Psychotherapy in Pain Management

Psychotherapy in Pain Management:

New Perspectives and Treatment Approaches Based on a Brain Model

By Robert Moss

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ABBREVIATIONS

 α -amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid receptor (AMPAR); anterior cingulate cortex (ACC); anterior insula (aIns); anterior MCC (aMCC); basolateral amygdala (BLA); bed nucleus of the stria terminalis (BNST); central nucleus of the amygdala (CeA); blood oxygen level dependent (BOLD); Clinical Biopsychological Model (CBM); default mode network (DMN); dentate gyrus (DG); Dimensional Systems Model (DSM); dopamine active transporter (DAT); dorsal ACC (dACC); dorsolateral prefrontal cortex (dlPFC); dorsomedial PFC (dmPFC); dorsal posterior insula (dpIns); Emotion Focused Therapy (EFT); Emotional Restructuring (ER); frontopolar cortex (FPC); gamma-aminobutyric acid (GABA); globus pallidus (GP); intralaminar nuclei (ILN); Heschel's gyrus (HG); lateral PBN (IPBN); medial nucleus of the posterior group (POm); medium spiny neuron (MSN); meta-analytic connectivity modeling (MACM); middle cingulate cortex (MCC); middle insula (mIns); nucleus accumbens (NAc); neural correlates of consciousness (NCC); Neurologic Pain Signature (NPS); Nmethyl-D-aspartate receptor (NMDAR); orbitofrontal cortex (OFC); parabrachial nucleus (PBN); parahippocampal cortex (PHC); periaqueductal gray (PAG); posterior part of the ventral medial nucleus (VMpo); posterior cingulate cortex (PCC); posterior ACC (pACC); posterior MCC (pMCC); posterior PCC (pPCC); prefrontal cortex (PFC); pregenual PFC (pgACC); primary care physician (PCP); pyramidal cells (PCs); pyramidal-interneuron gamma (PING retrosplenial cortex (RSC); rostral ACC (rACC); rostromedial PFC (rmPFC); subgenual PFC (sgACC); substantia nigra pars reticulata (SN); subthalamic nucleus (STN); superficial amygdala (SF); tactile C fibers (CT); temporoparietal junction (TPJ); theory of mind (ToM) :Universal Cerebellar Transform (UCT): ventral PCC (vPCC): ventrocaudal mediodorsal nucleus (Mdvc); ventrolateral PFC (vlPFC); ventromedial PFC (vmPFC); ventral posterior complex (VPC); ventroposterior inferior (VPI) nuclei; ventral posterolateral (VPL); ventral posteromedial (VPM)

CHAPTER 1

INTRODUCTION

The management of noncancer chronic pain has been an area of great interest and controversy for decades. Prior to the Covid-19 pandemic, the opioid crisis was a frequent topic covered by the news media. Over the past two years sweeping changes have occurred in relation to the use of opioid medications, including stricter laws on the use of pain medications in isolation and in combination with other medications. such as benzodiazepines. The growing number of lawsuits and disbanding of the American Pain Society are examples of the fallout of the growing public awareness of opioid addiction and new governmental regulations. Due to the fact that many patients treated with opioids are already receiving antidepressant medications and other psychotropic drugs, there is a need for additional treatment approaches with an opportunity for psychology to make significant contributions in the pain management arena.

In support of the need for involvement of psychological treatment in chronic musculoskeletal pain cases, Tseli et al. (2019, 148) performed a systematic review of prognostic factors for long term physical functioning following multidisciplinary rehabilitation. They found that pain chronicity and intensity did not predict physical functioning following multidisciplinary treatment. Instead, better outcome was predicted by low levels of pretreatment emotional distress and low levels of cognitive and behavioral risk factors, as well as high levels of protective cognitive and behavioral factors. The authors suggest that treatment targeting these factors "may perhaps provide an opening for yet untapped clinical gains."

In 1977, Engel proposed a new conceptual model of illness that included social, psychological, and behavioral dimensions. The biopsychosocial approach (Turk, 1999) is commonly used to both conceptualize and guide treatment in psychotherapy with pain disorders. Meints and Edwards (2018, 169) explained "the biopsychosocial approach describes pain and disability as a multidimensional, dynamic interaction among physiological, psychological, and social factors that reciprocally influence one another, resulting in chronic and complex pain syndromes."

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Gatchel, McGeary, McGeary and Lippe (2014) discussed the importance of interdisciplinary chronic pain treatment, with involvement of physicians, nurses, psychologists, physical therapists, and occupational therapists. In his book on pain management, Gatchel (2005) discussed a comprehensive approach to treatment of chronic pain patients using a biopsychosocial perspective with an emphasis on psychological approaches to treatment. He suggested the possible use of group therapy and marital/relationship counseling due to the importance of a patient's social support. In relation to individual psychotherapy, he discussed the use of relaxation/biofeedback, attention/distraction, cognitive-behavioral interventions, assertiveness/social skills, increasing reinforcing activities, life planning, managing secondary gains, secondary loss issues, and motivational enhancement. Little has changed from the overall program he discussed in 2005 (cf., Gatchel et al., 2014).

In a detailed review of psychosocial factors contributing to chronic pain outcomes, Meints and Edwards (2018) provide information that can serve to outline the potential targets of psychological treatment. In relation to affective factors, it is noted that pre-morbid psychological dysfunction is a risk factor for the development of chronic pain symptoms. They commented that this interpretation contrasts to the frequent one that psychological symptoms are a consequence of chronic pain. This is one factor leading to the recommendation for early interdisciplinary intervention for at risk populations in the prevention of disabling chronic pain conditions (Bevers, Watts, Kishino, and Gatchel, 2016).

The next area discussed by Meints and Edwards (2018) is trauma. They note there are strong prospective links between early-life trauma (i.e., physical, psychological, and sexual abuse) and the later development of chronic pain. Additionally, they discuss the evidence that later posttraumatic stress disorder (PTSD) amplifies the predictive effects of childhood abuse on chronic pain, making the observation of just how entrenched and enduring are the damaging effects of such abuse. A recent study (Stahlschmidt, Rosenkranz, Dobe, and Wager, 2020) demonstrated that as early as childhood and adolescence, there is a high prevalence of posttraumatic stress disorder (PTSD) associated with severe chronic pain. In their literature review, Burke, Finn, McGuire, and Roche (2017) indicate there is evidence of alterations in some neurobiological substrates (e.g., hypothalamic-pituitary-adrenal axis, neurotransmitter systems) as a result early-life stress that are correlated with chronic pain conditions. Similarly, Meints and Edwards (2018) discuss the correlational date on brain cortical and subcortical areas involved with chronic pain. However, to my knowledge, there has been no theoretical discussion involving brain

Introduction

mechanisms on the possible manner early-life trauma or other psychological factors contribute to chronic pain. In total, it appears that early-life trauma is seen as a static mechanism used primarily as a risk factor as opposed to being considered a dynamic, but ongoing and persistent, mechanism that may be modifiable with appropriate psychological treatment. This will later be discussed in more detail. Examples of clearly static and non-modifiable psychosocial mechanisms related to chronic pain are gender and race that Meints and Edwards (2018) also discuss.

Another area covered by Meints and Edwards (2018) involves social/interpersonal factors. The studies reviewed focused on either nonpain-related global social support or pain-related social responses. They note it is clear that interactions between chronic pain patients and their significant others can both facilitate and impair adjustment. Such factors as a partner's depressive symptoms or avoidant and anxious attachment styles are associated with increased problems related to pain. Patients with anxious or insecure attachment styles have been shown to be at elevated risk for poorer outcomes in pain treatment. In relation to the work environment, the authors note the importance of social support and satisfaction with coworkers as predictors of pain-related disability. It appears that the main proposed manner in which the social area has been addressed involves couple's interventions interpersonally and via occupational or vocational counseling related to the work situation. Thus, there is a lack of discussion of how important social and work relationships can be addressed in individual psychotherapy despite the role interpersonal relationships play in chronic pain syndromes. Sturgeon and Zatura (2016) noted more explicit focus on addressing interpersonal distress and enriching one's relationships are underexplored areas of chronic pain treatment. In relation to interpersonal factors in individual psychotherapy, Meints and Edwards (2018) also note the importance of establishing a sound therapeutic relationship, although there are few related studies to date.

This book presents a brain-based model with the potential for explaining brain mechanisms involved with chronic pain and a discussion of psychological treatment targets that have received little to no attention. I first want to provide insights as to pain management from the perspective of a practicing psychologist involved in pain treatment for many years. There will then be an introduction to an applied treatment model based on the brain theory, followed by a discussion of cortical pain processing based on previous research. This will be followed by an explanation of a brain model based on cerebral cortical columns and how this relates to the prior pain research. The second part of the book discusses in detail the applied Clinical Biopsychological Model (CBM). Following a discussion of how the model explains psychotherapy process variables and its relationship to current treatment approaches, there is a discussion related to identifying and treating influential negative emotional memories with the goal of reducing the psychological and physical impact of those memories. There is next an explanation of the two meta-traits (i.e., plasticity and stability) of the Five Factor Theory of personality based on the CBM and how the new viewpoints translate into specific recommendations on the most effective ways for pain patients to behaviorally interact with those in their social network. Information on loss-related depression will be discussed within an opponent-process framework with proposals on how this important area may be identified and addressed. The final section of the book provides the treatment manual with detailed information on assessment, conceptualization, and treatments. It is hoped that the book provides useful information to neuroscientists and clinicians related to chronic pain conditions, with emphasis on the belief that psychotherapy is the only treatment that can address important cerebral cortical processes in associated depression and anxietv.

PART 1.

CHRONIC PAIN NEUROPHYSIOLOGY AND THE DIMENSIONAL SYSTEMS MODEL

CHAPTER 2

PSYCHOLOGICAL TREATMENT WITH CHRONIC PAIN PATIENTS FROM A SEASONED CLINICIAN'S PERSPECTIVE

My first experience with pain patients began in 1977 while in my first year of graduate school. The focus was on using blood volume pulse biofeedback with migraine headache patients, being an alternative to the more frequently used hand temperature biofeedback. My thesis (Moss et al., 1982) involved using a conditioning program in the treatment of nocturnal bruxism and led to my developing an awareness of facial pain issues. By my last year of graduate school, I branched out to treating facial pain patients referred to as myofascial pain dysfunction or temporomandibular (TMJ) dysfunction patients (Haber, Moss, Kuczmierczyk, and Garrett, 1983; Moss and Adams, 1984a; 1984b; Moss, Garrett, and Chiodo, 1982; Moss, Wedding, and Sanders, 1983). Over the next few years, and with a grant through the National Institute of Dental Research, our research provided some new views based the overlap of facial pain and headache patients (Moss, 1987a; 1987b; Moss, 1988a; 1988b; Moss, Lombardo, et al., 1987; Moss, McClure, Jackson, and Lombardo, 1987; Moss, Ruff, and Sturgis, 1984; Ruff, Moss, and Lombardo, 1986; Villarosa and Moss, 1985), which I will briefly discuss at the end of this chapter.

While on internship at the University of Mississippi Medical Center, I had a rotation in the pain program which introduced me to chronic low back pain patients, none of which were involved in litigation. In 1986, I began working with chronic low back pain patients at a South Carolina inpatient pain treatment program that mainly treated worker's compensation patients. Two of the stated goals of that program were to eliminate the use of opiate medications and return the work injury patients to their jobs. A year later, I began a private practice in which treating worker's compensation chronic pain patients was a major component. Although there were four years that I did not see pain patients after closing my practice in 2011, I found myself in another pain treatment program that I was first asked to do risk assessment

ratings related to opiate medication treatment. As can be seen, I had the opportunity to deal with the psychological assessment and treatment of chronic pain patients in academic, multidisciplinary program, and solo practice settings over the past 43 years.

My co-major in graduate school was biopsychology which had become a major interest of mine when I was an undergraduate psychology major. It was during my graduate neuroanatomy class that I was introduced to the cortical column as a basic unit in cortical processing, although it was unclear as to its possible role outside of primary sensory reception. My graduate neuropsychology course was taught from a Lurian (1966) viewpoint which I found to be fascinating and quite sensible. In 1984 I taught a neuropsychology course while an assistant professor at the University of Mississippi. I used Luria's Higher Cortical Functions in Man (1966) as the text. While preparing for a lecture. I found myself considering how the cortical column might possibly fit into Luria's proposal of primary, secondary, and tertiary cortices. This reasoning was facilitated by my familiarity with logic programming (e.g., AND-gates, one-shots, universal timers) on a Colburn rack system that I learned from a fellow graduate student, David Hammer, while doing the bruxism conditioning program study (Moss et al., 1982). That led to the development of the Dimensional Systems Model (DSM) that has guided my thinking in both neuropsychology and clinical psychology across my career.

Despite my writing a paper on the model in 1984, it was consistently rejected for publication as being "too speculative" and "untestable." It was in 2006 that the field had progressed to allow the first peer-reviewed article to be published on the DSM (Moss, 2006). A year later I had an article published on the applied Clinical Biopsychological Model (CBM) related to influential negative emotional memories (Moss, 2007). I have continued in the expansion and explanation of the DSM and CBM, with this book representing the compilation of my work as it relates to the understanding and treatment of chronic pain patients. This book has given me the opportunity to use the DSM in explaining research on brain processing of pain and how the cerebellum, basal ganglia, cingulate cortex, and insula are theoretically involved with cortical column circuitry. Moreover, I provide information on how CBM-related treatment approaches addressing relevant negative emotional memories, personality patterns, and loss-related depression may provide important additions to the currently employed psychological treatments in chronic pain.

Over the years I had the opportunity to see many transitions in the pain management field. Obviously, pain management means pain elimination is not possible, a fact that is an important mutual understanding for both

Chapter 2

patient and clinician. Chronic pain patients with psychological problems are a difficult population because they have an ongoing aversive, and usually progressively worsening, condition for the remainder of their lives. Until both the patient and clinician have a mutual understanding of the permanent nature of the pain, it is difficult to move beyond the search for a pain cure. As will be explained, at the point of acceptance that one's pain condition is permanent, there will be a worsening of depression symptoms for a time. Improvement in the patient's quality of life is the primary goal of chronic pain management, of which teaching ways to reduce one's pain is only one component.

The initial chronic pain program with which I dealt was researchoriented and done on an outpatient basis, excluding those patients with litigation that might influence treatment outcome. It involved the use of relaxation procedures, biofeedback, and behavioral pacing, in addition to education and working with patients to accept the permanency of their condition. I was introduced to the "real world" in 1986 in which worker's compensation carriers were primarily the ones paying for treatment. It became clear to me that for most patients on the front end of a chronic pain condition, their goal is pain elimination, not management. That inpatient program lasted four weeks, gradually reducing opiates via a pain cocktail delivery system, such that the patient was unaware of dosage changes. Each morning involved a brief gathering of both staff and patients doing stretching exercises. Progressive muscular relaxation was done as a group twice daily. Motivational group therapy was done, selectively attending to "adaptive" statements by patients while selectively ignoring pain complaints. Educational classes on various topics (e.g., stress, nutrition, pain physiology) were done for an hour each day. Physical therapy (PT) was done twice daily, mainly involving exercise, with occasional use of modalities and myofascial release. Each patient had an individual psychotherapist with whom they met weekly, involving supportive therapy with encouragement to do all aspects of the program. Interestingly, at one point there was an education class done by a vocational specialist on the state's worker's compensation system's history and operation. This was helpful to staff and patients alike, but stopped within a few months after patients confronted their attorneys with uncomfortable (for the attorney) factual information. The director was told by plaintiff attorneys that the class was interfering with the attorney-client relationship and must stop or there would be a lawsuit against the hospital system. It stopped immediately. The knowledge I gained from that class proved invaluable in my practice over the years. This brings me to an important point. If one chooses to provide psychological treatment to chronic pain patients, expect ongoing litigation to be a frequently encountered issue that can greatly complicate matters. Let me briefly explain.

In worker's compensation cases, the insurance carriers inevitably create multiple problems, such as failure to pay for medical assessment and treatment procedures, "accidently" dropping patients from computer systems resulting in delayed weekly benefits, and having patients approaching settlement followed by private detectives. Case managers are sometimes involved to facilitate the most efficient treatment, but they work for insurance carriers who pay them based on their ability to keep treatment costs at a minimum. This results in an obvious conflict of interest. The problems frequently result in a patient's hiring an attorney, only to find additional frustration with the attorneys who often do little until the patient reaches maximum medical improvement and the case is approaching settlement. In South Carolina, where my practice was located, the insurance carrier chose the treating doctors with the injured worker having no control. That meant the "company" doctors (i.e., those chosen by the business and/or carrier to initially see an injured patient) find themselves in a position that carriers expect to have the least amount of overall medical costs and subsequent "permanent" loss upon which settlements are based. Accordingly, referrals to specialists were often discouraged, often not happening for an extended period of time if they were made. The result is that worker's compensation patients deal with ongoing victimization with very little ability to control any aspect. Therefore, I believe it is imperative that psychotherapists educate themselves on their state's system (and note the federal employees' system is different) so they have the ability to educate their patients on the reality of what the patient can expect. A truly effective therapist will avoid jaded comments and discuss information in a factual manner. Overall, the discussion allows the patient to become aware of each player in the system which typically results in the patient's having anger. However, patients are always appreciative for the knowledge and the therapist can then assist the patient in deciding how to deal with each aspect in her/his own best interest.

I also recommend therapists becoming familiar with the Social Security disability process because of the high probability that their patients will deal with that system as well. For example, I saw a work injury patient whose case was drawn out for 7 years, with her attorney never suggesting until after settlement that she apply for Social Security disability (which can be done even if the patient is receiving worker's compensation benefits in which case the Social Security Administration simply reduces benefits if the patient is judged to be disabled). When she applied, she was approved quickly, but was unable to draw on what she paid into the system because it was over 5 years since she last worked. She received much less money because she qualified only for Supplemental Security Income. An additional issue is that Medicare is not immediately provided to patients awarded disability. By applying while still on worker's compensation (in which case most patients no longer have an employer's health insurance benefits) means that badly needed health insurance may be obtained in a little over 2 years, as well as the additional benefit of receiving monthly payments for any of the patient's non-adult dependents. Another issue is that many patients file for disability only on the basis of the pain which is viewed as a subjective experience and seldom leads to favorable decision. Patients are typically unaware of the need to list in the application all physical and psychological problems that exist or these will not be considered despite evidence in records of such issues. Psychological problems are often what results in the awarding of benefits to chronic pain patients. Failure to be cognizant of and addressing these critical litigation issues will certainly compromise a patient's ability to benefit from any manualized treatment approach.

Until I began my current position, I dealt only with chronic pain patients who had psychological issues. Due to my current program's requirement that all patients being considered for treatment have a psychological evaluation, I have been pleasantly surprised at the number of patients with chronic pain who do not have depression, anxiety, or psychological factors playing an obvious role in their pain experience. This leads to the conclusion that many, if not most, pain patients show resilience when dealing with chronic pain. Compared to my previously seeing patients who were usually in the early stages of dealing with chronic pain, I now see many patients who have dealt with the pain for many years. There are two obvious factors associated with fewer psychological issues being present with long-term pain patients; they have completed the grief process related to the various pain-related losses and they are either working or receiving disability benefits.

I hope the foregoing comments drive home a very important point to those who treat or plan to treat chronic pain patients experiencing psychological issues; every patient is different and needs to be evaluated in that light. That includes the patients' psychosocial history and current situational factors. Although manualized treatment programs for various psychological problems have been a step forward, I propose that it is possible to do much more with the CBM approach based on a brain model. As opposed to viewing patients with the same diagnosis as somehow representing a uniform population, I discuss how a similar set of symptoms may have differing, but identifiable, factors leading to the symptoms. Those

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factors involve current factors, loss-related issues, and influential negative emotional memories. In an expansion of the biopsychosocial viewpoint, the CBM provides an explanation to the patient of the manner (based on brain operations) in which all factors (including past emotional memories) fit together and are logically expected to result in her/his psychological symptoms (Chapter 12). Based on the factors identified, a treatment approach follows to address each factor. Therefore, within the first two sessions it is possible to assess and conceptualize for the patient how and why problems exist, allowing formalized treatment to start by the third session.

Returning to the discussion of transitions in pain management treatment, by the early 1990's, the multidisciplinary programs with which I was familiar had closed. I understand this was a financial decision because insurance carriers began refusing to pay for that treatment due to its failure to reduce overall medical expenses for injured workers. After discharge, patients failed to return to work and were often back on their opiate pain medications within a year. At that time each specialty became independent with little communication. There was the development of outpatient "back schools" and "work hardening programs" to provide education (typically PT-related subjects, stress management, and nutrition) and exercise, but these were usually independent from treating physicians and psychologists. My observation was that each of these outpatient programs lasted for about 5 years, at which time I believe insurance carriers again determined that patients did not reduce their use of medical treatments and failed to return to work. It was after that point that I discovered most of my patients were never educated on body mechanics for activities and other PT strategies (e.g., ways to create a pelvic tilt such as pulling car seats closer, using foot stools while sitting, propping up one foot while standing). Any such education, if done, involved giving handouts with brief instruction. As will be discussed, that does not provide a means for such adaptive behaviors to be rehearsed frequently that would allow the possibility that the behaviors become automatic (i.e., cerebellum controlled as will be explained).

In the 1990's, a major shift occurred when the chronic use of opiates was allowed. The American Pain Society was instrumental in that change. The justifying concept I heard communicated was that many chronic pain patients could not reasonably function in daily activities and opiates allowed enough pain reduction for those patients to have a better quality of life. I am in complete agreement with that concept for patients with disabling pain conditions with identifiable causes. Over time, the use of risk assessment was proposed; low-risk patients could be treated by primary care providers (PCP's), moderate-risk patients meant PCP's would treat in consultation with an addiction specialist, and high-risk patients were referred to a pain clinic. I am unsure as to how often this was actually done, but I suspect it was very little (cf., Gallagher and Rosenthal, 2008).

When I arrived, I found my current program used a five-point rating system (low, low to moderate, moderate, moderate to high, and high) that was not objectively defined by my predecessors. My first step was to provide such a definition based on: the presence of depression (and anxiety to a lesser extent) and whether it was adequately treated; history of substance abuse and treatment; physical and/or sexual abuse in childhood/early teen years; cognitive impairment; and 2 screening assessments¹ for opiate misuse risk. As I explained to our providers and program manager, my risk ratings are now being used in a different context than was originally intended because there is no consideration of PCP pain treatment. I strongly recommended that the ratings not be used to determine program eligibility because a high-risk rating simply means the patient should be treated in a pain program which will happen if accepted into our program. However, each provider is the one who makes treatment decisions based on program rules (e.g., urine drug screen is consistent with the patient report, honesty about prescriptions obtained, not using benzodiazepines, objective medical testing supporting the basis for pain) and the psychological evaluation at her own discretion. I consider the psychological evaluation to be a very important component for identifying the presence of psychological issues and making treatment recommendations, although my preference would be to do so without the risk rating system. However, I believe it is important to note that our program was well-designed with clearly defined rules for dealing with aberrant behavior and dismissal, and includes required urine drug screens at each visit and using the prescription monitoring program (pharmacies in the state report opiate prescriptions and their sources) for verification purposes.

In addition to aggressive drug manufacturers' marketing that has been reported in the news, it appears that a lack of provider training and monitoring greatly contributed to the current opioid crisis. For example, a recent review (Hossain et al., 2020) shows poor adherence by prescribers related to opioid prescribing guidelines for chronic noncancer pain. With the current scrutiny and new regulations, I suspect that similar to ours, most chronic pain programs have seen a large increase in referrals for those longterm pain patients who were previously treated by PCP's. Although a few pain patients will continue to be treated by their PCPs, Mississippi's state

¹ Current Opiate Misuse Measure (COMM; Butler, et al., 2007) and Screener and Opioid Assessment for Patients with Pain - Revised (SOAPP-R; Butler, et al., 2008)

medical board has imposed limitations on the number. Thus, I now see a large number of pain patients with good psychological resilience and no depression or anxiety.

I hope the foregoing discussion is helpful for clinicians, particularly for those with limited exposure to chronic pain management. In the next chapter I will discuss some basic information on the DSM and CBM for the readers who have limited understanding of the brain prior to my detailed discussion of how the DSM applies to pain processing. However, I want to complete this chapter with a discussion of our past findings related to headaches.

Nontraditional Behavioral Approaches in the Assessment and Treatment of Chronic Headaches

Although most practicing psychologists do not address chronic headaches as a primary presenting problem, it is expected to be an issue in a number of their patients (10 to 30% of the general population were reported to experience chronic headache pain in one form or another when I last wrote on the subject; Moss, 1988). The two areas I discuss relate to morning onset headaches that may be related to sleep longevity and temporal (temple) headaches (typically classified as common migraines) that appear to often being associated with parafunctional oral habits.

Common migraine headaches are the most frequent form of migraine and do not have an associated aura prior to pain onset. Although there is no study to my knowledge that provides general population information on the frequency of common migraine with concurrent temporomandibular disorder (TMD) and/or parafunctional oral habits, there have been studies showing a high concurrence. For example, Goncalves et al. (2013) found in 38 women with episodic migraines that 86.8% had TMD, while 91.3 % of the 23 women with chronic migraines had TMD. That compared to 33.3% of 30 women without headaches having TMD. In a study (Didier et al., 2014) of parafunctional oral habits and gum chewing in 125 women with chronic daily headaches who were undergoing a withdrawal protocol to treat medication overuse, 80% showed oral parafunctions and 48% reported chewing gum for extended times. The most frequent parafunctional oral behaviors were combined clenching and grinding (44%), clenching (25.6%), and grinding (10.4%).

In relation to chronic morning headaches in a European general population sample of 18,980 individuals, Ohayon (2004) found a prevalence of 7.6%. The two highest associated factors were comorbid anxiety and depressive disorders (28.5%) and major depressive disorder alone (21.3%). The prevalence for subjects with an anxiety disorder was 10.8%. Based on

the foregoing information, it seems reasonable for psychologists to briefly assess for headaches with their clinical populations.

I was involved in research with TMJ disorder patients when we did a study to examine self-reported parafunctional oral habits (e.g., teeth clenching) comparing facial pain, tension headache, migraine headache, combined migraine and muscle tension headache, and non-headache groups (Moss et al., 1984). A surprising finding was that migraine headache and combined headache patients reported more frequent oral habits than did tension headache and non-headache subjects, while not differing from the frequency reported by facial pain patients. After several years of additional research, I believed it likely that many of the patients who experienced temporal headache pain had internal derangement of the TMJ in conjunction with frequent oral habits (Moss, 1987a). It appeared that individuals who had only TMJ problems or only oral habits were much less prone to headaches and facial pain.

In relation to morning headaches, we (Moss et al., 1987) reviewed the research literature and found overlap in the pain and sleep neurophysiological systems. At that time (in retrospect I consider the theory too simplistic), we suggested that excessive sleep may cause decreased serotonin levels with an associated decrease in pain threshold. For two classic migraine patients (having visual auras) and one morning onset "tension" headache patient, we found a history of increased headaches with increased sleep duration. All patients eliminated their headaches with a sleep manipulation involving their arising at a consistently earlier time.

Based on these early studies, I want to provide the relatively simple way we treated each type of headaches. A detailed discussion on the manner in which TMJ problems are assessed and dental treatments is available in a past article (Moss, 1988). I typically inquire about headache location and, if in the temple(s), how often the patient perceives they engage in parafunctional oral habits (clenching, grinding, lip/mouth biting, resting chin on the hands, chewing ice, pencils, resting jaw on one's hand, etc.) and chew gum. If you wish to have the patient verify that oral habits may contribute to pain, having them clench their teeth until pain begins or for a maximum of 5 minutes should determine if the typical pain can be induced.

The intervention involves a program to reduce the detrimental oral habits. Obviously, if the patient chews gum, ice, etc., they need only be instructed to refrain from doing this due to its being under conscious, voluntary control. Parafunctional oral habits are addressed by having the patient place small stickers (e.g., stars) in all locations/areas that she/he often passes or views. The stickers are to be placed on a large number of objects, such as a refrigerator, mirror (bathroom, rear-view), computer

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screen, car dashboard, etc. Each time the patients sees the sticker, it is a cue to check if her/his jaw is relaxed. If engaged in detrimental oral habits, she/he takes 15 to 30 seconds of keeping the jaw relaxed (i.e., lips together and teeth slightly apart with hands away from the face). It is also suggested that patients practice relaxation techniques a couple of times a day which is probably a procedure known to most clinicians. Although each clinician can decide how to approach relaxation, in my research with facial pain patient treatment I used a hybrid of the progressive relaxation approach I learned in graduate school (Bernstein and Borkovec, 1973). I added a brief three second jaw jutting muscle tightening because the pterygoid muscles are often prone to spasms in facial pain patients. My experience has been that my clinical patients have been more compliant with the habit reduction procedure than with practicing relaxation on a consistent basis.

In reference to sleep-related headaches that are unrelated to sleep apnea. baseline monitoring of the time of awakening and headache days can be done for 2 weeks for patients without daily headaches. If the patient shows more headaches on days with later awakening times, then there is a basis for a sleep intervention. If the patient experiences daily headaches, it is reasonable to start an intervention immediately. Using the awakening times on non-headache days, the patient is instructed to consistently arise 30 minutes earlier. For daily headache patients, they are instructed to arise an hour earlier than their earliest awakening time. The patients should get out of bed immediately and avoid drifting back to sleep which is a pattern I have seen with many morning-onset headache patients. If the sleep manipulation leads to improvement, on days (e.g., weekends) the patient may wish to sleep later, they may stay awake a few minutes and then go back to sleep which appears to be effective for some in avoiding headaches. If patients wish to consistently arise at a later time, they can consistently go to bed later.

I hope the information can be of benefit to some of your patients who experience headaches. I do not see any possible detrimental effects that will occur and these simple procedures may allow some to have many fewer headaches without the use of medications. Obviously, for patients showing a favorable response to these treatments, any decision on the continued use of medications (particularly those used prophylactically) should be discussed with the prescribing physician.

CHAPTER 3

AN INTRODUCTION TO THE CLINICAL BIOPSYCHOLOGICAL MODEL

The concept of a "Grand Unified Theory" was born when Georgi and Glashow (1974) first suggested the possibility of a unified theory of all elementary-particle forces in physics. Stricker (2013) applied this term in relation to true "theoretical integration" in psychotherapy, suggesting a grand unified theory would necessarily have the ability to explain all current single-school approaches that have basic philosophical differences. He indicated that such a theory would require the ability to explain both the stability of behavior and the ready changeability of behavior. He noted the fact that, to date, both physicists and psychotherapists have failed to provide such a theory in their respective fields.

Since the central nervous system is involved in all cognitions, emotions, and behaviors targeted by psychotherapists, it seems logical that a unified theory has to be based on a brain model. There has been a growing interest in the neuroscience field as applied to psychotherapy. Notable authors include Cozolino (2017), Ecker (Ecker, Ticic, and Hulley, 2012), McGilchrist (2012), Northoff (2012), Panksepp (2010), Schore (2002), and Siegel (2007, 2012). Despite the laudable efforts of so many, there has been a glaring missing component in all prior work. There has not been mention of how psychotherapy integration can occur based on a viable model of the manner in which the brain processes information and stores the memories of those processes.

There are major efforts underway in the pursuit of a better understanding of the human brain. In the past decade there have been two major efforts, the United States led Brain Research through Advancing Innovative Nanotechnology (BRAIN; Alivisatos et al., 2013) and the massive European Union's Human Brain Project (Markram, 2012). Additionally, the Research Domain Criteria (RDoC) approach of the National Institute of Mental Health (Cuthbert and Kozak, 2013; Sanislow et al. 2010) steers researchers toward identifying brain areas and systems as opposed to the traditional classifications of the Diagnostic and Statistical Manual-5 (DSM- 5; 2013). However, there is a frequently mentioned criticism of these efforts that promise to develop new technologies and generate massive amounts of data – an absence of neuroscience theory (Moss and Martin, 2014).

Miller (2010) adamantly stated that we do not know how biologypsychology causation works and that studies routinely offer completely inadequate interpretations. The result of this theory void is that attempts to merge neuroscience with psychotherapy have largely failed to produce new insights into the development and maintenance of psychological problems. Moreover, the emergence of a grand unified theory that can explain the observations and treatment findings of the current major approaches to individual therapy (i.e., psychodynamic, humanistic/experiential, and behavioral/cognitive-behavioral; Greenberg, 2002) has appeared improbable. Within this book, I hope to convince psychotherapists and neuroscientists alike that a theoretical model currently exists that has the potential to explain how cortical processing and memory occur, as well as how this leads to a unified theory of psychotherapy.

A summary statement of the Clinical Biopsychological approach is as follows:

We each have a brain. We each have two minds, as does everyone with whom we have a relationship. We verbally think and form verbal memories. We emotionally think and form emotional memories. Verbal and emotional processing occurs independently, but each can influence the other internally, and by controlling the external world perceived by the brain. It is possible to use a brain model to guide assessment, conceptualization, and treatment with patients.

Overview

The CBM was first described in two treatment manuals (Moss, 1992, 2001). I (2007, 2010, 2013a, 2015) subsequently discussed the theoretical aspects of the model as related to negative emotional memories and loss issues. At its core is the proposition that a group of several thousand neurons that comprise a *cortical column* is the fundamental binary unit (i.e., bit) involved in all cerebral cortical processing and memory storage (Moss, 2006). To assist psychotherapists lacking a strong neuroscience background, a simplified explanation of the brain model will first be provided.

First, all nervous system activities (i.e., barring more generalized circulatory system hormonal influences) work by connections. If there are no connections, then two areas cannot communicate directly. This is critical in an understanding of "conscious" versus "unconscious" processing and responses. This also indicates that the vague "chemical imbalance"

explanation of psychological disorders is wrong. I acknowledge that the major neurotransmitter systems (e.g., serotonin, dopamine, acetylcholine) influence both cortical and subcortical functioning, but imbalances in these systems cannot explain the manner in which negative emotional memories or loss of something positive lead to specific negative mood/affective states (e.g., sadness and depression, anxiety).

The brain is designed for one obvious purpose; the survival of the individual to allow the survival of the species. This requires that the brain have an accurate representation of both external and internal stimuli. Thus, sensory information that arrives at the cerebral cortex is as an accurate reflection of those stimuli, followed by the processing of the information which allows meaningful action. The same cortical areas involved in the original processing of information are the same areas reactivated when the memory is recalled. This means that all the sensory memories, including those emotional in nature, involve direct connections at the cortical, and not the subcortical, level.

The two cortices are considered to be semi-independent functioning minds. Within the suggested parallel processing design, the side that can best respond to an ongoing situation is the one that assumes control of the ensuing response. Both hemispheres receive similar sensory input. The posterior lobes (i.e., parietal, temporal, and occipital) are involved with processing and memory storage tied to incoming sensory information, while the frontal lobes are involved with analysis, planning, and response initiation, as well as associated memories of such activities (see Figure 3-1 for lobe locations). The left cortex processes sensory information in a detailed manner, resulting in its being slower than the right. The right cortex processes the information much faster (in milliseconds), but in a global, less-detailed manner. There is exchange of information between the sides, although this exchange can be both excitatory and inhibitory. From a developmental perspective, there is initially only very limited information exchange between lobes within each side, and between the hemispheres. This logically allows each cortical area to develop fully its memories and associated processing prior to influence from other areas. Additionally, left hemisphere functions (e.g., receptive and expressive speech) will develop slower than those of the right hemisphere (e.g., non-verbal emotional analyses and responses) because there are a greater number of information units (i.e., cortical columns) and interconnections in the circuits associated with left hemisphere processing. A final point is that the right hemisphere's global processing allows for faster responses if confronted with outside danger; suggesting this side is best designed biologically (i.e., for survival) to respond and assume behavioral control while in a negative emotional state.



Figure 3-1. A depiction of the cerebral cortical lobes.

In addition to the well-known distinction of simultaneous right and left cortical activity, there are also multiple streams (i.e., circuits of columns) of information processing that occur in the same hemisphere (e.g., internal/self-referential information involves the medial cortex while external information involves the lateral cortex). Whichever information streams are the most relevant to an ongoing situation are the ones leading to action associated with attention and behavioral responses. Because these processes occur in fractions of seconds and have been present for one's lifetime, it gives us the subjective impression that in most circumstances the "mind" is a uniform whole. In reality, whichever circuits can most effectively analyze and respond to a given situation are the ones that lead to the response. This means that there is often a lack of accurate verbal awareness as to why one's own actions occur.

The left cortex primarily handles language functions because of its capacity to do highly detailed processing. Thus, the left posterior areas are involved in comprehending (including memory storage) both spoken and written language, while the left frontal lobe controls spoken language, including the motor memories of language. Thus, thinking verbally is a left

cortical process involving the ventral lateral frontal lobe and is called the "verbal interpreter" (Moss, 2007; 2013a; 2015). I proposed the concept of that the left hemisphere was responsible for verbal-thinking in a clinical treatment manual in 1992 (Moss, 1992). Gazzaniga (2002) discussed a similar concept and he called it the "interpreter." He theorized that the interpreter attempts to make sense of things. He considered it a device, system, or mechanism that seeks explanations for event occurrence. He saw the advantage of an interpreter as allowing more effective coping with similar future occurring events. Notably, Gazzaniga viewed it as only one of the cortical "modules" that exist. Due to my belief in the accuracy of his concept I adopted the use of the term "verbal interpreter" (Moss, 2006).

Several papers can be used in support of this concept. Wagner et al. (1997) provided data that the left inferior prefrontal cortex may act as a semantic executive system that mediates retrieval of long-term conceptual knowledge, regardless of perceptual form (i.e., words and pictures). In a review of studies, Badre and Wagner (2007) discussed evidence supporting that, in relation to cognitive control of memory, the left anterior and left middle ventral lateral prefrontal cortices provide controlled retrieval and post-retrieval, respectively. The left frontal cortex has been shown to be involved in successful word recognition of both shallow and deep level of processing, as was the left hippocampus (Schott et al., 2013). Another study (Barredo, Öztekin, and Badre, 2013) provided support for the existence of a ventral frontal to temporal pathway involved with the cognitive control of episodic memory retrieval. In macaques, the ventral lateral prefrontal cortex has been shown (Hage and Nieder, 2015) as involved in response to speciesspecific calls. This was suggested to be a precursor to audio-vocal integration that ultimately gave rise to human speech. Based on their recording of task-related neurons with two macaques, Bruni et al. (2015) proposed that this frontal region may host an abstract "vocabulary" (author's italics) of the intended goals pursued by primates in their natural environments. In relation to declarative or episodic memories, these are defined as the individual being capable of providing relevant verbal information. The DSM views the verbal interpreter cortical circuitry as a sequential action processor and just one of many parallel circuits that exist. Many of these parallel circuits connect to the verbal interpreter. However, there are multiple parallel cortical circuits involved in complex memories (e.g., spatial learning) that do not include the verbal interpreter. Such nonverbal memories are not considered to be qualitatively different in the way the columnar circuits are formed and memory occurs.

A point that will be discussed in Chapter 5 is that the DSM indicates there is a corresponding right ventral frontal area that handles sequential