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Chemical Dependency, Anger Management and Depression: A Triumvirate Approach.[1]

SUMMARY

The issue of chemical addiction has recently garnered more and more national attention. It appears, at least on the surface, that the major health issue of chemical addiction has broken through the "wall of denial," if you will, regarding America and its love affair with mind-altering chemicals. With this overwhelming issue now gripping America by the "throat," so to speak, other, ancillary issues have emerged from the depths of chemical addiction.

One of the major mental health issues that has become closely associated with the issue of chemical addiction is anger management. By way of America's law enforcement, judicial and correctional systems, respectively, the issue of anger management has gained national recognition via the disease of chemical addiction. This is not to suggest, by explicit or implicit means, that one needs to be "chemically addicted" to have issues with their anger.

Rather, it appears, at least from the statistical evidence, that when mind-altering chemicals are involved, the issue of one's anger and the management thereof comes to the fore. For example, it is well known within the law enforcement community that one of the most dangerous and potentially explosive calls is the "domestic" where drugs, alcohol and violence expose themselves in a most unruly and ugly manner. Oftentimes, these domestic calls lead to someone getting critically injured or even killed.

Needless to say, the issue of anger management has been and continues to gain increasing national attention – especially when associated with chemical addiction.

Perhaps one of the most notorious examples of this particular association occurred back on March 3, 1991 when Rodney King was literally beaten by several officers, simultaneously, as he continued to resist the process of being arrested by apparently not complying with their orders to assume the prone position to secure his arms and legs in this incident.

Naturally, at least by all appearances via the videocam that caught most of the incident on tape, all of America witnessed an African-American male get a vicious, unrelenting beating from mostly Caucasian, male policemen. Although the use of violence and police tactics in this particular instance were unequivocally taken too far, most of the general public was not aware of the fact that Mr. Rodney King, who stands about six-foot three and weighed, at that particular time, about two-hundred and fifty pounds, was not only legally drunk, but was additionally high on PCP.

The combination of these two chemicals, combined with the intensity and adrenaline-rush that came along with the whole process of being chased by the police because he was doing about 100 mph before they were able to stop him, is a recipe for potential disaster.

Of course, most of the general public who witnessed the brutal "beat-down" did not fully understand that Mr. King was a proverbial "keg of dynamite" when you consider he was intoxicated with both alcohol and PCP – a combination that may precipitate psychotic, irrational, violent behavioral outbursts that are unprovoked.

Therefore, when you mix the combination of drugs and alcohol and one's unresolved anger, the end results could potentially be lethal when mixed together.

Finally, this paper will examine the critical third piece of this particular complex puzzle – that of depression. The literature has revealed, most unfortunately, that not only is depression the least treated diagnoses in America, but oftentimes does get overlooked and/or undiagnosed by professionals when they are dealing with the chemically dependent patient with presenting concurrent issues of anger management.

The fact of the matter is that many, many clinically trained professionals, even those of us in the specific field of chemical addiction, mistakenly think that the depression is a by-product of one's chemical addiction and/or a combination of one's anger turned inward.

As a direct result, there are multitudes of patients who come into various treatment facilities, clinics, offices, etc. who are quickly, and most obviously, diagnosed with some type of chemical abuse or dependence who may have some unresolved issues that are expressed as "angry outbursts" – especially when under the influence of drugs and/or alcohol.

However, when the "smoke has cleared," so to speak, and the patient has achieved early full remission along with managing to keep his anger "in check," it seems, at least from my clinical experience the last seventeen and a half years, that "something still is not right" with the patient. This is where many clinicians fail, unintentionally, to properly diagnose and treat the depression as a separate diagnosis and not as a by-product of either one's addiction/abuse and/or anger.

In summary, this paper will examine the relationship between chemical addiction, anger management and clinical depression in the chemically dependent patient. Additionally, this paper will attempt, based on the current literature and the seventeen and a half years of this author's clinical experience, to make the common connection between these three issues.

Also, once the common denominator has been established between these three above mentioned clinical issues, the paper will examine the difficult and ever-changing dynamics that may cause some health care providers to misdiagnose, or even, at times, not recognize these presenting issues as one's that deserve the diagnostic recognition that are clinically warranted.

Finally, the paper will take a close look at the various treatment approaches that have been utilized in the past and present regarding addiction, anger and depression. With respect to the different treatment modalities and/or interventions, this paper will examine the pros and cons of the different types of treatment approaches, including this author's, and what may be some of the more effective and not-so-effective types, respectively.

ARTICLE

The disease of chemical addiction has been around virtually as long as man has inhabited the earth. Needless to say, the problem has been around a lot longer than we in the industrialized world have made it out to be. In other words, it appears, at least from a media perspective, that the issue of chemical addiction is a "relatively new phenomenon that is slowly and insidiously taking control of 'us' more and more."

The direct and blunt truth of the matter is that as long as man has been able to walk upright, the desire to alter one's consciousness appears to more innate than anything else. Although there have been documented cultures and even certain societies using mind-altering chemicals as part of their religious belief systems, it appears, more now than ever before, that the disease of chemical addiction is more a by-product of the deficient hardwiring in the brain.

David Marley, PharmD, RAS, has articulated how the chemically addicted brain operates on a cellular level that keeps the disease alive and well throughout the course of one's entire life span:

Evolution of the Brain

In order to truly understand addiction, we have to look at the evolution of the human brain. Human beings, being highly evolved, have an incredibly large neo-cortex compared with other living beings. This neo-cortex can be described as our "new" or "thinking" brain. However, we still share with other beings an "old" or "instinctual" brain that controls all of our involuntary functions, including our instinctual drives (eg, hunger, sex, and nurturing). It is in this old instinctual brain that the pathophysiology of addiction resides, an area within the limbic system that controls our moods, a reward pathway within the medial forebrain bundle that includes the nucleus accumbens (NA) and the ventral tegmental area (VTA).

The Reward Pathway

It is the reward pathway that governs our instinctual drives, and when this pathway malfunctions, any number of instinctual drives may also malfunction. We now know that this malfunctioning process has been associated not only with drug addiction, but also food addiction, sex addiction, and a number of other compulsive disorders including attention-deficit hyperactivity disorder. In an otherwise normal individual, there is a cascade of neurotransmitters, which includes dopamine, that acts as a satiation of "off switch" when fulfilling an instinctual drive. Individuals who are predisposed to become addicted have been shown to have a variant gene that causes this reward cascade to malfunction. These people do not experience the same level of neurochemical reward when fulfilling an instinctual drive, and there is no satiation mechanism or "off switch" telling them that they have become satiated by the behavior.

The reward pathway, like all neurochemical pathways, is an electrochemical process involving electrical signals being transmitted across billions of nerve cells by way of neurotransmitters. As a nerve impulse is sent to a presynaptic neuron, the vesicles containing neurotransmitters merge with the presynaptic membrane and spill their contents into the synaptic junction. These neurotransmitters then bind with the postsynaptic receptor, which allows the signal to continue. At the same time, there are neighboring neurons that can potentate or inhibit the response. In the individual who is predisposed to addiction, this process is malfunctioning in the NA and VTA of the brain.

Early animal models showed that when a catheter containing cocaine was placed into the NA of a rat, the animal became addicted to the drug and learned to press a lever to get a dose of the drug. Once the animal was addicted, an electrical stimulus was connected to the lever that provided an ever-increasing shock each time the animal attempted to get his "fix." In every case, the animals electrocuted themselves rather than cease the drug use. When this study was conducted again with food instead of a drug, the animals starved to death rather than endure the electrical stimulus. Finally, this study was replicated with the drug-containing catheter placed in the spinal cord, and no addictive behaviors were observed. The findings of this study

> included: (1) the drugs of abuse have a specific site of action in the brain; (2) this area of the brain must have a similar area in human beings (ie, the instinctual brain); (3) no amount of unpleasant stimuli was great enough to get the animal to stop seeking the drug; and (4) this process was greater than other instinctual drives, including the drive to eat.
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> The addictive process manifests itself in human beings as the behaviors that we normally associate with being an addict (eg, inability to control drug use, replacement of important activities by drug use). When the reward pathway is exogenously stimulated by drug use, it sends exaggerated signals to the prefrontal cortex, an area of the brain that control reasoning and judgment. This signal highjacks the prefrontal cortex into believing that, with drug use, an instinctual drive was just fulfilled and should be repeated. Unfortunately, while many people are able to experiment with psychoactive drugs with little of no adverse effects or continuous use, the person predisposed to addiction has the previously described malfunctioning satiation process, and therefore progresses into uncontrolled drug use.
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> Understanding this process at a cellular level is not overly complicated. Medical science has already unlocked many of the secrets of brain function. We have identified hundreds of neurotransmitters that control brain function. We know that too little serotonin within the limbic system can lead to depression, and we prescribe selective serotonin reuptake inhibitors (SSRIs) to treat depression. It can be argued that drug addiction is nothing more than individuals taking a substance that they have found to medicate their dopamine deficiency within the reward pathway. However, when treating depression, we use highly researched compounds, prescribed at the lowest dose necessary and under close medical supervision (pp. 1-2).

Based on the above article that elaborates the intricacies of the disease of addiction from a neurochemical perspective, it appears that the disease of addiction is mostly inherited from one generation to the next. This is, however, not to imply that one's environment does not play any role whatsoever; rather, it is the by-product of complex variables that interact with one another that produces the disease of addiction.

For example, is someone is "genetically wired," if you will, for the disease of addiction, then perhaps all one would need then is the "correct" environmental cues and/or triggers to set-off this disease of addiction. Although this is a simplistic version of the complexity of the disease of addiction, it would seem, at least from my professional experience of seventeen and a half years in the Connecticut correctional system, that once the disease has been unleashed within the brain circuitry, the power of the disease seems to "take on a life form of its own."

In fact, one would think that the emotional, psychological ad spiritual pain one goes through while incarcerated would, at the very least, give a second thought before using his or her drug of choice. However, and astonishingly similar to the experiment that demonstrated that the rat would rather self-medicate with the drug versus being electrocuted or starve itself to death. This is powerful research that puts a different spin on the disease of addiction.

With this research data in mind, one of the major variables that we humans have over rats and other animals that are instinctually driven is that we humans have the intellectual capacity to "rationalize." In spite of our [humans'] ability to rationalize, the disease appears to supercede all the rules set forth by biology. What is even more frightening than the disease superceding one's ability to "rationalize" with forethought, is that the disease literally overtook the rat's basic instinctual drive to eat, and hence, survive.

If the disease of addiction can override instincts, it stands to reason that humans' ability to rationalize will not abate this profoundly powerful disease that seems to invade our minds to the point of losing total control. And if there is any question about how the disease literally overtakes one's person, just take a look into your local correctional, mental health and inpatient drug/alcohol facilities.

The numbers do not lie. Last time I looked, America had approximately ***two million*** people incarcerated for various felony and misdemeanor offenses – most of which are drug and alcohol related. These are staggering numbers that should get the attention of every politician – least we become a nation under the curse and scourge of drug addiction.

According to the DSM-IV-TR, the following are the clinical diagnostic features and criteria used for assessing a person with substance dependence:

Substance Dependence

Features

The essential feature of Substance Dependence is a cluster of cognitive, behavioral, and physiological symptoms indicating that the individual continues use of the substance despite significant substance-related problems. There is a pattern of repeated self-administration that usually results in tolerance, withdrawal, and compulsive drug-taking behavior. A diagnosis of Substance Dependence can be applied to every class of substances except caffeine. The symptoms are less salient, and in a few instances not all symptoms apply (e.g., withdrawal symptoms are not specified for Hallucinogen Dependence). Although not specifically listed as a criterion item, "craving" (a strong subjective drive to use the substance) is likely to be experienced by most (if not all) individuals with Substance Dependence. Dependence is defined as a cluster of three or more of the symptoms listed below occurring at any time in the same 12-month period.

Tolerance (Criterion 1) is the need for greatly increased amounts of the substance to achieve intoxication (or the desired effect) or a markedly diminished effect with continued use of the same amount of the substance. The degree to which tolerance develops varies greatly across substances. Individuals with heavy use of opioids and stimulants can develop substantial (e.g., tenfold) levels of tolerance, often to a dosage that would be lethal to a nonuser. Alcohol tolerance can also be pronounced, but is usually much less extreme than for amphetamine. Many individuals who smoke cigarettes consume more than 20 cigarettes a day, an amount that would have produced symptoms of toxicity when they first started smoking. Individuals with heavy use of cannabis are generally not aware of having developed tolerance (although it has been demonstrated in animal studies and in some individuals). It is uncertain whether any tolerance develops to phencyclidine (PCP). Tolerance may be difficult to determine by history alone when the substance used is illegal and perhaps mixed with various diluents or with other substances. In such situations, laboratory tests may be helpful (e.g., high blood levels of the substance coupled with little evidence of intoxication suggest that tolerance is likely). Tolerance must also be distinguished from individual variability in the initial sensitivity to the effects of particular substances. For example, some first-time drinkers show very little evidence of intoxication with three or four drinks, whereas others of similar weight and drinking histories have slurred speech and incoordination.

Withdrawal (Criterion 2a) is a maladaptive behavioral change, with physiological

and cognitive concomitants, that occur when blood or tissue concentrations of a substance decline in an individual who had maintained prolonged heavy use of the substance. After developing unpleasant withdrawal symptoms, the person is likely to take the substance to relieve or to avoid those symptoms (Criterion 2b), typically using the substance throughout the day beginning soon after awakening. Withdrawal symptoms vary greatly across the classes of substances, and separate criteria sets for withdrawal are provided for most of the classes. Marked and generally easily measured physiological signs of withdrawal are common with alcohol, opioids, and sedatives, hypnotics, and anxiolytics. Withdrawal signs and symptoms are often present, but may be less apparent, with stimulants such as amphetamines and cocaine, as well as with nicotine. No significant withdrawal is seen even after repeated use of hallucinogens. Withdrawal from phencyclidine and related substances has not yet been described in humans (although it has been demonstrated in animals). Neither tolerance nor withdrawal is necessary or sufficient for a diagnosis of Substance Dependence. Some individuals (e.g., those with Cannabis Dependence) show a pattern of compulsive use without any signs of tolerance or withdrawal. Conversely, some postsurgical patients without Opioid Dependence may develop a tolerance to prescribed opioids and experience Withdrawal symptoms without showing any signs of compulsive use. The specifiers With Physiological Dependence and Without Physiological Dependence are provided to indicate the presence or absence of tolerance or Withdrawal.

The following items describe the pattern of compulsive substance use that is characteristic of Dependence. The individual may take the substance in larger amounts or over a longer period than was originally intended (e.g., continuing to drink until severely intoxicated despite having set a limit of only one drink) (Criterion 3).

The individual may express a persistent desire to cut down or regulate substance use. Often, there have been many unsuccessful efforts to decrease or discontinue use (Criterion 4). The individual may spend a great deal of time obtaining the substance, using the substance, or recovering from its effects (Criterion 5). In some instances of Substance Dependence, virtually all of the person's daily activities revolve around the substance. Important social, occupational, or recreational activities may be given up or reduced because of substance use (Criterion 6). The individual may withdraw from family activities and hobbies in order to use the substance in private or to spend more time with substance-using friends. Despite recognizing the contributing role of the substance to a psychological or physical problem (e.g., severe depressive symptoms or damage to organ systems), the person continues to use the substance (Criterion 7). The key issue in evaluating this criterion is not the existence of the problem, but rather the individual's failure to abstain from using the substance despite having evidence of the difficulty it is causing.

Criteria for Substance Dependence

A maladaptive pattern of substance use, leading to clinically significant impairment or distress, as manifested by three (or more) of the following, occurring at any time in the same 12-month period:

(1) tolerance, as defined by either of the following:

(a) a need for markedly increased amounts of the substance to achieve intoxication or desired effect
(b) markedly diminished effect with continued use of the same amount of the substance

(2) withdrawal, as manifested by either of the following:

(a) the characteristic withdrawal syndrome for the substance (refer to Criterion A and B of the criteria sets for Withdrawal from the specific Substances)
(b) the same (or a closely related) substance is taken to relieve or avoid withdrawal symptoms

(3) the substance is often taken in larger amounts or over a longer period than was intended

(4) there is a persistent desire or unsuccessful efforts to cut down or control substance use

(5) a great deal of time is spent in activities necessary to obtain the substance (e.g., visiting multiple doctors or driving long distances), use the substance (e.g., chain-smoking), or recover from its effects

(6) important social, occupational, or recreational activities are given up or reduced because of substance use

(7) the substance use is continued despite knowledge of having a persistent or recurrent physical or psychological problem that is likely to have been caused or exacerbated by the substance (e.g., current cocaine use despite recognition of cocaine-induced depression, or continued drinking despite recognition that an ulcer was made worse by alcohol consumption) (pp. 192-197).

In summary, there are a plethora of different clinical definitions for the disease of addiction that ultimately end-up in the same place: personal pain for those not only using the chemical but also family, friends, employers, etc. who may be connected to the person afflicted with the disease of addiction.

In the final analysis, at least from my professional experience of seventeen and a half years, I have yet to find a person, afflicted with the disease of addiction, who has reported to me positive experiences that would make a convincing argument to continuing using and feeding their ever-progressing disease of addiction.

In fact, all of the personal stories I have heard over the years from my patients and inmates that I have been exposed to through the years, some have reported some anecdotal “good times”; however, invariably, the disease ultimately caught up to them and left them in a position to either quit altogether or continue and risk isolation, depression, loss of family and friends, unemployment, poor psychological, spiritual and physical health – among a list of damages that is frankly too long to enumerate for our purposes herein.

Suffice to say that institutions, jail or death await those who refuse to stop feeding their disease of addiction.

Dovetailing the disease of chemical addiction and all of its clinical vicissitudes has emerged the ever-increasing issue of anger management. The term "anger management" is presumably derived from those patients and/or inmates who clearly have a demonstrated inability to control his/her anger. Although the issue of anger management has now become sort of "pop culture," it has been another clinical issue that has been with humans as long as we could walk upright.

The last ten to fifteen years have really brought this issue to the forefront – especially as it relates to domestic violence and chemical addiction. Perhaps one of the most compelling arguments made for anger management coupled with chemical addiction occurred back on June 10, 1983 in a rural town of Torrington, Connecticut.

According to the information I have gleaned over the years via news stories, having treated her brother at Osborn Correctional Institution for heroin addiction (he has since died of an overdose of heroin) and even a "made for television movie," the Tracy Thurman case virtually changed the domestic violence laws in the state of Connecticut.

In brief, Tracy Thurman (her married name at the time) was being verbally, emotionally and physically abused by her chemically addicted husband, Charles Thurman. After this had been going on for some time, Tracy apparently asked for a divorce. During their time of separation, Charles Thurman would come around the house making verbal threats that he would kill Tracy if she proceeded with the divorce. When he did not physically come around the house, he would constantly call her at home and continue to mentally torment her by telling her he would "hurt or kill" her if she follow-through with the divorce.

It was during this timeframe of constant terror by Charles Thurman that Tracy Thurman made several phone calls to the Torrington Police Department relaying all of the pertinent information and obviously expressing her very serious concern about her physical well-being an how they (Torrington Police) could prevent the threats from being carried-out – in spite of the restraining order that was placed on Charles Thurman.

According to most news accounts, and even speaking with her brother Donald Bodner, the Torrington Police "could not do anything" at the time because the laws prevented them from taking any type of precautionary measure.

Shortly after Tracy Thurman's multiple phone calls to the Torrington Police, without any action on their part whatsoever, the worst occurred on June 10, 1983. On this particular date, Charles Thurman followed-through with his multiple threats and beat Tracy Thurman at her home so badly that she almost died as a result of her injuries.

When she did recover from her vicious beating at the hands of Charles Thurman, who incidentally was intoxicated at the time of the offense, incurred permanent physical damage in the form of a partially paralyzed left side of her body.

Needless to say, Tracy Thurman sued the Torrington Police Department and was awarded a 2.3 million dollar settlement along with attracting the attention of the entire country. As a result of this notoriety, the laws were literally changed in the state of Connecticut regarding domestic violence and taking proactive, preventative measures to ensure that violence does not occur in our homes.

Although this case is high profile and tragic with the eventual outcome, it serves to illustrate a very important point that I have seen consistently throughout the course of my seventeen and a half year career: violence is ***inextricably*** linked to the disease of addiction. The next logical question that should be posed is how, why and when?

Before I begin to unravel this very difficult, complex and often misunderstood subject matter, I would like to outline a working, clinical definition of what anger consists of and its various manifestations.

Throughout the course of my career in corrections and in the private sector, I have had the clinical benefit of working in places that most clinicians would either never dare set foot in or enter with a fair amount of trepidation and plenty of apprehension: a maximum security prison that houses inmates for very long (sometimes a lifetime) periods of time.

It is, and continues to remain, a place where anger, along with chemical addiction and depression, can be seen up close, personal and very intimately. Having worked my entire career on the line with direct inmate contact every day for the last seventeen and a half years, you are exposed to someone's anger several times daily throughout the course of your shift. Frankly speaking, if you are squeamish about other people's unbridled anger, then working in a correctional facility, even as a treatment provider, is not the place for you.

With this factor in mind, it seems that although there are several varying definitions of anger within the treatment field, the following article appears to highlight the major aspects of anger that I have come to know, rather intimately, during my correctional and private sector career:

What is Anger?

The Nature of Anger

Anger is "an emotional state that varies in intensity from mild irritation to intense fury and rage," according to Charles Spielberger, PhD, a psychologist who specializes in the study of anger. Like other emotions, it is accompanied by physiological and biological changes; when you get angry, your heart rate and blood pressure go up, as do the levels of your energy hormones, adrenaline, noraadrenaline.

Anger can be caused by both external and internal events. You could be angry at a specific person (such as a coworker or supervisor) or event (a traffic jam, a canceled flight), or your anger could be caused by worrying or brooding about your personal problems. Memories of traumatic or enraging events can also trigger angry feelings.

Expressing Anger

The instinctive, natural way to express anger is to respond aggressively. Anger is a natural, adaptive response to threats; it inspires powerful, often aggressive, feelings and behaviors, which allow us to fight and to defend ourselves when we are attacked. A certain amount of anger, therefore, is necessary to our survival.

On the other hand, we can't physically lash out at every person or object that irritates or annoys us; laws, social norms, and common sense place limits on how far our anger can take us.

People use a variety of both conscious and unconscious processes to deal with their angry feelings. The three main approaches are expressing, suppressing, and and calming. Expressing your angry feelings in an assertive – not aggressive – manner is the healthiest way to express anger. To do this, you have to learn how to make clear what your needs are, and how to get them met, without hurting others. Being assertive doesn't mean being pushy or demanding; it means being

respectful of yourself and others.

Anger can be suppressed, and then converted or redirected. This happens when you hold in your anger, stop thinking about it, and focus on something positive. The aim is to inhibit or suppress your anger and convert it into more constructive behavior. The danger in this type of response is that if it isn't allowed outward expression, your anger can turn inward – on yourself. Anger turned inward may cause hypertension, high blood pressure, or depression.

Unexpressed anger can create other problems. It can lead to pathological expressions of anger, such as passive-aggressive behavior (getting back at people indirectly, without telling them why, rather than confronting them head-on) or a personality that seems perpetually cynical and hostile. People who are constantly putting others down, criticizing everything, and making cynical comments haven't learned how to constructively express their anger. Not surprisingly, they aren't likely to have many successful relationships.

Finally, you can calm down inside. This means not just controlling your outward behavior, but also controlling your internal responses, taking steps to lower your heart rate, calm yourself down, and let the feelings subside.

As Dr. Spielberger notes, "when none of these three techniques work, that's when someone – or something – is going to get hurt."

Anger Management

The goal of anger management is to reduce both your emotional feelings and the physiological arousal that anger causes. You can't get rid of, or avoid, the things or the people that enrage you, nor can you change them, but you can learn to control your reactions.

Are You Too Angry?

There are psychological tests that measure the intensity of angry feelings, how prone to anger you are, and how well you handle it. But chances are good that if you do have a problem with anger, you already know it. If you find yourself acting in ways that seem out of control and frightening, you might need help finding better ways to deal with this emotion.

Why Are Some People More Angry Than Others?

According to Jerry Deffenbacher, PhD, a psychologist who specializes in anger management, some people really are more "hotheaded" than others are; they get angry more easily and more intensely than the average person does. There are also those who don't show their anger in loud spectacular ways but are chronically irritable and grumpy. Easily angered people don't always curse and throw things; sometimes they withdrawal socially, sulk, or get physically ill.

People who are easily angered generally have what some psychologists call a low tolerance for frustration, meaning simply that they feel that they should not have to be subjected to frustration, inconvenience, or annoyance. They can't take things in stride, and they're particularly infuriated if the situation seems somehow unjust: for example, being corrected for a minor mistake.

> What makes these people this way? A number of things. One cause may be genetic or physiological: There is evidence that some children are born irritable, touchy, and easily angered, and that these signs are present from a very early age. Another may be sociocultrual. Anger is often regarded as negative; we're taught that it's all right to express anxiety, depression, or other emotions but not to express anger. As a result, we don't learn how to handle it or channel it constructively.
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> Research has also found that family background plays a role. Typically, some people who are easily angered come from families that are disruptive, chaotic, and not skilled at emotional communications.
>
> **Is It Good To "Let it All Hang Out?"**
> Psychologists now say that this is a dangerous myth. Some people use this theory as a license to hurt others. Research has found that "letting it rip" with anger actually escalates anger and aggression and does nothing to help you (or the person you're angry with) resolve the situation.
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> It's best to find out what it is that triggers your anger, and then to develop strategies to keep those triggers from tipping you over the edge (pp. 1-3).

In brief summary, the literature appears to reveal the fundamental fact that virtually every human being that has ever walked this God-given earth has experienced the powerful emotion we have identified as "anger." As the article so accurately pointed out, the very essence of anger is not bad in-and-of-itself; rather, it becomes problematic when people express their anger in ways that are harmful to not only themselves, but others who are usually innocent bystanders.

Obviously, most of our penal institutions are filled with people who have lacked, among other things, the necessary coping skills to deal effectively with their anger – wherever and however it emanated from. With this factor in mind, the issue of anger is often a portal, if you will, to the final piece of the clinical triumvirate puzzle: Clinical Depression.

Before the paper begins to explore the relationship between chemical addiction, anger management and clinical depression, the issue must be diagnostically explored and understood more comprehensively as there are several myths, misconceptions and a fair amount of social stigma associated with the diagnostic label of "Clinical Depression."

As mentioned previously in the paper, clinical depression continues to remain the number one diagnosis in America that goes untreated. The reasons are as varied as they are complex. Unlike chemical addiction and/or anger management, clinical depression presents itself in sometimes very subtle ways that even trained professionals oftentimes misdiagnose or miss altogether.

In my professional clinical experience, there have been multiple occasions (too many to enumerate) where the patient presented with issues centering around chemical addiction, anger management, marital problems, employment difficulties, etc. that, on the surface, preoccupy the clinician to such a degree that they simply overlook and/or do not probe deep enough to unfurl the layers, if you will, to the underlying issue(s) that may be masked by another.

For example, I have had a plethora of referrals from other health care providers (private practitioners, primary care physicians, probation officers, family court clinicians, EAP clinicians, treatment facilities, etc.) where they thought that the patient had unresolved issues with chemical addiction as their only presenting issue(s).

Unfortunately, it has been by clinical experience that most (at least seventy to seventy five percent) of the patients that I have treated in my practice had an underlying, co-occurring disorder of clinical depression that had been masked or covered-up by one's addiction for as long as the addiction was visible to the patient and others around him.

In other words, I have had multiple patients referred to my office for chemical addiction only to discover, in relatively short order, that they had clinical depression for most of their adult life but never sobered-up long enough for it to be diagnostically recognized by most treatment providers.

Additionally, I have had many occasions when patients were referred to my office for "anger management" who not only had issues with controlling their anger but also had difficulties with chemical abuse/addiction and/or clinical depression that was not noticed because the anger simply masked the symptoms.

With some of the above information in mind, Charles B. Nemeroff, MD, PhD and David Gutman, BS wrote an article entitled, ***Neurobiology of Depression and Treatment Strategies*** that was posted on Medscape from WebMD:

The Neurobiology of Depression

Charles Nemeroff, MD, PhD, from the Emory University School of Medicine, Atlanta, Georgia, presented a review of the neurobiology of depression and current treatment strategies. He began by stressing the importance of continuing research in understanding depression and development of better drugs to target the disorder. Unipolar depression is currently the fourth major cause of disability in the United States and is likely to become the second major cause of disability by 2020. Antidepressants nonresponders are among the heaviest users of healthcare, highlighting the pressing need for newer and better pharmacologic approaches to the disease. Less than half of patients with major depression are recognized as being depressed and only half of patients diagnosed with depression receive treatment. Of those treated, only about a third achieve remission from all symptoms, signified by a Hamilton Depression Rating Scale score of less that 7. It is clear that physicians and scientists must make an effort to better diagnose and better treat patients with major depressive disorder.

Disturbances in neurotransmission are the neurobiologic hallmark of depression. Changes have been found in monoamine systems, such as serotonin (5-HT), norepinephrine (NE), and dopamine (DA), as well as other systems, such as corticotropin releasing factor (CFR) and somatostatin. Clinically, the 5-HT and NE systems have been the most thoroughly studied, and it is in these systems that most currently prescribed antidepressants function. Depletion of both 5-HT and NE has been linked to depression; all 5-HT reuptake inhibitors are highly effective antidepressants, and NE reuptake inhibitors are similarly effective for depression. Antidepressants binding to receptor targets results in both the desired clinical outcomes as well as the observed side effects.

Serotonergic pathways are believed to function largely in mood, while NE is likely involved with drive and energy state. Both systems function in appetite, sleep regulation, and anxiety. The selective serotonin reuptake inhibitors (SSRIs) are now the most common treatment for depression. Although 5-HT depletion is related to depressive symptoms, depression is also strongly linked with stress, and stress systems in the brain are largely mediated by changes in NE transmission. A study performed by Lambert and colleagues measuring monamines and their

> metabolites from internal jugular vein catheters showed large decreases in NE in depressed patients vs healthy subjects. Smaller changes were seen in the 5-HT measurements.
>
> Due to the overlap between the 5-HT and NE systems, it is likely that a drug targeting both the 5-HT and NE systems would have better clinical efficacy than more selective compounds. Paroxetine and clomipramine are two currently prescribed antidepressants with strong affinity for the NE transporter (NET) that have strong 5-HT transporter (SERT) binding as well. Duloxetine, a newer compound, has greater affinity for the NET and strong SERT affinity in vitro. Microdialysis from rat frontal cortex indicates that duloxetine induces dose-dependent increases of extracellular 5-HT and NE. Venlafaxine also shows a dose-effect relationship to increase synaptic NE and is effective as a dual reuptake inhibitor at high doses.
>
> Current research in the field is aimed at developing a “throat culture” for depression using either imaging or blood markers. Positron emission tomography imaging has been used to observe changes in SERT occupancy with antidepressant treatment. Ligands specific for the SERT are injected, and decreased ligand binding following treatment suggests that the SSRI is bound to the receptor site, which is an obvious prerequisite for therapeutic efficacy. There is currently no NET ligand available. Another method employed uses a transfected cell assay to determine how much reuptake blockade the patient has. Both techniques are still in research stages, but represent promising advances in the diagnosis and treatment of depression (pp. 1-2).

Although the above mentioned is a medical description of the disease of depression and the treatment thereof, it is a comprehensive examination into the biological, brain-based disease of depression. And much like the disease of chemical addiction that is also a disease of the brain, it too has serious and oftentimes devastating consequences for those who suffer from such.

Additionally, it should be noted that there are thousands of cases where clinical depression can and is situational due to various circumstances in one’s life. For example, I have treated hundred of patients over the years who presented with clinical depression, among other clinical issues, only to discover after a few sessions that they were in the midst of a divorce, grieving the loss of a loved one, etc. that presented itself as the brain-based disease of depression but was, in fact, a transient, situational type of depression that is not permanent and chronic in nature.

As mentioned briefly in the medical definition of depression and the treatment for such, the most effective way to deal with clinical depression is the administration of psychotropic medication(s) coupled with some type of psychotherapy. However, there are some circumstances where just the administration of antidepressants may be indicated and entirely sufficient.

Regardless of the approach, even if it is just psychotropic medication, it should be monitored, at the very least, by that patient’s primary care physician (PCP).

To clarify this issue even further from a diagnostic standpoint, the DSM-IV-TR, the clinicians primary source of diagnostic information, has comprehensively outlined clinical depression the following way:

Diagnostic Features

The essential feature of Major Depressive Disorder is a clinical course that is characterized by one or more Major Depressive Episodes (see p. 349) without a history of Manic, Mixed, or Hypomanic Episodes (Criteria A and C). Episodes of Substance-Induced Mood Disorder (due to the direct physiological effects of a drug of abuse, a medication, or toxin exposure) or of Mood Disorder Due to a General Medical Condition do not count toward a diagnosis of Major Depressive Disorder. In addition, the episodes must not be better accounted for by Schizoaffective Disorder and are not superimposed on Schizophrenia, Schizophreniform Disorder, Delusional Disorder, or Psychotic Disorder Not Otherwise Specified (Criterion B).

The fourth digit in the diagnostic code for Major Depressive Disorder indicates whether it is a Single Episode (used only for first episodes) or Recurrent. It is sometimes difficult to distinguish between a single episode with waxing and waning symptoms and two separate episodes. For purposes of this manual, an episode is considered to have ended when the full criteria for the Major Depressive Episode have not been met for at least 2 consecutive months. During this 2-month period, there is either complete resolution of symptoms or the presence of depressive symptoms that no longer meet the full criteria for a Major Depressive Episode (In Partial Remission).

The fifth digit in the diagnostic code for Major Depressive Disorder indicates the current state of disturbance. If the criteria for a Major Depressive Episode are met, the severity of the episode is noted as Mild, Moderate, Severe Without Psychotic Features, or Severe With Psychotic Features. If the criteria for a Major Depressive Episode are not currently met, the fifth digit is used to indicate whether the disorder is In Partial Remission on In Full Remission (see p. 412).

If Manic, Mixed, or Hypomanic Episodes develop in the course of Major Depressive Disorder, the diagnosis is changed to a Bipolar Disorder. However, if manic or hypomanic symptoms occur as a direct effect of anitdepressant treatment, use of other medications, substance use, or toxin exposure, the diagnosis of Major Depressive Disorder remains appropriate and an additional diagnosis of Substance-Induced Mood Disorder, With Manic Features (or With Mixed Features), should be noted. Similarly, if manic or hypomanic symptoms occur as a direct effect of a general medical condition, the diagnosis of Major Depressive Disorder remains appropriate and an additional diagnosis of Mood Disorder Due to a General Medical Condition, With Manic Features (or With Mixed Features), should be noted.

Associated Feature and Disorders

Associated descriptive features and mental disorders. Major Depressive Disorder is associated with high mortality. Up to 15% of individuals with severe Major Depressive Disorder die by suicide. Epidemiological evidence also suggests that there is a fourfold increase in death rates in individuals with Major Depressive Disorder who are over age 55 years. Individuals with Major Depressive Disorder admitted to nursing homes may have a markedly increased likelihood of death in the first year. Among individuals seen in general medical settings, those with Major Depressive Disorder have more pain and physical

illness and decreased physical, social, and role functioning.

Major Depressive Disorder may be preceded by Dysthymic Disorder (10% in epidemiological samples and 15%-25% in clinical samples). It is also estimated that each year approximately 10% of individuals with Dysthymic Disorder alone will go on to have a first Major Depressive Episode. Other mental disorders frequently co-occur with Major Depressive Disorder (e.g., Substance-Related Disorders, Panic Disorder, Obsessive-Compulsive Disorder, Anorexia Nervosa, Bulimia Nervosa, Borderline Personality Disorder).

Major Depressive Disorder may begin at any age, with an average age at onset in the mid-20s. Epidemiological data suggest that the age at onset is decreasing for those born more recently. The course of Major Depressive Disorder, Recurrent, is variable. Some people have isolated episodes that are separated by many years without any depressive symptoms, whereas other have clusters of episodes, and still others have increasingly frequent episodes as they grow older. Some evidence suggests that the periods of remission generally last longer early in the course of the disorder.

The number of prior episodes predicts the likelihood of developing a subsequent Major Depressive Episode. At least 60% of individuals with Major Depressive Disorder, Single Episode, can be expected to have a second episodes. Individuals who have had two episodes have a 70% chance of having a third, and individuals who have had three episodes have a 90% chance of having a fourth. About 5%-10% of individuals with Major Depressive Disorder, Single Episode, subsequently develop a Manic Episode (i.e., develop Bipolar I Disorder).

Major Depressive Episodes may end completely (in about two-thirds of cases), or only partially or not at all (in about one-third of cases). For individuals who have only partial remission, there is a greater likelihood of developing additional episodes and continuing the pattern of partial interepisode recovery. The longitudinal course specifiers With Full Interepisode Recovery and Without Full Interepisode Recovery (see p. 424) may therefore have prognostic value. A number of individuals have preexisting Dysthymic Disorder prior to the onset of Major Depressive Disoder, Single Episode. Some evidence suggests that these individuals are more likely to have additional Major Depressive Episodes, have poorer interepisode recovery, and may require additional acute-phase treatment and a longer period of continuing treatment to attain and maintain a more thorough and longer-lasting euthymic state.

Follow-up naturalistic studies suggested that 1 year after the diagnosis of a Major Depressive Episode, 40% of individuals still have symptoms that are sufficiently Severe to meet criteria for a full Major Depressive Episode, roughly 20% continue to have some symptoms that no longer meet full criteria for a Major Depressive Episode (i.e., Major Depressive Disorder, In Partial Remission), and 40% have no Mood Disorder. The severity of the initial Major Depressive Episode appears to predict persistence. Chronic general medical conditions are also a risk factor for more persistent episodes.

Episodes of Major Depressive Disorder often follow a severe psychosocial

stressor, such as the death of a loved one or divorce. Studies suggest that psychosocial events (stressors) may play a more significant role in the precipitation of the first or second episodes of Major Depressive Disorder and may play less of role in the onset of subsequent episodes. Chronic general medical conditions and Substance Dependence (particularly Alcohol or Cocaine Dependence) may contribute to the onset or exacerbation of Major Depressive Disorder.

It is difficult to predict whether the first episode of a Major Depressive Disorder in a young person will ultimately evolve into a Bipolar Disorder. Some data suggest that the acute onset of severe depression, especially with psychotic features and psychomotor retardation, in a young person without prepubertal psychopathology is more likely to predict a bipolar course. A family history of Bipolar Disorder may also be suggestive of subsequent development of Bipolar Disorder (pp. 369-373).

Criteria for Major Depressive Espisode

A. Five (or more) of the following symptoms have been persistent during the same 2-week period and represent a change from previous functioning; at least one of the symptoms is either (1) depressed mood or (2) loss of interest or pleasure.

   Note: Do not include symptoms that are clearly due to a general medical condition, or mood-incongruent delusions or hallucinations.

   (1) depressed mood most of the day, nearly every day, as indicated by either subjective report (e.g., feels sad or empty) or observation made by others (e.g., appears tearful). **Note:** In children and adolescents, can be irritable mood.
   (2) markedly diminished interest or pleasure in all, or almost all, activities most of the day, nearly every day (as indicated by either subjective account or observation made by others)
   (3) significant weight loss when not dieting or weight gain (e.g., a change of more than 5% of body weight in a month), or decrease or increase in appetite nearly every day. **Note:** In children, consider failure to make expected weight gains.
   (4) insomnia or hypersomnia nearly every day
   (5) psychomotor agitation or retardation nearly every day (observable by others, not merely subjective feelings of restlessness or being slowed down)
   (6) fatigue or loss of energy nearly every day
   (7) feelings of worthlessness or excessive or inappropriate guilt (which may be delusional) nearly every day (not merely self-reproach or guilt about being sick)
   (8) diminished ability to think or concentrate, or indecisiveness, nearly every day (either by subjective account or as observed by others)
   (9) recurrent thoughts of death (not just fear of dying), recurrent suicidal ideation without a specific plan for committing suicide

B. The symptoms do not meet criteria for a Mixed Episode (see p. 365).

C. The symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

D. The symptoms are not due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication) or a general medical condition (e.g., hypothyroidism).

E. The symptoms are not better accounted for by Bereavement, i.e., after the loss of a loved one, the symptoms persist for longer than 2 months or are characterized by marked functional impairment, morbid preoccupation with worthlessness, suicidal ideation, psychotic symptoms, or psychomotor retardation (p. 356).

Now that the definitions have been comprehensively examined and defined for our purposes herein, this final part of the paper will examine a particular case study that involved all of the above mentioned clinical issues: chemical dependency, anger management and clinical depression.

It should be noted that this case study was actual and taken, with written permission by the patient, from my private practice.

The Case of Peter T.

Peter, who had been under my clinical care for almost eighteen months, managed to get my name from another patient of mine who happened to be very much involved within the fellowship of AA. When Peter called my office and left me a detailed message on my confidential voice mail, he related that he was involved in the fellowship, had recently been successfully discharged from an inpatient drug and alcohol program where he spent 28 days and was looking to follow-up with clinical counseling for his issues.

Upon my return phone call, he shared that he had "gotten your name from Francisco in the fellowship who said you were 'pretty good.' " Peter proceeded to briefly tell me his story to which I responded that we should make an appointment to get some more information and take it from there.

Upon Peter's arrival to my office, he presented as a married male with two dependent children ages four and two, respectively. Peter informed me that he had multiple issues occurring simultaneously that prompted him to seek further clinical help after his 28-day inpatient drug and alcohol stay.

Among some of the issues Peter identified during our first session included his wife's recent filing for divorce and to secure full-time custody of the two children, living with his aunt (he's thirty-eight years old), trying to figure-out what to do with his business (he's self-employed), filing for bankruptcy in Federal Court and having to deal with his second DUI charge that included the very real possibility of losing his license for as much as one calendar year. Talk about a train-wreck!

Needless to say, these issues took about two to three session to accurately sort-out and prioritize from a clinical standpoint. Once this was done, it gave me a chance to peel back the layers, if you will, regarding Peter's diagnostic issues that needed to be addressed and worked on sooner rather than later.

Peter related to me over the course of our early sessions that he had been "drinking and drugging since I was about twelve or thirteen." Peter shared that he started by drinking alcohol and smoking pot for the most part until he was about fifteen or sixteen years old. After the age of sixteen, he experimented with hallucinogenics (e.i., blotter acid, mushrooms, etc.) and cocaine. According to Peter, he never tried heroin or any other types of illicit drugs.

However, by the time Peter was about nineteen years old, he "pretty much stuck to the booze…that became my drug of choice." It appeared, at least according to his self-report, that he would drink "every day…I never did the hard stuff, though." Peter related that by the time he entered into treatment back in August, 2002, he was consuming "20-24 beers a day…I would usually start in the morning to 'calm' my nerves."

Peter reported that his childhood was largely unremarkable but did state that he was "very close to both my mother and father – especially my mother." Additionally, Peter informed me that he had one older brother and one younger sister – both of whom he was "pretty close to." Peter denied experiencing any type of childhood abuse from either parent during his formative years.

Once all of the necessary information had been disseminated and sifted-through, it became obvious that ***if*** Peter had any other presenting issues such as depression, anger, grieving, etc., they had ***all*** been masked over by his alcoholism of approximately nineteen years. This is where most clinicians diverge and continue on the "blind" path that if a patient has an Axis I of Alcohol Dependence, then more than likely the only thing to treat is just that one diagnosis.

I have actually gotten into heated debates with other treatment providers who adamantly cling to the version that all this patient would need is to do "ninety-and-ninety" and find himself a "good sponsor" and the rest will take care of itself. Frankly speaking, this is ***very dangerous clinical advice!*** There have been too many times in my career where the primary clinician, PCP (Primary Care Physician), psychiatrist or even the psychologist have singularly or collectively failed to properly diagnose these two prominent, yet at times, very subtle issues: anger management and depression.

In Peter's particular case, the inpatient program that he stayed in for 28-days was astute enough to diagnostically ascertain that he had suffered for most of his adult life with Bipolar Mood Disorder. However, because Peter had self-medicated over half of his natural life span, it was never properly diagnosed, or for that matter, recognized as a ***separate and distinct diagnosis*** apart from his alcohol dependence.

Once Peter had been detoxified and administered a thorough neuropsychological examination, it was then clearly and accurately diagnosed that he had been long-suffering from a major mood disorder that oftentimes, if not always, ***disguised itself as symptoms emanating from his alcohol dependence.*** In fact, in some bizarre and yet strangely accurate way, Peter described the alcohol as "something that calmed me down; I never got too up or down when I was drinking."

As strange and bizarre as it may sound, I ***clinically*** understand why several of my patients continue to imbibe alcohol and/or abuse illicit drugs; usually to not only satiate the addiction itself, but concurrently the symptoms of a mood disorder like clinical depression. Peter was no exception to this theory that I have seen itself played-out too many times to enumerate herein.

In Peter's case, he was put on Neurotin, 1200 mg., Zyprexa, 30 mg., Wellbutrin, 300 mg., and Seraquil, 30mg. to stabilize his Bipolar Disorder. Once his major mood disorder was clinically stabilized, this is when some of the other clinical issues emerged and needed to be addressed and worked through.

Two of the issues that began to manifest themselves ***after*** Peter was stabilized on the psychotropic medications and was sober from all mind-altering substances, were anger management and grieving. It became very apparent, at least to myself and those who were closest to Peter that his anger was "bubbling over," so to speak, as a result of years and years of suppressing issues that were important to him but had difficulty dealing with them appropriately.

Peter's anger management, much like the many hundreds I have seen over the span of my career, usually represent at least one, if not multiple, issues that have been repressed/suppressed into the subconscious mind for a multitude of reasons. One of the primary reasons that Peter repressed so many issues was that he did not have the emotional and psychological capacity to deal with these issues without feeling completely overwhelmed and hence, out of control.

As paradoxical as this sounds, Peter felt he had better control of his drinking than he did his mood disorder and anger management issues. And with Peter's tolerance being so incredibly high, he felt better, at least from an emotional and psychological standpoint, that he could control his drinking rather ***risking*** the unknown; emotional fear stemming from an incident that occurred when he was sixteen years old.

According to Peter's aunt, with whom Peter gave me written permission to speak to openly about his clinical issues, she related to me the following: "You know, Jay, Peter witnessed a murder at Mr. Steaks when he was sixteen years old. He was working there as a short-order cook and the police actually arrested him and brought him into the station to be booked and questioned. As it turned-out, they let him go after several hours of interrogation because he was not the prime suspect in the case. All of the family, especially Peter's mother, think that was the 'turning point' for Peter; that's when he really began to drink heavily and became somewhat estranged from the family and some of his friends."

Needless to say, being a witness to a murder at such an impressionable age and then being arrested, booked and interrogated for it certainly left a lasting impression upon Peter that later in life manifested itself diagnostically as Post Traumatic Stress Disorder.

Although this is not the only issue that Peter had not dealt with from his past, it certainly was one of his foremost issue that needed to be dealt with. Also, the recent death of his mother just added more "fuel to the fire," so to speak, regarding his anger issues.

Once Peter and I began to explore and work-through the multiple issues that Peter had just repressed for the majority of his adulthood, these issues lost some of the power over him through the shared clinical experience. In other words, the power of the "secrets" lost their grip on his psyche and began to have less and less power upon his emotional and psychological disposition.

As a direct result of the aforementioned, Peter's ability to manage his anger became that much easier not because he learned to manage his symptoms better, but rather as a result of unraveling and peeling away the layers and layers of pain, loss, fear, trauma, etc. ***that actually manifested itself as angry emotions and behaviors.*** Once the underlying issues were aggressively addressed and worked-through, the anger significantly dissipated because the underlying issues that disguised themselves as anger were once and for all clinically and appropriately dealt with.

In summary, the case of Peter T. represents well over a third of the patients that I currently treat in my private practice. This is being conservative, too. As I mentioned previously in the Peter T. case, he was ***fortunate*** to have a consulting psychiatrist on staff who was keen enough to look past the obvious presenting issues of alcohol dependence. Once Peter detoxified from the ill effects of alcohol, some of his other issues such as his Bipolar Disorder, anger management, PTSD became more discernable, at least to the clinically trained eye, that then became ***separate*** diagnostic issues to be treated and worked-through just as aggressively as the alcohol dependence itself.

As a follow-up to the case of Peter T., once he became abstinent from all mind-altering drugs, he continued to attend AA fellowship every day (at least for the first three months), as well as securing a sponsor and several phone numbers from other fellowship participants. Once his mind was cleared of the "cobwebs," so to speak, he continued to visit with his psychiatrist to manage and monitor his psychotropic medications. It should be noted that Peter

signed a release of information allowing me to communicate with his psychiatrist to ensure continuity of his clinical care.

After Peter was completely abstinent from the alcohol, attending AA fellowship regularly, talking with his sponsor daily and spend time with other fellowship members in social settings, we (Peter his psychiatrist and myself) were able to "tweak," if you will, his psychotropic medications to get him not only stabilized regarding his Bipolar Disorder, but comfortable as well.

The reason I address the issue of "comfort" regarding Peter and his psychotropic medications, is that I have found it ***imperative*** that if the patient, and in this case it is Peter, does not feel comfortable with his psychotropic medication, there is a very high probability that he will relapse because of the discomfort he may be experiencing. In other words, if Peter's mood continues to cycle excessively and remain labile, there stands a very, very high probability that he will consume alcohol as a way of self-medicating.

Although I certainly never condone any of my patients relapsing to their particular drug choice, I certainly ***understand, at least from an objective, clinical point of view,*** that self-medicating through one's drug of choice is far better, at least from their particular perspective, than ***suffering from the debilitating effects of one's mood disorder.***

This is why I have to really emphasize the ***critical importance*** of making sure that not only Peter, but my other patients as well who suffer from some form of clinical depression, maintain a regiment of psychotropic medication that makes them feel comfortable and not like jumping out of their skin!

Finally, once Peter was stabilized on the psychotropic medications for his bipolar and was abstinent from all mind-altering substances (in this case alcohol), we laid the clinical foundation to address and work on his unresolved issues that manifested themselves as "problematic angry emotions and behaviors."

My clinical approach to Peter's underlying emotional and psychological issues were eclectic in nature. For example, utilizing insight-oriented therapy with Peter was somewhat useful but limited since he tended to be less introspective in nature. Also, Peter was by trade a mechanic and tended to be more visual and concrete in his approach to the way he dealt with the world. As a result, I mostly utilized Rational-Emotive Therapy to help Peter reframe and look at some of the events in his past with a "different set of lenses," if you will.

The cognitive-behavioral approach was certainly more effective versus the insight-oriented therapy as Peter enjoyed doing things "hands-on" and was much less cerebral when it came to most things – especially when dealing with past issues that were emotionally and psychologically difficult to process.

At the time of his discharge from my practice, Peter had spent approximately eighteen months in clinical psychotherapy and had made some significant gains in his personal growth. For example, he was able to sort-through some of his issues that overlapped and intertwined with his alcohol dependency, bipolar disorder and anger management.

Once Peter became abstinent from alcohol via the help of the fellowship, this gave him an opportunity for he and his psychiatrist to work-out, through much experimentation I might add, the difficult and complex task of stabilizing his Bipolar Disorder with some very powerful psychotropic medications.

As I mentioned previously, once these two, but very distinct diagnostic issues, were stabilized, we were then only able to address and work on his anger management issues that were representative of much deeper, complex and powerful issues that Peter had ***never addressed up until this point in his life.*** Once Peter had the courage and fortitude to finally address and work on his past traumatic issues, it proved very therapeutic because the alcohol and major mood disorder had been effectively removed from the equation.

Although Peter did experience a fair amount of psychic pain reliving the witness to a murder at the impressionable age of sixteen, the death of his mother to whom he was very close to and other perceived traumas throughout the course of his life span, he was able to process this information, along with the powerful emotions that they evoked, in such a way that no longer had such terrific power over his mind and emotions the way the once had.

In other words, because Peter was of sound mind (sober) and emotions (stabilized via the psychotropic medications), he was able to ***process, with a clear mind and stable emotions,*** all of the "unfinished business," if you will, that expressed itself in the form of "anger management problems."

Last I heard, Peter was doing very well and was continuing with his program and self-growth in a positive, productive fashion.

In conclusion, this paper has examined, rather thoroughly, the various clinical aspects of chemical dependency, anger management and clinical depression. Although these three separate diagnostic issues have gained more and more popularity in the clinical field, the novel concept that these three particular issues are oftentimes intertwined and overlapping with one another is no mistake.

The fact of the matter, if I may be so bold in my particular assertion, is that I have seen these three issues concurrently in ***hundreds of inmates*** over the course of my seventeen and a half years in five different correctional facilities throughout the diminutive state of Connecticut. And while the fact of the matter that most of these issues are seen in our criminal populations more so than the non-offending populations, functional people in our society have these issues, too.

Additionally, the paper has closely examined not only these particular issues separately, but also together and how they interact to create a new, complex, and at times, perplexing diagnostic puzzle that the clinician must solve – at least for the well-being of the patient. Unfortunately, as notated in the paper, is the very real fact that many, many clinicians fail to unravel the this diagnostic enigma, if you will, that only leads to further pain and suffering for the stricken patient.

This is by no means denigrating to those professionals whose passion for their patients' well-being is unquestionable, but rather a wake-up call for the those who have to clinically deal with these patients and their presenting issues – as confusing and, at times, maddening, to the well-intentioned, yet ill-prepared clinicians.

In closing, the paper has brought to the forefront diagnostic issues that the majority of chemical dependency clinicians will face, at some point during the course of their career, these three major diagnostic issues that must be dealt with in a thorough and comprehensive manner. And, these three issues, chemical dependency, anger management and clinical depression, while overlapping and mixed-together at times, must be treated separately yet concurrently with one another.

To ignore one of these three issues, even with the best of clinical intentions, will oftentimes lead to relapse, whether it be illicit chemicals, alcohol, mood swings and/or angry outbursts for the patient at some point during their treatment.

Hopefully, this issue will become more prominent in the clinical treatment field and subsequent seminars will be held for those clinical professionals who must deal and effectively treat the increasing number of patients presenting with the chemical dependency, anger management and clinical depression.

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