SMGroup

Reasons Why Schizophrenia-specific Medication and Psychotherapy are Essential for Treatment of Schizophrenic Psychosis

Andreas Laddis[1]*

[1]Private practice, Framingham, MA, USA

***Corresponding author:** Andreas Laddis, M. D., 18 Independence Ln., Shrewsbury, Massachusetts 01545-6242, USA, Email: aladdis@gamil.com

Published Date: February 26, 2016

INTRODUCTION

The meaning of the term "psychosis" has been remarkably stable for more than the past one hundred years; however, explanations for it have diverged and overlapped repeatedly, in ways important for the treatment of psychosis. Explanations are still the object of much controversy [1-4]. Freud introduced the current meaning of the term "psychosis" as the presence of hallucinations and delusions. For him, those phenomena were evidence of psychological disorder qualitatively more severe than "neurosis". There is reasonable consensus about defining what a hallucination or a delusion is. A hallucination is the experience of perceiving with one's senses an object that does not exist in immediate reality. A delusion is a strange belief to which a person returns persistently, despite others' logical and empirical disproof of it. Typically, delusions are explanations for good and bad turns of events in reality or in the person's abilities. Delusional explanations are always in terms of individuals, social institutions or fantastic beings who can know the person's desires and intentions and who can furthermore manipulate reality or the

sufferer's power to control the outcome. The term "neurosis", as a lower grade of mental disorder, has been mostly deleted from diagnostic manuals; but "psychosis" continues to mean a higher grade of mental disorder, when the sufferer's "reality testing" function fails [5,6]. While suffering hallucinations and delusions, patients cannot transition from *thinking of* what exists in reality to *knowing* it. That pertains specifically to thinking of desired or feared objects in immediate reality, or ones with power to influence the sufferer from afar.

Current diagnostic manuals [7,8] compile a handful of mental disorders together with schizophrenia in the category of "psychotic disorders". They treat this handful of disorders as if their potential for great severity, which they share with schizophrenia, were more important than their etiology, which they do not share. Looking more closely at the descriptions in those manuals, these kinds of psychosis are, in fact, just a phase of disorders classified elsewhere, mostly dementia, delirium or disorder during extreme stress. Etiology matters for choice of treatment. Some treatments are symptom-specific, for alleviation of particular symptoms, e.g., irrational negative emotions, fear, anger and anxiety, that are common in various mental disorders. Some treatments are disorder-specific; they repair or otherwise remedy the fundamental abnormality for each kind of psychosis, which, in turn, alleviates all symptoms. To understand and treat schizophrenic psychosis, it is necessary to a) diagnose it correctly and exclude other kinds of psychosis; b) discriminate between treatments generic for all kinds of psychosis and those that are etiologically specific for schizophrenic psychosis.

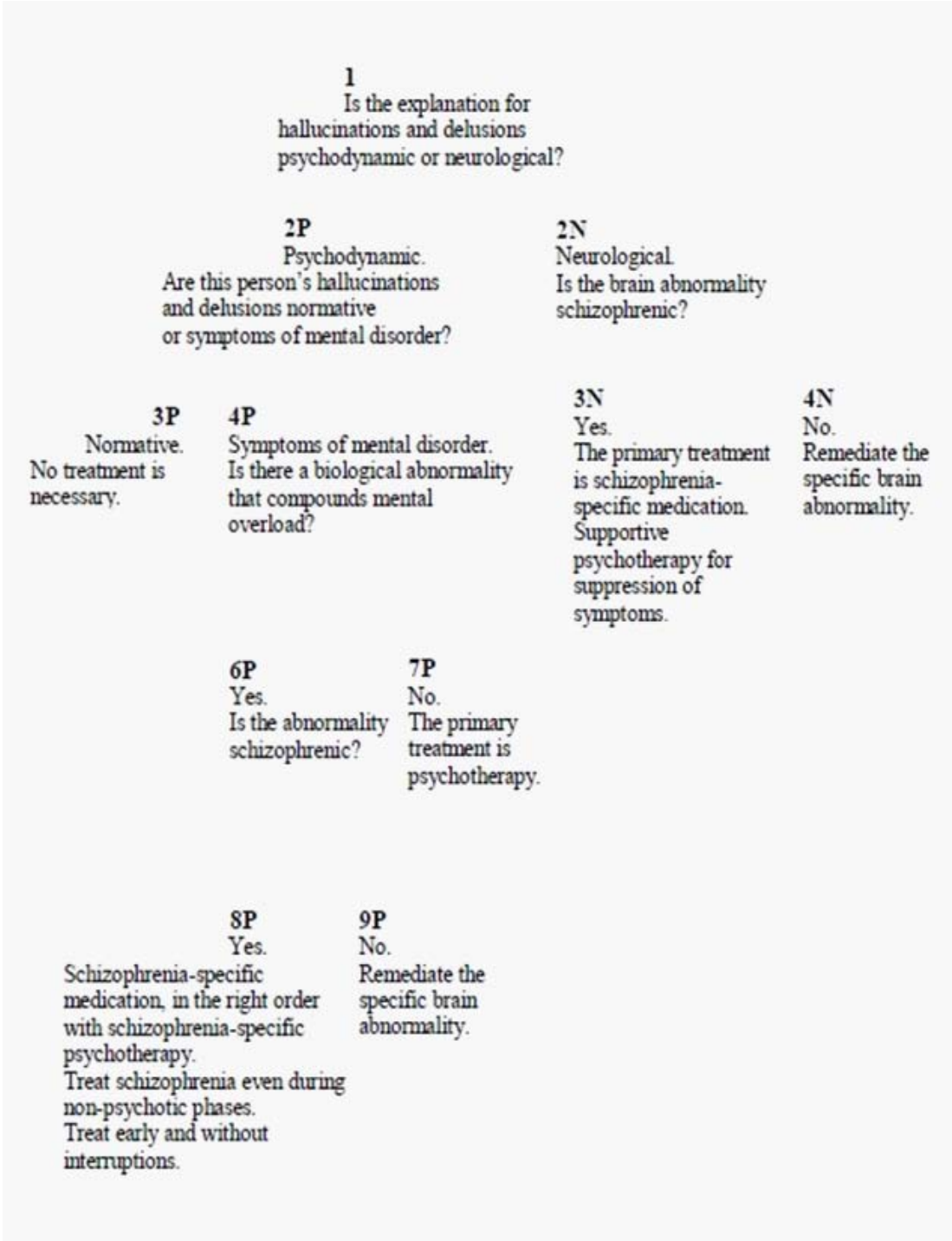

Figure 1: Diagnostic reasoning leading to schizophrenia-specific treatment.

Figure 1 below depicts an algorithm, i.e., the stepwise logic for differential diagnosis among kinds of potentially psychotic disorders, pertinent for the first part of this chapter. The author will present the reasoning for judgments to make at each fork of the algorithm. In the second half, at Step 8P, he will provide evidence for the schizophrenia-specific psychological impairment and for the biological abnormality that underlies it. The algorithm proceeds on two paths, beginning at Steps 2P and 2N, according to two dichotomous explanations for the generation of hallucinations and delusions; one explanation is in psychodynamic (P) and the other in neurological (N) terms. Note that only the psychodynamic explanation, which the author of this chapter adopts, allows for generation of normative hallucination-like and delusion-like experiences, i.e., without mental disorder (Step 3P). It also explains how people may suffer mental disorder without significant biological abnormality (Step 7P), as is the case in certain posttraumatic disorders, which occasionally may even become of psychotic severity.

The second part of this chapter proposes a) medication that remedies the schizophrenia-specific biological abnormality, and b) psychosocial treatments that remedy the schizophrenia-specific psychological impairment. It provides reasons why the medical and psychosocial interventions are most effective if given in a certain order, early in the course of the biological abnormality and without interruption of medication.

The Reasoning for Differential Diagnosis

Step 1

What causes people to perceive an object that does not exist in immediate reality or to persist defending a belief irrationally, which others can easily disprove? Good answers to this question are emerging; they are composed of well-tested concepts from cognitive psychology [1,2,3,9,10] These are mainly concepts from studying how strange perceptions and beliefs may be generated normatively, i.e., as the result common errors by people without mental disorder. They are subsequently applied to hallucinations and delusions suffered by people in mental disorder.

For example, a man without mental disorder may hear a voice that is not spoken in immediate reality (hallucination-like) in the mental context of anticipating to hear it with much desire or fear. Ordinarily, people choose cues of the speaker's anticipated presence consciously, which they subsequently detect automatically; they "prime" their senses to selectively detect such cues in anticipated reality. The outcome may be a hallucination, if the hearer chooses signals of the speaker's presence from memory of similar events, instead of signals relevant to immediate reality [3,11]. Normally, people get startled momentarily, then they scope reality broadly to ground themselves better. In contrast, hallucinations in mental disorder occur recurrently and are difficult to extinguish (see explanation at Step 4P).

Similarly, people without mental disorder may persist with promoting a strange belief, despite others' logical and empirical disproof of, delusion-like [1,12]. Like children, adults commonly begin explaining strange events (a piece of mail that arrived unsealed) with arbitrary correlations

among facts, some of them beyond empirical observation, like others' intentions ("the government spies on people like me"). Subsequently, people routinely find reasons to doubt such a crudely reasoned belief and they occasionally debate and test it reasonably and thoughtfully. However, they do not relinquish it until they may construct a better one [1]. For that reason, they may defend the odd belief irrationally, if someone challenges it while debating and revising is not a priority for themselves. Then, their urgency to defend it biases their attention automatically, effortlessly, in favor of ideas and evidence that bolsters it [12]. Differently from people without mental disorder, sufferers of schizophrenia often lack the ability to carry out the intricate thinking, debating and testing activity that it takes to revise a failed belief, even when they find motivation to do so (see explanation at Step 4P).

Steps 2P and 2N

The above explanation for hallucinations and delusions, at Step 2P, is a psychodynamic one. It is about perceptual and reasoning operations gone errant, askew of the purpose they were initiated to serve, because of insufficient attention while the person became absorbed in something else.

People initiate a perception or an explanation to serve a purpose in relation to an object in reality, but then they startle themselves with having brought those performances to closure errantly and crudely, unfit for the purpose that they initiated them to serve.

Insufficient attention to perceptual and reasoning operations is due to mental overload. It happens when a person is pursuing an intricate goal passionately and urgently, while a rapid stream of tangents in thought and action are generated and must be given priority of attention. Mental overload and inattention, in turn, is much more likely if the person suffers errors because of psychological shortcomings pertinent to perception and reasoning. Psychological shortcomings may be due to: a) medical effects (dull hearing or vision, drugs); b) situational (sensory deprivation); maladaptive learning (false or outdated judgments and methods).

According to the psychodynamic explanation, symptoms are meaningful [13]; they are performances initiated in the course of a goal which the person has ended unfinished, unfit and unwanted.

In contrast, *neurological* explanations treat hallucinations and delusions as meaningless, unrelated to the sufferer's current concerns and conflicting motives (Step 2N). These symptoms are "endogenous" psychological phenomena, i.e., born of errant biological processes. [3,14] They are either a) experiences generated by focal excitation of a brain part, e.g., of the cortex that stores the auditory experience of past events, or b) experiences of a purposeful initiative (detection of an anticipated event or reasoning about one's troubles), performed errantly because of deranged feedback among brain parts. These two kinds of neurological explanation for hallucinations and delusions derive from explanations for symptoms of certain neurological diseases, e.g., epilepsy and the so-called disconnection syndromes, improperly to symptoms of mental disorder.

Steps 3P and 4P

It is well-documented that about 4-15 % of people without mental disorder occasionally perceive objects that do not exist in immediate reality (Step 3P) [12]. That is more likely to occur in people with intense religious beliefs. For unknown reasons, scores were even higher among college students. It is similarly well-documented that 2-8% of people without mental disorder have bizarre beliefs of being persecuted or owning special powers, despite the fact that those beliefs have served their purposes badly and are often disproven by others. People who hold bizarre beliefs, whether with or without a mental disorder, show similarly faulty reasoning to defend them [12]: they conveniently overvalue or undervalue evidence in favor or against those bizarre beliefs and rush to foreclose further argument. It is remarkable that, compared to sufferers of mental disorder, people without mental disorder experience less distress and motivation to remedy beliefs rejected by others.

These facts about hallucinations and irrational beliefs among people without mental disorder create the impression of a continuum, that the difference from people with mental disorder is merely a matter of degree [12]. However, the difference is qualitative (Step 4P). In mental disorder, the psychological shortcomings are so persistent and uncontrollable that they impair correction itself, and then the correction of correction [15]. The persistent psychological shortcomings, the sources of error, are various, but they all result in mental overload, a shortage of attention and of reasoning. Some shortcomings are because of biological abnormality (Step 6P below); some are from fixations on certain dysfunctional beliefs and methods (Step 7P below). Psychodynamically speaking, a person in mental disorder acts compelled to repeat a failed performance despite growing awareness of futility, i.e., despite one's inability to learn from recurrent failures fast enough, while the outcome still matters. The person then may suffer hallucinations and illogical arguments over delusional beliefs repeatedly and uncontrollably, as symptoms of that irrational urge.

Steps 6P and 7P

It is important to note that: a) The psychodynamic definition of mental disorder allows for similar hallucinations and delusions to ensue from different psychological shortcomings; psychotic symptoms may occur in etiologically various mental disorders. b) The psychodynamic definition incorporates biological abnormality as one factor, a vulnerability, great or small (Step 6P).

Many biological dysfunctions cause errors of little consequence for most goals, thus contributing little to proliferation of significant errors and mental overload. Others cause pertinent errors recurrently, whose correction overtakes the capacity of working memory, the psychological function of making order out of complexity: reasoning about the stepwise fulfillment of a priority, while also reasoning about ending it because of competing motives. Working memory functions as the mind's "workbench". It preserves a handful of activity streams outside consciousness

selectively, as pertinent to the conscious one, e.g., an alternative course, a tangent, a reason to end it. It seems that, among all mental disorders, schizophrenia presents with psychotic symptoms most often because it ensues from dysfunction in the biology of working memory itself [2].

People may suffer uncontrollable mental overload and disorder from errors due to fixation on dysfunctional beliefs and methods, even without significant brain abnormality (Step 7P). They make errors by repeatedly foreclosing certain logical steps in the pursuit of a goal, taking logical shortcuts in reasoning how to learn from failure. Various fixations have been postulated as the fundamental psychological shortcoming in chronic posttraumatic disorders, in the aftermath of extreme stress in mistrusted caretaking relationships during the patients' childhood [16]. Such history correlates highly with the diagnoses of Dissociative Identity Disorder (DID), Complex Posttraumatic Stress Disorder (CPTSD) and Borderline Personality Disorder (BPD). It is well documented that patients with BPD and DID suffer psychotic symptoms episodically. Remarkably, the resemblance between DID and schizophrenia is greater when they become chronic and severe, because of similarly high scores in psychotic symptoms [17]. Because those facts are not highlighted in diagnostic manuals, patients with BPD and DID are often misdiagnosed with schizophrenia or schizoaffective disorder. Psychotherapy is the primary, possibly reparative treatment for these disorders. In BPD and DID, antipsychotics are modestly effective, which compounds reasons to misdiagnose those disorders as schizophrenia. However, patients with BPD and DID benefit from the antianxiety properties of antipsychotics, coincidental with those drugs' apparent reparative effect for schizophrenia and bipolar disorder [18] Still, as antianxiety agents, antipsychotics are not the drug of first choice, even when psychotic symptoms are present.

Steps 8P and 9P

"Schizophrenia-*like*" psychotic symptoms are present in sizable minorities of patients with bipolar disorder, dementia, delirium, epilepsy and Parkinson's disease; they are also present less frequently in a range of other neurological disorders (Step 9P). Treatment of psychotic behavior in dementia with psychiatric medication of any kind is "of limited efficacy and should be used only after environmental and non-pharmacologic techniques have been implemented" [19]. Psychiatric medication is similarly of limited efficacy for psychosis in delirium [20,21] Here, the basic psychological impairment is low alertness and dullness of all mental functions, because of widespread encephalopathy from infection, metabolic toxicity, etc. Medication also entails high risk of compounding the encephalopathy. Low alertness and mental dullness set the stage for psychosis also during the *peri*-ictal and *post*-ictal phases of epilepsy. That may be compounded with toxicity from antiepileptic drugs. Clinicians should keep in mind that people with epilepsy may suffer another kind of psychosis, which occupies the *inter*-ictal phases, i.e., in the presence of alertness and sharp senses [22]. Its median duration is four months. This kind of psychosis has the characteristics of schizophrenia, including looseness of associations. Its etiological relation to schizophrenia and epilepsy is under study. Treatment recommendations are as for schizophrenia.

In Parkinson's disease (PD), hallucinations are in various modalities and surge steadily over the course of the illness; delusions are rare and correlate with the development of dementia [23,24]. Drugs used for treatment of schizophrenia are likely to worsen the motor symptoms of PD. Studies of the converse correlation, i.e., between duration of medication for PD and severity of hallucinations, remain inconclusive [24].

Step 8P relies on discerning a schizophrenia-specific psychological impairment. It is caused by degeneration of certain brain component, which is possible to remedy, more or less, with schizophrenia-specific medication. It is also possible to help patients with incompletely medicated schizophrenia recover a good measure of their social roles with schizophrenia-specific pyschotherapy and rehabilitation. As early as in 1994, research findings indicated that a defect in working memory is the fundamental psychological impairment in schizophrenia [25]. Working memory consists of the ability to preserve a number of perceptions, thoughts and urges activated concurrently in short term memory, "the mind's workbench", for immediate recall. It preserves them selectively, as related to the person's conscious priority, to the exclusion of unrelated mental developments. Since then, supporting evidence has proliferated [26,27]. Neuroimaging has visualized the neural correlates of working memory as brain -wide networks systems recruited variously in the successive stages of a task [26] Working memory dysfunction is found in almost all persons with schizophrenia, to some degree in the stage before the onset of clinical symptoms, as well as in unaffected relatives [2]. The schizophrenic defect of working memory shows as looseness of associations in the course of thinking (thought disorder) and acting. Sufferers are unable to adhere mentally to a priority while also contemplating and testing alternative ones. Instead, they become incoherent, drifting from one mental development to the next with no awareness or memory of what they left unfinished. Unlike ordinary slips of attention and memory, schizophrenic incoherence occurs recurrently, even while sufferers are motivated to avoid it.

Slight schizophrenic impairment shows as incoherence only in complex tasks. Metaphorically speaking, a hairline fracture in the leg may amount to disorder of dancing but not of walking. People with untreated mild schizophrenia learn to avoid complexity and novelty; they forego goals that become complex and rush to finish simple goals crudely. While speaking, they avoid tangents for fear of losing their sentence, e.g., searching for an exact word, observing others' reaction to make adjustments in mid-sentence, comparing and hypothesizing. Two vignettes will illustrate looseness of associations, one while handling a material object and the other while rethinking a delusional belief. Making a cup of coffee entails choosing among substitutable steps, going back to repeat a step, minding intervening unrelated priorities, etc, all that while still aiming to finish making the coffee in time. It is about mentally organizing the succession of one-step acts. A man with untreated mild schizophrenia might leave making coffee unfinished while searching for sugar; or, he would add sugar a second time, if he is reminded incidentally by the sugar-bowl on the table.

The second vignette is more relevant to the clinician-patient relationship. Patients with mild schizophrenia can debate their delusions coherently as long as they control the terms of the debate [9]. They recite lame explanations in circles and question the motives for every challenging question. However, if, for whatever reason, they must answer to the point, they will show a "surge of cognitive slippage" [9]. For example, a man with schizophrenia of moderate severity will drift with each next question, unable to return to his own earlier thread of logic that prompted the clinician's latest question. He may lose track of his grammar, interchanging "he" and "she" in a sentence; if the clinician points that out, the patient will start arguing hotly about pronouns, without memory of having wanted to reason about his delusion. Eventually, the man ends it all abruptly, citing for a reason the clinician's motives, now incorporated in his delusion. The patient says, "Huh! The FBI sent you to learn what I know about them." Next, he looks back at past events and finds strange justifications for this revelation. He does that mostly coherently, as long as he can disregard challenges to his reasoning.

Schizophrenia-Specific Treatment

Antipsychotic drugs were devised to remedy the specific biological abnormality of schizophrenia, but they have done so incompletely, especially in cases where medication starts late in the course of the illness or is interrupted often [20,28]. Similarly, certain psychosocial interventions were tailored to remedy schizophrenic incoherence, for patients who are incompletely medicated or refuse medication because of its bothersome side-effects [20]. These interventions help patients stay the course of a current goal to its completion, e.g., a conversation, making coffee or stacking shelves at a store. They help patients finish making sense of their initiatives in relation to immediate reality, contemplate priorities and commit to this or that goal. Making sense of the need for medication is usually necessary before patients may comply with the principle of early and uninterrupted medication. That benefit of medication is often not evident to patients; instead, others must draw the patients' attention to improvement in their ability to fulfill their intentions against adversities and distractions [13]. Patients' motivation to persevere through lengthy and bothersome trials of antipsychotics hinges on their judgment that medication will help them become useful and affable again. Remedial psychotherapy and opportunities for the patients' rehabilitation in a workshop and in the family are modalities integral to the treatment of schizophrenia.

Medication and other medical interventions

Antipsychotic drugs: Much research suggests that antipsychotic drugs are reparative for the biological dysfunction of schizophrenia, albeit usually incompletely and so variably among individual patients [20,29]. These references call antipsychotic drugs "the mainstay" of the treatment for patients with schizophrenia. They cite abundant evidence that medication with these drugs correlates with some correction of the cognitive impairment that is characteristic of schizophrenia. i.e., looseness of association. By remedying the root cause of the mental

disorder, more or less, antipsychotic drugs improve symptoms of schizophrenia globally, not just hallucinations and delusions [20]. Improvement includes thought disorder, autistic behavior (inability to stay tuned-in to others) and negative symptoms, such as blunted affect, withdrawal and psychomotor retardation. Compared to all other antipsychotic drugs, clozapine is the only one shown to have a small statistical advantage in terms of efficacy in treating schizophrenia and its subtypes. Antipsychotic drugs are more effective if used early, preferably in the prodromal phase of the illness, before it may manifest with the severity of psychotic symptoms. Even after psychotic symptoms begin to emerge, sometimes the benefit from medication is dramatic, rapid and virtually complete. Antipsychotic drugs lessen incoherence in non-psychotic phases of mild schizophrenia and in simple schizophrenia, which presents without hallucinations or delusions.

However, within a year, one quarter to one third of patients suffer some worsening of symptoms while still taking their medication [20,28]. Eventually, many patients end medication because of bothersome side-effects and three quarters of those suffer serious relapse within 6-24 months. On closer look, patients object to side-effects that outweigh the benefit from medication, which they measure by their ability to resume their social roles, not merely by attenuation of hallucinations and delusions. Even after the most crippling symptoms subside, patients often remain disabled enough to fail social tasks that others carry out effortlessly time and again, like thinking of what a joke meant while also keeping track of the other's next sentence. Worse yet, remissions become less complete than before each time medication is restarted, after intervals without medication. Laboratory tests with instruments measuring working memory show that incoherence of untreated schizophrenia generally stabilizes at a low point within a few years. It seems, however, that elements of the underlying degenerating biology remain responsive to medication for a little longer. That is the opportune time for clinicians to cultivate their patients' faith in medication. Even moderately effective medication improves patients' ability to participate in schizophrenia-specific psychotherapy and rehabilitation greatly. But the potential for a better quality of life that incomplete medication creates must be demonstrated to them. Better quality of life, in turn, motivates them to maintain medication, despite side-effects.

Many clinicians do not recognize the reparation of the fundamental schizophrenic deficit as the effect of antipsychotic drugs. Instead, they understand the effect of antipsychotic drugs as limited to certain symptoms, like hallucinations and delusions [30,31]. Clinicians then often generalize this understanding and use antipsychotic drugs stubbornly to target hallucinations and delusions in other kinds of psychosis, mostly with little or no effect (*Footnote*). From this chapter's outlook, "anti-schizophrenic" would be a more exact name for antipsychotic drugs. Bipolar disorder is one notable exception, where antipsychotic drugs have a similarly reparative effect. The explanation for that similarity will be probably found in the large overlap of genetic vulnerabilities between schizophrenia and bipolar disorder [32].

Non-specific medical interventions: Other medications used for schizophrenia are not schizophrenia-specific. Those often include antidepressants; less often, anticonvulsants and

polyunsaturated fatty acids (PUFA) [20]. It is important to remember that it is more likely to get improvement of depression, anxiety and impulsivity by searching for yet another, more effective antipsychotic and improving patient compliance than with antidepressants and anticonvulsants. Lingering or explosive negative moods (fear, anger, shame, anxiety, depression) are the consequence of frustrations while living with residual schizophrenia. The physiology of negative emotions is normal in schizophrenia, but current and anticipated challenges and failures rekindle it almost with no pause and with no end, as long as reparation of incoherence remains incomplete and patients face unaided. That is why medication that moderates negative emotions is never sufficiently effective and the need for it never ends. On the other hand, affective symptoms of residual schizophrenia cease reliably if someone helps the patient finish reasoning about ways to cope with challenges, despite residual incoherence, one challenge at a time. In the longer term, many patients with schizophrenia have a chance for better remission of incoherence, less disability, frustration and excessive emotions by trying a different antipsychotic. It is indicated that clinicians persist with testing alternative antipsychotics for greater improvement of incoherence once psychotic symptoms subside.

For schizophrenia patients with concurrent substance abuse also, "antipsychotic medications remain the mainstay of pharmacological treatment" [20]. Any degree of success with treatment of substance abuse disorders hinges on good treatment of schizophrenia. Evidence that antidepressants energize patients, to counter schizophrenia's negative symptoms, and alleviate secondary depression and anxiety, is mixed at best. There is better evidence that these outcomes are more likely by trying a next antipsychotic drug alone, by means of further life improvement and removal of sedation and extrapyramidal motor symptoms [20]. For anticolvusant drugs, carbamazepine and valproate, "the evidence is quite convincing that neither agent, used alone, is of significant value in the long-term treatment of schizophrenia" (excluding cases with schizo *affective* disorder) [20].

Polyunsaturated fatty acids are thought to be essential for neural development and lack of them has been implicated in a number of mental health conditions [33]. They have been used in schizophrenia alone or concurrently with antipsychotic drugs. "The… data [for PUFA] are intriguing but far from definitive" (20). Besides medication, other medical interventions introduced from the treatment of affective disorders are electroconvulsive therapy (ECT) and magnetic brain stimulation (rTMS). ECT offers a short-lasting benefit for urgent reversal of the most debilitating symptoms in cases resistant to all antipsychotic medication [34, 35]. Studies of rTMS have shown mixed results for reduction of hallucinations [34,36].

Development of new schizophrenia-specific drugs: Growing evidence that antipsychotic drugs remediate the fundamental biological abnormality in schizophrenia, more or less, has generated much investment in understanding the pathophysiology of schizophrenia and in developing drugs that act more selectively. Research begins with identifying "protein-encoding" genes that are a

"potential susceptibility" for schizophrenia (37). These genes are the template for the production of proteins necessary for transmission of signals between functionally related brain systems. Then, researchers test drugs that show affinity for the "molecular mechanisms and processes" that result from faulty protein production [38].

Psychoeducation, Cognitive Remediation, Psychotherapy and Social Rehabilitation: Medication is necessary but often not a sufficient treatment for schizophrenia. It depends on how much rehabilitation a patient may need in order to recover a measure of social fulfillment despite mild incoherence that often remains after diligent trials of antipsychotics. Patients are unlikely to persevere with medication merely for the benefit of symptom reduction; they will take their medication if it helps them also become loved and useful in a few relationships with the help of psychosocial interventions. What makes psychosocial treatment schizophrenia-specific? Its essential feature is that others serve as the patient's mental crutches, so to speak, when events unfold rapidly and intricately. For a man with residual schizophrenia, others can mind fulfillment on his behalf, i.e., as they understand his concerns and intentions. They can help him stay the course of choosing priorities and then bringing them to closure; they can lend him the function of working memory, of having drifted on tangents and distractions. First, psychosocial treatment should help patients complete practical goals for the benefit of their relationships, e.g., watching the news in someone's company or making dinner at the group home. Then, it should help patients complete thinking goals, e.g., contemplation of priorities, what they mean to others and others' intentions and trustworthiness. Most patients with residual schizophrenia are able to participate in this kind of *psychodynamic* psychotherapy, focused on judgments about immediate social reality. Still, clinicians who have not discerned incoherence as the fundamental psychological shortcoming and have not discovered how remediable it is do not offer psychodynamic psychotherapy. For patients, contemplating such judgments entails all kinds of thinking ramifications, like appraising priorities with a long-term view, side-stepping initiatives that troubled them before, rethinking their beliefs and values, computing others' feedback in mid-course, etc. During such intricate contemplation, patients suffer multiple discontinuities of attention, which show as hallucinations and other misperceptions of immediate reality, unfinished statements, approximate choice of words and emotions aroused for reasons that they can no longer know, etc. That is when they think and speak of explanations for all that experience in delusional terms. There are all kinds of compensation measures that can make much difference in mild or residual schizophrenia [13,39]. Others can help patients retrace their thinking and reality-testing initiatives, to selectively prune some of those tangents, and complete the rest. All that is effortful, but it is often sufficient for a degree of rehabilitation to the person's roles. Attainment of belonging again, one collaborative goal at a time, becomes the source of motivation for adherence to medication and to the rehabilitation routine. For patients, the effectiveness of medication is measured by the kind of social rehabilitation that it makes possible.

Unfortunately, much psychosocial treatment has been devised and delivered as isolated

modalities, whereas success with one modality is obviously an asset necessary for the next one. There is clear advantage for combinations of them as "comprehensive treatment programs", always with medication added [20,31,39]. Even then, programs often lack the element of psychodynamic psychotherapy that helps patients complete the thinking task of a) understanding their social priorities and, consequently, b) choosing treatment modalities, including medication, that in fact serve those priorities. What follows is a quick review of studies about the efficacy of isolated components of psychosocial treatment. It demonstrates those modalities' limited impact, for lack of generalization to the quality of the patients' social life. Then, this section about psychosocial treatments closes with stating the principles of schizophrenia-specific psychodynamic psychotherapy.

Psychoeducation: This modality consists of explaining to patients and their families the nature of the cognitive shortcoming, the life-course of the brain illness and the value of medication. It temporarily reduces severe relapses, especially when family participates [40]. But it has no effect on symptoms, social functioning and compliance with medication.

Social skills training: It entails training in elementary social skills with modeling, rehearsal and feedback [30]. Benefits are not likely to generalize.

Cognitive remediation therapy: This intervention is based on the idea that "training targeting basic sensory processes and simple cognitive functions will ultimately benefit higher-level cognitive operations" [41]. Patients undergo drills and practices on such simple tasks with modest improvement, which however generalizes to more complex tasks and social functions. Remarkably, the generalization is attributed to empirically measured regeneration of the schizophrenic brain's residual plasticity. It is likely that neural regeneration and task generalization occur when those basic practices are "combined with some other form of rehabilitation and… included strategy coaching" [41]

Cognitive psychotherapy: It consists of helping patients simplify challenging tasks or ask for help with them. It also helps patients rethink delusional explanations for their troubles [30]. One model [42] aims to reduce patients' worry by demonstrating to them reasons to doubt their persecutory delusions. It improves frequency of worry, rumination and irrational suspiciousness, but not relapses during crises in important relationships.

Psychodynamic psychotherapy: The trademark of this therapy is that it attributes errors and discontinuities in thought and action to latent motives and urges that interfere with the person's conscious activity from outside the person's awareness. Psychodynamic psychotherapy treats symptoms as expression of both at once, the conscious and the latent initiatives [30,13]. For schizophrenia specifically, therapists treat hallucinations as expression of greatly desired or feared developments while contemplating solutions for a particular problem. Then, they help the patient restart contemplating solutions with sufficient attention to anticipating developments strictly

in immediate reality. To treat delusions, therapists presume that, like everyone else, patients cling to a crude explanation for failing to make sense of events while, nonetheless, yearning to join someone else's reasoning for a better explanation. Then, they help the patient through the intricacies of such reasoning, instead of challenging the patient's crude conclusions. The author of this chapter finds that the model for "supportive psychodynamic therapy" by Rosenbaum and his colleagues [13] well articulated and well tested. That model emphasizes "development of self-agency" (p. 334), i.e., sustaining the patient's attention to fulfilling priorities relevantly to one's social roles.

Early intervention

1. The decline of responsiveness to medication over patients' life time inspired emphasis on treatment early in the course of the illness. Several programs tested treatment before or immediately after the emergence of psychotic symptoms [20,28,31,43,44]. The overall impression is that it delays the onset of psychotic symptoms and unresponsiveness to medication. But to attain, treatment must combine medication and psychosocial interventions for several years.

CONCLUSION. RATIONALE FOR TREATMENT PRIORITIES

The effectiveness of treatment for schizophrenic psychosis hinges on choosing schizophrenia-specific medical and psychosocial modalities. Patients measure the effectiveness of treatment initially by reduction of symptoms, but, in the longer term, they measure it by how treatment helps them recover their social roles, even despite residual symptoms.

It seems that antipsychotic drugs are schizophrenia-specific, reparative of the schizophrenic biological abnormality and root cause of all schizophrenic symptoms, although often incompletely and less effectively over the life course of the illness. Adjunct medication (for irrational fear, anger, depression and impulsivity) is less likely to be effective than testing yet one more antipsychotic drug is. Antipsychotic drugs are the mainstay of medication, therefore, it is essential to take advantage of their potential, which depends on using them early and without interruption. Patients comply with that medication principle if they have faith that medication will help them become useful in their family's and and the wider community's eyes again.

Cultivating that faith is accomplished with a combination of psychosocial interventions in the following *logical* order, although it may not be the order in which those steps are *in fact* taken. It begins with educating patients and their families about the rehabilitation potential with the medication's benefit and how to weigh that against the immediate adverse experiences and risks from the medication. Then, clinicians must demonstrate to the patients the difference that medication makes in their ability to use the clinicians' working memory as a crutch, to finish practical and reasoning tasks with others' help. Usually, that difference is not evident to patients, just as it is not evident to many clinicians, beyond the reduction of hallucinations and delusions. Finally, patients need opportunities for rehabilitation to their social roles staged directly in their

relationships in their family, a therapeutic residence or a workshop, with non-clinical partners who become the patients' mental crutch now and then.

Clinicians treating patients with schizophrenia periodically face a difficult predicament for which, however, there are good solutions. Patients begin treatment according to others' advice who see its potential on the patients' behalf. They are likely to refuse treatment later when they experience the adversities of medication. The adversities consist of side-effects, lengthy medication trials with little social progress, the effort and heartache of having to refashion their roles, sometimes against a trend to stigmatize and marginalize them. Patients are likely to resume medication in as much as they are able to understand why others see its potential and, furthermore, if they can have quick proof of it. Ironically though, untreated patients are too disabled to participate in reasoning about the merit of that potential, as others see it on their behalf, let alone getting quick proof of it. The solution usually lies in asking patients to give treatment a good chance because someone they love believes in it and rekindles their trust in that advice every day. Sometimes, patients comply for an interval of treatment because someone they fear believes in its potential on their behalf. For example, a judge may commit them to treatment after they acted dangerously. Intervals with medication fashioned in this manner must be exploited for proof of progress in social rehabilitation. Patient's memory of relief from certain symptoms and dangerous behavior will not suffice to keep them in treatment later, when adversities from medication and social stigma may worsen.

Schizophrenia cripples patients' future, as they had come to expect it, in a way that often remains incomprehensible to them. Still, recovering expectations for their future by means of treatment, as loved ones see the future on the patient's behalf, can become the patient's motive to persevere with the discomforts of medication and painful trials of social rehabilitation. Accumulating proof of social fulfillment, in turn, is for patients the necessary lesson for lifelong commitment to medication.

FOOTNOTE

In addition to their power to remedy the biological impairment of patients with schizophrenia, antipsychotic drugs mitigate anyone's normative or irrational fear, anger and anxiety. In that manner, they contribute to averting the emergence of hallucinations and delusions in posttraumatic disorders. Still, antianxiety drugs do the same more effectively and with fewer side-effects [18]. That contribution is a less important factor for reduction of psychotic symptoms in neurological disorders.

References

1. Maher BA. Anomalous experience and delusional thinking. The logic of explanations. In Delusional Beliefs, Oltmanns, T. F. , Maher, B. A, Eds. John Wiley , Sons, Toronto. 1988; 15-33.
2. Barch DM. The cognitive neuroscience of schizophrenia. Annu Rev Clin Psychol. 2005;1:321-353.
3. Aleman A, Larøi F. Hallucinations: The science of idiosyncratic perception. Washington, DC: American Psychological Association. 2008.
4. Langdon R, Ward PB, Coltheart M. Reasoning anomalies associated with delusions in schizophrenia.Schizophr Bull. 2010;36:321-330.
5. Bentall RP, Baker GA, Havers S. Reality monitoring and psychotic hallucinations. British Journal o f Clinical Psychology. 1991;30: 213-222.
6. Landa Y, Silverstein SM, Schwartz F,Savitz A. Group cognitive behavioral therapy for delusions: helping patients improve reality testing. Journal of Contemporary Psychotherapy.2006; 36: 9-17.
7. WHO (World Health Organization) International Statistical Classification of Diseases and Related Health Problems. 10th Revision, 2010.
8. APA (American Psychiatric Association) Diagnostic and Statistical Manual of Mental Disorders (Fifth ed.). Arlington, VA: American Psychiatric Publishing. 2013.
9. Chapman LJ, Chapman JP. The genesis of delusions. In T. F. Oltmanns, B. A. Maher (Eds.), Delusional beliefs. New York, NY: John Wiley , Sons.1988; 167-183.
10. Clark DA,Rhyno S. Unwanted intrusive thoughts in nonclinical individuals: Implications for clinical disorders. In D. A. Clark (Ed.), Intrusive thoughts in clinical disorders: Theory, research, and treatment. New York, NY: Guilford Press. 2005, 1-29.
11. Honig A,Romme MA,Ensink BJ, Escher SD,Pennings MH,DeVries MW. Auditory hallucinations: A comparison between patients and nonpatients. J NervMent Dis.1998; 186: 646-651.
12. Van Os J. Is there a continuum of psychotic experiences in the general population? Epidemiologia e PsichiatriaSociale.2003; 12: 242-252.
13. Rosenbaum B, Harder S, Knudsen P, Køster A, Lindhardt A, Lajer M, et al. Supportive Psychodynamic Psychotherapy versus Treatment as Usual for First-Episode Psychosis: Two-Year Outcome. Psychiatry . 2012; 75: 331-341.
14. Catani M, ffytche DH. The rises and falls of disconnection syndromes. Brain. 2005; 128: 2224–2239.
15. Liberman N,Dar R. Normal and pathological consequences of encountering difficulties in monitoring progress toward goals. In G. B. Moskowitz, H. Grant (Eds.), The psychology of goals (pp. 277-303). New York, NY: Guilford Press. 2009.
16. Laddis A. The Pathogenesis and Treatment of Emotion Dysregulation in Borderline Personality Disorder. The Scientific World Journal.2015; 11.
17. Laddis A, Dell PF. Dissociation and psychosis in dissociative identity disorder and schizophrenia. Journal of Trauma. Dissociation. 2012; 13: 397-413.
18. Laddis A. Medication for Complex Posttraumatic Disorders. Journal of Aggression, Maltreatment , Trauma. 20: 6: 645-668.
19. AGS (American Geriatric Society). A Guide to the Management of Psychotic Disorders and Neuropsychiatric Nymptoms of Dementia in Older Adults. 2011.
20. APA (American Psychiatric Association). Practice guideline for the treatment of patients with delirium. Am J Psychiatry. 1999; 156:1-20.
21. Henderson D. Delirium: Common Treatment No Better Than Placebo. Medscape Medical News. 2013.
22. Adachi N, Kanemoto K, de Toffol B, Akanuma N, Oshima T, Mohan A, et al. Psychiatric Disorders in Epilepsy. Basic treatment principles for psychotic disorders in patients with epilepsy. Epilepsia, 54: 19–33.
23. Ravina B, Marder K, Fernandez HH, Friedman JH, McDonald W, Murphy D, et al. Diagnostic criteria for psychosis in parkinson's disease: report of an NINDS, NIMH work group. MovDisord. 2007;22:1061–1068.
24. Holroyd S, Currie L, Wooten GF. Prospective study of hallucinations and delusions in Parkinson's disease. J NeurolNeurosurg Psychiatry. 2001;70:734–738.
25. Goldman-Rakic PS.Working memory dysfunction in schizophrenia. J Neuropsychiatry ClinNeurosci. 6:348-357.

26. Manglam MK, Ram D,Praharaj SK,SarkhelS. Working Memory in Schizophrenia. German J Psychiatry.2010; 13: 116–120.

27. Park S, Gooding DC.Working memory impairment as an endophenotypic marker of a schizophrenia diathesis Schizophrenia Research: Cognition. 2014; 1127-1136.

28. McGorry PD, Killackey E, Yung A. Early intervention in psychosis: concepts, evidence and future directions World Psychiatry. 2008; 7: 148–156.

29. Tandon RJ. Antipsychotics in the treatment of schizophrenia: an overview. Clin Psychiatry. 2011; 72:4-8.

30. Gaudiano BA. The Cognitive-Behavioral Treatment of Schizophrenia: The State of the Art and the Evidence. International Journal of Behavioral Consultation and Therapy. 2: 1-11.

31. van der Gaag M, Nieman DH, RietdijkJ, Dragt S, Ising HK, Klaassen RM, et al. Cognitive behavioural therapy for subjects at ultra high risk for developing psychosis: A randomized controlled clinical trial. Schizophr Bulletin. 2012; 38: 1180–1188.

32. Cross-Disorder Group of the Psychiatric Genomics Consortium, Lee SH, Ripke S, Neale BM, Faraone SV, Purcell SM, et al. Genetic relationship between five psychiatric disorders estimated from genome-wide SNPs. Nat Genet. 2013; 45: 984-994.

33. Amminger GP,Schäfer MR,SchlögelhoferM,Klier CM,McGorry PD. Longer-term outcome in the prevention of psychotic disorders by the Vienna omega-3 study. Nature Communications 6.2015; 7934.

34. Greenhalgh J, Knight C, Hind D, Beverley C, Walters S. Clinical and cost-effectiveness of electroconvulsive therapy for depressive illness, schizophrenia, catatonia and mania: systematic reviews and economic modelling studies. Health Technol Assess. 2005:1-156, iii-iv.

35. Petrides G,Malur C,Braga RJ,et al. Electroconvulsive Therapy and Other Brain Stimulation Therapies, Schizophrenia and Other Psychotic Disorders. American Journal of Psychiatry. 172: 52-58.

36. Voineskos D, Daskalakis ZJ. A Primer on the Treatment of Schizophrenia Through Repetitive Transcranial Magnetic Stimulation. Expert Rev Neurother. 2013; 13:1079-1082.

37. Tcheremissine OV, Castro MA, Gardner DR. Targeting cognitive deficits in schizophrenia: a review of the development of a new class of medicines from the perspective of community mental health researchers. Expert OpinInvestig Drugs. 2012; 21:7-14.

38. Lencz T, Malhotra AK. Targeting the schizophrenia genome: a fast track strategy from GWAS to clinic. Molecular Psychiatry. 2015;20**:** 820–826.

39. Kane JM, Robinson DG,Schooler NR, Mueser KT, Penn DL, Rosenheck RA, et al. Comprehensive Versus Usual Community Care for First Episode Psychosis: Two-Year Outcomes From the NIMH RAISE Early Treatment Program. American Journal of Psychiatry. 2015.

40. Lincoln TM, Wilhelm K,Nestoriuc Y. Effectiveness of psychoeducation for relapse, symptoms, knowledge, adherence and functioning in psychotic disorders: A meta-analysis Schizophrenia Research 2007; 96: 232–245.

41. Kaneko Y,KeshavanM. Cognitive remediation in schizophrenia. Clinical Psychopharmacology and Neuroscience. 2012; 10: 125-135.

42. Freeman D, Dunn G, Startup H, Pugh K, Cordwell J, Mander H, Černis E, et al. Effects of cognitive behaviour therapy for worry on persecutory delusions in patients with psychosis(WIT): a parallel, single-blind, randomised controlled trial with a mediation analysis. Lancet Psychatry. 2015; 2: 4305–313.

43. Marshall M, Rathbone J. Early intervention for psychosis. Cochrane Database Syst Rev. 2006;18:CD004718.

44. Hutton P, Taylor PJ. Cognitive behavioural therapy for psychosis prevention: a systematic review and meta-analysis. Psychol Med. 2014; 44: 449–468.