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Attachment-Based Treatment for Anxiety Disorders Use of spontaneity as a mode of relating to resolve anxiety

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Summary

Although we have established effective treatments for anxiety disorders,the problem is on the rise worldwide, and the effect sizesfor treatments suggest that advancements are needed. Part of the reason for the limitations in our treatments may be because they are designed to address the disorder's symptoms rather than its underlying cause. This paper is geared toward presenting a likely neurobiological cause and a proposed treatment.

There is significant evidence that the basis for anxiety is a combination of stress, especially interpersonal stress, and an inability to find solutions to resolve it. Anxiety is the result of this lack of coping ability. The part of the brain involved in designing these coping strategies, especially when the stressor is complex and emotion-based, is the dorsolateral prefrontal cortex. That is because it is the brain's "sketch pad," in that it allows us to think of solutions on a "symbolic" rather than a concrete basis.Anxiety arises when the person is unable to use this brain region effectively in formulating solutions, and instead designs ineffective solutions. The reason for that deficit may stem from early childhood, during a phase of attachment in which parents do not engage spontaneously in child-induced play. The mother is unable to be spontaneous in sharing the child's play,which is needed for the child to generate his or her own solutions to problems. This leads to an inability of the child's dorsolateral prefrontal cortex structure to properly develop. This will manifest as a lack of "insight," or ability to generate spontaneous solutions to problems instead focusing on routinized solutions to emotion-based problems later in life that often do not work. Evidence is provided in a case report of a patient with an anxiety disorder who could not solve his interpersonal problems because he lacked the necessary insight. This deficit was so profound that he could not use insight-based psychotherapy and found use of medication too sedating. By modelling a more spontaneous give and take between the therapist and the patient's wife, who served as a "co-therapist,"the patient was able to learn to develop his own insight. His wife would tell the therapist the problems the patient was unable to tell him and would help the patient to learn to respond to the interpretations offered by the therapist. The patient learned how to imitate his wife and describe his problems, and then to imitate the therapist and generate his own insight. As this occurred, his anxiety was resolved. The basis of the attachment that developed with his therapists was based on the use of spontaneity, from which the patient learned his own insights resolving his anxiety.

Keywords: Cardiac Percussion, Left Ventricular Geometry, Cardiothoracic Ratio

Introduction

Anxiety Disorders are the most prevalent psychiatric disorder and are associated with the highest burden of illness of any mental disorder. According to the World Health Organization (2019), 1 in 13 adults globally suffers from anxiety. Furthermore, according to large population-based surveys (Bandelow and Michaelis, 2015) up to 33.7% of the population will be affected by an anxiety disorder during their lifetime.Based on the Global Burden of Disease study, anxiety disorders are the sixth leading cause of disability in terms of years of life lived with disability in both high-income and low- and middle-income countries, accounting for 390 disability-adjusted life years per 100,000 persons [1].

With regard to the effectiveness of treatment for these disorders, Bandelow conducted a metanalysis on the efficacy of current treatments for anxiety, including different types of psychotherapy, pharmacotherapy, and combination therapies [2]. They compared 234 studies involving 37,333 patients. Medications were associated with a significantly higher average pre-post effect size than psychological therapies. Further, they found a higher

effect size for mindfulness therapy than the most effective form of psychotherapy, cognitive behavioral therapy. The effect size for psychotherapy and medication combined was a bit better than pharmacotherapy alone (2.12 vs. 2.02); however, there is clearly room for improvement, especially in psychotherapy.

No form of treatment, psychotherapy, or medication has demonstrated a clear relationship between the genesis of a disorder and the treatment of that disorder. Although the effectiveness of our treatments for anxiety is significant, improvements in treatment may need to focus more on possible etiologies which can be reversed, rather than on strictly concentrating on reversing symptoms. One potential avenue for advancement might be based on the established underlying neurobiologically-based susceptibility factor, which could precipitate anxiety when one is under stress. Recent investigations in neurobiology have established this potential neurobiological vulnerability. Further, this vulnerability can potentially be reversed by psychotherapy resulting in a resolution of the disorder when stressful conditions are presented.

Body

In a previous study, Monk et al found that, in adolescents with generalized anxiety disorder, when responding to angry faces, the ventrolateral prefrontal cortex was hyperactivated,and they showed an increased attentional bias to angry faces,compared with controls [3]. Moon found that in patients with generalized anxiety, a lower level of metabolic activity in the dorsolateral prefrontal cortex and the degree of hypoactivity were directly correlated with symptom severity [4]. Similarly, in a metanalysis of proton magnetic resonance spectroscopy studies in generalized anxiety disorder, Delvecchio et al found altered metabolic levels in the dorsolateral prefrontal cortex and hippocampus [5]. Moon and Jeong also found reduced metabolic activity in the dorsolateral prefrontal cortex and reduced white matter volume in that same region, as well as in the midbrain, precentral gyrus, and anterior limb of the internal capsule [6]. These investigators further reported a correlation between morphological deficits and reduced brain activity in the dorsolateral prefrontal cortex. When faced with emotional distractors and measuring working memory task performance, patients with generalized anxiety disorder demonstrated reduced metabolic activity in the dorsolateral prefrontal cortex and elevated activity in the hippocampus [7].

Dolcos and McCarthy demonstrated a possible mechanism for generating anxiety in a model using young adults [8]. They recorded the activity in various brain regions when participants were exposed to different types of distractors while performing working memory tasks. The distractors were emotional stimuli, in that participants were shown pictures of different emotional scenes, contrasted with pictures that were similar but had no emotional content. The researchers found that emotional distractors produced the greatest reduction in activity in the dorsolateral prefrontal cortex, in which action plans would be generated to accomplish the task. Concurrently, there was the greatest increase in the ventromedial prefrontal cortex, where emotions are generated and organized. These investigators concluded that it was the shift between these two brain regions thatmay be associated with the processing of emotion-based information. Based on these experiments, we can see that the dorsolateral prefrontal cortex,which is involved in the process of finding “solutions” to emotional issues,may be deficient, and the ventromedial prefrontal cortex may become over activated during the process of experiencing emotions. Anxiety may then occur when the person is overloaded with too many emotional distractors and cannot shift that activity to the dorsolateral prefrontal cortex to effectively generate solutions. Thus, it may be that the brain region in which deficits may be present in these patients may be in the processing of emotions may be the transition region between the ventromedial and dorsolateral prefrontal cortex.

Data on the decrease in dorsolateral prefrontal cortex activity in generalized anxiety disorder are considerable [6, 9-11]. Moreover, there is significant evidence that these same patients have lack of insight [12]. Newman et al provided evidence that these patients avoid having insight through their excessive worry [13]. Crouch provided further support for the notion of a lack of insight in these patients by demonstrating that, in a naturalistic setting, they used “contrast avoidance,” or the inability to cope with shifts in emotions from positive to negative with this use of excessive worry, in lieu of insight.

Warren et al showed that stress and anxiety alter decision-making ability by altering the balance between the amygdala, which is increasingly activated by stress and anxiety, and decreasing activation of the dorsolateral prefrontal cortex, where decision-making processes to generate solutions to stress occur in children [14]. The evidence from a neurobiological perspective supports that a failure of insight may be associated with the failure to activate the dorsolateral prefrontal cortex in generalized anxiety disorder patients. This failure may be associated with the excessive buildup of emotions in the ventromedial prefrontal cortex, which is the cause of the anxiety. That may be the reason why helping patients develop insight is so critical to the effectiveness of psychotherapy. However, paradoxically, generalized anxiety is the least of the anxiety disorders amenable to treatment [15]. This may be because the neurobiological deficit that may produce this lack of insight is not being addressed by treatment.Often, these patients “appear” to have insight, but do not really have the capacity for it. These treatments address the symptoms but not the likely underlying cause. Siegal, in developing the concept of mindsight, outlines this failure. However, no one has ever discussed that this failure may be due to a neurobiologically based structural deficit in the process of transition between the ventromedial and dorsolateral prefrontal cortex, nor have they offered a potential remedy.

Rudorf and Hare studied context-dependent valuation to determine the role of switching from the ventromedial to the dorsolateral prefrontal cortex in decision-making [16]. Since the ventromedial prefrontal cortex is mostly involved in choices based on reactivity

to environmental circumstances, while the dorsolateral prefrontal cortex is involved in self-directed choices, these investigators sought to determine if this choice was related to the context in which the choice is made. They found that the more participants were forced to deviate from the basic context to accommodate a second variable, in addition to the primary one of choosing between two simple options, the more the dorsolateral prefrontal cortex was involved. What this means is that the more confusing the context of a situation is, the more the person has to make choices based on self-directed objectives, as opposed to in situations that require less complex decision-making.

Thus, the shift from the ventromedial to the dorsolateral prefrontal cortex was designed to deal with more complex decisions requiring self-directed goals that are more sophisticated than what is generally required in complex emotional situations. The implication here is that this advance from the basic response system to more complex environmental social conditions that provide context for decisions is involved.

The reason that the dorsolateral prefrontal cortex is needed for these more complex decisions is that it is capable of dealing with a more advanced, more symbolic thought processesthan are the ventromedial prefrontal cortex or any other brain region. This is because the dorsolateral prefrontal cortex has a layer of pyramidal cells, which enable it make connections in an effective and flexible manner not found in any other brain region. This is why it is called the brain's sketch pad [17]. The brains of mammals other than primates and humans do not have an equivalent to this brain region [18, 19]. Evidence suggests that the reason why patients with generalized anxiety disorder are unable to successfully process emotional stimuli in the prefrontal cortex when under stress may be failure of the activation of the dorsolateral prefrontal cortex resulting in a lack of insight.

It might be a failure of the region between the ventromedial and dorsolateral prefrontal cortex, which is involved in the "switch" in activation between these two regions, that is responsible for the deficits seen in patients with anxiety. The anterior cingulate has been shown to be the transition point between the ventromedial and dorsolateral prefrontal cortex, and stability of attachment may be reflected in improved anterior cingulate function when threatened by social stressors, and attachment stability is needed to buffer that stress [20]. Karremans et al demonstrated this in an experiment using a group rejection paradigm as a social stressor. Those who were more securely attached had a decrease in fMRI brain reduction of activity in the anterior cingulate, ventromedial, and dorsolateral regions, relative to more insecurely attached individuals. They further found that the presence of an attachment figure further reduced hypoactivation of the anterior cingulate, especially in more securely attached individuals [21]. Dewall et al found that the type of attachment deficit distinguished anterior cingulate response to social rejection. More anxiously attached individuals showed hyperactivation of their anterior cingulate, likely because they were more sensitive to rejection, while avoidantly attached individuals showed a decrease in anterior cingulate activity in response to this same cue. This would be expected, as this group is buffered against sensitivity to social rejection.

This concept of attachment-based synchronicity between mother and child serving as a vehicle that enables the child to make the brain-based transition between the ventromedial and dorsolateral prefrontal cortex is supported by brain-to-brain synchronicity studies between bonded mothers and children. In a synchronicity study using functional near infrared spectroscopy (fNIRS), Reindl et al found that, during cooperative activities, bonded mothers and children found synchronization between the brain dorsolateeral prefrontal cortices of both partners, that this synchronization predicted the efficiency of their performance, and that this synchrony was directly correlated with the child's ability to regulate his or her emotions [22]. In a similar brain-to-brain synchronicty study between bonded mothers and children, Miller et al found that this synchronicity was directly related to not only the effectiveness of cooperation but also the quality of the attachment relationship and predominated in the right dorsolateral and frontopolar lobes [23]. Thus, there is a brain-to-brain synchronicity between mother and child which reflects the unconscious connectivity-based communication between them, supporting the activation and likely development of the child's dorsolateral prefrontal cortex. This form of attachment is generated in spontaneous interactions between mothers and children that are associated with play. The absence of this mode of communication may be responsible for a lack of this type of attachmentand, in axiety patients,may result in a failure to use insight and to activate the dorsolateral prefrontal cortex. This is especially true in those who lack insight completely.

Oxytocin receptors, which are associated with attachment processes, have been found in the anterior cingulate in chimpanzees and humans [24]. When oxytocin was administered to generalized anxiety disorder patients viewing fearful faces, it enhanced the connectivity of the amygdala and the middle cingulate and dorsal anterior cingulate cortex and the insula, which was not seen in the control group without generalized anxiety disorder. These investigators also found that the greater the connectivity, the less the anxiety [25]. Similarly, oxytocin administered to generalized anxiety disorder patients at rest increased the amygdala rostral anterior cingulate cortex–medial prefrontal cortex connection and, in doing so, reversed the deficit in this connectivity found in those patients relative to controls [26]. Thus, oxytocin, which is involved in attachment processes, can improve the connectivity of the amygdala-based fear system,especially by improving the connectivity of the anterior cingulate with the dorsolateral prefrontal cortex in order to enable patients with anxiety-based disorders manage stress.

Can this capacity be restored by forming an attachment in psychotherapy similar to that which was deficient in a patient's childhood? If we look at the data from the brain-to-brain synchrony in cooperative spontaneous play, it seems likely that it can. It is the spontaneity of that cooperative play that appears to be the most

salient aspect of that communication and the attachment process inherent in it. In the case report to follow, I demonstrate that this can occur if the treatment set-up is modified to facilitate its occurrence.

In the case presentation to follow, I show how it can be attained by altering the structure of psychotherapy to include the wife of the patient, with whom I could have coopertive spontaneity-based interaction,thus providing the patient with a model for developing his own insight into the meaning of his actions. This could be viewed as me providing the insight, or the function of the dorsolateral prefrontal cortex, and his wife providing the input from his daily life, or serving the role of the ventromedial prefrontal cortex. It was the spontaneity of our interactions that ultimately engaged him in an attachment process with us. That resulted in his first imitating his wife by providing his own input, and then imitating me by generating bhis own insight. As this occurred, his anxiety symptoms resolved without any psychotropic medication.

Case History: Sam

Sam had retired after practicing law for over forty years when he was referred to me. He had been very successful. Toward the end of his career, he shocked his family by divorcing his first wife and going to live with another woman. His new partner was not controlling, as his first wife had been, and she showed him real affection, of which his first wife was not capable.

Sam had two children from his first marriage, both of whom were gradually able to accept his decision to dissolve his marriage. However,after his retirement, he found that, alone with his new partner and without the admiration of his coworkers, he became far more anxious. He had no interests or hobbies, and an automobile accident had left his left arm weakened. Although doctors were unable to find anything wrong with his arm, he often used this injury to seek attention from others. Most people, including his wife, felt that anxiety was responsible for most of his issues, but Sam preferred to blame everything on hisaccident.

After I began working with Sam, he told me that when he was four, he was playing quietly on his living room floor with a new toy when his mother, who was in the same room cleaning out a closet nearby, suddenly shrieked, “A mouse!” and began wildly swiping a broom in its direction. The commotion interrupted Sam’s play, and he remembers wondering what the big deal was over a little mouse. Although his father came to the rescue, Sam was unable to go back to concentrating on his game or on the new discovery he was about to make. He remembered the event vividly, but this was not the only time he was distracted by his mother’s supercharged displays of emotion. One could say that Sam’s ability to have self-generating activities that were stress-free and could be shared by his mother never materialized. Instead, he was overloaded with excessive emotion-based stimulation, mostly generated by his mother, which produced anxiety. He learned how to put out these hysterical-based behaviors, which he did on becoming a very successful attorney.

However, Sam had no self-generated insight, no ability to direct his own actions and plans, and was unable to be assertive or manage his emotions. He was constantly motivated by putting out fires generated by pseudo-stressful situations. As he was not capable of self-generated activity of any sort, he experienced anxiety when there were no fires to put out.

Sam was born in Baltimore into an Irish-American family, the oldest of eight children. As he described it, everyone in the family had a role that they played constantly—there was little or no variation or spontaneity. Sam was the prodigal son, his father was his mother’s enforcer, his sister was the family failure, his youngest brother was the priest, and so on. His mother ruled the roost through her overly dramatic actions.

Sam’s first wife’s behavior closely aligned with thatof his mother. She ruled the roost with the same iron hand his mother had, using the same histrionics. Sam was supposed to be the enforcer like his father. When he went to California for law school, his wife went with him until their first child was born, when she returned to Baltimore so her parents could help raise the child. Later, after he returned east to practice law and they had two more children, Sam became their primary caregiver because his wife was so preoccupied with her anxiety. He would come home after work to cook dinner and attend to the children. There was no gratification in any of this for him, and he eventually found a much more satisfying life with his new girlfriend. All went well for him until his retirement.

Although Sam did not have insight into his situation, his partner, Gwen, did. He would come to his sessions with nothing to say, asking, “What should I talk about?” Then, Gwen would tell me what was going on, I would provide interpretations based on her input, and she would help him apply what I said to his life. Using a brain model, Gwen served as the ventromedial prefrontal cortex, standing in for Sam. I was providing her dorsolateral prefrontal cortex with insight, which she could use to organize the ventromedial prefrontal cortex, serving as a model for him. As Sam saw how the model worked, he began to imitate Gwen by providing the input himself. He also began to use my interpretations himself, without her mediation. Then, he started to generate insight by himself as well.

If we look at Sam’s early life, we can see the root of his problem. His mother, instead of helping him develop insight by playing with him spontaneously, was overstimulating his perceptual apparatus with “noise,” by creating overly dramatized “scenes” in which someone, usually Sam’s father, had to come to her rescue. Sam was never able to engage in spontaneous play with either of his parents. He never learned how to develop self-generated ideas about how he wanted to copewith his emotional reactions to others. Instead, he lived his life constantly reacting to the emotions of others, especially women like his overly dramatic first wife. Play was never fun or spontaneous. Using this treatment modification, with Gwen’s assistance, we were able to reverse the process. Gwen did

not flood me with input the way Sam's mother had flooded him, enabling me to provide useful interpretations.

The excessive input the world provided for Sam—input he saw as meaningless distraction—kept his system at a biological level. The attachment he formed with Gwen and me helped him transform input at a psychological level. He started to developinsight into his problems and initiate actions based upon those insights. He began to recognize when he was being overstimulated.

A key turning point came when, in discussing his mother's hysteria, Sam continued to insist that his mother really was frightened by the mouse and Gwen, amazed at his naivete, challenged him. I spontaneously remembered a situation like his from my own childhood, and told him a story that occurred when I was eight. It was Yom Kippur in the Jewish calendar, the day everyone was expected to fast and pray all day in the synagogue to atone for their sins. My father had tickets for a Brooklyn Dodgers baseball game that day. He swore me to secrecy, and we snuck out of the synagogue, ate hotdogs at the game, and returned to the synagogue at sundown—just in time for the rabbi to dismiss the congregation so that they could break their fast. When we got home, my mother and grandmother heaped praise on me for fasting all day, while my father watched me carefully to make sure I kept our pact. As with Sam's mother, my father's behavior was not a one-time event. In many variations, my father enacted this same scene over and over, expecting my complicity and punishing me when he did not get it. Although I, too, liked going to the ballgame, the demand in my childhood to constantly play along with his strategies for avoiding a more authentic relationship with his religion, our family, me, and himself was a continual burden.

I was inspired to share this story with Sam after he kept insisting that his mother really was anxious and this was not just hysterical behavior. In a sense, his mother and my father were like puppeteers, pulling the strings of everyone else. Sam, however, laughed at this anecdote, and said my father was deliberately fooling everyone, to which Gwen responded: "And your mother—wasn't she deceiving you about how frightened she was?"

Ultimately, Sam admitted that his mother had overreacted, just as my father always did. "Maybe she was play-acting a bit," he said. This was a critical breakthrough. From this point on in his treatment, he started to develop insight. Our spontaneity with each other, and with Gwen, formed the basis of a real attachment between us, upon which he could develop a sense of self-direction, initiative, and insight ability, none of which he had in the past. His anxiety started to recede. His attachment enabled him to drop the histrionic, overly dramatized way he related, blaming everything on his accident—a strategy that he realized was not credible.

In sharing my story, I had not departed that much from my psychotherapist role, but it was enough. Defined roles, including that of psychotherapist, are important and helpful to many, but we must also be aware of when that role needs to be modified to connect in a real way with the patient in order to make that role work. Knowing when to apply this principle is critical for all therapists.

Sam knew his mother was playing him, just as he was playing me. By telling a story from my own life that directly corresponded to his, I was able to have a spontaneous moment with Sam, thereby allowing him to have one with me. We could think that Sam was just "going along" with Gwen and me in admitting his mother's behavior, but the "real connection" between us at that time was considerable. He was spontaneous with us, and from then on would think about what he really thought instead of just reacting to what others thought. It was the exact opposite of the overly dramatic and unspontaneous behavior of his mother and the rest of his family. It was not a "strategy"—it was what I said without thinking. That experience, and many others like it which he had during treatment, restructured his attachments, enabling him to develop insight and self-directed behaviors.

After about five months, the dynamic interplay in treatment started to accelerate in intensity and sophistication. Sam went from being withdrawn to much more engaged in the process. His interest was piqued, and he began to read articles on anxiety and depression. At one point, he read an article asserting that anxiety was genetic. He was clearly baiting me. Gwen, who realized this, said she had read the article, too, adding that genetics was only one of many possible causes of anxiety and wondering why he had only chosen that one. I asked, "Are you indicating that we can't help you because your anxiety is due to genetics?" Sam's face lit up because I had taken the bait, and he declared, "Prove to me that my anxiety is not genetic!" He felt triumphant.

Gwen chimed in that Sam did this all the time—making others the straight man for his jokes. I compared this to the situation with the mouse and his mother, when as a child he felt victimized—the "straight man" for his mother's overdramatization. Sam pretended not to understand, but he knew very well what I was saying. His lack of insight had morphed over time into a game he played to master his anxiety, a game at which he had become very artful. To play this game, he would become passive and appear to be helpless until he pulled the rug out from under the other person.

Gwen came to my assistance. If I had been alone, he would have seen me as discredited. He would have mastered his anxiety by mastering the authority of the therapist. *Sam's way of dealing with the repeated helplessness he felt at being constantly victimized by the world was to prove to himself that they, not him, were the "victims." However, we were gradually shifting. He began to come to his sessions thinking about what he wanted to talk about, instead of waiting for Gwen to tell us what he was doing. Then, he started to try to think about what I might say about any situation, gradually learning to use insight himself.*

We can see from this why traditional treatment would not work for Sam. He was busy making the therapist into the straight man for

his jokes; psychotropic medications had the same fate, because the so-called anxiety that they were treating was his "false anxiety," which could never be cured. However,above all, traditional psychotherapy would not work because, as he lacked insight, Sam was incapable of understanding any interpretation of his behavior.

In the next group of sessions, he persisted along the same track. He asked me, for example, what he had been talking about in the last session, declaring he was trying to initiate the conversation. Again, neither Gwen nor I took the bait. Sam continued to try to bait me. He indicated that I might feel that he was not so sharp in the last session, but that today he was sharper. I refused to take the bait again. He had expected me to say that he was sharp the last time, too. He wanted me to tell him what we said the last time, which he remembered perfectly well.

Gwen again interceded on my behalf, saying that Sam always pretended to be unable to do things so that others would helphim, then left them looking foolish for trying to assist. Gwen's "modeling" the needed insight was enough to move the treatment forward. I suggested to Sam that perhaps it would be better if he recognized this and did not replay the mother/mouse story. The patient was play-acting, just as his mother did all the time, and just like her, he was unable to see beyond this play-acting. Only because I structured the treatment in this manner, we able to transcend the limits of the traditional setting to find a way of forming a real attachment. The model I used was based on the type of brain deficit I believed was underlying his bottom-up to top-down brain imbalance.

As Gwen and I worked to help him accept his anxiety, instead of getting others to appear to be the anxious ones, Sam became more assertive. He came to sessions without his walker (even though he had no real reason to use the walker in the first place), and started sessions himself instead of demanding that we start for him. He acknowledged, for the first time, that it was his own anxiety that was generating some of his physical symptoms. Instead of becoming frustrated with us, he acknowledged that he had been trying to get us to be the butt of his jokes.

I want to skip ahead another year into Sam's treatment, when he fell and fractured his hip and had to go to a nursing home for rehabilitation. There, instead of being his former passive self, he told the staff what he expected from his treatments and complained until they took good care of him. They knew he was an intelligent and informed patient and paid attention to his needs. Sam's assertiveness, spontaneity, and capacity for insight had evolved dramatically. He was truly participating in his physical therapy. As all these changes occurred, his anxiety diminished considerably.

A year later, as the treatment had progressed further, he had a dream in which a woman was preparing a dinner, and he was asked to stay to eat. He wanted to go home, but they told him he must stay, and he felt trapped. When I suggested that represented his feeling of being forced to do things against his will by his family, Sam readily agreed. "That's the story of my life," he said. He then accused Gwen and me of doing the same thing to him. "When do I do that to you?" Gwen said. We were both able to show him that we were encouraging just the opposite, constantly reinforcing his own initiative, and he was able to see this.

Another incident occurred shortly afterward in Sam's treatment. Gwen mentioned that a TV show she had been watching, *Antiques Roadshow*, displayed the bat of a famous baseball player. She told me that Sam had represented him after the player's car accident years before. I remembered this accident vividly from my own childhood, because I very much admired this player. I allowed myself to feel awe that my patient had worked on behalf of one of my childhood heroes. Gwen mentioned that he had also represented the family of another famous ballplayer, and Sam chimed in with his memories while I continued sharing my admiration. These moments of spontaneity drove the treatment and made our attachment work, and that attachment, in turn, made the treatment work. That spontaneity would never have occurred, and the treatment could not have succeeded, without his partner. It istrue that the fortuitous combination of my interest in baseball, Sam's having worked with two players I admired, and his having an insightful partner who could enable the treatment was unusual. However, often, if we seek to find a combination like this to make treatments of this type work, we can do so.

Sam had resolved his inability to have insight and was able to apply his newly developed insight to his life. This enabled him to become assertive and no longer passive. His anxiety resolved. His relationship with his wife markedly improved, as didhis health. We could consider Sam as having resolved his anxiety symptoms and his passive personality trait without any psychotropic medication through this attachment-based treatment.

In ensuing sessions, the patient was able to apply his ability for insight to help his son, who also had difficulties with insight, using my and his wife's advice. He was able to encourage himself to do things he wanted to achieve by self talk, a capacity he had never had previously.

Conclusion

By using neurobiological-based modeling of the structure of the treatment setting, we were able to develop a model by including his wife. She and I developed a "model" psychotherapy which he could imitate. This model contrasted with the behavior of his mother, who never developed any spontaneous play interchange with him to support his development of a successful dorsolateral prefrontal-based insight system. That was the reason for his anxiety disorder. As he established his own spontaneity-based actions, his anxiety dissipated. This treatment model may be very helpful for patients with anxiety disorders. Future research may enable us to further advance this model and apply it to different clinical situation.The basic mode of communication, that of spontaneity, is the basis of this level of attachment. The critical turning point in treatment was when I could connect with Sam brain-to-brain, as I spontaneously

blurted out an experience from my own life,which is something I almost never do. He responded to my spontaneity with his own, and this formed an attachment which generated his development of his own spontaneity, insight ability, and self-directed actions, and ultimately stopped his anxiety.

References

1. Baxter AJ, Vos T, Scott KM, Ferrari AJ, Whiteford HA (2014) The global burden of anxiety disorders in 2010. Psychol Med 44: 2363-2374.
2. Bandelow B, Michaelis S (2015) Epidemiology of anxiety disorders in the 21st century. *Dialogues in Clinical Neuroscience* 17: 327–335.
3. Monk CS, Nelson EE, McClure EB, Mogg K, Bradley BP, et al. (2006) Ventrolateral prefrontal cortex activation and attentional bias in response to angry faces in adolescents with generalized anxiety disorder. Am J Psychiatry 163: 1091-1097.
4. Moon CM, Jeong GW (2016) Brain morphological alterations and cellular metabolic changes in patients with generalized anxiety disorder: a combined DARTEL-based VBM and (1) H-MRS study. MagnReson Imaging 34: 429-436.
5. Delvecchio G, Stanley JA, Altamura AC, Brambilla P (2017) Metabolic alterations in generalised anxiety disorder: a review of proton magnetic resonance spectroscopic studies. Epidemiol Psychiatr Sci 26: 587-595.
6. Moon CM, Kang HK, Jeong GW (2016a) Metabolic change in the right dorsolateral prefrontal cortex and its correlation with symptom severity in patients with generalized anxiety disorder: proton magnetic resonance spectroscopy at 3 Tesla. Psychiatry Clin Neurosci 69: 422-430.
7. Moon CM, Sundaram T, Choi NG, Jeong GW (2016b) Working memory dysfunction associated with brain functional deficits and cellular metabolic changes in patients with generalized anxiety disorder. Psychiatry Res Neuroimaging 254: 137-144.
8. Dolcos F, McCarthy G (2006) Brain systems mediating cognitive interference by emotional distraction. J Neurosci 26: 2072-2079.
9. Mathew SJ, Mao X, Coplan JD, Smith EL, Sackeim HA, et al. (2004) Dorsolateral prefrontal cortical pathology in generalized anxiety disorder: a proton magnetic resonance spectroscopic imaging study. Am J Psychiatry 161: 1119-1121.
10. Mohlman J (2004) Psychosocial treatment of late-life generalized anxiety disorder: Current status and future directions. *Clinical Psychology Review* 24: 149-169.
11. Moscovitch M (2014) Multiple dissociations of function in amnesia Human Memory and Amnesia 4: 338-370.
12. Castrogiovanni A, Iapichino S, Castrogiovanni P (2004) Insight in anxiety disorders. *Italian Journal of Psychopathology* 10: 123-130.
13. Crouch TA, Lewis JA, Erickson TM, Newman MG (2017) Prospective investigation of the contrast avoidance model of generalized anxiety and worry. BehavTher 48: 544-556.
14. Warren SL, Zhang Y, Duberg K, Mistry P, Cai W, et al. (2020) Anxiety and stress alter decision-making dynamics and causal amygdala-dorsolateral prefrontal cortex circuits during emotion regulation in children. Biol Psychiatry 88: 576-586.
15. Newman MG, Llera SJ, Erickson TM, Przeworski A, Castonguay LG (2013) Worry and generalized anxiety disorder: a review and theoretical synthesis of evidence on nature, etiology, mechanisms, and treatment. *Annual Review of Clinical Psychology* 9: 275–297.
16. Rudorf S, Hare TA (2014) Interactions between dorsolateral and ventromedial prefrontal cortex underlie context-dependent stimulus valuation in goal-directed choice. J Neurosci 34: 15988-15996.
17. Arnsten AF, Wang MJ, Paspalas CD (2012) Neuromodulation of thought: flexibilities and vulnerabilities in prefrontal cortical network synapses. Neuron 76: 223-239.
18. Sallet J, Mars RB, Noonan MP, Neubert FX, Jbabdi S, et al. (2013) The organization of dorsal frontal cortex in humans and macaques. *The Journal of Neuroscience* 33: 12255-12274.
19. Koechlin E (2011) Frontal pole function: what is specifically human?*Trends in Cognitive Sciences* 15: 241.
20. Karremans JC, Regalia C, Paleari FG, Fincham FD, Cui M, et al. (2011) Maintaining harmony across the globe: The cross-cultural association between closeness and interpersonal forgiveness. *Social Psychological & Personality Science* 2: 443-451.
21. DeWall CN, Masten CL, Powell C, Combs D, Schurtz DR, et al. (2012) Do neural responses to rejection depend on attachment style? An fMRI study. *Social Cognitive and Affective Neuroscience* 7: 184-192.
22. Reindl V, Gerloff C, Scharke W, Konrad K (2018) Brain-to-brain synchrony in parent-child dyads and the relationship with emotion regulation revealed by fNIRS-based hyperscanning. Neuroimage 178: 493-502.
23. Miller JG, Vrtička P, Cui X, Shrestha S, Hosseini SMH, et al. (2019) Inter-brain synchrony in mother-child dyads during cooperation: An fNIRShyperscanning study. Neuropsychologia 124: 117-124.
24. Rogers CN, Ross AP, Sahu SP, Siegel ER, Dooyema JM, et al. (2018) Oxytocin- and arginine vasopressin-containing fibers in the cortex of humans, chimpanzees, and rhesus macaques. *American Journal of Primatology* 80: e22875.
25. Gorka SM, Fitzgerald DA, Labuschagne I, Hosanagar A, Wood AG, et al. (2015) Oxytocin modulation of amygdala functional connectivity to fearful faces in generalized social anxiety disorder. *Neuropsychopharmacology* 40: 278-286.
26. Dodhia S, Hosanagar A, Fitzgerald DA, Labuschagne I, Wood AG, et al. (2014) Modulation of resting-state amygdala-frontal functional connectivity by oxytocin in generalized social anxiety disorder. Neuropsychopharmacology 39: 2061-2069.