ALCOHOLISM AND DRUG ABUSE IN PATIENTS WITH PTSD

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The combination of PTSD and substance abuse is both common and problematic. In this chapter we review the following questions: 1) What is the relationship between PTSD, Alcoholism, and Drug Abuse? 2) Can the general "dual diagnosis" literature be of help? 3) Is the phenomenology of PTSD combined with alcoholism and/or drug abuse either unique or specific? 4) Does current pathophysiologic data allow conceptualization of a neurobiological model of PTSD, alcoholism, and drug abuse? 5) Drawing on these ideas, on the limited treatment literature, and the "dual diagnosis" literature, can we develop rational assessment and treatment approaches? Available literature suggests that diagnoses can be validly applied to these patients; that the illnesses must be treated simultaneously as co-primary illnesses; that extreme psychological symptoms reduce the efficacy of alcoholism or drug abuse treatment; and that effective control of these symptoms improves treatment outcome.

The combination of PTSD and substance abuse is a common and complex clinical problem. A number of reports indicate that individuals meeting diagnostic criteria for post-traumatic stress disorder (PTSD) are likely to also meet DSM-III-R criteria for alcoholism and/or drug abuse. Among Vietnam veterans seeking treatment for PTSD, 60–80% exhibit concurrent diagnoses of drug or alcohol abuse or dependence (1–7). These clinical data are bol-

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stered by two major epidemiologic surveys. In the Center for Disease Control's Vietnam Experience Study (8), 39% of veterans meeting criteria for PTSD during the month before examination also meet criteria for alcohol abuse or dependence. Higher rates of comorbidity were found in the National Vietnam Veterans Readjustment Study (9) which revealed that among Vietnam veterans who met criteria for a current or lifetime PTSD diagnosis, 20% and 75% respectively met criteria for alcohol abuse or dependence.

There is a more extensive but less conclusive literature on lifetime and current prevalence rates of alcoholism and drug abuse among Vietnam veterans with combat exposure. Since subjects in these reports are only differentiated according to degree of combat exposure and not presence or absence of PTSD, results are difficult to interpret. In general, data from these studies tend to support the impression that veterans with high combat exposure are more likely to suffer alcoholism or drug abuse than other veterans. One well done study based on NVVRS data (10) reports significantly higher current and lifetime rates of alcohol or drug problems among male Vietnam veterans with high war zone stress than among low war zone stress veterans, other Vietnam era veterans, or civilian controls. Similar results were found for female veterans. Since high war zone stress in this sample has been shown to be associated with PTSD (9) these findings are consistent with the presumption of an important relationship between PTSD and alcohol or drug abuse. Similar results were obtained in a recent study by Fischer (11). In a national probability sample of 1176 Vietnam veterans he found exposure to heavy combat more than doubled a typical Vietnam veteran's risk of reporting a postdischarge substance abuse problem as compared to Vietnam era noncombat veteran controls. Because these studies did not specifically exclude PTSD, it is not known whether combat exposed veterans without PTSD differentially experience alcohol or drug abuse, or whether effects seen are due solely to inclusion of subjects with PTSD.

Statistical association cannot prove cause-and-effect. The relationship between PTSD, alcoholism, and drug abuse seems especially complex. For example, Helzer (12) has suggested that pre-service variables (drinking and other risk factors) are better predictors of post-service drinking than combat exposure. Robins et al (13) found high rates of in-country narcotic use by Vietnam

veterans, but found highly varied outcomes after return to the U.S. Clearly there is a strong likelihood that PTSD sufferers will also experience alcoholism or drug abuse (A-DA). However, the likelihood of such comorbidity is not unique to PTSD, but is seen in most groups suffering from serious psychiatric illnesses (14). We are led in this chapter to review the following questions:

1. What is the relationship between PTSD, Alcoholism, and Drug Abuse?
2. Can the general "dual diagnosis" literature be of help?
3. Is the phenomenology of PTSD/A-DA either unique or specific?
4. Does current pathophysiologic data allow conceptualization of a neurobiological model of PTSD/A-DA?
5. Drawing on these ideas, on the limited PTSD/A-DA treatment literature, and the "dual diagnosis" literature, can we begin developing rational assessment and therapeutic approaches to this problem?

We will evaluate the literature on the relationship of traumatic experience to A-DA. We will proceed with an overview of the general literature on dual diagnosis, emphasizing diagnostic, prognostic, and treatment planning issues. Focusing on PTSD/A-DA, we will review theoretical models, basic and clinical research, clinical experience, and outcome studies. Finally, we will offer a summary of current knowledge, recommend possible treatment approaches, and outline needs for future research.

WHAT IS THE RELATIONSHIP BETWEEN PTSD, ALCOHOLISM, AND DRUG ABUSE?

There may be no simple answer to this important question. Much as many different factors can produce fever, a variety of complex interactions may lead first to PTSD. Even more factors must interact to compound PTSD with A-DA. Although exposure to a toxic environment is a necessary condition for development of fever or PTSD, the impact of exposure for both conditions is modified by previous experience, current vulnerability, family history, individ-

ual adaptive responses, and the post-exposure environment. These same iterated factors will determine whether a PTSD sufferer will either previously or subsequently develop A-DA.

Self-medication and tension reduction models postulate that PTSD develops first and A-DA is reinforced secondarily. Kosten and Krystal (15) suggest the unique importance of hedonistic aspects of drug use in PTSD, since the euphoric effects of alcohol or other drugs may partially counter the emotional numbing so prominent in many PTSD sufferers. As noted in the review of dual diagnosis literature following, evidence for these related models is ambiguous and often contradictory.

Other models emphasize pre-service variables, the nature of the trauma, cognitive attributional style, generational/cohort differences, and biological factors.

Helzer's (12) data suggest that pre-service variables such as family history and pre-service drinking behavior are better predictors of post-service alcoholism than combat exposure. Fischer (11) has criticized Helzer's methodological approach both with respect to sampling and his operational measure of combat exposure. Fischer did not find that premilitary factors predicted later A-DA. He found instead that younger combatants exposed to heavy combat were at greatest risk for post-discharge A-DA.

Green et al (16) suggest that important differences in the nature of the trauma itself predict which comorbid diagnoses will most likely be associated with PTSD. Citing research on civilian disaster survivors (The Beverly Hills Supper Club Fire) (17) as well as studies of Vietnam veterans (18) Green and associates suggest that exposure to grotesque deaths and atrocities is likely to result in A-DA being associated with PTSD, while comorbid anxiety disorders are more prevalent among Vietnam veterans with PTSD exposed to high risk combat assignments.

McCormick et al (19) offer a cognitive modeling approach, postulating that addicted patients with PTSD utilize a learned helplessness attributional style in framing their experiences.

Davidson et al (1) underscored cohort differences based on comparison of World War II and Vietnam veterans with PTSD. Despite many similarities, the Vietnam veterans with PTSD/A-DA had an earlier age of onset for alcoholism. In fact, alcoholism *preceded* PTSD by 3.1 years in his Vietnam sample but *followed* PTSD by 6.9 years in the WWII sample.

Biological models suggest a vulnerability to comorbid A-DA based on the pathophysiology of PTSD, as will be reviewed below.

Many different combinations and sequences of factors may eventually result in the PTSD/A-DA syndrome. For some patients, genetic loading and pre-trauma factors seem paramount. In others experiential, cognitive, generational, and/or biological variables may be decisive. In most, pre-trauma and traumatic factors interact complexly and iteratively to determine whether or not PTSD will be associated with A-DA.

OVERVIEW OF OTHER "DUAL DIAGNOSIS" LITERATURE

There is an interesting and growing literature regarding the co-existence of A-DA and other psychiatric disorders. This literature includes discussions of diagnosis, etiology and sequence, and treatment. While much of the literature focuses on diagnosis, a particularly interesting and pertinent group of papers focuses on global psychiatric symptom severity as a prognostic factor in substance abuse treatment. This current of literature has received little notice in published works on PTSD and substance abuse; we will provide an overview here.

The concept of "self-medication" is commonly seen in the general dual diagnosis (20,21) as well as the PTSD/A-DA literature. Those treating patients with substance abuse and PTSD have generally assumed "self-medication" as a principal etiology for co-existing disorders (15,22,23). This hypothesis suggests that substance use relieves specific dysphoric symptoms of PTSD and reinforces further use. Self-medication models can be considered as diagnosis or symptom specific modifications of more general "tension reduction" models. Evidence for tension reduction models of substance abuse is mixed and inconsistent (24). Such models have been used to argue that treating the "primary" disorder (in this case PTSD) will lead to resolution of the "secondary" disorder. There is no empirical, and little anecdotal, support for this position. The hypothesis of "self-medication" assumes a similar relationship between psychiatric diagnosis and drug of choice across patients (20,21). The non-PTSD literature examining diagnosis/drug of preference combinations does not often support such relationships;

much of what is found seems counter-intuitive (for instance increased rates of stimulant abuse in schizophrenic samples) (21). In PTSD there is little evidence of specificity of drug choice. Robins et al (13) in an early study of narcotic use in Vietnam returnees found that although almost half their subjects had used narcotics in Vietnam, relatively few continued narcotic use after their return. Subsequently some changed to other drugs, and alcohol use was quite high but unaffected by pattern of narcotic use. Current clinical samples report use of a wide variety of drugs with very different pharmacologic actions including alcohol, cocaine in various forms, and marijuana. Since there is little specificity of drug choice in PTSD, it appears the self-medication hypothesis is so nonspecific as to be of little help.

McLellan and colleagues developed the Addiction Severity Index (ASI) to investigate variables pertinent to outcome of substance abuse treatment. This structured interview provides global severity ratings in several potentially important problem areas, including medical, legal, family, employment, alcohol use, drug use, and psychiatric problems (25). In studies evaluating the ASI as an assessment instrument, McLellan and colleagues found that the most powerful prognostic variable was the global severity rating of psychiatric problems. They further noted differential effectiveness of various substance abuse treatment approaches when subjects were stratified by psychiatric severity ratings (26). In general, subjects with low psychiatric severity scores seemed to benefit from most treatment approaches; those with midrange scores demonstrated differential treatment responsiveness across programs; those with high scores benefited least from any treatment. These findings were later confirmed in a prospective treatment matching study (27), and strongly suggest the value of global psychiatric severity ratings in treatment planning and differential therapeutics.

Questions about the validity of psychiatric diagnoses in the presence of substance abuse have often been raised. Impractical recommendations for extended sobriety before attempting psychiatric diagnosis are often seen. Still, discussion of the implications of diagnosis requires some evidence that psychiatric diagnoses can be validly made in the presence of substance abuse. Some argue that psychiatric symptoms in substance abusing patients are largely due to direct effects of alcohol or drugs and/or result from conse-

quent social and economic dysfunction (28). Penick et al, however, provide evidence suggesting that in alcoholic men diagnoses made soon after detoxification remain quite stable when followed up after a year, with overall agreement rates ranging from 86% to 99% (29). Most differences across time were due to reduced, rather than qualitatively different, symptoms. They conclude that "symptom patterns appear to reflect additional psychiatric disorders that are stable over time and a potential target of treatment." Thus available data suggest that both severity and psychiatric diagnosis may be validly assessed early in the course of treatment.

Other studies have examined the relationship of diagnosis, severity, and treatment completion with somewhat mixed results. This literature becomes difficult to interpret because of probable interactions between diagnosis, severity, and treatment variables. Rounsaville et al (30) found that high psychiatric severity was a predictor of poor treatment results; however they found that specific diagnoses were also predictive. Antisocial personality or depression were associated with poor outcome in men; depression was associated with improved outcome in women. They suggested that diagnosis could also be an important predictor of treatment outcome; while the principle seems valid it is not clear whether their specific findings can be generalized to other treatment settings. We will suggest later that diagnosis is primarily important for differential therapeutic assignment.

Given the negative prognosis of psychiatrically symptomatic patients, Woody et al (31) studied the efficacy of psychotherapy to minimize symptomatology in a group of non-psychotic opiate dependent subjects beginning drug counseling treatment. The addition of psychotherapy to drug counseling produced little additional benefit in subjects with low psychiatric severity scores. Subjects with mid-range scores benefited from drug counseling alone, but additional psychotherapy produced further benefits. High-severity patients showed minimal improvement in drug counseling alone, but showed improvement when also given psychotherapy. Believing this implied the importance of psychiatric symptom reduction in conjunction with substance abuse treatment, Kofoed et al (32) established a pilot program which simultaneously treated substance abuse and major psychiatric illness. They found that patients with personality disorders dropped out quickly, but patients with DSM-III axis I disorders often completed several months of

stringent treatment. Since a retrospective measure of psychiatric severity was not associated with treatment drop-out, Kofoed et al suggested that effective concurrent treatment of psychiatric illness removed the negative prognostic implications of the "severity effect."

We believe that the literature on other concurrent psychiatric illness and substance abuse problems suggests the following major points: First, that diagnoses can be validly applied to patients with co-morbid substance abuse; Second, that the relationship of the concurrent illnesses is variable, and the illnesses must be treated simultaneously as co-primary illnesses (33); Third, that significant psychiatric symptoms seriously reduce the efficacy of traditional substance abuse treatment approaches (26); and Fourth, that effective control of these symptoms improves the possibility that patients will benefit from treatment (32,34). Thus the more general literature implies that successful treatment of concurrent PTSD and substance abuse may require prompt control of PTSD symptoms, combined with simultaneous substance abuse treatment.

IS THE CLINICAL PHENOMENOLOGY OF PTSD/A-DA DIFFERENT THAN PTSD COMORBID WITH DEPRESSION OR AN ANXIETY DISORDER?

We believe that co-morbid A-DA is more likely to affect the clinical course of PTSD than other co-morbid diagnoses. In our opinion, this is partly because some diagnoses shown to occur frequently among PTSD patients (such as depression or other anxiety disorders) may be artifacts of the decision rules used to make DSM-III-R diagnoses. There are no exclusionary rules to prevent clinicians from diagnosing additional anxiety or affective disorders in patients whose primary problem is PTSD. In this regard Keane and Wolfe (35) have observed that "the presence of multiple distinct disorders . . . may simply be a function of the severity or intensity of a single primary disorder that is adversely affecting many areas of psychological functioning."

In addition to methodological questions about sensitivity and specificity of the DSM-III-R classification scheme, there are unique biological issues concerning comorbid depressive and anxiety disorders when PTSD is involved. For example recent findings (36) on

the hypothalamic-pituitary-adrenal (HPA) system suggest that the pathophysiology of major depressive disorder (MDD) involves subsensitivity (downregulation) of glucocorticoid receptors whereas PTSD is associated with glucocorticoid supersensitivity (upregulation). Despite this important biological distinction it appears that DSM-III-R criteria cannot distinguish between melancholia (MDD) and depression associated with PTSD. As Friedman argues elsewhere (37) it is possible that PTSD/MDD is a specific affective subtype of PTSD and that there should be an exclusionary rule so that patients with PTSD and depressive symptoms will not receive a diagnosis of MDD. The high comorbidity among PTSD patients for anxiety disorders such as panic, phobic, and generalized anxiety disorders (1,3,10,16) raises a similar question. It is possible that PTSD/anxiety disorder is an anxious variety of the general PTSD syndrome; if so, there should be an exclusionary rule reflecting this. Mellman and Davis (38) have already pointed out that PTSD flashbacks meet DSM-III-R criteria for panic attack; perhaps phobic and generalized anxiety symptoms observed with PTSD patients simply reflect PTSD avoidant and hyperarousal symptoms respectively.

We do not believe that alcoholism or drug abuse bear such a potentially specific relationship to PTSD. These problems are not simply predictable aspects of the PTSD syndrome, nor due to "symptom counting" decision rules. Many traumatized patients develop PTSD but not A-DA. Perhaps most importantly, whereas there is currently little evidence that treatment for PTSD/MDD or PTSD/anxiety disorders differs markedly from PTSD alone, this is certainly not true for PTSD/A-DA, which clearly requires specific multi-modal treatment.

CAN WE CONCEPTUALIZE A UNIQUE NEUROBIOLOGICAL MODEL FOR PTSD/A-DA?

As argued elsewhere (37) there may be a neurobiological reason for high comorbidity rates between PTSD and A-DA. Adrenergic, opioid, and possibly serotenergic system dysregulation associated with PTSD may make affected individuals particularly susceptible to A-DA. Some of the abused drugs appear to have effects on the endogenous opioid system, partially reversing the chronic opioid

deficiency observed in animals exposed to inescapable shock and postulated to also occur in PTSD patients (15,25,39,40). This model provides a more specific approach to application of tension reduction hypotheses to PTSD/A-DA.

It also follows "that once the vicious addiction-withdrawal cycle is established, PTSD patients may have even more difficulty achieving (and maintaining) abstinence than chemically dependent individuals without PTSD . . . because the rebound hyperarousal experienced by . . . PTSD patients undergoing withdrawal may itself trigger a conditioned emotional response associated with PTSD symptoms" (37). Kosten and Krystal (15) had predicted that withdrawal induced hyperarousal could serve as a Pavlovian conditioned stimulus capable of eliciting PTSD symptoms. This prediction was confirmed by Risse et al (41) who reported intrusive recollections, nightmares, and even flashbacks among patients with PTSD undergoing withdrawal from alprazolam.

CLINICAL EXPERIENCE AND TREATMENT STUDIES

The clinical impact of comorbidity is summarized by Boudewyns et al (42) who report that in a cohort of Vietnam veterans admitted to a specialized VA PTSD treatment program, 91% met criteria for a lifetime diagnosis of substance abuse or dependence. The authors suggest that the disorders are "inextricably intertwined" so frequently that almost all PTSD treatment must be dual focused, providing simultaneous treatment for those frequently co-occurring disorders. There are few articles specifically addressing what effective treatment might be. Available literature includes descriptions of treatment directed towards Vietnam veterans with PTSD or women substance abusers with a history of rape-incest trauma. Most authors have created hybrid treatment models based on knowledge and experience accumulated in the separate treatment of PTSD or A-DA. A similar process is described in the general dual diagnosis literature. Decisions on how to blend the strategies into meaningful treatment have been based on theoretical models, clinical experience and clinical judgment. There is little outcome data to differentiate the effectiveness of these approaches.

In general, clinicians have combined group and individual therapies for PTSD with aspects of "Twelve Step" programs such as Alcoholics Anonymous. Goals of treatment involve both relief of PTSD symptoms and reduction and eventual cessation of addictive behaviors. In the more comprehensive descriptions (23,43,44) treatment begins with a stabilization period during which treatment priorities include detoxification, treatment of psychotic symptoms, suicidal or homicidal ideation, and medical stabilization. The next step involves nurturing a motivation for further substance abuse focused treatment in the individual patient. Jelinek (44) describes this as "hooking the patient"; Abueg (43) called it the "commitment stage." A similar focus, here called "persuasion," was shown to improve treatment acceptance in a general dual diagnosis patient population (45). This period usually entails education about PTSD and substance abuse in both general and individualized terms. Given a degree of willingness by the patient, combined substance abuse treatment, psychotherapy, and symptom focused behavioral or pharmacologic therapies can then proceed.

Substance abuse treatment is usually offered in group format. All authors agree that treatment is long-term, including inpatient and outpatient services, with a need for dual-focused aftercare and contingency plans for relapse. Jelinek (44) and Schnitt & Nocks (23) present treatment strategies which combine aspects of Twelve Step recovery programs with group and individual psychotherapy. The dynamics of group support and confrontation are a cornerstone of their treatment approaches. Individual psychotherapy is used to help patients cope with difficult feelings generated by sobriety-induced awareness and working through of past traumatic events. A main objective of these approaches is to validate and integrate the emotional and cognitive experiences related to trauma while simultaneously nurturing motivation towards and interpersonal rewards for an abstinent lifestyle. Bellerud (46) presents a similar approach for treatment of chemically dependent women with concurrent trauma related syndromes.

All these authors report improved outcomes, but only two have provided outcome data (43,47). Abueg et al (43) distinguish themselves by giving a comprehensive assessment of this clinical problem citing empiric evidence, clinical experience, and behavioral

and biologic theory about A-DA and PTSD. Their approach, described in detail, draws from well developed behavioral models, including relapse prevention technologies. Their sequential treatment goals include building and sustaining motivation for treatment, decreasing active PTSD symptoms including disruptive intrusive recollections and outbursts of rage, and building more effective social and problem solving skills. They include specific individualized behavioral and cognitive strategies for relapse prevention. They utilize group therapy, direct therapeutic exposure, and cognitive problem solving training to cope with the complex mix of symptoms and interwoven relapse risk factors of the combined disorders.

In the same chapter, Abueg et al (43) report a controlled study of relapse prevention training as an adjunct to inpatient treatment on a specialized PTSD unit. One group of 42 patients received relapse prevention training in addition to residential PTSD treatment; the control group of 42 patients received only residential PTSD treatment. At six month follow-up 63% of the experimental group reported sustained abstinence, compared to 41% of controls. Relapse rates converged at 9 months (44% of experimental vs. 38% of control group), though overall alcohol consumption was self-reported as lower in the experimental group.

Kuhne et al (47) performed a study evaluating the benefit of specific trauma-oriented therapy offered concurrently with residential alcoholism treatment. They provided a readjustment group for veterans with high combat exposure. At one year follow-up abstinence rates were the same for non-theatre, light combat exposure, and heavy combat exposure subgroups. The authors argue that readjustment group therapy ameliorated the negative effects of heavy combat exposure. Unfortunately they had no heavy combat exposure controls. Furthermore they did not differentiate by PTSD diagnosis; they reported little difference in MMPI scores or profiles between the different combat exposure groups, suggesting they had inadvertently pre-selected a group with low psychiatric symptom severity despite heavy combat exposure. Thus their results must be considered inconclusive.

Pharmacologic treatments are touched upon briefly in this literature. Jelinek (44) and Schnitt & Nocks (23) generally discourage psychotropic medications, especially benzodiazepines, in an at-

tempt to avoid cross-addiction and to maintain the integrity of the Twelve Step recovery model.

The reports summarized have been written by thoughtful clinicians attempting to address a very complex clinical situation by developing treatment strategies based upon their clinical knowledge, skills, and theoretical understanding of the disorders. They raise important issues about the interaction of the co-occurring disorders and propose treatment approaches. However convincing evidence of the efficacy of any specific approach has not appeared. The literature does not specifically address the differential therapeutic process, does not guide specific treatment selection, and offers little guidance for integrating pharmacotherapeutic approaches such as disulfiram (Antabuse), naltrexone (Trexan) or symptom reducing psychotropics.

HOW CAN WE BEGIN TO DEVELOP A RATIONAL APPROACH TO THE PROBLEM?

Assessment

Practically, our review suggests the importance of both severity *and* diagnosis in initial assessment and treatment planning. Assessment of symptom severity implies the necessity of additional treatment approaches to reduce symptoms. Timely diagnostic assessment is critical, however, in the differential therapeutics decision making process. Even with the uncertainties about the etiology of major depressive disorders or panic disorder in complex patients with PTSD, these diagnoses do seem to have specific treatment implications. Major depressive symptoms will respond to antidepressant medications even in the presence of PTSD; panic disorder similarly responds to appropriate treatment with tricyclic antidepressants, MAO inhibitors, or benzodiazepines. This review implies that an important part of assessment should be delineation of the relative contributions of each diagnostic entity to the current symptom acuity picture, and prioritizing treatment accordingly.

Symptom Reduction

The clinical phenomenology and interaction of the addiction/withdrawal process with PTSD symptoms (15,41) suggests the unique interaction between the pathophysiology of PTSD and the addiction and recovery process. The essential implication is the requirement for simultaneous treatment of PTSD and A-DA symptoms from the very first stages of treatment. PTSD and A-DA must be treated simultaneously because the complex self-sustaining interrelationship between intra-psychic, behavioral and biological aspects of PTSD and concurrent A-DA demands a comprehensive treatment approach (48). This conclusion is consistent with the general literature on dual diagnosis, where it is suggested that disorders must be treated simultaneously as co-primary illnesses (33). Reduction of severe symptoms must be attempted to make substance abuse treatment components acceptable and psychologically accessible, otherwise treatment will fail (26). Effective control of such symptoms improves the chances patients will benefit from such treatment (34). Effective behavioral (49), psychotherapeutic (50) and pharmacologic (51) approaches to symptom reduction in PTSD need to be systematically applied to and studied in the PTSD A-DA population.

A particularly controversial issue is the use of benzodiazepine anxiolytics in this population. Most authors suggest use of non-benzodiazepine anxiolytics, including buspirone or adrenergic beta-blocking agents, because they fear development of "cross-addiction" to benzodiazepines in these patients. Certainly available data suggest a feasible mechanism for such risk. Ciraulo et al (52,53) found that both alcoholic patients and men with a family history of alcoholism were more likely to have euphoric responses to the benzodiazepine alprazolam (Xanax) than controls. In some patients, however, adequate control of anxiety without benzodiazepines cannot be achieved. Failure to control intractable anxiety symptoms may be a significant factor in treatment failure. Further, available clinical studies do not convincingly demonstrate increased risk of benzodiazepine abuse in alcoholic patients compared to other psychiatric patients. Ciraulo et al (54) published an exhaustive review of the data on liability for benzodiazepine use among alcoholics. The studies they cite suggest that benzodiazepine *use* is frequent among alcoholics entering treatment,

generally between 33 and 40%. This rate of use is similar to that reported in outpatient psychiatric populations (55,56,57) and is interpreted as reflecting appropriate treatment of co-morbid psychiatric conditions, including anxiety disorders. Rates of abuse and misuse, quite variously defined, ranged from 5% (58) to 17.8% (59). Ciraulo et al concluded from their review that "there is a body of literature which suggests that alcoholics as a group may be more susceptible to benzodiazepine abuse than nonalcoholics, but there is little evidence to suggest that all or even more alcoholics abuse them. Clearly, more definitive work is needed to clarify the issue, but in the meantime, we feel that benzodiazepines are relatively safe drugs with many uses in the treatment of alcoholics when prescribed rationally" (54, p.1505). At this time the balance of the risk of cross-addiction versus the negative prognostic implications of uncontrolled severe anxiety must be made individually by the clinician. Benzodiazepines may vary in risk and efficacy; we recommend clonazepam because of its efficacy and because slow absorption and elimination reduce euphoric responses and hence abuse potential.

Primary Substance Abuse Treatment

Available literature suggests application of either relapse prevention technologies (43) as outlined by Marlatt & Gordon (60) or group focused treatment incorporating the principles of Twelve Step recovery programs such as Alcoholics Anonymous and Narcotics Anonymous. Generalizing from other dual diagnosis literature, it seems likely that some success may be achieved with a variety of techniques if psychiatric symptoms are well controlled. There is clearly a need for studies of the effectiveness of the variety of described treatments, with special attention to possible differential benefit for patients with varied symptom severity and co-diagnoses. Such studies should also examine whether achievement of "sobriety" is associated with other improvements in functioning, or whether additional individualized rehabilitative treatments including family, educational, social skills, and occupational interventions are required.

Patient choice among treatment options has been shown to be associated with improved satisfaction and improved treatment completion in primary alcoholics (61); this common sense but little

honored principle should be studied in our complex, bitter, and disenfranchised patients.

Aftercare

Ongoing supportive treatment involvement characterizes effective treatment for both substance abuse and PTSD individually; it seems likely this will also be true for the juxtaposition of the disorders (23).

WHERE SHOULD FUTURE CLINICAL RESEARCH EFFORTS FOCUS?

Assessment

Available data suggest that it is often possible to gather diagnostic data for treatment planning soon after detoxification. There are also a variety of substance abuse screening and diagnostic tools available. These include the CAGE (62,63) and Michigan Alcoholism Screening Test (MAST) (64) or the more specific Veterans Alcoholism Screening Test (VAST) (65) as screening instruments, and the Addiction Severity Index (ASI) (25) or Alcohol Use Inventory (AUI) (66) as aids to treatment planning. The Inventory of Drinking Situations (67) or Alcohol Expectancy Questionnaire (68) are particularly helpful in identifying relapse risk factors that may be amenable to behavioral, cognitive, or social skill training interventions. Further study of the reliability, validity, and perhaps most importantly stability of diagnostic and assessment tools in this particular population is needed.

Symptom Reduction

Continued study of pharmacologic methods of symptom reduction is likely to be valuable. Studies of the role of benzodiazepines in reducing anxiety in these patients will be valuable. Relapse prevention techniques may prove to be applicable to aspects of both substance abuse and PTSD; these and the related cognitive therapies seem likely to prove valuable. Studies of variable symptom presentations and more specific treatments will be required.

Primary Treatment

The relative roles of inpatient and outpatient programs is still controversial even in treatment of primary substance abusers. Still, studies of this issue are essential to provide treatment that is maximally effective and minimally intrusive or disruptive. The prognostic implications of symptom severity and various co-diagnoses must be specifically studied in this population, with special attention to interactions between prognostic factors and treatment modalities. Examination of clearly described treatments in well characterized populations, similar to studies of manual guided psychotherapies, will be essential in order to achieve findings which can be generalized with confidence. Recent psychotherapy studies suggest that therapist qualities may have more impact on treatment success than type of therapy offered; studies of the helping relationship and qualities of therapists successful with this population may be ultimately more productive than studies of various treatment types. The role of patient choice and involvement in treatment planning should also be formally examined.

Functional Improvement

The relationship between symptom reduction and improved functioning in the family, workplace, or community has not been established. It probably varies under different circumstances. The overall functional implications of symptom reduction and/or sobriety need to be monitored in future studies.

Aftercare

While aftercare is probably important in maintaining treatment improvements for both PTSD and substance abuse, duration and frequency of aftercare needed for effective maintenance of treatment benefits probably varies both with type of individual treatment and with variations in the individual sociocultural and clinical picture. Studies to determine an efficient and effective duration of aftercare for PTSD/A-DA patients are needed. The role of the Twelve Step self-help fellowships also requires further examination, though rigorous study of these programs is difficult due to their essential anonymity. These fellowships may be particularly

helpful to some PTSD sufferers because of their explicit but non-sectarian attention to spiritual issues.

SUMMARY

There are suggestions and some data, both specific to PTSD/A-DA and in the broader dual diagnosis literature, allowing clinicians to develop somewhat informed treatment plans for their patients. However much remains in the realm of clinical lore or even frank guesswork. We hope that those developing and offering new treatment programs to these challenging patients will also assume the mantle of clinical science, and attempt to characterize, evaluate, and report the results of their experience. With such efforts, our patients can surely benefit.

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