrounding his sequestered nidus of psychosis were quite normal if not exemplary. At such times, Dr Schreber was simultaneously psychotic and remitted.

It is clear, however, that his illness could erupt periodically into an all-encompassing psychotic state, often at times of significant personal gains or losses. We have a vivid description of Schreber's second illness at the end of 1893. Freud adds the following about this.

At the climax of his illness, under the influence of visions which were 'partly of a terrifying character, but partly, too, of an indescribable grandeur', Schreber became convinced of the imminence of a great catastrophe, or the end of the world. Voices told him that the work of the past 14,000 years had now come to nothing and that the earth's allotted span was only 212 years more; and during the last part of his stay in Flechsig's clinic he believed that the period had already elapsed. He himself was 'the only real man left alive', and the few human shapes that he still saw—the doctor, the attendants, the other patients—he explained as being 'miracled up, cursorily improvised men'.

Freud continues:

A world catastrophe of this kind is not infrequent during the agitated stage in other cases of paranoia. If we base ourselves on our theory of libidinal cathexis, and if we follow the hint given by Schreber's view of other people as being 'cursorily improvised men', we shall not find it difficult to explain these catastrophes. The patient has withdrawn from the people in his environment and from the external world generally the libidinal cathexis which he has hitherto directed on to them. Thus everything has become indifferent and irrelevant to

him, and has to be explained by means of a secondary rationalization as being 'miracled up, cursorily improvised'. The end of the world is the projection of this internal catastrophe; his subjective world has come to an end since his withdrawal of his love from it.

At the apex of acute psychosis, relatedness appears moribund, and the patient feels alone without connection. But the instinct of self-preservation fills the void with created thoughts, feelings, and relationships nonetheless.

And the paranoid builds it again, not more splendid, it is true, but at least so that he can once more live in it. He builds it up by the work of his delusions. *The delusional formation, which we take to be the pathological product, is in reality an attempt at recovery, a process of reconstruction.* Such a reconstruction after the catastrophe is successful to a greater or lesser extent, but never wholly so; in Schreber's words, there has been a 'profound internal change' in the world. But the human subject has recaptured a relation, and often a very intense one, to the people and things in the world, even though the relation is a hostile one now, where formerly it was hopefully affectionate.

Freud finishes his essay with thoughts about the distinction between paranoia and DP.

Abraham has very convincingly shown that the turning away of the libido from the external world is a particularly clearly-marked feature in dementia praecox Here once more we may regard the phase of violent hallucinations as a struggle between repression and an attempt at recovery by bringing the libido back again on to its objects This attempt at recovery, which observers mistake for the disease itself, does not, as in paranoia, make use of projection, but employs a hallucinatory (hysterical) mechanism. This is one of the two major respects in which dementia praecox differs from paranoia The second difference is shown by the outcome of the disease, in those cases where the process has not remained too restricted. The prognosis is on the whole more unfavourable than in paranoia The regression extends not merely to narcissism (manifesting itself in the shape of megalomania) but to a complete abandonment of object-love and a return to infantile auto-erotism.

Freud recognized that Schreber's illness included periods where the clinical picture mimicked DP or hallucinatory psychosis. Overall, however, in between episodes of global psychosis, the picture was one of paranoia. He concluded this in part because of the better prognosis, although ultimately he professed ignorance as to what actually determined Schreber's relatively positive outcome.

Discussion

The Schreber case and Freud's analysis of it raise questions about the diagnostic manifestations and the psychological dynamics of psychosis as well as the experience of deficits in mental functioning, questions that are as

relevant today as they were at the beginning of the 20th century. Each of these will be addressed in turn.

Diagnosis of Psychosis

At the turn of the 20th century, disorders of insanity were handled largely by neurologists. The European expert at the time was Emil Kraepelin, and he had recently consolidated the psychotic disorders into 3 distinct categories: DP, dementia paranoides, and manic depressive insanity. Using Kraepelin's system, Freud, himself a practicing neurologist, diagnosed the patient Schreber as a case of dementia paranoides who nevertheless, at times, displayed a collection of symptoms of hallucinatory psychosis that was similar to DP.

The major elements of Schreber's illness suggesting paranoia rather than DP were the later onset of disorder, the long periods of remission (sufficient to allow functioning outside of asylums), and the ability during these times to maintain psychotic thinking and delusional systems in a sequestered or compartmentalized fashion. In this phase, Schreber's special (psychotic) identity and mission remained alive and real to him (eg, his memoirs) but did not interfere with everyday functioning and relationships.

Schreber also clearly had periods of greater disorganization and functional paralysis, 3 in all, each lasting months to years. During these, he experienced florid hallucinations in addition to bizarre and nonbizarre delusions, and he was unable to care for himself and required long-term hospitalization. Schreber's clinical picture at these times was more compatible with DP, and Freud was careful to extend his theoretical discussions of the case to include this disorder.

Today, according to *Diagnostic and Statistical Manual of Mental Disorders* (Fourth Edition) (*DSM-IV*), Schreber would meet criteria for schizophrenia, the Kraepelinian "equivalent" of DP. Although the *DSM-IV* diagnosis of delusional disorder carries the nosologic legacy of Kraepelin's dementia paranoides, Schreber would not meet these criteria because some of his delusions could easily be considered bizarre and because he was for considerable periods functionally incapacitated by psychotic symptoms.

Early in the 20th century, when Kraepelin created the term DP and Freud analyzed Schreber's dementia paranoides, the etiology of these conditions was unknown. For both disorders, it was clearly perplexing that post-mortem brain examination did not suggest organic processes, i.e., revealed no changes compatible with other disorders labeled dementia. Kraepelin nevertheless assumed that the etiology, especially of DP, was "organic" because of the deterioration that he considered to be its diagnostic hallmark. Dementia paranoides represented a greater challenge to a purely organic pathophysiology because of its preservation of significant islands of sanity and object relatedness. It was the latter that suggested

more functional “psychodynamic” elements, at least to the clinical picture, if not to the etiology. It appears to be this element that attracted Freud to the case to begin with.

Psychological Dynamics of Psychosis

Freud followed Kraepelin’s system for classifying the psychoses. Nevertheless, his main interest in the Schreber case was to apply and further develop his emerging theory of psychological dynamics. Freud’s observations from 20 prior years of psychoanalyzing cases of hysteria and neurosis led him to postulate a “libido” theory of thinking and behavior. This theory focused upon an individual’s libidinal psychological dynamics or the mental expressions of love and sexual drive, “the psychical representative of organic forces”¹. Freud felt that the objects or targets of this libidinal instinctive interest were developmentally differentiated. During infancy, eg, a major proportion of each day is filled with experiences of one’s body and its appetites, and this leads to rich psychological investments in or cathexes of the body relative to cathexes of other people and things. Part of this also happens because brain capacities for self-object differentiation and for abstract conceptualization are not yet matured developmentally.

The strength and qualitative nature of one’s psychological cathexes, according to Freud, could vary as a result of experiential complexity and organic brain development and capacity. In fact, he regarded both as sources of variations in the nature and targets of an individual’s libidinal instincts. Freud’s model to explain Schreber’s symptoms has been labeled a deficiency or deficit model.² Clinically, Schreber’s illness started with hypochondriacal preoccupations followed by apocalyptic panic, leading to catatonia, personality change, and symptoms of psychosis, particularly grandiose and paranoid delusions. Freud postulated the following psychological dynamics to explain this sequence. The patient first withdraws libidinal cathexes from the real world and its people. These cathexes, in part, are invested in fantasied (hallucinatory) images of the world and its people, but much of the withdrawn cathexes (psychic energy) become redirected to the self, seen as megalomania, and to the body, seen as hypochondriasis. The withdrawal of cathexes from the world is so profound that breaks occur with external realities and relationships. The patient still has an active mind and libidinal cathexes to invest, but now the investments attach to created objects that are not part of the real world. The patient has reinvested his libidinal cathexes but in objects that the observer now recognizes as the well-known symptoms of psychosis. Freud considered this to be the patient’s attempt at “recovery,” however distorted and dysfunctional.

The brain aberrations generating psychosis essentially stem from paralysis of the integrative circuits of the high-

er central nervous system, resulting in a form of deafferentation syndrome.³ As in the phantom limb syndrome, where the brain creates in mind what has been severed from the body, in psychosis the brain creates persons and relationships in mind to fill the blankness created by the brain’s diminished capacity to gather and process daily social experiences and relationships. Freud described it as inadequate “normal” libidinal cathexes, normal meaning libidinal cathexes (in mind) of real persons in real life.

Which comes first, deficit processes or positive symptoms? From Freud’s analysis of the Schreber case, the primary psychopathology is deficit, ie, a withdrawal of cathexes for reasons that were largely unknown (perhaps experiential, perhaps organic, probably both). Positive symptoms then emerge in the form of hallucinations and delusions as a way to “reverse” this process and restore a world of objects and relationships. The problem, of course, is that this world is now psychotic, unreal, and ultimately orthogonal to the commerce of everyday life.

Psychosis as a Disorder of Deficit

Freud’s theory of reduced libidinal cathexis was by no means the first or the last deficit theory of psychosis. Kraepelin considered deficit to constitute the essence of DP in its “loss of mainsprings of volition” and in its relentless deterioration to “weak-mindedness.”⁴ Deficit has been prominent in most labels and descriptions of psychosis through the 20th century, eg, acute vs chronic, reactive vs process, acute vs residual, and accessory vs fundamental. The clearest latter day articulation of the deficit theory comes from the work of Carpenter and colleagues^{5,6} on the deficit syndrome in psychosis. Particularly important has been this group’s articulation of the symptomatic expressions of deficit such as alogia, avolition, anhedonia, and lack of social drive that are not a result of or secondary to the positive/productive symptoms of psychosis. That is, they are “primary” negative symptoms of psychosis and reflect an absence of mental activity per se rather than reactive behavior that is avoidant and defensive.

The concept of psychological deficits, like the presence of absence, is difficult to grasp conceptually. The legacy of Freud’s theory of psychological libidinal cathexes, however, may be helpful here. Deficit in this context is more clearly the absence of something basic. That something is cathexis, the neurobiological substrate of investment and meaning. From this perspective, deficit is more clearly a product of a hypofunctioning neuronal syncytium, ie, a paralysis of the higher central nervous system.⁷

The question often arises as to how absences or deficits in cortical connectivity can generate positive symptoms and other forms of “actively” dysfunctional psychology. Computer simulations of parallel distributed processing neural networks can be manipulated to study effects of

reducing communication between network modules. One consequence is that some cortical circuits become functionally autonomous and hyperactive, generating “parasitic foci” that repetitively produce the same cognitive output. Most of the positive symptoms of psychosis are postulated manifestations of parasitic foci located at different cortical levels of language processing. The theory, mechanics, and dynamics of those processes are detailed elsewhere.^{8–12}

Is Deficit Experienced and if so How?

Deficits in libidinal cathexes leading to psychosis may be a form of higher central nervous system paralysis, but it is not likely to be experienced as such (ie, nothingness). Schreber’s psychosis, in fact, was a very lively and enthralling cacophony of delusions and hallucinations, interesting enough that he often seemed enraptured by his created world. His memoirs of this world were a best seller and captured much public interest, including Freud’s. Schreber’s interactions with his psychotic objects, especially those with God, were very rich, interactive, and sexy. His remissions, in contrast, seemed quiet and tame compared with the intense excitements connected to his living delusions with Flechsig and with God.

It is tempting to guess what Schreber’s clinical picture and course would look like with “modern” treatment, ie, antipsychotic pharmacotherapy. I speculate that his severe attacks of hallucinatory psychosis would have lasted for days, not years. With medicine, Schreber would quickly become less fascinated by and interactive with his hallucinatory world. In fact, he probably would start complaining that his world was uninteresting, unidimensional, and flat, and that his personal life was empty and missing *joie de vivre*. He would start wanting to stop the medication in order to feel alive, related, and important again. In short, he would “feel” deficits as an unnatural absence.

At this point, Schreber, like many psychotic patients in remission from acute positive symptoms, would become acquainted with his deficits in psychological cathexes as an all-encompassing absence of interest, a weakened capacity to engage with or grasp the world psychologically, and a disconnection from personal commerce and everyday life. For many patients like Schreber treated in the 21st century, rapid diminution of positive symptoms often unveils an emerging postpsychotic state of diminished interest and activity termed postpsychotic depression or dysphoria or anhedonia.^{13,14} This state is frequently blamed on the drug and labeled “neuroleptic dysphoria.” From this perspective, the positive symptoms appear to come first, and, with treatment, the deficit symptoms come second. The direction originally postulated by Freud gets reversed, and the primacy of deficit processes to the pathophysiology of psychosis gets lost. For this reason alone, the Schreber case is important for its dead reckoning of direction in the causal chain to psycho-

sis, the initial factor being the developmental emergence of inadequate libidinal cathexes to everyday life.

Lingering Questions

A case like Schreber’s is sufficiently removed from our contemporary clinical scene to be useful in germinating questions that would otherwise never ever occur to us as we run from one modern patient to another. For example, do we ever see cases like this, and if not, why not? I suggest we do not see cases like Schreber because of pharmacotherapy that has truncated extended periods of active positive psychotic symptoms.

Schreber also illustrates that varying levels of remission from acute psychosis are possible to achieve without drugs. Furthermore, it appears that Schreber used every ounce of his intellect and judgment to overcome the chaos of his hallucinatory states by creating an extensively elaborated delusional system suffused with intricate paranoid and grandiose content. This system, while itself psychotic, was coherent and consistent enough to provide for Schreber a template around which to organize his daily thinking and activity in a more normal fashion. Such a process is sometimes labeled compartmentalization, but we still do not know how it “works.” In fact, we probably see this form of adaptation less frequently now because contemporary pharmacotherapy quells acute psychosis so rapidly.

Schreber’s case raises the question of whether the long-term course and outcome of psychotic disorders are any better now than at the turn of the 20th century. The question clearly cannot be answered, but I suggest that this case challenges any assumptions that pharmacotherapy-aided long-term outcomes are likely to be superior. Drugs clearly restrain the positive symptom elaborations of deficit process in the short term, but whether they restore the loss of brain connectivity that creates the positive symptom generating deficits to begin with is another matter. The continuous use of drugs may dampen the generation of “parasitic attractors,” but fail to prevent further cortical connectivity attenuation resulting from the original *praecox* diathesis or the ongoing aging process or both.

Concerning the psychological dynamics of psychosis, the Schreber case raises the question of whether the concept of psychological libidinal cathexes adds anything to our description or understanding of psychotic states and/or disorders. Could the concept simply be another way of describing what we already articulate as clinical signs and symptoms? Perhaps, but the latter focus truncates our perspective to the pathological products and can distract our attention from the primary pathological agent. Freud’s dynamic meta-analysis helps us to see that what is missing, ie, adequate psychological cathexes, may be the critical issue, and, if it is, then an exclusive focus on controlling positive symptoms will never lead

to understanding and treating the source of these symptoms, ie, the deficit core of the disorder.

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