EMDR Therapy for Schizophrenia and Other Psychoses

Paul William Miller
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for Schizophrenia
and Other Psychoses
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Ad Dei Gloriam (To the Glory of God)

For my family: Dad, who taught me the meaning of hard work, love, and perseverance; my wife Nicola and children Jessica and Joshua, my heart, my home, and my safe place. Thank you for giving me the space and time for this project.

For Helen and Rosie: without you this book would never have been written; thank you for all your support.

E Pluribus Unum
An individual having unusual difficulties in coping with his environment struggles and kicks up the dust, as it were. I have used the figure of a fish caught on a hook: his gyrations must look peculiar to other fish that don’t understand the circumstances; but his splashes are not his affliction, they are his effort to get rid of his affliction and as every fisherman knows these efforts may succeed.

—Karl Menninger, psychiatrist
(Asylum to Action, Helen Spandler, 2006)
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Foreword

No psychiatric disorder is more shrouded in mystery, misunderstanding, and fear than schizophrenia. As Paul Miller notes, the original intention of Eugen Bleuler was for the diagnosis of “the schizophrenias” to represent a group of disorders. Upon examination of our contemporary scientific assumptions regarding schizophrenia in 2015, it is apparent that we may need to come full circle and carefully revisit the ideas Bleuler published in 1911.

The pendulum of mainstream psychiatry has, since the 1950s, swung overwhelmingly in the direction of endogenous and genetic models of schizophrenia. This mode of thinking has persisted despite the fact that the scientific data do not support a primarily genetic cause. As a specific genetic identifier is yet to be discovered, the current gold standard of evidence for genetically induced traits is monozygotic concordance. The classic twin study design relies on observing sets of twins raised together in the same family environment. Monozygotic (“identical”) twins share 100% of their genes, whereas dizygotic (“fraternal”) twins share only approximately 50% of their genes. Therefore, if a researcher compares the similarity for a particular trait between a set of identical twins to the similarity for said trait between a set of fraternal twins in that same family, then any excess resemblances between the identical twins should be attributed to genetics rather than to the environment.

So, for example, if we examine traits that are obviously genetic, such as race, eye color, or gender, we find 100% monozygotic concordance. Similarly, in medical diseases that have clearly shown genetic causation, such as Huntington’s chorea, cystic fibrosis, and Tay-Sachs disease, we also find 100% monozygotic concordance. In studies examining schizophrenia, in contrast, we find only 30% monozygotic concordance. In fact, more recent studies with refined methodologies have found only approximately 22.4% concordance. Thus, the data do not support the claims that schizophrenia is predominantly genetic in origin. They support the conclusion that 22.4% to 30% may have genetic causation, whereas 70% to 78% of schizophrenia’s causation is, therefore, nongenetic. This calls into question the confidence with which the medical and scientific communities continue to make these claims despite decades of empirical evidence to the contrary.

Although Eugen Bleuler’s stature as one of the fathers of the schizophrenia field has endured, his descriptions of schizophrenia have been forgotten. Many of his phenomenological descriptions are almost identical to modern portrayals of dissociative identity disorder (DID): for example, what Bleuler defines as “splitting” is synonymous with today’s definition of dissociation. He was furthermore aware that this group of schizophrenias also contained cases wherein...
a formal thought disorder, rather than splitting/dissociation, was manifest. Bleuler observed that in the cases that he described as dissociative, histories of trauma were often evident. In contrast, some cases that he described as manifesting florid thought disorders were not overwhelmingly driven by traumatic histories. We’ve celebrated Bleuler, and honored many of his conceptualizations, but have managed to completely forget some of his most cogent and important observations.

Further examination of the amnesia and confusion in our medical and scientific communities requires an exploration of the mainstream criteria for the diagnosis of schizophrenia: Schneider’s first rank symptoms. First rank symptoms (FRS) were first defined by Kurt Schneider as diagnostic of schizophrenia in 1959, at which point in time schizophrenia was already considered to be a purely genetic, endogenous thought disorder. Bleuler’s phenomenological descriptions of splitting, dissociation, and trauma were forgotten, whereas his classifications of schizophrenia were still considered the gold standard.

To date, despite the lack of consistent empirical support, modern diagnostic criteria of schizophrenia continue to give particular emphasis to Schneider’s FRS. Recent empirical explorations have noted numerous methodological flaws in previous studies that supported the diagnostic strength of FRS. The overwhelming majority of said studies suffered from insufficient sampling and methods of interview. An example: When examining whether FRS are predominantly features of schizophrenia, empirical standards dictate that it is necessary to examine whether the symptoms under examination are also found in nonschizophrenic patients, yet the overwhelming majority of studies lacked such a nonschizophrenic control group.

So, let’s examine what this means in real life. Although Schneider, in the original German text, did not explain in detail his 11 FRS, they are considered to be as follows: voices commenting, voices arguing, made feelings, made impulses, made actions, made influences on the body, thought withdrawal, thought insertion, voice or thought broadcasting, delusions, and hallucinations.

Reflecting on the foregoing, it should come as no surprise to anyone at this point that people properly diagnosed with DID consistently exhibit a majority of the first eight FRS (i.e., voices commenting, voices arguing, made feelings, made impulses, made actions, made influences on the body, thought withdrawal, thought insertion). They would thus appear to be symptoms of dissociation, not psychosis. They are often manifestations of alters speaking and of uncontrolled switching. In contrast, people properly diagnosed with the nondissociative schizophrenia thought disorder tend to overwhelmingly exhibit the FRS of voice or thought broadcasting, delusions, and hallucinations.

If this is known in the trauma and dissociation field, why is it ignored in the mainstream of psychiatry? Why do diagnostic interviews omit phenomenological explorations, such as “tell me about your life… what happened to you?” Is there some societal pressure causing us to cast a blind eye?

The author notes that Rolf Carriere, formerly of the United Nations, UNICEF, and the World Bank, has spoken of the “staggering global burden of trauma.” Indeed, recent epidemiological studies suggest that with the increasing rates of trauma worldwide, posttraumatic stress disorder (PTSD) is on track to
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become a major global public health problem. Despite its wide prevalence, PTSD continues, nonetheless, to be ignored or relatively underrecognized, with proper diagnoses complicated by stigma, comorbidity and symptom overlap, rigid onset criteria, and questionably high diagnostic thresholds.

Frank Putnam has argued that the study of dissociation, and DID in particular, appears to have been held to a different standard than that of any other disorder. Nowhere else has such a body of research, consisting of clinical case histories, series studies with structured interview data, and studies of memory, prevalence, neurobiology, and neuroimaging, utilizing samples of children and adolescents from North America, Europe, Latin America, Turkey, and Asia, been so entirely discounted.

Richard Lowenstein has opined that when viewed within a larger sociopolitical context, dissociation theory intersects with many of the most controversial social issues of modern times. The role of trauma in our culture, particularly intergenerational violence and sexual abuse, intersects with historically taboo subjects such as rape, incest, child abuse, and domestic violence, and their actual pervasiveness in our society. In addition, the study of trauma forces us to confront greater legal, social, and cultural questions related to peace and war, the implications of violence in our society, the meaning of good and evil, and even divergent religious views about the relationship between men, women, children, and the nature of the family.

Bessel van der Kolk, McFarlane, and Weisaeth (2007) contend,

A hundred years of research have shown that patients often cannot remember, and instead reenact their dramas in interpersonal misery. The professionals attending to these patients have had similar problems with remembering the past, and thrice in this century have drawn a blank over the hard-earned lessons. It is not likely that these amnesias and dissociations will be things of the past; they are likely to continue as long as we physicians and psychologists are faced with human breakdown in the face of overwhelming stress, which flies in the face of our inherent hubris of imagining ourselves as masters of our own fate, and as long as we need to hide from the intolerable reality of “man’s inhumanity to man.” (p. 67)

Thus, the understatement of the impact of the vast epidemic of world PTSD—driven by centuries of history written in the blood of colonialism, wars, slavery, pogroms and holocausts, global economic and natural disasters—should hardly surprise us. Is it any wonder that on a global level, science ignores Bleuler’s observations regarding trauma and dissociation, and their relationship to psychotic processes?

From a clinical perspective, several questions are raised. If we reach back to Bleuler and bring his phenomenology forward to the present, how would this affect our diagnoses of schizophrenia? One possibility is that Bleuler was referring to both what are currently considered cases of DID as well as nondissociative schizophrenic thought disorders. Another possibility, as Colin Ross has suggested, would be that Bleuler was addressing a whole spectrum of disorders, such as nondissociative schizophrenia, dissociative schizophrenia, schizo-
dissociative disorder, and DID. A third possibility, also proposed by Colin Ross, suggests that Bleuler intended to distinguish between dissociative schizophrenia and nondissociative schizophrenia; this would imply that we may not need the diagnosis of DID—we would simply follow Bleuler and discuss the various forms of schizophrenias. That would truly honor Bleuler’s recommendations.

The good news is that we need not immediately answer these questions in order to implement Dr. Miller’s clinical recommendations. We need only, as Colin Ross recommends, to begin to view these cases through a new lens, without necessarily reaching a definitive conclusion about their diagnoses. If we abide by Bleuler’s emphasis on a careful phenomenological exploration, it becomes rather clear as to what specific treatment these different cases require.

Dr. Miller notes that, with respect to eye movement desensitization and reprocessing (EMDR) treatment, the following presentations can be discerned: people diagnosed with psychosis who are affected by identifiable trauma that appears etiologically linked; people diagnosed with psychosis who experience the psychotic phenomena and/or their treatment as traumatic; and those in whom the PTSD and psychosis are comorbid, with the comorbidity acting as a perpetuating factor in their presentation. In this book, perhaps the most seminal and promising recommendation that Paul Miller makes is that EMDR treatment can address psychotic symptoms without needing to identify a dysfunctional memory network, but rather by processing directly the core beliefs that are driving the psychosis.

This scholarly offering is a clarion call for us to listen to our patients and to let their stories inform their treatment. This book is a major contribution to furthering the understanding of trauma in general, and the schizophrenias in particular. It is written with a wonderful warmth and an ever-so-subtle twinkle of humor that lurks just below the surface. Paul Miller’s ideas bring a healing sunlight to an area that has been encased for so long in darkness, and will open the doors to tens of thousands of people suffering from schizophrenia and other psychoses who have been denied effective comprehensive treatment. It is with the greatest of pleasure and admiration that I write this foreword.

Uri Bergmann, PhD
Past president, EMDR International Association
Author, Neurobiological Foundations for EMDR Practice

REFERENCE
Preface

I asked a dear friend and mentor to comment on what he thought of this book, and with his consent I share what he sent to me in reply:

Readers of this text will eventually end up in two categories:

1. Finally! A new approach, something radically different.
2. No! This is too radical, too different, too unproven.

My hope for the reader is that you avoid either category!

Let this text create arguments leading to a constructively improved adjunctive therapy for those who suffer and have not received sufficient help through traditional treatments.

Bring forward the doubts, the questions, the arguments: the constructive differentiations of these diagnostic categories.

Bring on the good that comes forth as you tentatively explore, implement, and personalize some of these ideas.

Stay united, as well, on the goal of all healers: to bring understanding, adaptation, comfort—a sense of wholeness—to those for whom suffering has been so poorly understood, so difficult for both the individual and for those whom he loves.

Walter Bahn, MSW

The biggest impact of any person on my clinical practice as a psychiatrist was a patient I will call Janus, for he was the ending of my thinking that schizophrenia and psychoses were untreatable with psychotherapy and the beginning of my journey that witnessed a person heal and get better, who met the strict DSM-III-R (Diagnostic and Statistical Manual of Mental Disorders [3rd ed., revised]—the diagnostic “bible” used for classifying mental illness) criteria for schizophrenia, which I learned to use as a member of Professor Kenneth Kendler’s Irish Schizophrenia Triad Study. At the time of writing, Janus is approaching 8 years symptom-free and medication-free; he has restarted his working life and is contented. I hope that these pages will entice you to make a similar journey.

I have always loved stories, and one of my mentors in psychiatry taught me that good psychotherapists are often good storytellers. Have a beginning, middle, and end . . . and have a point (i.e., meaning): These are the essential rules of narrative and they never need to be learned. We know them intuitively—from our earliest days we do not need to be taught how to understand or follow a
story; we are simply able to do so. This ability to generate narrative is a core part of the human condition and one that wreaks havoc when it ceases to function as designed.

I want to invite you to join me in a story, a journey through knowing and understanding a narrative that is in general about psychosis and in particular schizophrenia. If I follow the innate “rules” of narrative, then this journey will have a beginning, middle, and end and it will have meaning. I hope you will agree that I have stuck to this model when we finish our journey together. This story, like all good stories, has a number of subplots that create something that is bigger than any individual element: the story of psychosis and schizophrenia; the story of a client who changed my practice; the story of a woman, a “walk in a park,” and a community of open-hearted healers—Francine Shapiro and the EMDR therapy community.

This community introduced me to a wonderful and powerful psychotherapy that I have witnessed helping people in my community who have suffered from the internecine violence of what we in typically understated Ulster terminology called “the Troubles.” I have also seen it help bring wholeness and healing to clients with anxiety disorders, phobias, obsessive-compulsive disorder, and depression, but the most powerful impact on me was when I realized its efficacy in schizophrenia and psychosis.

One day, while listening to a radio program on my commute to work, I heard the host interview an expert who made the following comment: “We wanted Jack, who was a six-year-old boy, to ask some of the big questions about the universe and life; so that we could record them.” But they were unable to get the boy to do so, as he simply wanted to tell them what he thought the answers to such questions were. Jack already had a narrative in place that explained the universe as he saw it, “the sky isn’t black; it is just, really . . . really blue.” This process of narrative generation is what we do throughout life and it begins when we are children. We are born to narrate the world around us; this is what allows us to feel our way through life and to place ourselves in the world. Random observations and data taken into the mind are sorted and assimilated to generate meaning, and this process helps us to orientate and feel safe. This is essentially the adaptive information processing (AIP) system that Francine Shapiro describes, which underpins eye movement desensitization and reprocessing (EMDR) therapy (Shapiro & Maxfield, 2002). AIP is a natural, in-born system, and the AIP model is the soil in which we sow the stories of our clients. The AIP mechanism is like a cartographer making a map, which allows us to navigate our way through the unknown landscape of life ahead, and AIP is the map-making system with which we are all born. People and societies have always told stories to help understand themselves and to allow them to find their place in the world. Dáithí O’Suilleabhain, a friend of mine who is a cartographer, once explained how many indigenous peoples have stories and songs that teach them about their environment (O’Suilleabhain, personal communication, 2013). Often, as an elder walks a person through the landscape, the elder will sing his or her journey. These songs orientate individuals, allowing them to find their way, and also teach them how to look after the land.
Dáithí explained to me, following a trip to Belfast,

When place, story, and song come together, indigenous people call it singing the land. We sing it into existence by story and song. It then fills our awareness. We can sing it as we journey through naming all the features we see left and right as we pass, Slieve Scroob & Dromara, Slievenaboley & Aughnaskeagh, Lappoges & Dromore Donaghloney & Maheralin, Moira, Hillsborough & The Maze, Lisburn & Lambeg, Ballynahatty’s Giant’s Ring, Belvoir Forest. Queen’s City, hidden Blackstaff River and The Docks. Mountains and hills of Collin, White, Divis, Black, Wolf, Squires, Cave and Knockagh, and Mariners naming each rocky crag of coastline Lough. Singing it into existence, singing a journey through, singing it back to health. (O’Suilleabhain, personal communication, 2013)

However, what happens when the narrative process fails or flounders? Do we get meaningless jibber-jabber, or do we get a different form of narrative? In 1913, Jaspers built the foundation of psychiatric nosology on the dichotomy of neuroses and psychoses (Jaspers, 1913). For a long time, in the post-Jaspers age, we viewed psychosis as unintelligible and meaningless babble spewed out from the “broken mind” of the psychotic individual. No more worthy of study than spilled milk; all it represents was that a spillage had occurred. However, for those of us who are prepared to really listen, we discover that there is symbol and meaning in the psychotic material; this can at times be understood, and often it can provide access to the place where healing can be found. Schizophrenia is generally considered the most disabling form of psychosis; however, contrary to the assumptions of Jaspers, the mind of a person suffering with schizophrenia is not just a complex, broken machine with meaningless output; there is meaning in the symbols contained in the phenomena (delusions and hallucinations), and I believe that this principle is the key to seeing the negative cognitions and dysfunctional memory networks in psychosis as amenable to the psychotherapies such as EMDR therapy. We are creatures of symbols; they give us meaning, form the basis of language, and facilitate connection with each other. I want to invite us to think of the phenomena in psychosis as Indicating Cognitions of Negative Networks (ICoNN), and it is these negative networks that the EMDR therapist ultimately wants to reprocess; they are what Shapiro calls “dysfunctional memory networks” (DMNs) in her AIP model.

Example:

A man who comes to the clinic with a dog phobia presents his phenomena, and its functional impact, as an icon (ICoNN) of his underlying problem: the dysfunctional memory network relating to a time in childhood when a dog attacked him. Thus in EMDR therapy, considering his presentation within an AIP formulation, we identify and target the dog attack with the resulting functional outcome being that he is no longer afraid of dogs in the present.

In the standard eight-phase treatment model of EMDR therapy, the target is the past event that connects us to the dysfunctional memory network, its affect and negative cognition. In the previous example, the target would be
an aspect of the original dog attack. Yet, as the EMDR therapy model has been evolving clinically, we see authors delineating protocols when the target is in the present. We see this in Elan Shapiro’s Recent-Trauma Episode Protocol (R-TEP; Shapiro & Laub, 2013) and Robert Miller’s Feeling-State Addiction Protocol (FSAP; Miller, 2010).

The R-TEP is an adaptation of the basic EMDR therapy protocol for treating recent traumatic memories—it is useful when the traumatic memory has not yet been consolidated or integrated into memory. The R-TEP protocol is a brief intervention (possibly on successive days) that may be used not only to treat acute distress, but also to provide a window of opportunity to prevent future complications from occurring and to strengthen resilience. Early intervention with EMDR therapy seems to reduce the sensitization and accumulation of trauma memories by means of rapid reduction of intrusive symptoms and a de-arousal response. R-TEP incorporates and extends existing EMDR therapy protocols together with additional measures for containment and safety.

The FSAP allows clinicians to work with substance and behavioral addictions, such as gambling compulsions, sex addictions, and smoking, that have been notoriously resistant to treatment. The feeling-state theory (FST) of addiction presents a new understanding of the etiology of addiction, hypothesizing that addictions are caused by a fixation of a positive feeling event. Afterward, whenever the person wants to experience that feel-good feeling, the link with that particular behavior is triggered. By utilizing this model of addictive behavior, Robert Miller has delineated the FSAP as a modified form of EMDR therapy that helps the client to break the fixation, resulting in the resolution of behavioral addictions with the elimination of the urges and cravings of substance addictions.

This book introduces the ICoNN approach, which is an adaptation to the standard eight-phase protocol that is helpful in working with psychosis. The ICoNN approach is similar to the R-TEP and FSAP, as the modification of the standard eight-phase, three-pronged protocol is in respect to target identification, which allows psychotic phenomena to be used as targets for reprocessing by a clinician using EMDR therapy. Once we repatriate schizophrenia into the spectrum of disorders with which it was originally associated, which is to say, “those which can be interpreted by a dissociation model” (Moskowitz, Schäfer, & Dorahy, 2008, p. 62), we can more understandably apply a trauma-focused formulation to schizophrenia in particular and psychoses in general; then the potential effectiveness of the psychotherapies, such as EMDR therapy, in psychotic disorders becomes all the more apparent.

We have been speaking about psychosis and are also mentioning dissociation, seemingly interchangeably, and I have spoken of returning schizophrenia to the category of dissociative disorders. Dr. Colin Ross looks at the clinical phenomenological conundrum of “psychosis or dissociation” and states, “Because of the way the two disorders are defined in the DSM-IV-TR and the clinical and research literatures, they cannot be separated into two discrete categories. They are too much and too often the same thing” (Ross, 2004, p. 16). I concur with his opinion.

Essentially, from a psychological perspective, if trauma results in a failure of narrative generation, this results in the formation of a DMN; this in turn is
adapted to through dissociation, which suggests that if the DMN driving the dissociative (psychotic) psychopathology can be processed, then the pathology should resolve. This theory is in keeping with the AIP model of EMDR therapy. We will see from the small amount of case material that is presently available and from current international research that this is indeed what can be observed clinically in some, and those patients who respond positively can achieve long-term symptom control without the need for medication. Despite having more than 100 years of experience with the mental disorder formulated as schizophrenia, only a minority of cases can be said to make a full recovery; this observation invites us as clinicians and scientists to be, at the very least, curious about the apparent response to psychotherapeutic interventions. In many fields of science we have suffered from a them-and-us form of trench warfare: psychology versus psychiatry, nature versus nurture, and drug therapy versus talk therapy. I want to invite us to be more integrative and to consider allowing the content of this book’s journey to inform a third space where we can allow dissonance to form and, I hope, to eventually bring clarity. Richard Rohr describes such a position well, and I share a section from a recent daily contemplation from his blog to give us a context for the “third space” in which I am inviting you to join me:

The House that Wisdom Builds—“Paradox” comes from two Greek words: para + dokos, meaning beyond the teaching or beyond the opinion. A paradox emerges when you’ve started to reconcile seeming contradictions, consciously or unconsciously. Paradox is the ability to live with contradictions without making them mutually exclusive, realizing they can often be both/and instead of either/or. G. K. Chesterton said that a paradox is often a truth standing on its head to get our attention! “Dialectic” is the process of overcoming seeming opposites by uncovering a reconciling third. The third way is not simply a third opinion. It’s a third space, a holding tank, where you hold the truth in both positions without dismissing either one of them. It often becomes the “house that wisdom builds” (Proverbs 9:1–6). It’s really the fruit of a contemplative mind. (Rohr, 2014)

So, with this in mind, let us indulge our curiosity in our own third space.

“Curiosity has its own reason for existing. One cannot help but be in awe when he contemplates the mysteries of eternity, of life, of the marvellous structure of reality.”

—Albert Einstein

(Recollection of a statement to William Miller, an editor, as quoted in LIFE magazine, May 2, 1955)

My own professional journey with EMDR therapy began in 1997 when a colleague, Dr. Michael Curran, invited me to attend an EMDR therapy training organized by Humanitarian Assistance Programs (HAP), which was being run to help the local mental health professionals in Northern Ireland in dealing with the violence of the previous decades: “the Troubles.” The training was delivered
in the city of my birth, its very name a shibboleth—Derry/Londonderry—as it remains so for many, and I have witnessed too many people who have been attacked and injured just for calling it the “wrong” name. All these things meant that it was a very appropriate birthplace for an area of my professional practice that is responding to the pain and hurt of the violence of the Troubles and for trauma in general. In that training I was excited to hear and experience this “new” therapy that was linking in with a developing neurobiological understanding of the psychological impact of a traumatic event. It also appeared readily scalable, as it built upon the existing professional skills of mental health professionals and has at its core the innate information processing of the human mind. I believed that it could provide much-needed treatment for posttraumatic stress disorder (PTSD) and the other psychological sequelae of trauma to a community that needed healing. EMDR therapy’s potential as a readily scalable psychotherapy for the treatment of posttraumatic psychological conditions was to be later picked up famously by Rolf Carriere, a development economist who worked for the United Nations and the World Bank. After he read Dr. Shapiro’s book, while a UNICEF representative in Bangladesh in the 1990s, he saw EMDR therapy’s potential for the people of Bangladesh who had been traumatized through a violent war of independence; 54 Bangladeshi psychiatrists and psychologists were initially trained after he serendipitously picked up Dr. Shapiro’s first book on the therapy (Carriere, 2013).

As in the United States (Manfield, 1998), Northern Irish psychiatry in the early 1990s was not generally accepting of EMDR therapy, which was treated with much suspicion; many viewed it as a dressed-up and repackaged form of cognitive behavioral therapy (CBT) at best. I recall a time, while at a revision course for my exams for membership in the Royal College of Psychiatrists, when trainers were asking with which psychotherapy models we had experience; they looked with derision on EMDR therapy. I was repeatedly told that EMDR therapy was just a technique, and a colleague recently told me that a senior colleague had once told him that as a practitioner of EMDR he ought not to consider himself a psychotherapist.

At the same time, in the field of psychotherapy, CBT was the rising star, and there was little space for other modalities. Nonetheless, I began to see people recovering from their traumas as I applied an EMDR therapy paradigm to their presenting problems, which largely consisted of PTSD. One of my favorite quotes that characterizes that period in my professional development is by Dag Hammarskjöld, a Swedish diplomat, the second United Nations Secretary-General, and Nobel Peace Prize recipient: “Never, ‘for the sake of peace and quiet,’ deny your own experience or convictions” (Hammarskjöld, 1966, p. 84). When working with victims of the violence of the Troubles and of civilian trauma—road traffic collisions and childhood sexual abuse, for example—I began to see remarkable healing: I witnessed patients’ recovery and was powerfully impacted by this. Whereas some mental health professionals were negative or merely apathetic, Dr. P. S. Curran was supportive and encouraged me, saying, “the patients don’t read the textbooks.” As a consequence, they didn’t know that they were not supposed to improve, and so despite there being no strong published evidence at that time, I found my patients getting better. As I write this, there are now
more than 30 randomized controlled trials (RCTs) worldwide examining EMDR therapy; the patients still haven’t read them.

The battle for the recognition of the efficacy and validity of EMDR therapy was led in Northern Ireland by pioneers in the field such as Dr. Des Poole, who brought EMDR therapy to Northern Ireland; and the advocacy of Dr. Michael Paterson OBE was key in having the Clinical Resource Efficiency Support Team (CREST) include EMDR therapy in its 2003 guidance for the psychological treatment of PTSD, guidance that preceded the wider UK National Institute for Health and Care Excellence (NICE) Guidelines. Many people are now benefiting because of these endeavors. The EMDR therapy community in the island of Ireland is now healthy and growing, with many active and openhearted participants.

We are also at a time of a highly significant paradigm change for the EMDR therapy community. Pathfinders forge ahead and make the path easier for those who follow. In the United Kingdom (UK), enormous efforts and commitment by Dr. Derek Farrell have resulted in the development and delivery of the first university-based EMDR therapy training at the University of Worcester, where it is delivered as a Masters in Science course (MSc). I am fortunate to be in the first group of EMDR Europe Trainers-in-Training who are being mentored by Dr. Farrell; the group has been drawn from the UK, Ireland, Greece, and Pakistan, and as a group we will be endeavoring to develop the research and further study and growth of EMDR therapy through academic training. We as trainers will be contributing to the teaching of the course, and many of us believe that this shift from an essentially entrepreneurial model of EMDR therapy training to an academically focused one will mark a paradigm shift for EMDR therapy training that is as vital and as important as the delineation of EMDR therapy itself. These are exciting times, and this book is written for such a time as this, where innovation can be tested and cupellated in the crucible of academia for the benefit of humankind and not for seeking fame, reward, or a bigger bank balance.

I have now had the great honor to hear Dr. Francine Shapiro, the originator of EMDR therapy, speak many times, and she has been a source of great support and encouragement for me. She has called me the “father” of this area of development for EMDR therapy: the application of EMDR therapy in schizophrenia and psychosis. If I am the father, she must surely be the “mother,” for I could never have birthed this area of innovation without her. At the start of her keynote addresses, she asks the audience to indicate with a show of hands to which areas of clinical practice they are applying EMDR therapy. I have witnessed responses that cover depression, obsessive-compulsive disorders, addictions, pedophilia, the reduction of self-harming behaviors, and, of course, psychosis; her response is always the same: “publish.” There is now a solid research literature examining EMDR therapy for PTSD, but the other clinical areas continue to lag behind. This is something that I believe will change fundamentally as we move EMDR therapy training and study into mainstream academia. I hope that this book will further encourage the exploration and research of the clinical applications of EMDR therapy, and to achieve that goal I believe that we need to take some “radical” approaches. I believe that this radical change will come through a return to the roots of the phenomenology of schizophrenia, with a repatriation of “the schizophrenias” into the category of psychiatric illnesses that can be framed within a
dissociation model (Moskowitz et al., 2008) that allows a trauma-focused formulation of cases to be made (Miller, 2014). Such a formulation sits readily within the architecture of the AIP model if we postulate that trauma leads to a derailment and failure of the AIP system, resulting in a dysfunctional memory network. I propose that by targeting and reprocessing the dysfunctional memory network through the biological facilitation of memorial processing, by the actions of dual attention stimulation/bilateral stimulation (DAS/BLS) acting via stochastic resonance (SR), we can achieve resolution of the psychotic phenomena; this is indeed what I have observed. As the AIP model is the central paradigm upon which EMDR therapy is built, I wish to look at it in more detail next, and we will similarly explore how the proposed innate mechanism of SR, which is believed to be ubiquitous throughout nature, has a key role too.

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Acknowledgments

My early experience in psychiatry attracted me to the multidisciplinary team environment of psychogeriatrics (the psychiatry of old age), with the integration of mind and body formulations and treatments. Psychiatric colleagues such as E. Anne Montgomery, Stephen Compton, Jill Gilbert, and Noel Scott, to name a few, stoked my love for working with the over-65-years age group, and I am grateful for that. I found the field of old-age psychiatry to be richly multidisciplinary, maintaining a healthy connection with the brain, thus retaining a holistic approach.

The first consultant psychiatrist under whom I worked was Peter S. Curran, a local Northern Irish expert in psychological trauma, based at the Mater Hospital. I am grateful for his support and mentorship across the years. He taught me to respect every member of the team when I started in the Mater, Belfast. Subsequently, F. A. (Tony) O’Neill and Professor Kenneth Kendler gave me a wonderful opportunity within the GEMINI team, the Northern Irish arm of the Irish Schizophrenia Triad Study. This was a genetic epidemiological exploration of schizophrenia on the island of Ireland, and the training in the phenomenological assessment of schizophrenia that these men and the wider team gave me continues to be a wonderful gift.

To my many friends and colleagues in the EMDR therapy community—where to start and end is nearly as big a challenge as writing this book, but I must name a few: Francine Shapiro, Robbie Dutton, Udi Oren, Uri Bergmann, Mark Dworkin, Jim Cole, Robin Shapiro, Jim Knipe, Frank Corrigan, Arne Hofmann, Ulrich Lanius, Carol Forgash, Katie O’Shea, Zona G. Scheiner, Derek Farrell, Derek McLaughlin, Anabel Gonzalez Vazquez, Des Poole, John Swift, Mary Mitchell, Marshall Wilensky, Sue Genest, and Peter Mulhall. My fellow EMDR Europe Trainers-in-Training at the University of Worcester have also been wonderful team members who have walked alongside me throughout this project; they are Saleem Tareen, Rashid Qayyum, Lorraine Knibbs, Lynn Keenan, Paul Keenan, Gus Murray, Penny Papanikolopoulos, and Tessa Prattos—thank you all.

I have been encouraged greatly by those in the EMDR therapy and wider trauma community who are utilizing psychotherapy for psychosis and schizophrenia: Akiko Kikuchi, Anabel Gonzalez, Andrew Moskowitz, Colin Ross, Daeho Kim, Jim Knipe, Karen Forte, and Martin Dorahy. You are an inspiration. In particular I must mention Colin Ross; he opened my eyes to the potential role of EMDR therapy for people diagnosed with schizophrenia. I stand on the shoulders of giants.
ACKNOWLEDGMENTS

This journey has led me to explore how we, as humanity, have approached these experiences. I am especially grateful to the following people who were gracious enough to give me time and the benefit of their experience. I was blessed to talk with the following about their work and gained some wonderful insights: Phil Borges, Walter Bahn, Hal Stone, Sidra Stone, and Professor Marius Romme. I must also thank in particular some people whose writing has greatly influenced me in my journey: Joseph Campbell, Richard Rohr, M. Scott Peck, and Robert Johnson.

To those who proofread and helped with shaping the chapters—Helen Harbinson, Alastair Clarke-Walker, Aaron Brady, and Derek McLaughlin—thank you for giving so generously of your time. I am especially grateful to those who coauthored chapter sections with me: Remy Aquarone, Mark Dworkin, and Derek Farrell. Of course, none of this input would have been necessary were it not for Marilyn Luber and her kind introduction to Sheri W. Sussman at Springer Publishing Company. Sheri has “tickled” me over the years this project has taken, with the recent assistance of Alina Yurova. Thanks for your patience with me and for your tenacity with this project.

This book is a testament to a community of people; I hope that you find something in it that is of value. My prayer is that in the crucible of clinical practice and peer review, the “hay and straw” will burn up and the “gold and silver” remain as a good work, well done.

“The way to get things done is not to mind who gets the credit.”

Introduction

EYE MOVEMENT DESENSITIZATION AND REPROCESSING (EMDR) THERAPY AND THE ADAPTIVE INFORMATION PROCESSING (AIP) MODEL

Eye movement desensitization and reprocessing (EMDR) therapy is an integrative psychotherapy developed by Dr. Francine Shapiro (Shapiro, 2001). In 1989, she published the first research data examining and delineating the therapy, while a Senior Research Fellow in Palo Alto, California (Shapiro, 1989). Through the endeavors of the clinical community, EMDR therapy has gained recognition as an efficacious therapy for the treatment of posttraumatic stress disorder (PTSD). In the United Kingdom (UK), it has been recommended as a gold standard in the psychological treatment of those suffering from PTSD, being first recommended by the Clinical Resource Efficiency Support Team (CREST) in its document that gave guidance on the psychological management of PTSD, The Management of Post-Traumatic Stress Disorder in Adults (CREST, 2003). CREST operated in Northern Ireland, a UK region that has seen substantial loss of life and experienced severe trauma within its relatively small community of around 1½ million people. This was due to the internecine violence colloquially referred to as “the Troubles.” The work of CREST was duly followed in the rest of the UK with a similar recommendation by the National Institute for Health and Care Excellence (known as NICE; NICE, 2005); and most recently the World Health Organization (WHO) published WHO Guidelines on Conditions Specifically Related to Stress, which recommends the use of EMDR therapy in the treatment of PTSD for children, adolescents, and adults—see specifically recommendations 14 and 15 (WHO, 2013).

ADAPTIVE INFORMATION PROCESSING, PSYCHOPATHOLOGY, AND MALADAPTIVE ENCODING OF TRAUMATIC LIFE EXPERIENCES

Rolf Carriere has spoken of the “staggering global burden of trauma” (Carriere, 2013), and I believe that it is our duty to respond to this huge area of need. I have heard this articulated in many forms, but essentially it comes down to the same thing: hurt people, hurt people. As we will see later from the discussion of the epigenetics and neurobiological effects of trauma on the body, this cycle need not continue (Pembrey et al., 2006; Waterland & Jirtle, 2003; Weaver et al., 2004). A foundational postulate of the AIP model and of EMDR therapy is that we are
all born with an innate information processing system that takes the experiential data of our lives and processes it into a cohesive, coherent, and contiguous narrative that allows us to make sense of the world around us and of our place in it. This system need not be learned or studied by the patient before he or she can benefit from its functioning. However, it is this same information processing system that can get derailed in a trauma, resulting in dysfunctionally stored material, which results in the pathologies we see clients presenting with in our offices and clinics. In the late 1880s, pioneering French psychologist, philosopher, and psychotherapist Pierre Janet developed detailed and comprehensive models of dissociation and traumatic memories (van der Hart & Dorahy, 2006). Although some have sought to erroneously state that he came to later repudiate his theories on dissociation, the evidence does not support this (Dorahy & van der Hart, 2006). Janet stated that traumatic memories are distinct from normal “bad” memories and postulated that they are stored differently in the brain and have differing properties—something that to my mind is very much a foundational aspect of the AIP model. These unprocessed, state-specific, frozen memories are conceptualized as dysfunctional memory networks (DMNs) by Shapiro (2007), and I believe that just as Jung’s “complex” derives from Janet’s “fixed idea,” the DMNs of the AIP model belong to the same lineage. Consider Jung’s description of a complex in his 1934 review, quoted by Moskowitz (2006, p. 14),

What then, scientifically speaking, is a “feeling-toned complex”? It is the image of a certain psychic situation which is strongly accentuated emotionally. . . . This image has a powerful inner coherence, it has its own wholeness, and in addition, a relatively high degree of autonomy . . . and therefore behaves like an animated foreign body in the sphere of consciousness. (Jung, 1934/1960)

In this same review in 1934, Jung describes complexes as having a trauma at their genesis: “The aetiology of their origin is frequently a so-called trauma, an emotional shock or some such thing, that splits off a bit of the psyche (Jung, 1934/1960)” (Moskowitz, 2006, p. 14). This is also how Janet described the formation of his “fixed idea,” and Jung acknowledges his debt to Janet, as I believe we must do too in regard to the DMN.

PIERRE JANET—ONE OF PSYCHOTRAUMATOLOGY’S GIANTS

I believe that we stand on the shoulders of giants; indeed, it is our duty to do so, and one such giant in the area of psychotraumatology is undoubtedly Pierre Janet. We will learn later in the book that Janet’s work had a significant influence on Bleuler and Jung (Moskowitz, 2006; Moskowitz, Schäfer, & Dorahy, 2008). The AIP model allows us to see further along this course of study, as it explains the basis of pathology, predicts successful clinical outcomes, and guides the clinician in case conceptualization (formulation) and treatment procedures (Shapiro, 2007). Within this innate information processing system, we take the experiences of the outside world and process them, stripping them of extraneous data, automatically linking the perceptions of current situations with associated memory networks already in existence.
Everyone reading this book will know that one plus one equals two—it forms a part of our mathematical understanding—but very few of us, if any, will recall where we were and who we were with when we learned it. We simply do not need that level of information. When the AIP system processes new experiences, the incoming sensory perceptions are integrated and connected to related information that is already stored in the person’s memory networks. This conceptualization informs the intention and sequencing of the eight phases of EMDR therapy, and we know that the more closely treatment adheres to the eight-phase protocol, the better the clinical outcome (Maxfield & Hyer, 2002).

THE DOG BITE

If we explore a clinical example, this will aid our understanding of how the AIP model benefits us. Think of an individual who has had the experience of being bitten by a dog as a young boy; such an experience can be sufficiently negative and emotionally charged to overwhelm the innate processing system, resulting in unprocessed material. This material, we conceptualize, becomes stored as a DMN containing emotions and perceptual information in state-specific form. This DMN is cut off from the processed, functionally encoded, coherent memories that already exist in the person’s mind—perhaps happy memories of playful interaction with a dog—but it also remains cut off in the person’s future experiences. The unprocessed material can remain walled off like an abscess within a patient’s body, and it is this “psychic abscess” that can be triggered by idiosyncratic present experiences, manifesting disorder. The boy grows into a man and the DMN remains walled off from any new learning. So when he sees a dog of the same breed that bit him as a boy, this acts as a trigger to summon the DMN. The key characteristics of this DMN are that it exists outside of context and chronology and is stored in state-specific form—meaning it is frozen in time in its own neural network, unable to adaptively connect with other memory networks (Solomon & Shapiro, 2008). In this unprocessed form, it is relived rather than remembered, in line with Pierre Janet’s model for dissociated traumatic memories (Janet). So considering our current example, even if a strong adult male was bitten by a very small dog such as a Chihuahua when he was a boy, the DMN—with its strongly negative state-specific perceptions, feelings, and cognitions—is triggered in the present by seeing a Chihuahua; this results in an intense emotional response. He may become frozen in fear in the presence of this breed of dog even though the actual risk to him is negligible in the present. In this example we can see how the AIP model predicts pathology and helps us to understand the client’s current presentation—that is, a severe fear response to and avoidance of Chihuahuas. The DMN is fear-laden and associated with “adaptive” avoidant behaviors triggered by this specific breed of dog.

GETTING THE RIGHT TARGET FOR EMDR THERAPY

The AIP model directs the clinician to target the original trauma, which, like a pollutant entering a river, poisons everything downstream of its point of entry. In this metaphor of a polluted river, we can see that the best response is to
remove the pollution at the source, rather than to merely decontaminate the river downstream, and this is what the AIP model directs us as EMDR clinicians to do; we do so within the eight phases of the treatment model. The processing of the original trauma (the source of pollution) links the previously unprocessed material with existing functional memory networks and so removes the drive for pathology in the present and future, and this is indeed what we observe (Shapiro, 2007; Shapiro & Forrest, 1997; Shapiro & Maxfield, 2002). In EMDR we examine the past, present, and future within what is called the “three-pronged process.” This three-pronged process sits within the AIP model, and essentially the clinician processes the past “unmetabolized” DMN that generates the presenting pathology, processes the present situations that cause disturbance, and generates an adaptive future template to allow the individual to facilitate effective future action (Manfield, 1998). As already mentioned, the growing neurobiological understanding of memory processing and the effects of trauma help us to understand the nature of the eight phases in the standard protocol and the logic for their sequencing. We can unpack this further if we consider the neurobiology of normal information processing according to the AIP model. Those wishing a deeper understanding of the current research on the neurobiological foundations of EMDR practice are directed to read Uri Bergmann’s book *Neurobiological Foundations for EMDR Practice*, which I consider a seminal text in this area of study and that I recommend to you (Bergmann, 2012).

**SCHIZOPHRENIA/PSYCHOSIS AND THE TRAUMA MODEL**

As the nature/nurture debate continues, I hope that we can hold both these considerations in a “third space,” as ultimately this will provide clarity and give the best hope of healing to this patient group. When we examine schizophrenia by formulating it within a trauma model, this allows us to consider the application of psychotherapies with a trauma focus. EMDR therapy is one of the current international gold-standard psychotherapies for PTSD, and early outcomes of its application to schizophrenia have been encouraging (Kim et al., 2010; Miller, 2010, 2014); however, more work is warranted. Of course, by holding to the principle of third space we can choose not to get stuck in the debate of talking therapy versus drug therapy (either/or), and instead we can embrace a both/and approach. This makes sense clinically, as psychotherapy will not necessarily exclude the need for drug therapy completely in all cases. As clinicians we commit ourselves to lifelong learning; psychiatrists and doctors refer to our work as a medical “practice”—we are not yet getting it perfectly right. If you think you are, perhaps you ought to think again.

**LIFELONG LEARNING**

Francine Shapiro and the works of Colin Ross (2004, 2013), Jim Knipe (personal communication, 2014; 2015), and Carol Forgash (Forgash & Copeley, 2008) have been extremely influential. Their work encourages me to explore this area of EMDR therapy. I still recall the conversation that Jim Knipe and I had in Philadelphia, sitting outside in the sun during an EMDR International Association
INTRODUCTION

Research is not a simple and straightforward endeavor; it is difficult, complex, and challenging. However, seeing the commitment of individuals like Tony O’Neill and Kenneth Kendler encouraged me to take the risks and work toward getting the necessary research done and published. At the start of my research MD thesis (Miller, 2007), I quoted the words of Barbara W. Tuchman (American author and two-time Pulitzer Prize winner): “Research is endlessly seductive: writing is hard work” (Tuchman, 1979, p. 34).

Research and the statements based upon it have consequences—unintended as well as intended. We see this in the nosological journey that the Diagnostic and Statistical Manual of Mental Disorders (DSM) itself has taken with disorders such as PTSD. I believe that it is in community that we heal and can be healers; therefore, we need to understand mental disorder as it is experienced within and through a community context. The importance of the intersubjective within the EMDR therapy method is greatly enriched through the teaching of Mark Dworkin (2005). We are required to be fully present and connect with one another to undertake good therapy. It will therefore come as no surprise that I believe that healing takes place, in psychosis, schizophrenia, and dissociative disorders, within the intersubjective space. This is where we connect in the milieu of the very nature of our consciousness. At the start of a book that explores shell shock, the following appears: “A French doctor has said, ‘Il n’y a pas de maladies’ [There are no sicknesses, there are only sick people]” (Smith & Pear, 1917). I have been taught that those who are wounded in the crucible of community must heal in community, and this is an important consideration for the people who seek my help. The EMDR therapy community is eclectic, and, like the function of rapid eye movement (REM) sleep, I found that upon reaching out and forming new associations, I was able to advance the development of the Indicating Cognitions of Negative Networks (ICoNN) model. The other communities where I have witnessed healing work are mythopoetic support groups, the community of faith, and the ManKind Project (MKP). These communities are all rich with story, and I have come to appreciate through them the power of mythos as a healing dynamic. When we complete research, as we ought to, or even reflect upon our clinical work, we should share the outcomes—not keep the research to ourselves, because that is shortsighted. We need to share it, present it at conferences, and publish it, so that it can be examined and debated. I am committed to lifelong learning and continuing professional development. Colleagues teach me much; books and journals teach me something else, but it is the journeys that I make with people, like Janus, that teach me the most. I see their courage to share and seek healing in a safe community. This, more than anything, encouraged me to explore EMDR therapy’s applicability to schizophrenia and psychosis.

LIGHTING A BEACON FIRE

I have, within these pages, the opportunity to explain and articulate my position on EMDR therapy for schizophrenia and the other psychoses. EMDR therapy is a powerful psychotherapy, but it is not a panacea. Neither ought it to be undertaken by those unfamiliar with the treatment of schizophrenia/psychosis. As the motto of the Royal College of Psychiatrists states, “Let Wisdom Guide.” This
work in the area of psychosis and schizophrenia is fledgling and requires more research and critical examination. I heard the following story about a theologian who once visited a university chaplain:

The theologian observed that the chaplain would preach to the students by standing on a soapbox and haranguing them. At dinner that night in the University College the theologian was critical of the chaplain’s “style” and method of communication. In response the chaplain asked the theologian how he preached the Gospel. The theologian responded that he did not preach the Gospel at all; he lectured on the theology of scripture. The chaplain responded then if that was the case, he liked the way he did it, better than the way the theologian didn’t do it.

This book is not a declaration of complete and final knowledge as it pertains to the application of EMDR therapy for the treatment of psychosis and schizophrenia. This book is a beginning. When I lecture, I always begin with the following quotation:

“The mind is not so much a vessel to be filled, as a fire to be kindled.”

—Plutarch

I hope that this book will act as academic kindling, and I hope that researchers and clinicians will add their wisdom and clinical experience to this fire. Hopefully, the light of this beacon will illuminate a path through the fog of battle.

THE ROAD AHEAD

In the first chapter we will explore the links between trauma, psychosis, and schizophrenia. This connection is one that was known and accepted from the earliest days of the characterization of the mental disorder (Bleuler, 1911, 1950; Kraepelin, 1881; Kraepelin, Barclay, & Robertson, 1919) that we now know as “schizophrenia” (Moskowitz et al., 2008). Then, with the passage of time, nosologically we lost our way for a season, choosing to see schizophrenia as an entirely organic illness that was psychologically incomprehensible (Jaspers, 1913, 1963). However, the wheel turns and we are returning once again to acknowledge the connection between trauma and psychosis/schizophrenia (Knipe, 2015; Lanius, Paulsen, & Corrigan, 2014; Moskowitz, 2006; Moskowitz et al., 2008; Ross, 2004, 2013).

Next, we look into the phenomenology and diagnostic entities of dissociation, psychosis, and schizophrenia. This is important because I believe that if we can see beyond and through the current labels of diagnosis, we can apply the healing power that EMDR therapy can bring to people with these experiences. I hope to guide you through the limitations of the current categorical diagnostic nosology of the DSM and International Classification of Diseases (ICD) systems, as I believe we need to move through the current focus on diagnostic labels. By refocusing on the phenomenology beyond a mere label and through a therapeutic awareness of the intersubjective nature of these disorders, it is my belief that we will be more capable of helping the people who present to us seeking
assistance. Will it help all people with these experiences? I doubt that. Will it help some? In my opinion, it will. I hope that this book will make people think about for whom it might be helpful and why. We ought not to be so wedded to our research or clinical “findings” that we cannot reappraise them in the light of new knowledge. Sticking to a position in the face of new experience may be the same dynamic that we propose occurs in the information processing system when overwhelmed by trauma: No new learning occurs. Sometimes we simply see research from a different perspective, one that comes from having traveled some distance further down the road of experience. It is not so much that I am suggesting that the current diagnostic labels are wrong as saying that they are mere labels. They are pale representations of the complex person who has joined us in therapy. We need labels at some level. Indeed, we can think of language itself as a collection of labels that we use to bring narrative to life. Language is the externalization of the inner experience of our minds and consciousness.

The next chapter explores the phenomenology of dissociation and psychosis. This naturally leads on to a suggested model for gathering the necessary information and thus assisting the person seeking our help. This is done through the outlining of a semistructured model of history taking and a review of how to examine the mental state.

After equipping ourselves with what to look for and how to look for it, we will look briefly at the current psychotherapies that are applied to psychosis and schizophrenia. In the light of these other paradigms, we will then explore in particular the work around EMDR therapy for psychosis and schizophrenia. All good structures need a sound foundation, and so we will recap the standard EMDR therapy model first (Shapiro, 1989; Shapiro & Maxfield, 2002; Solomon & Shapiro, 2008). The ICoNN paradigm is a methodology that adapts and adds to the standard EMDR therapy model, so knowing where and why we are making a change is professionally and clinically important. As we have seen in the important work done by the Dutch team, there are occasions when EMDR therapy can be applied in psychosis without any modification to the standard eight-phase, three-pronged protocols (van den Berg & van der Gaag, 2012; van den Berg, van der Vleugel, Staring, de Bont, de Jongh, 2014; van der Vleugel, van den Berg, & Staring, 2012). There is a small but growing literature around the use of EMDR therapy in and for the treatment of psychosis/schizophrenia; this is reviewed and summarized for the reader. This literature and clinical experience are used to present the logic and argument in favor of performing EMDR therapy in people with these experiences. I will also provide some guidance on how to identify those who are most likely capable of engaging with and benefiting from EMDR therapy. The next chapter will assist in the step from academic exploration to the clinical environment: the so-called “translational step.” We will look at how to generate a case formulation and develop a treatment plan in general before looking at the specifics of the ICoNN model’s methodology, which we will do with the aid of clinical examples. There are four key cases:

1. In the first case, we will explore a formulation in which the trauma is clearly known and believed to be etiologically connected to the psychosis that is manifesting (Miller, 2010). It is a case of Cotard’s syndrome (also known as
“walking corpse syndrome”)—this is not specifically contained in the DSM-5 (American Psychiatric Association, 2013). In the ICD-10 we can diagnose this as F32.3: “Severe depressive episode with psychotic symptoms” (World Health Organization [WHO], 1992, 1993). We will see that the case formulation allows the application of the standard model with a resulting resolution of psychotic phenomena and depression. Interestingly, during a dip in mood during the recovery, there was no return of psychosis (Miller, 2010).

2. The second case (Miller, 2010) is one of a body dysmorphic disorder, which the ICD-10 classifies under the rubric of F45, “Somatoform Disorders.” Specifically, it is coded as F45.2, “Hypochondriacal Disorder” (WHO, 1992, 1993). In this case, a young man presents with the belief that he has female breasts; in his case this was a delusional belief. Here I treat the emotional impact of the belief within EMDR therapy rather than challenging its veracity. The specifics of targeting and processing within the ICoNN method are described and discussed.

3. The third case is one of complex PTSD with marked dissociation. There are heard voices that can be engaged in dialogue—these are the “peopled wound” (McCarthy-Jones, 2012), and talking with them acts as a proxy for accessing the DMN.

4. The fourth and final case (Miller, 2010) is the one that prompted me to write this book. I introduced Janus to you in the preface. Janus fulfilled the strict DSM-III-R criteria for schizophrenia used by the GEMINI team (American Psychiatric Association & American Psychiatric Association Work Group to Revise DSM-III, 1987). Janus was given the usual medication for the treatment for schizophrenia, but failed to respond over a suitable duration of time. Sensing that Janus possessed the capacity to engage in psychotherapy, we discussed the possible benefit of EMDR therapy, to which Janus consented. We outline the formulation and treatment plan as delivered. Janus is now 8 years symptom-free and medication-free and has been able to reenter the workplace successfully.

THE PERSON IN THERAPY

Those who live with experiences of trauma, dissociation, and psychosis and those who have been given the label of schizophrenia are, first and foremost, people. The most human thing in our life journey is our innate desire to tell stories, to find meaning in our lives (Frankl, 1988, 1992). If we are to find a solution to the challenge of mental disorder, then I believe that we need to have all the information before us. A diagnosis is epistemologically a reduction and characterization of the complex phenomena of a person’s conscious experience. How on earth would a label fully encapsulate that? I believe it cannot. So, am I saying that we should eschew labels altogether? No, but we must never forget that they are labels. In the ICoNN model, the labels can lead us to the real material that needs to be targeted in therapy. I use the analogy of talking to “the man behind the curtain” in the scene where Dorothy goes to meet the Wizard of Oz. We will never solve the problem fully by talking to the big scary green face of the “wizard”; that will send us on quests to do battle with witches and flying monkeys, but we will not reach home until we look behind the curtain. It is the little dog that leads
Dorothy to look behind the curtain. I believe that in the treatment of psychosis the little dog can be EMDR therapy when applied through the methodology of the ICoNN model. So let us begin our journey as Dorothy and Toto did: one step at a time. The journey continues.

Journey Well.

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INTRODUCTION


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ONE

The Link Between Trauma, Psychosis, and Schizophrenia

**Intention:** To equip the reader with a working knowledge of the relationship between trauma, schizophrenia, and the other psychoses.

**TRAUMA**

The word *trauma* comes from the Greek term meaning “to wound.” The one thing that we all experience is wounding. I was born in Northern Ireland at the start of the internecine violence known colloquially as “the Troubles,” a phrase concocted by the stoic Ulster personality. I grew up in a city whose very name was a shibboleth—Derry/Londonderry—and I later treated people assaulted just for calling it the “wrong” name. As a medical student I witnessed the aftermath of the Shankill Road bombing, carried out by the Provisional Irish Republican Army (IRA), and during the following week, while on rotation in psychiatry in Londonderry, I saw the psychological aftermath of the retaliatory Ulster Defence Association (UDA) shootings that occurred at the Rising Sun Pub in Greysteel. After initially qualifying as a doctor, I completed my general training and joined a central surgical training rotation, but was soon to return to my first love: psychiatry. My first consultant, whom I worked under, was Dr. Peter S. Curran, a local expert in the psychological impact of trauma, and I gained from him a deep and abiding interest in this area of psychiatry (Curran & Miller, 2001).

**THE HISTORY OF THE NOMENCLATURE OF THE PSYCHOLOGICAL IMPACT OF TRAUMA**

Trauma and its consequences have been a part of society for a very long time. It is a central theme in the archetypal hero’s journey (Campbell, 2004), occurring time and time again throughout the myths and stories of humanity. Herodotus describes an Athenian soldier, Epizelus, who had been “behaving with valor” (Herodotus, 1837, p. 118) at the Battle of Marathon (490 BCE). The historian describes Epizelus
as suddenly losing his sight in the midst of the fight, although he had not been wounded. We would frame this as a somatic response to trauma. Such things have occurred throughout military history, but unfortunately the people who experienced them have not always received a sympathetic response. Herodotus goes on to tell us about the Spartans, who were known to be fierce warriors and made more famous still by their exploits at the Battle of Thermopylae (480 BCE). He mentions Aristodemus, who, although one of the elite “300” warriors, was so affected by the battle that he chose not to rejoin the fray; as a consequence, he was judged by the people as “being faint-hearted” (Herodotus, 1837, p. 235). Upon his return home he became known as “Aristodemus the Trembler.” He was to later redeem himself at the Battle of Plataea (Herodotus, 1837). Others, too, have experienced rejection and have had negative judgments made upon their character when really they were suffering a mental illness. The Caesars chose the bravest of their men to be standard-bearers, but even these men could break down, demonstrating that, as in the case of Epizelus, personal valor is not protective against “war neurosis” (Sargent, 1976). In 1678 Swiss physician Hofer coined the term *nostalgia*—an illness that afflicted soldiers on campaign—and by 1755 one directory of diseases stated that it was found most often in the Swiss, as they were exceptionally fond of their country (Anderson, 2010; Babington, 1997; Wilson & Hohman, 1959). Larry, who was a surgeon in Napoleon’s army, thought nostalgia to be a form of madness and stated so in a paper published in 1821, but many of his contemporaries believed it to be a severe form of melancholia (Babington, 1997). Nostalgia was also recognized and diagnosed during the American Civil War period (Anderson, 2010; Wilson & Hohman, 1959). In the early part of the 20th century, World War I was raging, and hundreds of men fighting on the Western Front were being diagnosed with “irritable heart.” The psychological impact of the trauma of war became most widely known as “shell shock” in World War I (Babington, 1997; Myers, 1940). In the United Kingdom (UK), Wilson named this “soldier’s heart” and focused on the cardiac symptoms and signs of the malady (Wilson, 1916). MacCurdy, who came from the United States and made a study of men suffering shell shock on the Western Front, thought it was a form of anxiety neurosis, given that heart function could be affected by anxiety (MacCurdy, 1918a, 1918b). These developments in nomenclature were occurring in the context of a society that was deep in the thrall of the psychological repercussions of the war. Jacoby in Russia and British-based Salmon remarked on the increase in asylum admissions for insanity during wartime (Babington, 1997; Metcalf, 1940; Raftery, 2003). Experts began to conceptualize that these disorders could be the physical consequences of the war environment and proposed that perhaps it only *seemed* that no physical injury had occurred. This was similar in thinking to the concept of “railway spine” that had been described by Sir John Erichsen, who postulated a physical concussive injury as the mechanism of injury in railway accidents (Erichsen, 1875). However, the theory of railway spine was linked neither with the developing concept of soldier’s heart nor with that of shell shock. Charcot was critical of Erichsen and stated that he believed railway spine to be nothing more than hysteria (Babington, 1997). The British medical establishment was becoming increasingly confronted by the psychological impact of battle from the time of the Boer War (Anonymous, 1904). War neurosis was mostly diagnosed as neurasthenia or hysteria before Myers coined the term
shell shock in 1915, assuming that there was a form of brain damage resulting from the concussion of exploding shells. This was a delicate time, during which investigation and professional discussions about shell shock were difficult. The authorities were so worried about Myers writing on this sensitive subject that they insisted on a delay in his book being printed; it finally reached publication many years after World War I (Myers, 1940). First in the post of consulting neurologist and later as consulting psychologist to the British Army, the then-Colonel Myers stated as late as 1916 that there were only one of two options for men presenting as deserters: the asylum or the firing squad (Babington, 1997). By the 1920s the British public was unsettled by the military executions during the 1914–1918 war. However, it was not until 2007 that the Armed Forces Act of 2006 passed an order that allowed soldiers to be pardoned posthumously (The Guardian’s corrections & clarifications column, 2007), although section 359(4) of the act states that the pardon “does not affect any conviction or sentence.” Society is changing: In 2009, in commemoration of the first Armed Forces Day in the UK, the Bolton council added the name of Private James Smith to the Roll of Honor. Private Smith had been executed in 1917 for military misconduct, but had been suffering from shell shock (Reid, 2010).

MODERN NOSOLOGICAL CLASSIFICATIONS

Wartime features heavily in the development of the nomenclature of the psychological impact of trauma. William C. Menninger was a member of the famous Menninger family of psychiatrists and the director of the Psychiatry Consultants Division in the Office of the Surgeon General of the U.S. Army. He chaired the committee that produced “Medical 203” (Lieberman, 2015), which defined psychiatric nomenclature and became hugely influential in the development of subsequent classification systems (Medical 203, 1946). This document acknowledged the psychological impact of trauma, but framed it as largely due to a diathesis—that is, predisposing factors in the person who is presenting with the disorder. The first edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-I) in 1952 acknowledged the role of trauma in psychological reactions (Blashfield, Keeley, Flanagan, & Miles, 2014; Conti, 2014). However, the DSM-II completely ignored the concept of trauma resulting in psychological disorders. This has been attributed to the Vietnam War that was raging at the time (Lieberman, 2015). It may well have remained the case for the DSM-III, were it not for the lobbying undertaken by Chaim F. Shatan, a Polish-born Canadian psychiatrist. Shatan realized that in 1972, the nomenclature gross stress reaction, which was used to classify psychological reactions to trauma, had been removed from the DSM. Moved to action by this observation, he and a group of colleagues began to tirelessly advocate for a return of this nosological concept. Among others, they reached out to Mardi J. Horowitz, who was a renowned researcher in the area of stress reactions (Horowitz, 1973, 1975a, 1975b; Horowitz & Becker, 1973; Horowitz, Becker, & Malone, 1973; Horowitz, Becker, Moskowitz, & Rashid, 1972). However, it was apparently Shatan’s lobbying of the influential Robert Spitzer, chair of the task force of the DSM-III (American Psychiatric Association, 1980) that made the difference. Spitzer is surely the “master builder” of the modern nosology of mental
disorders, but even he has been critical of what he feels have been the excesses of the later iterations of the DSM (Carey, 2008; First & Spitzer, 2003).

THE "BIRTH" OF PTSD

The DSM-III saw a paradigm change with the creation of posttraumatic stress disorder (PTSD), which appeared after Spitzer appointed Nancy Andreasen to scrutinize Shatan’s proposed “post-Vietnam syndrome” (Lieberman, 2015). Given her own experience of treating the psychological impact of severe burns, she found his ideas very resonant, and the DSM-III ushered in PTSD. Instead of the focus of the diathesis–stress model being on the diathesis, the DSM was now saying that the essential criterion for making a diagnosis of PTSD was something that lay outside the person, an experience “outside the normal range of experience” (American Psychiatric Association [APA], 1980). This was Criterion A, and it became known as the “gatekeeper” criterion. PTSD was also delineated in the International Classification of Diseases (ICD-10; World Health Organization, 1993, F43.1). Rather than the problem being an innate weakness in the person experiencing the disorder, the DSM was now stating that the event was so devastating that it was sufficient to overwhelm a person’s normal coping strategies. This was not referring to the normal vagaries of everyday life, such as divorce or losing your job; the authors were thinking of events of such magnitude as the Nazi Holocaust, the atomic bombs’ devastation in Hiroshima and Nagasaki, war, torture, sexual abuse, natural disasters, and other disasters such as severe industrial injuries and road traffic collisions (Friedman, 2007). The latest version, DSM-5 (APA, 2013), retains this “gatekeeper” criterion for PTSD. The other criteria fall into four symptom clusters: intrusion, avoidance, negative alterations in cognitions and mood, and alterations in arousal and reactivity. A sixth criterion details the duration of symptoms required to make the diagnosis, the seventh considers whether there is functional impairment, and the eighth makes clear that the symptoms are not attributable to a substance or co-occurring medical condition (APA, 2013). It is clear that the evolution of the understanding of the psychological effects of trauma has been largely driven by our experiences of war. This has, unsurprisingly, led to the evolution of the diagnosis of PTSD. A person who is involved in a trauma may, however, as a consequence, suffer from a range of affective disorders, or none, and not reach case criteria for PTSD. For example, an electrician I saw following an accidental electrocution, in which he was thrown several feet across the room he was working in, did not fulfill all of the criteria necessary for a diagnosis of PTSD to be made. He did, however, fulfill all of the criteria for a specific phobia because he was terrified of even seeing electrical wiring when I first met with him. Indeed, this event had a severe functional impact upon him, for he was unable to do his job. The recognition of the diagnosis of PTSD in 1980 came after Congress finally passed Senator Alan Cranston’s bill in 1979, which saw the creation of outreach centers for Vietnam veterans suffering from the psychological sequelae of their service. PTSD is, at least in the public mind, the archetypal response to a traumatic event, and the concept soon expanded from the military to all of society as potential sufferers. In my opinion, much of this has been driven by a culture of compensation and legal actions, more so than any public health endeavor. The only
disorders in our current diagnostic systems that have an etiological factor specified in their diagnostic criteria are PTSD and the substance misuse/addictions. This is because the traditional emphasis of the diathesis–stress model is upon the diathesis and not the stress. We will see this as we examine schizophrenia.

SCHIZOPHRENIA: A GROUP OF DISORDERS

The evolution of the diagnosis of schizophrenia was characterized by a move away from a trauma/dissociation model (Moskowitz, 2006; Moskowitz, Schäfer, & Dorahy, 2008) and toward a biological diathesis model, which resulted in schizophrenia’s phenomena being viewed as psychologically incomprehensible (Jaspers, 1913, 1963). We will explore this further in the next chapter when we examine the phenomenology of schizophrenia. This view of schizophrenia as a mental disorder with a pathophysiological predisposition that interacts with psychological factors returns us to the same considerations that were being debated in 1908, around the time of the first public mention of “the schizophrenias” by Bleuler (1906, 1911). Moskowitz observed that Jung compared his work to Bleuler’s, stating, “The chief difference between us is as to whether the psychological disturbance should be regarded as primary or secondary in relation to the physiological basis [italics added]” (Jung, 1960/1914, p. 155; quoted in Moskowitz, 2006). As we see in more detail in the following chapter, Bleuler’s original intention was to describe a group of disorders (Bleuler, 1911), and a return to the dissociative origins of this group of disorders is also being called for (Gonzalez, Mosquera, & Moskowitz, 2012; Moskowitz et al., 2008). Ross, for example, proposes the following spectrum: from nondissociative subtypes of schizophrenia, to dissociative schizophrenia, through schizodissociative disorder, to dissociative identity disorder (DID) (see Figure 1.1) (Ross, 2007; Ross & Halpern, 2009).

• FIGURE 1.1 The Dissociative Spectrum of the Schizophrenias. Includes ‘Grid 1’ (Oil on canvas, 48” X 72”) by Julie Leff (2008). Owned by the Author.

Source: Ross (2007).
This is a very important realignment of our current paradigm. Schizophrenia is not one disorder; it is a group of disorders.

**POSITIVE AND NEGATIVE “SCHIZOPHRENIAS”**

In 1980 Tim Crow published a landmark work delineating two syndromes of schizophrenia: positive and negative (Crow, 1980; Crow, Cross, et al., 1980; Crow, Frith, Johnstone, & Owens, 1980; Crow & Johnstone, 1980). Essentially he described an acute “positive” syndrome that consists of active delusions, hallucinations, and thought disorder. He proposed that this was related to a pathological process involving increased numbers of dopamine receptors, and thus it would be responsive to antipsychotic drugs (Crow, 1981), which are largely antidopaminergic (Frith, 1992). The negative syndrome is a chronic syndrome characterized by poverty of speech and flattened or “wooden” affect with social constriction. He stated that it is associated with structural brain abnormalities (Crow, 1995) and has a generally poor response to antipsychotic drugs (Crow, 1981; Frith, 1992).

**THE DISSOCIATIVE HEART OF “THE SCHIZOPHRENIAS”**

This group of disorders has at its core the dynamic of dissociation, and the force that drives this is trauma. Ross opines that toward the nondissociative-subtypes-of-schizophrenia end of the spectrum there are more negative and fewer positive symptoms, with less psychological trauma and less responsiveness to psychotherapy, with the voices becoming much less interactive. At the other end of this spectrum we have DID, with the voices manifesting as distinct ego-states, with more positive and fewer negative symptoms of schizophrenia (Ross, 2007; Ross & Halpern, 2009). This is clinically what people who have been working with this group of individuals see; as Japanese researcher Akiko Kikuchi noted, “the hallucinations of the hi-DES [Dissociative Experiences Scale] patients are more understandable. I don’t hear devils and gods as common interpretations of their voices. The origin of voices are [sic] thought by the patients to be someone from the past, such as the bully” (Kikuchi, personal communication, 2012).

**OVERVIEW OF THE NOSOLOGY OF SCHIZOPHRENIA**

Bentall suggests that the orthodox position of believing that mental illness is capable of characterization as a discrete number of diagnoses is wrong (Bentall, 1992, 2003, 2009). He makes a similar statement about the erroneous assertion that “madness” cannot be understood in terms of the psychology of the person (Bentall, 1992, 2003, 2009). This latter statement is an inheritance from the work of Karl Jaspers (1913). Bleuler first mentioned the term “the schizophrenias” in a public lecture in April of 1908, and in print shortly thereafter (Moskowitz, 2006). He had continued the discussion group at the Burghölzli that his junior, C. G. Jung, had run while there (1900–1909) and demonstrated an interest in both the biological and psychological etiologies of his patients’ symptoms (Bleuler, 1906, 1911, 1931). However, Jaspers’s influence was to forge the mold for “modern nosology,” and Kurt Schneider, of “First Rank Symptoms” fame, also stated
something similar when he emphasized that the clinician ought to pay more attention to the “form” rather than to the actual “content” of a patient’s experience (Schneider, 1950, 1957). There were those who still emphasized the dissociative mechanisms at play in schizophrenia and took an active interest in their phenomena, as we see from this passage:

In that most frequent and disastrous of all the mental disorders, namely, dementia praecox or schizophrenia, this condition becomes confirmed; the patient lives apart in his dream-world, loses all emotional rapport with his fellows, and interprets such impressions as he continues to receive from the world about him in terms of the world within. (Emphasis in original text; McDougall, 1935, p. 249)

When we return to the dissociative roots of schizophrenia and psychosis, we are inextricably also drawn to examine the relationship with trauma. For if there is a dissociation of the mental processes, something must be causing that to happen, and trauma has long been acknowledged as the key that opens that door (Foote & Park, 2008).

EPIGENETICS

Many have thought that genetic epidemiology has come to the aid of biological psychiatry—a cavalry charge to fight off those who would seek to assail the edifice of schizophrenia as a biological entity. The models for the genetic transmission of schizophrenia have become increasingly complex since the realization that we were not going to find a single gene of major effect in causing it. The International Schizophrenia Consortium described an oligogenic model for schizophrenia in 2009 (i.e., lots of genes of small effect working together), but noted that very large numbers of genes were required to make the model work: 1000 genes only explained 30% of the liability for schizophrenia. It has been estimated that the human genome has 26,000 genes. Modeling also considers how the expression of one gene can be modified by one or more other genes, referred to as epistasis (Khoury, Beaty, & Cohen, 1993). This epistatic effect works in addition to any other physiological and epigenetic force (Sapolsky, Uno, Rebert, & Finch, 1990; Sweatt, 2009; Weaver et al., 2004). Epigenetics can involve a variety of various mechanisms, including the methylation of DNA, which can affect gene stability and has been observed to switch gene expression on or off (Habl et al., 2012; Kuang, Sun, Zhu, & Li, 2011; Shifman et al., 2008; Teixeira et al., 2011; Wedenoja et al., 2010; Yang, Kang, Liu, & Yang, 2013). However, the most recent work on genetics that includes epigenetics demonstrates further how a person’s experiences can result in biological change (Tsankova, Renthal, Kumar, & Nestler, 2007; Yehuda et al., 2014). These marks, which influence the expression of the person’s genetic code, have also been shown to be capable of being passed on to subsequent generations (Sweatt, 2009). As an identical twin, genetics has always fascinated me. The effects of epigenetics are one of the factors that mean genetically identical individuals can look different. In work carried out on mice it was shown that genetically identical mice appear different in color and size (brown and thin cf. yellow and obese) because of epigenetic effects on the agouti gene, which is turned “off” by methylation in the
thin mice and “on” in the obese ones (Duhl, Vrieling, Miller, Wolff, & Barsh, 1994). In my opinion, the focus on heritability research has not given adequate consideration to the environmental factors, and I believe that epigenetic forces may be much more significant and substantial than once thought. However, are we “mice or men” (sorry—couldn’t resist that)? We are, of course, the human race—so what do we know of epigenetic marks in humans?

EARLY-LIFE CHOICES, EXPERIENCES, AND EPIGENETIC TRANSMISSION OF RISK

Consider that each cell in the body—apart from the sperm and the egg—contains the same complement of genetic material. If we stretched out the DNA from one cell into a long string, it would be over 6 feet of linear DNA. The problem of packaging this into each cell is solved by winding this DNA around “spools” called histones that, with the DNA, form chromatin. Although each cell has the same DNA, one becomes a neuron, for example, and the other a muscle cell; how is this happening? This is related to environment in two senses of that term. There is the immediate environment that the cell is in, but there is also the wider environment: the milieu that the embryo is developing within. As we will see, a father can pass on “epigenetic marks” in his sperm (Pembrey et al., 2006; Pembrey, Saffery, & Bygren, 2014), and these marks can switch “on” or “off” gene expression. A mother can affect the developing child epigenetically too—through lifestyle choices, such as smoking and diet, as well as through her experiences, such as stress levels, depression, and the like.

SMOKING AND DIET

Long-term studies have now demonstrated that children and grandchildren can be affected epigenetically by the choices that a predecessor makes. In one study of the sons and grandsons of men who chose to smoke and who had poor diets in their prepubescent years, researchers found that those offspring had shorter life spans than their peers (Pembrey et al., 2006, 2014). Here we see the epigenetic risks being passed down through the paternal line.

THE CHILDREN OF HOLOCAUST SURVIVORS

Rachel Yehuda, a professor of psychiatry and neuroscience and director of the Traumatic Stress Studies Division, and her team at the Mount Sinai School of Medicine have completed work that looks at the intergenerational effects of the trauma of the Nazi Holocaust on subsequent generations. As we have already observed, methylation is an epigenetic mechanism that can switch “on” or “off” genes, controlling what of the genome is expressed. The levels of PTSD in Holocaust survivors were evaluated, and the degree of methylation of a gene-promoter region of the glucocorticoid receptor (GR-1F) gene (NR3C1) was assessed. These measures were then examined against data on the glucocorticoid receptor sensitivity in the offspring (Yehuda et al., 2014). This work built upon the important glucocorticoid receptor work of Michael Meaney’s team in Canada,
completed on rats (Weaver et al., 2004). His team had found that better nurturing by the mother (e.g., licking of the pups by the rat mother) resulted in a lower level of methylation of a gene-promoter region (i.e., a region that enables gene expression for the glucocorticoid receptor). They found that the rat pups were born with a number of epigenetic silencing marks that can be removed through nurturing, grooming, and licking, and this change stays with the pups throughout their lives. (See also TEDxOU Talk by Courtney Griffins at https://www.youtube.com/watch?v=JTBg6hqeuTg; Griffins, 2012.) Yehuda’s team, which was also looking at glucocorticoid receptors, proposed that PTSD in the father resulted in higher levels of methylation and consequently a decrease in receptors. This would lead to higher circulating levels of cortisol that could in turn be associated with higher risk of developing depression or “chronic stress responses” (Yehuda et al., 2014). The team noted a different effect when the mother had PTSD. In these cases the researchers noted an increased attachment of the mother to the child, which they propose is due to the mother’s fear of a loss of attachment. The presence of maternal PTSD alongside paternal PTSD was found to be associated with lower levels of methylation, and Yehuda proposes that the epigenetic forces in the mother may be preconception as well as in utero (Yehuda et al., 2014). She argues that the epigenetic marks could not have been simply laid down in childhood, as a consequence of experiencing a parent with PTSD, because that would fail to explain the different findings that depend on whether it was the mother or the father with PTSD. It is acknowledged that more work is needed, but remember that you can positively affect your epigenome.

TRAUMA AND SCHIZOPHRENIA

When those in our field speak to members of the general public about the relationship between a history of exposure to trauma and the development of psychosis/schizophrenia, they generally express the belief that adverse life events can lead to the development of these disorders (Tarrier et al., 2004). Indeed, when I asked my wife this question she stated exactly that. Work by John Read’s research group has found that people in the following countries believe that mental health disorders are caused primarily by adverse life events and therefore enter mental health services with this set of beliefs in their minds (Read & Dillon, 2013):

South Africa  China  Egypt  Turkey  Fiji
India  Mongolia  Japan  Malaysia  Switzerland
Ethiopia  Greece  Australia  Russia  Bali
Brazil  England  Ireland  Germany  Italy
New Zealand

Although many people, like my wife, generally believe that this connection appears obvious, Kingdon found that for every one psychiatrist who agreed with the thinking of the general public, there were 115 who thought it primarily due to biological factors (Kingdon, 2004; Tarrier et al., 2004). The issue is then compounded by the behavior of professionals within the system of treatment. People receive psychoeducation that they are “biologically broken” as a consequence of a genetically inherited set of risk factors. As professionals, by doing so, we make
them passive and incapable. We essentially tell them that they are not able to be responsible for their illness, and medical paternalism steps in and mediates this broken biology. People, as a consequence, fail to be given the opportunity to talk about the adverse life events. However, if we give people the opportunity, I have found that they are willing, and with time and the right assistance, able to do so. In medical school I was taught that the biological and indeed genetic etiology of schizophrenia was a universally accepted “fact.” I had the honor and great opportunity to work as a small part of the team that Professor Kenneth Kendler led in Ireland that examined the genetic epidemiology of schizophrenia and poor-outcome schizoaffective disorder. However, as we are learning, the nature/nurture debate is not a straightforward one. It is in every sense a Gordian knot, and pulling loose one piece seems only to tighten others.

“AM I CRAZY?”

I want to introduce at this point a concept that might be new to many: the healthy-voice-hearer. A substantial proportion of psychotic phenomena are auditory verbal hallucinations (AVHs). If we explore AVHs, the following figures have been quoted in the literature: 34% of voice-hearers have been given a diagnosis of schizophrenia, 31% have been diagnosed with borderline personality disorder, 21% have no psychiatric diagnosis (healthy-voice-hearers), and 10% have PTSD (McCarthy-Jones, 2012). Romme argues that hearing voices is not the pathology; rather, it is the failure to adapt to the experience of hearing voices that results in mental illness (Romme & Escher, 1993).

THE WIZARD OF OZ FALLACY

I agree with Romme that the “psychotic” material need not be viewed as the problem; it is actually how a person is adapting to the core pathology of the dysfunctional memory network (DMN): The DMN is the real problem. In the introduction I referred to the Wizard of Oz fallacy. I define this as the situation where we focus our treatment solely on the psychotic material rather than working to psychologically “metabolize” the DMN. In the Indicating Cognitions of Negative Networks (ICoNN) model, we view psychotic material as a bridge that connects us to the DMN, which is the primary target for our therapeutic endeavors. I have already said that we need to focus on “the man behind the curtain” (DMN) because only “talking to the big scary green face of the wizard” (psychosis) results in being sent on expeditions to battle witches and flying monkeys. This is misspent time in therapy, as getting “home to Kansas” requires us to look behind the curtain, and that is what we do by reprocessing the DMN through ICoNN methodology. Toto, the little dog that leads Dorothy to look behind the curtain, is eye movement desensitization and reprocessing (EMDR) therapy applied using the ICoNN model’s methodology.

ALLEGORY: “LET ME TELL YOU A STORY”

The function of the adaptive information processing (AIP) model of EMDR therapy is to generate a healthy memory network. A healthy memory is a coherent and contiguous narrative of the experience; this is in keeping with what we

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hypothesize in the AIP model (Shapiro, 2001, 2007; Solomon & Shapiro, 2008). The generation of narrative, as an adaptive response to one’s surroundings and experience, is a normal phenomenon. Once we discard the fallacy that psychotic phenomena are psychologically unintelligible, we begin to see the meaning and symbolism in them (Jaspers, 1913, 1963). We see this archetype in the allegory presented in Life of Pi, where a boy tells a story of being in a boat with a tiger, when what he really experienced was the horrors of having seen another person do terrible things to their shipmates (Martel, 2011). We see clearly that the generation of his version of his experience was adaptive. I believe that this is also what I observe in people who present with these experiences and who have been diagnosed with psychosis. These are adaptations to the experience of living. As Ross has indicated, there is a range of disorders within “the schizophrenias,” with differing levels of dissociative force. McCarthy-Jones offers the following paradigm shift: “It may be that the important transition is not from patient-voice-hearer to non-voice-hearer but from patient-voice-hearer to healthy-voice-hearer” (McCarthy-Jones, 2012, p. 148).

THE ROLE OF TRAUMA

PTSD and DID are known and accepted to have trauma as an etiological factor. We will also see that schizophrenia was born of dissociative mechanisms and trauma accepted as a causative force (Moskowitz, 2006; Moskowitz et al., 2008). The AIP model proposes that trauma derails this normal innate mechanism and generates a DMN. It is my hypothesis that this is then adaptively expressed as psychotic material. If, however, we remember that there is meaning in the “ICoNN-ography” of the psychosis, then I believe that by telling the person that there is meaning that can be decoded, we give them hope. When we assume this position as professionals, this is not an extremist position when the whole population is considered; it may be extreme for those of us who are psychiatrists, but I certainly think these data are compelling and confirm something that we appear to have known intuitively. Hope flows when meaning is present, and the mind is all about meaning (Campbell, 2004). At a lecture he gave in Cork, John Read described a story from his experiences of working in a U.S. hospital while in his 20s (Read, 2013). He told of a man, whom he called Bob, who had been admitted and was walking around with his eyes closed; he was “black and blue” as a consequence. At the end of his tale, Read explains that being naturally inclined to engage with the other man, and not being trained at the time, he did what came naturally: He asked him why he was keeping his eyes closed. Note that this “intervention” came from what Read felt natural to do, and I believe this is a nice example of working with an awareness of the intersubjective. Healing is a very relational endeavor; or, rather, it ought to be. The man opened his eyes and got right into Read’s face and angrily stated that it was about time someone had asked him that question. His answer: “My family put me in here to get insight and that’s what I am doing!” We can see in this example that the man was adapting, albeit concretely. He had to get insight; so he closed his eyes and cast his “sight” inward—literally, in-sight. Psychosis and the associated behaviors are not meaningless; they are iconic.

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“Neither can embellishments of language be found without arrangement and expression of thoughts, nor can thoughts be made to shine without the light of language.”

—Cicero (unknown source, 106–43 BCE)

I have had the fortune to hear both John Read and Richard Bentall speak, and my recollection is that they both used the same phrase to cameo the work that they have been undertaking. There is meaning in the psychosis, and they sum up the connections that they are shining a light upon through their publications as follows: “Bad things happen and can drive you crazy!”

Currently a large amount of research has been completed and published that confirms a link between adverse life events and trauma and the later development of psychosis/schizophrenia. This research is not saying that these trauma cause the psychosis; rather, research indicates that there is a very important relationship between the two. If we examine psychiatric inpatients with psychosis, we find that 59% will have experienced either childhood sexual abuse (CSA) or childhood physical abuse (CPA) (Read, Fink, Rudegeair, Felitti, & Whitfield, 2008). When people diagnosed with schizophrenia are examined, we find that 47% have experienced emotional abuse, 51% have experienced neglect, and 41% have experienced CPA (Read, 2013; Read & Dillon, 2013). These authors are not making extreme statements. Some people consider them “radicals” in making the connections noted earlier. Perhaps if we return to the origins of the word radical, which has been getting some bad press of late, people can feel better about this accusation. Radical comes from the Latin word radix, which means “root.” I think that we ought to go back to the fundamental root of psychosis, and that, as we shall see in the next chapter, is dissociation.

REMEMBER THE PERSON SEEKING HELP

There is no single cause of psychosis (Read & Dillon, 2013), just as there is no single gene (Owen, Cardno, & O’Donovan, 2000). Some have even gone as far as to say that there is no evidence of a genetic predisposition to psychosis and schizophrenia (Hamilton, 2008). As I have already noted, there is little to be gained in fighting from entrenched positions. At this point I want to move forward and keep in mind the needs of the person who is asking for assistance. We see that there is a growing modern literature linking adverse life events with increased risk of developing pathology-level psychosis (Read & Dillon, 2013). The strongest predictive factor for psychosis is actually poverty, or, more specifically, relative poverty (Pickett & Wilkinson, 2009). When biological psychiatrists were examining this phenomenon, they proposed that the biological illness preceded the social drift downward rather than poverty being the primary factor. However, when the relationship is examined, we see that higher rates of psychiatric disorder are found first, before poverty. Higher rates of mental disorder were also observed for those who were members of an ethnic minority, or of a colonized indigenous people (Pickett & Wilkinson, 2009). The research into this area of inquiry now consistently shows the importance of attachment and healthy
interpersonal functioning in the intersubjective space. When 390 people who were diagnosed with first-episode psychosis were studied, the researchers found that they were 2.4 times more likely to have been separated from one or both parents before the age of 16 years, they were 3.1 times more likely to have had a parent die, and they were 12.3 times more likely to have had their mother die (Morgan et al., 2007). Attachment is not an optional extra for us; it is a need, and needs are nonnegotiable. Attachment is so important that when a child is confronted by abuse at the hands of a parent, the child will dissociate in order to maintain the attachment (Freyd & Birrell, 2013). The importance of a nurturing attachment is seen in the research with Romanian orphans. Romanian orphanages were known for their harsh and overcrowded conditions. After the fall of the Romanian Communist politician Nicolae Ceaușescu, the plight of the orphans came to wider international awareness. In follow-up studies it was observed that those orphans who went to foster homes and had loving, nurturing mothers had hippocampal volumes that were 10% larger than those of the children whose mothers were not so nurturing, and these children suffered more wide-ranging neurocognitive impairments (Almas et al., 2012; Bos et al., 2011; Bos, Fox, Zeanah, & Nelson, 2009; Bos, Zeanah, Smyke, Fox, & Nelson, 2010; Gleason et al., 2011; Johnson et al., 2010; Levin, Zeanah, Fox, & Nelson, 2014; Marshall, Reeb, Fox, Nelson, & Zeanah, 2008; McGoron et al., 2012; McLaughlin, Fox, Zeanah, & Nelson, 2011; McLaughlin et al., 2010; McLaughlin, Zeanah, Fox, & Nelson, 2012; Moulson, Westerlund, Fox, Zeanah, & Nelson, 2009; Nelson, Fox, & Zeanah, 2013; Nelson et al., 2007; Slopen, McLaughlin, Fox, Zeanah, & Nelson, 2012; Smyke et al., 2007; Troller-Renfree, McDermott, Nelson, Zeanah, & Fox, 2014; Windsor et al., 2011; Windsor, Moraru, Nelson, Fox, & Zeanah, 2013). In 2004, a team led by Jim van Os published its findings that experiences of childhood abuse increase the likelihood of pathology-level psychosis (Janssen et al., 2004). Over a 3-year follow-up, the team found that children who had been abused had 9 times the likelihood of developing a pathology-level psychosis, but, most important, it identified that severe abuse increased this to 48 times (Janssen et al., 2004). This demonstrates a dose–response relationship and is a highly significant finding. Although this finding was received with some skepticism, the evidence is mounting in support. A later study that examined the sample group for the presence or absence of five types of trauma also found a dose–response relationship: For those who had experienced three traumas, the increased likelihood of being psychotic was 18 times; for those who had experienced five traumas, that increased likelihood went up to 193 times (Shevlin, Dorahy, & Adamson, 2007).

THE EFFECT OF TRAUMA ON THE BRAIN

If we acknowledge this important effect of exposure to trauma, then we will naturally consider the nature of the impact. We have touched upon there being physical changes to the brain; so, are there any similarities between the impact of trauma on the developing brain and the changes seen in psychosis? Original work by Read et al. reported a huge similarity between the pattern of brain abnormalities in people with schizophrenia and the changes found in the
developing brains of individuals who had gone through traumatic events (Read, Perry, Moskowitz, & Connolly, 2001):

1. Overactivity of the hypothalamo-pituitary-adrenal (HPA) axis, the body’s stress regulation system
2. Abnormalities in the neurotransmitter systems (especially dopaminergic systems)
3. Hippocampal damage
4. Cerebral atrophy
5. Reversed cerebral asymmetry

(Read, 2013)

TRAUMAGENIC NEURODEVELOPMENTAL MODEL OF SCHIZOPHRENIA

Read’s team described a traumagenic neurodevelopmental (TN) model of schizophrenia based upon the findings (Read et al., 2001; Read, Fosse, Moskowitz, & Perry, 2014). The TN model also helped in the development of models for how cognitive impairment and the positive and negative symptom clusters (Crow, 1981, 1995) occur as a consequence of exposure to trauma. Although Read discusses the relationship between dissociation and psychosis, as we will see in the following chapter, Ross believes it is not so much an issue of either/or versus both/and, because in his opinion they are frequently describing the same thing (Ross, 2004). Following publication of the TN model there has been much interest in this area of research, and when the same team revisited the model in 2014 they noted that 125 subsequent publications indirectly supported or directly confirmed their hypothesis (Read et al., 2014). As a consequence, they made the recommendation that clinicians and researchers should endeavor at all times to gather a comprehensive history that details a person’s experience of trauma, neglect, and loss. They argue that the implications for primary prevention are profound, given the role of trauma in the development of later mental conditions (Read et al., 2014). As already stated, this represents a return to the clear connection between trauma and psychosis that was acknowledged in the days of Bleuler (Bleuler, 1911; Moskowitz, 2006). Rather than the stress–diathesis model, with the emphasis being on constitutional vulnerabilities, conferred through an oligogenic model of risk (Fanous et al., 2005; Straub et al., 2002), they are proposing that the focus be on the stressor. Of 11 studies that have looked for a dose–response relationship, 8 have found it to be present (Read et al., 2008). This same group advocates a return to the stress–vulnerability model (Zubin & Spring, 1977), noting that the vulnerability need not be inherited genetically; the original work stated that the vulnerability may also be acquired through life events. The observation that more than 337 publications now demonstrate a link between trauma and increased likelihood to developing psychosis, and that many note a dose–response relationship, would appear to support this acquired vulnerability. Researchers such as Read and Bentall have invited the geneticists to now turn their attention toward the epigenome and cease looking for genes of small effect, to add to the oligogenic
models, which in the end of it all will still require thousands of interactions to explain the increased vulnerability in only a fraction of cases. We ought also to ask, what will we do if we identify all the genes of risk in any case? Compare this to the finding that people who have experienced five traumas have a 193-times-increased likelihood of developing psychosis (Shevlin et al., 2007). The work in the area of epigenetics is increasingly opening doors to understanding how these increased levels of vulnerability might occur. To continue to insist that mental illness requires an underlying genetic vulnerability is simply no longer in alignment with the research. When Read’s team looked at 41 of the most rigorous of studies in a meta-analysis, they found that people who had experienced childhood adversity were 2.8 times more likely to develop psychosis than the general public (Varese et al., 2012). In the 10 studies that looked for a dose–response relationship, nine found it to be present (Varese et al., 2012).

DOOMED FROM THE START

When we examine the nosological origins of schizophrenia, we see that psychiatry has struggled to hold out any hope for healing in the area of psychosis in general, but this is especially so for schizophrenia. Kraepelin actually included as part of the dementia praecox concept the statement that if someone got better, they could not really have had dementia praecox in the first place. This is precisely what some people have said about the Janus case study. Given how well Janus has done (now more than 7 years posttreatment and symptom- and medication-free), surely the original diagnosis could not have been correct. However, Janus was assessed with the same rigor and instruments that we used in the Irish Schizophrenia Triad Study—I completed his ratings and another colleague reviewed the Structured Clinical Interview for DSM-IV (SCID). Had we been recruiting for the genetic study, he would have been included. Now is the time to end our therapeutic nihilism for schizophrenia and psychosis.

HOPE SPRINGS FROM MEANING

Jim van Os, in his TEDx talk, spoke of the need for us to give people who are being given diagnoses of psychosis a “perspective of hope and the possibility of change” (van Os, 2014). This is the opposite of how many professionals are looking at psychoses. The population attributable risk for psychosis has been calculated as 33% for people who have experienced six childhood adversities while under the age of 5 years (Varese et al., 2012). This means that from a primary prevention perspective, if we can eliminate those six childhood adversities, we would reduce the incidence of psychosis by one third. By acknowledging the link between trauma and psychosis, we also naturally come to examine treatment, and where better to look than among the psychotherapies with a trauma-based formulation and a trauma-focused methodology of therapeutic intervention? Researchers exploring EMDR therapy in patients with psychosis have found that it is safe and efficacious in these people (de Bont et al., 2013). As clinicians and researchers who are working with people who are experiencing psychotic
disorders, it is very important that we have a high index of suspicion for the presence of early adverse life events. Our assessers need to form a strong therapeutic rapport, listen well, and gather information in a comprehensive manner, from a position of therapeutic neutrality. Our journey continues.

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1 THE LINK BETWEEN TRAUMA, PSYCHOsis, AND SCHIZOPHRENIA


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