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Chapter 39

Clinical management of alcohol use disorders in the neurology clinic

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INTRODUCTION

The link between alcohol use disorders (AUDs) and adverse health outcomes is well established. Alcohol affects 20–50% of hospitalized patients, 15–30% of patients seen in primary care settings, and upwards of 60% of patients with psychiatric illnesses. AUDs are a leading cause of early preventable death and are associated with a constellation of medical consequences (Saitz, 2005a).

Patients typically do not directly present their unhealthy alcohol use to their primary or specialty care provider but do present with alcohol-related conditions such as ataxia, cognitive impairment, tremor, gastritis, or psychogenic problems, including anxiety and depression (Aira et al., 2003). Adverse health consequences, particularly the neurologic sequelae, are common in AUDs.

Despite the fact that patients with AUD frequently present in ambulatory care settings, physicians and other healthcare providers seldom screen or intervene for this problem. The reasons are many, including the moral stigma of addiction, lack of physician training in addiction, and a pervasive belief that addiction treatment doesn't work.

The moral stigma associated with addiction continues to affect the care of the addicted patient. CASA Columbia's national survey of the attitudes of US adults vis-à-vis addiction and its treatment found that approximately one-third of Americans believe that addiction is a sign of lack of willpower or self-control, and approximately 50% of physicians believe that addiction is caused by a character defect (CASA Columbia, 2012). These beliefs are at odds with an ever-growing body of evidence showing that addiction is a chronic illness with strong genetic and biologic components. Given these preconceived notions, it is no wonder that physicians do not screen for substance use disorders: Many of them do not actually consider this a medical problem.

Lack of physician training in the area of substance misuse and abuse is another barrier to care. Only 20–30% of primary care physicians feel "very prepared" to detect risky substance use, yet 80% feel "very prepared" to tackle hypertension or diabetes (CASA Columbia, 2012). Instead of targeting addiction, most healthcare professionals address the medical complications of addiction without addressing its core cause.

Many people, including healthcare providers, express a sense of hopelessness about addiction treatment. Yet, the data demonstrate that addiction treatment works at rates on a par with treatment for other chronic illnesses, from depression to asthma (McLellan et al., 2000). In fact, treatments for alcohol addiction reduce alcohol use by 40–60%, as well as decreasing criminal activity, decreasing comorbid physical and mental illnesses, and improving other measures of well-being, such as housing status and employment.

To treat the medical problems related to substance misuse and to target the substance use disorder itself, providers should screen and intervene for substance use disorders within an ambulatory care clinic, even when resources for addiction treatment are limited. An evidence-based algorithm called screening, brief intervention, and referral to treatment (SBIRT) explains how at-risk drinking and AUDs can be identified, and how at-risk drinking can be effectively targeted within ambulatory care settings. In this chapter we review the components of SBIRT and how to implement SBIRT

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in clinical practice. We also describe clinical presentations of common neurologic disorders associated with acute and chronic alcohol toxicity.

WHAT IS SBIRT?

SBIRT is a method of intervention with individuals with alcohol and other substance use problems. SBIRT is best suited for primary care clinics, hospital emergency rooms, trauma centers, neurology clinics, and community health settings, and is a physician-led prevention approach.

The Substance Abuse Mental Health Services Administration (SAMHSA) recommends the routine use of SBIRT in primary and specialty care settings for the following reasons:

- It is brief. The initial screening is accomplished quickly (modal time about 5 minutes).
- Specific behaviors are targeted. The screening tool addresses a specific behavioral characteristic deemed to be problematic or preconditional to other diagnoses.
- The services occur in a healthcare or other non-substance abuse treatment setting. This may be an emergency department, primary or specialty care physician's office, including the neurology clinic.
- It is comprehensive. SBIRT includes a seamless transition between a quick screening and brief intervention.
- Research and substantial experiential evidence supports the SBIRT model. At a minimum, programmatic outcomes demonstrate a successful approach.

WHAT IS THE EVIDENCE THAT SBIRT WORKS?

Early identification and brief intervention in primary and specialty care settings have been shown to be effective in patients with at-risk alcohol use. Forty percent of patients with risky alcohol use who received screening and brief intervention from their primary or specialty care provider moderated their drinking to safe levels compared with only 20% in control groups. These differences between intervention and control groups were still present 4 years later. There is growing evidence from review studies as well as meta-analyses of randomized clinical trials that, using the SBIRT approach, reduces risky drinking in patients presenting in primary and specialty care settings.

From these and other studies, the World Health Organization, the Institute of Medicine, and other health authorities recommend asking (screening) all patients about their alcohol use patterns and providing brief intervention when necessary. Evaluation of lifestyle health risks should always also include an evaluation of alcohol use. The US Preventative Services Task Force (USPSTF) has recommended that "interventions for risky/harmful alcohol use among adult primary care patients can provide an effective public health prevention approach to reducing problematic drinking" (US Preventative Services Task Force, 2004). The USPSTF also concluded that physician-led brief interventions for risky drinkers should include education and advice to reduce current drinking; feedback about current drinking patterns; and explicit goal setting, usually for moderation and assistance in achieving the goals. The combination of screening all adult primary care patients to identify individuals with unhealthy use and a brief counseling intervention has been proposed as a population-wide, preventive intervention in primary care. Clinical trials support the use of screening and brief intervention in unhealthy alcohol use for adults (Saitz, 2013).

SCREENING

The purpose of alcohol screening is to identify patients with risky alcohol use so that interventions can be provided to reduce drinking and prevent harm (Bradley and Berger, 2013). Screening is not the same as diagnosis, nor should a positive screen be misinterpreted as an AUD. Rather, screening alerts the provider to patients who need follow-up and further assessment of drinking patterns and their association with adverse health effects.

Quantity and frequency of alcohol should be used as the initial method of determining who is engaging in at-risk alcohol use. Evaluation of alcohol consumption patterns should be included during medical consultations or physical examinations, as part of a more general evaluation of lifestyle health risks. It is important to introduce the alcohol discussion with the patient during the medical consultation and always connect that discussion with the presenting medical issues, the diagnosis, and the physical care plan goals.

When screening patients for quantity and frequency of alcohol use, it is important to know what constitutes one standard drink. One standard drink is defined in the alcohol literature as 0.5 fluid ounces of alcohol, which can be found in one 12-oz bottle of beer, one 5-oz glass of wine (there are usually five standard drinks in one bottle of wine), and one 1½-oz "shot glass" of hard liquor (vodka, whiskey, etc.).

An ever-growing body of evidence links quantity and frequency of alcohol consumption to overall morbidity and mortality. Those who drink above a certain level are at increased risk for numerous poor health outcomes, including gastrointestinal problems (Lembke

et al., 2011), trauma (Harris et al., 2009), and all-cause mortality (Harris et al., 2010); and one-third are at risk of developing addiction (Saitz, 2005b).

Risky alcohol use for an adult male, based on the evidence above, is defined as a pattern of alcohol use exceeding 14 standard drinks per week or four per occasion, and for an adult female as exceeding seven standard drinks per week or three drinks per occasion. The amounts differ for men and women because of differences in metabolism and vulnerability to alcohol toxicity, with women being more vulnerable to the toxic effects of alcohol than men, even with shorter drinking careers. Amounts below these are considered to be non-risky, or moderate alcohol use, because they do not correlate with adverse health outcomes.

Focusing on these quantifiable anchor points minimizes the problems of subjective report and patient denial. Also, although many individuals, addicted or not, are poor at recalling substance use accurately when asked to retrospectively assess their use over a long time period, such as the past year (even when they are comfortable disclosing it candidly to the clinician), data show that when asking about specific amounts and specific days in the recent past, patients are surprisingly accurate reporters of consumption. Finally, quantity and frequency lead to a number that can be regarded as a "fifth vital sign," easily charted and compared with other numbers past and future, a possible indicator of a problem but not a definitive diagnosis of a problem.

Two brief and easy-to-use screening tools to assess quantity and frequency of alcohol consumption are available and ideal for the neurology clinic, namely, the Timeline-Followback (TLFB) method and the AUDIT-C.

The TLFB method charts the amounts and patterns of substance use in the preceding week, independently of consequences or compulsivity of use (Sobell and Sobell, 1995). By keeping to specifics and a timeline, there is a markedly decreased chance of the patient minimizing consumption, mixing up drinking occasions, or mentally averaging drinking over the period of interest. Ask patients to begin with the day prior to the clinical encounter and move backwards, remembering how much alcohol they consumed on each day in the preceding week. Where appropriate, offer concrete events to stimulate memory, e.g., "Let's start with Monday, that was the first day of the week before mid-term exams." Then calculate the number of standard drinks consumed in a week.

The same principle applies to use of the AUDIT-C, which consists of three questions regarding frequency and quantity of alcohol consumption: (1) "How often do you have a drink containing alcohol?" (never, monthly, 2–3 times per week, etc.); (2) "How many drinks of alcohol do you drink on a typical drinking day?"; and (3) "How often have you had six or more drinks per day?" The last question captures binge drinkers, a well-established drinking pattern which might be missed with the TLFB method, and consists of heavy, condensed periods of consumption interspersed with longer periods of abstinence. Again, starting with quantity and frequency allows the clinician to tackle the issue of alcohol use in a quantitative and non-judgmental way.

If risky drinking is identified based on quantity and frequency, then the healthcare provider is prompted to delve deeper, specifically to address whether the patient meets *Diagnostic and Statistical Manual of Mental Disorders* (DSM-V) criteria for a substance use disorder (American Psychiatric Association, 2013).

The DSM defines a substance use disorder as use of nicotine, alcohol, and/or other drugs with two or more of the following symptoms within a 12-month period: (1) attempting to cut back on substance use without success; (2) consuming more of the substance than planned; (3) spending a lot of time and energy getting, consuming, and recovering from using the substance; (4) experiencing intense desire to consume the substance, often referred to as "craving"; (5) failing to fulfill major life obligations due to substance use; (6) continuing to use the substance despite consequences; (7) giving up or reducing important activities due to substance use; (8) using in dangerous situations; (9) developing tolerance; and (10) experiencing withdrawal.

The new edition of the DSM (DSM-V) has added spectrum qualifiers "mild," "moderate," and "severe," allowing providers to capture the variance in severity that is common with substance use disorders. Severity is rated as follows: If the patient endorses twoor three items on the list, then (s)he has a mild substance use disorder, four or five items and she has a moderate substance use disorder, and six or more items and she has a severe substance use disorder. For example, the patient who describes attempting to cut back without success, spending a lot of time using and recovering from the substance, failing to fulfill major life obligations due to substance use, and experiencing withdrawal has a moderate substance use disorder (American Psychiatric Association, 2013).

The DSM does not rely on frequency and quantity of substance use, not because it assumes these data points are unimportant, but because of the difficulty in standardizing across the wide variety in form (plant, pill, liquid, gas) and potency of addictive substances, not to mention the difficulty in knowing what is in the new synthetic drugs that have been flooding the market over the last decade ("legal highs," "bath salts," etc.). The absence of quantifiable measures in the DSM is an expression of the limitations of a phenomenologic guide

rather than a dismissal of its importance. Therefore, using DSM criteria alone, without also assessing quantity and frequency, is inadequate. A patient who drinks a fifth of vodka every night but denies all of the DSM criteria for a substance use disorder nonetheless likely has an AUD and could benefit from an intervention. A quantitative reference point allows clinicians to make the appropriate diagnosis, even in the absence of subjective recognition of addiction ("denial").

Once the patient has screened positive for at-risk alcohol use or an AUD, the algorithm below is useful in determining the "next steps" after an alcohol screen.

SCREENING RESULTS

1. No or minimal problems with drinking: Patient education and no further intervention.
2. Mild to moderate at-risk drinking: Patient education and brief intervention.
3. Moderate to high-risk drinking: Brief treatment.
4. AUD based on DSM criteria: Referral to specialty treatment.

Screening can be repeated at intervals, as needed.

In summary, alcohol use occurs along a continuum from at-risk drinking to AUD, ranging in severity from mild to moderate to severe. Appropriate interventions should be applied based on the severity of the patient's drinking determined through a screening process. The National Institute of Alcoholism and Alcohol Abuse (NIAAA, a division of the National Institutes of Health) focuses on two cohort groups of problem alcohol use: (1) at-risk drinkers (i.e., those who exceed recommended maximum daily levels of drinking but do not necessarily meet criteria for an AUD); and (2) drinkers who meet the DSM V criteria for AUD (American Psychiatric Association, 2013). Brief interventions are not effective for those with AUD who need referral to specialty alcohol treatment, but the process of doing SBIRT can provide a litmus test to identify those patients who have more significant substance use problems (i.e., addiction) and need specialty referral.

BRIEF INTERVENTION

Brief interventions are interactions with patients that are intended to induce a change in a health-related behavior. Brief interventions are generally used as a health management strategy for patients with at-risk alcohol use but who do not necessarily meet criteria for an AUD. Most patients in primary and specialty care medicine are in this at-risk category. The goal of a brief intervention (which usually involves one to five sessions lasting about 5–10 minutes) is to educate patients and increase their motivation to reduce risky behavior and embrace the healthier lifestyle as outlined in their physical care plan.

Research from smoking cessation and problem alcohol use has increased our understanding of the change process, giving new directions for improved health promotion and lasting lifestyle change in patients. Current views depict patients as being in a process of change. When physicians choose a mode of intervention, "one size doesn't fit all." Two important advances include the stages of change model and motivational interviewing. Developed by Stephen Rollnick at Cardiff University School of Medicine in the United Kingdom, motivational interviewing is a collaborative, goal-oriented style of communication with particular attention to the language of change (Miller and Rollnick, 2012). It is designed to strengthen personal motivation for and commitment to a specific goal by eliciting and exploring the person's own reasons for change within an atmosphere of acceptance and compassion. Motivation is multidimensional and not easily assessed. To assist with motivating the behavioral change process, psychologists James Prochaska and Carlo DiClemente identified five primary stages that people move through as they seek to make changes in their lives. Motivational interviewing can assist people with successfully moving through the first three stages of the change process. Through motivational interviewing, the physician can help a patient to consider the possibility of change, contemplate the risks and benefits of change, and prepare for moving forward with making changes in her or his life.

The creators of the stages of change model used factor and cluster analytic methods in retrospective, prospective, and cross-sectional studies of the ways people quit smoking. The "stage-based" approach has been validated and applied to myriad behaviors, including nicotine cessation, exercise, and dietary behavior (Grimley, et al., 1993; Glanz et al., 1994; Prochaska et al., 1994; Hellman, 1997). Simple and effective "stage-based" approaches derived from the stages of change model have demonstrated a widespread utility Additionally, brief interventions (a 5–10-minute exam room consult) are as effective as longer visits and result in significantly longer sustained behavioral change (Calfas et al., 1997).

Behavior change is generally not a discrete, single event. Physicians can sometimes see patients who, after experiencing a medical crisis and being advised to change the contributing behavior, readily comply. More often, physicians see patients who are unable or unwilling to change (Zimmerman et al., 2000). Recently, research has shown that behavioral change is a process of five identifiable stages through which people pass successively and that, for most people, a change in

behavior occurs gradually, with the patient moving from being uninterested, unaware, or unwilling to make a change (precontemplation), to considering a change (contemplation), to deciding and preparing to make a change (action). Physicians can facilitate movement through these stages by taking specific actions. Understanding this process provides physicians with additional tools to assist patients, who are often as discouraged as their physicians with their lack of change.

The stages of change model encompasses many concepts from previously developed models. The health belief model, the locus of control model, and behavioral models fit together well within this framework. During the precontemplation stage, patients do not consider change. They may not believe that their behavior is a problem or that it will negatively affect them (health belief model), or they may be resigned to their unhealthy behavior because of previous failed efforts and no longer believe that they have control (external locus of control). During the contemplation stage, patients struggle with ambivalence, weighing the pros and cons of their current behavior and the benefits of and barriers to change (health belief model – Janz and Becker, 1984). Cognitive behavioral models of change (e.g., focusing on coping skills or environmental manipulation) and 12-step programs fit well in the preparation, action, and maintenance stages. The five stages of change are described below:

1. Precontemplation: Patients do not consider changing. Overeaters who are "in denial" may not see that the advice applies to them personally. Patients with hypertension may feel "immune" to the health problems that strike others. Diabetic patients may have tried many times unsuccessfully to lose weight and change their diet habits, but have since given up.
2. Contemplation: Patients are actually ambivalent about changing. Letting go of an enjoyed behavior can result in a feeling of loss and depression or anxiety despite any promised gain. During this stage, patients assess the different and individualized risks and benefits of change.
3. Action: Patients prepare to make a specific change in the near future. They can experiment with small changes as the determination to make more and more change increases. For example, sampling two drinks per day instead of five may be an experimentation with a move towards reducing alcohol intake. Switching to a healthier diet, starting a blood pressure journal, or starting to walk a little for exercise are indicators that they have decided a change is needed. This is the stage that most physicians and other healthcare providers are most eager to get their patients to achieve. However, the provider's eagerness must be tempered and the interventions must be based in the stage where the patient is not in the stage the provider wants the patient in. For example, new year's resolutions broken in February provide evidence that, if the prior stages have been rushed through, the action stage is not enough to sustain.
4. Maintenance: Patients internalize new behavior as part of their lifestyle.
5. Relapse prevention: Discouragement over occasional setbacks may retard the change process and result in the patient giving up. Therefore, encouragement and recognition from the physician of the gains made by the patient are important.

When using motivational interviewing during brief interventions, it is helpful to consider the stage a patient currently is in regarding potential change (Prochaska et al., 1992). Physicians can carefully direct intervention efforts based on consideration of two important factors: (1) the severity of the alcohol problem; and (2) the patient's readiness to change drinking behavior (i.e., the patient's stage of change).

Well-intended advice, a practice all too familiar with physicians, works best with patients who are ready to prepare for change. Using direct persuasion with a patient who is too ambivalent risks generating patient resistance. Patient resistance is seen when the physician moves too fast with the patient in the change process. Note, however, an urgent intervention in patients at any stage when an immediate change is required, e.g., a pregnant woman who is still smoking, or a patient on anticonvulsant meds for traumatic brain injury (TBI) who continues to drink occasionally. In all circumstances, educate patients about the consequences of their substance use: "Smoking affects the developing fetus in this way. . ." The information on smoking, in this example, works best when it addresses the issue that directly concerns the patient. Therefore, using the medical diagnosis and the physical care plan as the basis for motivating the patient to change behaviors is essential. A lecture on the hazards of smoking, drinking, and drug abuse, separate from the medical care plan, is insufficient to create lasting and meaningful change.

Begin the interview with an attempt to understand how the behavior fits into the patient's life. This assessment is the complete intervention in patients who are in precontemplation stage. In the contemplation stage, physicians should discover the patient's ambivalence toward change, including risks and benefits of continued drinking. It is at this point that patients may be more receptive to information about alcohol's effects. In the later readiness stages, physicians can introduce patients to helpful community resources, including Alcoholics Anonymous

(AA) or specialty treatment programs, should the severity of their drinking warrant.

The overall goal to stage-based interventions is to help patients develop their own motivation for change and to guide them toward a healthier lifestyle. This guidance underscores the idea that behavioral change occurs incrementally rather than all at once and the physician can determine his/her patient's stage of change and provide an intervention appropriate to that stage for a best response. This intervention approach is non-directive and therefore removes much of the resistance the patient would otherwise have. Identifying patients' readiness to change through assessing the particular stage of change they are in allows them to do all of the work, including reflecting on the ways alcohol fits into their life, weighing the personal risks and benefits of continued drinking, providing the rationale for change and making the decision to stop drinking. The physician's job is simply to elicit information, encourage patients to reflect, and support their movement toward healthy change.

The wording of questions the physician asks during an intervention requires forethought because wording can bias reactions and responses. When precontemplative patients respond to questions, rather than quickly giving advice, reflect with empathy, instill hope, and empathetically point out discrepancies between patients' stated care plan goals and their statements. Asking patients, "Do you know this will ultimately kill you?" can be viewed as threatening and will result in greater resistance. However, asking patients, "How will you know that it's time to quit?" allows patients to be in charge of their lives and can help foster a meaningful thought process that extends beyond the exam room consult. Good questions from the physician inspirespatients to think about answers that are applied to them and advances them along the change process. Some examples of stage-based questions are shown below.

Precontemplation stage

Goal: To move the patient toward thinking about change

- "What would have to happen for you to know that this is a problem?"
- "What warning signs would let you know that this is a problem?"
- "Have you tried to change this in the past?"

Contemplation stage

Goal: To encourage the patient to examine the benefits and risks of change

- "Why do you want to change at this time?"
- "What were the reasons for not changing?"
- "What would keep you from changing at this time?"
- "What are the barriers today that keep you from change?"
- "What might help you with that aspect?"
- "What things (people, programs, and behaviors) have helped in the past?"
- "What would help you at this time?"

Patients who are in preparation stage begin to experiment with changing a behavior such as cutting down on smoking/drinking or starting to exercise or eat differently, and this is seen as transitioning into more decisive action. While continuing to explore ambivalence, intervention strategies need to shift from motivational to planned action-oriented behavioral skills specific to their stage of change or readiness. During the action and maintenance stages of change, physicians ask patients about their continued successes and challenges with praise, encouragement, and empathy as this can best reinforce the continuation of sustained healthy change.

REFERRAL TO TREATMENT

Patients who meet DSM criteria for an AUD or do not respond to a brief intervention may benefit from referral to an intervention specifically for alcohol use problems. There are many such specialty clinics, residential facilities, and intensive outpatient programs, but sometimes patients are unwilling or unable to participate in such programs often due, for example, to geographic or insurance restrictions.

This chapter focuses on just one of these options, namely AA, simply because it is the least expensive and most available and therefore the most utilized service for AUDs in the world.

AA is an organization founded in the mid-1930s by two confirmed alcoholics who decided to help each other get sober. They found that through mutual support and shared experiences they were able to remain abstinent from alcohol when all other treatments available at that time had failed. Today, AA boasts a membership of more than 4 million people from 400 different societies across the globe.

Despite its popularity, AA is not considered an "evidence-based treatment." This is not to suggest that AA does not work. Rather, AA is not considered "treatment" because it is not administered by professional healthcare providers. Second, it cannot be formally studied like other evidence-based treatments, in which people are divided into case and control groups, because by definition people who go to AA self-refer and cannot be assigned to groups. Nonetheless, more than three decades of accumulated evidence show that AA improves drinking outcomes and other markers of

health and well-being for those who actively participate, and the higher the level of participation, the better the outcome. AA involvement is a potent predictor of achieving and maintaining abstinence from alcohol (Emrick et al., 1993, Humphreys, 2004). AA participation also predicts improved psychologic and social functioning (Humphreys, 2004).

For effective referral of patients to AA, healthcare providers need to understand some fundamental facts about the organization, how it works, and which patients are appropriate for referral.

AA is an abstinence-oriented organization, which means that the goal for people in the group is to stop drinking, not "cut back." This approach is distinctly different from patients with alcohol use problems who are working to reduce drinking to healthy levels. The previous section, describing brief interventions for AUD, discusses the importance of establishing mutually agreed-upon goals. If the mutually agreed-upon goal between the healthcare provider and the patient with an AUD is to reduce drinking but not stop, then this patient would not be an appropriate referral to AA. On the other hand, the single criterion for membership in AA is the "desire to stop drinking," which means that patients who are still actively abusing alcohol but would like to stop would be appropriate for AA.

North American research has consistently found no relationship between AA affiliation and demographic variables such as age, social class, race, employment status, and parental socioeconomic status (Trice and Roman, 1970; Emrick et al., 1993). However, AA may work best for the most severely addicted (Trice and Wahl, 1958; Emrick et al., 1993). Therefore, the options for patients with the most refractory and severe alcohol use problems may indeed be AA. Often clinicians assume that a 30-day residential program is mandatory for those severely addicted to alcohol. There is no evidence to support this notion, and the costs of such a program are prohibitive for many people, hovering around $30 000 for a month's stay. AA has the advantage of helping individuals find recovery while still integrating the treatment into their everyday lives and without incurring the burden of high expense. Also, meetings can be found in almost every community center, church basement, or synagogue in almost every city in the United States. The absence of fees and the ubiquity of meetings reduce potential barriers to participation for patients who may not have health insurance or are otherwise limited in their ability to access care.

AA has a developed philosophy and program of change, outlined in a book titled *Alcoholics Anonymous*. The book was published in 1939 and is known universally by its members as *The Big Book*, because of the thick paper on which it was originally printed (Humphreys, 2004). AA views alcoholism as a chronic illness, from which one is never cured but remains in recovery. The core principles of AA are known as the 12 steps, and practicing them is called "working the program." Full sobriety is achieved when the individual is abstinent from alcohol and working the program, which, loosely summarized, consists of living out the ideals of honesty, humility, selflessness, and mindfulness. Although the achievement of abstinence alone is considered a central goal, it is not considered complete recovery in itself (Humphreys, 2004).

AA is not affiliated with any specific religion but does espouse the importance of spirituality and a higher power in the process of transformative healing. The higher power of AA is loosely defined and can consist of anything from a Christ-like deity to the ineffable mystery of the universe to the awe-inspiring support and acceptance of the AA fellowship. The spiritual aspect of AA can be an obstacle to participation for some would-be members. The use of the words "God" and "higher power" throughout the AA literature can be alienating for some persons who do not self-identify as religious. For these individuals in particular, it is essential to point out that God can be defined in any way the individual chooses and therefore need not interfere with membership.

Clinicians who refer patients to AA, or work with patients already active in AA, should monitor their progress in the organization, by asking them for example "What step are you working on now?" or "Are you working with a sponsor?" This communicates to patients that the provider is invested in their well-being outside of clinic time and reinforces a patient's attachment to a therapeutic community. It also helps monitor for relapse, because it has been shown that having a sponsor prevents against relapse (Witbrodt et al., 2012).

NEUROLOGIC MEDICAL COMORBIDITY

Physical examination

The physical examination does not provide much evidence that would suggest unhealthy alcohol use in the early stages of alcohol-related problems (Burge and Schneider, 1999). These patients may have mildly elevated blood pressure and few other abnormal physical findings. Later however, as the consumption and frequency of drinking increase, patients can develop significant signs of alcohol overuse, including gastrointestinal conditions such as an enlarged tender liver; cutaneous issues, including spider angiomata, varicosities, and jaundice; and neurologic signs, including tremor, ataxia, or neuropathies; and cardiac arrhythmias (Mersy, 2003).

Acute presentations

Alcohol intoxication is generally obvious, with the smell of alcohol, nystagmus, dysarthria, skin flushing, hypotension, and ataxia. Presentations depend on the blood alcohol concentration (BAC) but are individual-dependent. This individualized variation can be clinically informative. For example, the ability to maintain a conversation with a blood alcohol of 300 mL/dL indicates severe dependence; these individuals will exhibit withdrawal at alcohol levels associated with intoxication in non-tolerant individuals (i.e., around 100 mg/dL). In practice, treatment should be initiated when withdrawal symptoms occur, rather than waiting for the alcohol level to fall to near zero.

Quite common to emergency departments is the patient with reduced consciousness who reeks of alcohol. While this might be simple inebriation, exclusion of metabolic causes, poisoning, concomitant use of other drugs, or an underlying structural brain lesion needs to be considered. Alcoholics may have cerebral atrophy, predisposing them to subdural hematomas, and disordered coagulation, rendering them liable to intracerebral hemorrhage. These patients need a full examination, blood alcohol testing, and a computed tomography brain scan. Focal neurologic signs are not expected in alcohol intoxication, and a blood alcohol level of <200 mg/dL should not cause coma in the alcohol-tolerant individual. By comparison, serum ethanol levels for non-tolerant individuals, including binge drinkers, are as follows:

- Negative: no alcohol detected
- Lower limit of detection = 10 mg/dL
- >80 mg/dL (>17.4 mmol/L) is considered positive for driving under the influence in most states
- >300–400 mg/dL (65.1–86.8 mmol/L): potentially fatal

Blood alcohol concentration

To convert serum ethanol levels to BAC, move the decimal point three places to the left. For example, a 100 mg/dL serum ethanol level is equivalent to a 0.10 (g/dL) BAC, or 0.10% (weight/volume). This means that one-tenth of a percent of a person's blood volume is alcohol or that a person has 1 part alcohol per 1000 parts blood. Blood alcohol levels are easily quantified using a breathalyzer.

Alcohol withdrawal

Alcohol withdrawal occurs in those physically dependent on alcohol and is the result of the neuroadaptive compensatory changes that occur during prolonged exposure to alcohol's depressant effects (see Chapter 3). This neuroadaptive process represents the neurobiologic foundations of tolerance and includes the downregulation of γ-aminobutyric acid type A receptors and the upregulation of *N*-methyl-D-aspartate (NMDA) receptors (Kosten and O'Conner, 2003, Reoux and Oreskovich, 2006). If a physically dependent individual abruptly stops drinking, the inhibitory effects of alcohol are diminished, while the adaptive changes persist, resulting in NMDA-facilitated hyperactivity that generates alcohol withdrawal symptoms. The most common substance-related cause of seizures is alcohol withdrawal. Typically seizures occur 6–48 hours after the last drink and are generalized tonic-clonic, although partial seizures can also occur. They are usually self-limiting but may give rise to status epilepticus, where alcohol dependence accounts for 9–25% of cases (Daeppen et al., 2002; O'Brien et al., 2007).

Delirium tremens are not common and occur in about 5% of patients hospitalized for alcohol withdrawal. Alcohol-related seizures generally develop 24–72 hours after the last drink. Worsening agitation, distractibility, and delusions generally precede the onset, which is characterized by fluctuating disturbance of consciousness, changes in cognition, exacerbation of autonomic symptoms (sweating, nausea, palpitations, and tremor), and fear or terror (O'Connor, 2007). Persons with a history of hallucinosis (e.g., schizophrenia, bipolar disorders) have a greater susceptibility to delirium tremens than other alcohol-dependent cohort populations (Stanley et al., 2005).

Wernicke–Korsakoff syndrome

Alcohol withdrawal can precipitate Wernicke's encephalopathy. Wernicke's encephalopathy consists of psychomotor slowing or apathy, nystagmus, ataxia, ophthalmoplegia, impaired consciousness, and, if untreated, coma and death. Wernicke's encephalopathy results from inadequate intake or absorption of thiamine plus continued carbohydrate ingestion. Excessive alcohol intake interferes with thiamine absorption from the gastrointestinal tract and hepatic storage of thiamine; the poor nutrition associated with alcoholism often precludes adequate thiamine intake (Martin et al., 2004; Sechi and Serra, 2007). The disorder may remit with treatment, persist, or degenerate into Korsakoff's syndrome (KS), a late complication of persistent Wernicke's encephalopathy, and results in memory deficits, confusion, and behavioral changes. KS is an amnestic syndrome with impaired ability to consolidate new memories and relatively intact intellectual function, perhaps through an interruption of diencephalic–hippocampal circuitry, including between thalamic nuclei and mamillary bodies

(see Chapters 12, 12, and 16). KS occurs in 80% of untreated patients with Wernicke's encephalopathy. Wernicke–Korsakoff syndrome, which combines Wernicke encephalopathy and Korsakoff psychosis, occurs in some alcoholics who do not consume foods fortified with thiamine.

The risk factors and early manifestations of Wernicke's must be recognized and treated promptly and adequately with parenteral vitamins, notably, thiamine (B_1). Improvement in confusion usually occurs in 1–2 days, and in ocular abnormalities in days to weeks, while ataxia usually responds within the first week but can take months or much longer to resolve. Treatment consists of thiamine and supportive measures. Loading carbohydrates in patients with thiamine deficiency (i.e., refeeding after starvation or giving intravenous (IV) dextrose-containing solutions to high-risk patients) can trigger Wernicke's encephalopathy. Ensuring that dietary supplies of thiamine are adequate is important regardless of symptoms. Because IV glucose can worsen thiamine deficiency, alcoholics at risk of thiamine deficiency should receive IV thiamine 100 mg before receiving IV glucose solutions (O'Connor, 2013). Patients rarely have truly discrete deficits in forming new memories, generally exhibiting more global deficits along a spectrum of severity. KS is treatable and only about 25% of patients show no recovery.

KS can also present without a definable episode of Wernicke's, developing insidiously or following undiagnosed subacute episodes. This has led to prescribing oral thiamine supplements to chronic alcoholics. Magnesium is a cofactor for many thiamine-dependent enzymes, and deficiency may induce clinical signs of thiamine deficiency (Meier and Daeppen, 2005).

Pellagra

Niacin (vitamin B_3, nicotinic acid) and/or tryptophan deficiency results in skin, gastrointestinal, and mental abnormalities that often progress to memory impairment, delusions, hallucinations, dementia or delirium. Hypertonus and startle myoclonus may be present. Symptoms usually improve with nicotinic acid or nicotinamide (amide of vitamin B_3) replacement therapy (Kumar, 2010; So, 2012).

Blackouts (retrograde amnesia)

Blackouts are periods of amnesia during and after episodes of heavy alcohol consumption. They are similar to episodes of transient global amnesia (which occur in the absence of alcohol consumption) and are attributed to alcohol inhibition of NMDA receptor and impairing long-term potentiation (Eichenbaum, 2002, Hartzler and Fromme, 2003; White et al., 2004).

Central pontine and extrapontine myelinolysis

Hyponatremia is common in alcoholics, especially beer drinkers, due to hyperhydration of fluid intake. It is best treated by restoring normal hydration and diet while abstaining from alcohol. Attempts to correct the electrolyte disturbance with saline (particularly hypertonic saline) may result in demyelination, thought to be triggered by rapid osmotic shifts in the brain causing complement-mediated oligodendrocyte toxicity. This most commonly occurs in the pons (central pontine myelinolysis) but it can also occur elsewhere (extrapontine myelinolysis); the basal ganglia and thalamus are vulnerable (National Institute of Neurological Disorders and Stroke, 2012). If the pons is primarily affected, symptoms include dysarthria, dysphagia, and spastic quadriparesis. Lesions outside the pons have a much more variable presentation, which can include mutism, parkinsonism, dystonia, and catatonia (Kitabayashi et al., 2007; Yoon et al., 2008; Schneider et al., 2012).

Neuropathy

AUDs have been associated with what has been called the "Saturday-night palsy" due to compression of the radial nerve in deep or stuporous sleep (akinesic and mute). Chronic alcoholics can develop a symmetric, bilateral mixed sensory and motor peripheral neuropathy, usually of the lower limbs (Harati and Bosch, 2008). Patients may be asymptomatic or present with pain, numbness, burning feet, and hyperesthesia. This neuropathy is usually attributed to thiamine deficiency, although direct alcohol toxicity (Shy, 2007) may also contribute. Research suggests that, if thiamine deficiency is the predominant cause, the symptoms are motor, whereas direct alcohol toxicity produces more of a sensory-dominant presentation (Koike et al., 2003; National Institute of Neurological Disorders and Stroke, 2012). Recovery occurs with abstinence and thiamine supplements can be used. Chronic cerebellar ataxia affects about one-third of the chronically alcohol-dependent. It is related to a combination of malnutrition and alcohol's direct toxic effects. The ataxia consists of instability of gait and stance with severe lack of coordination of the knee–shin test, and relatively little involvement of the arms. In severe cases, there is evidence of neural degeneration in the anterior and superior portions of the cerebellar vermis with extension into the anterior lobes and flocculi (Shy, 2007).

Cognitive impairment

Alcohol-related brain damage has no single cause. Thiamine deficiency underlies KS on one hand, yet the

alcohol-dependent person repeatedly has episodes of drunkenness with subsequent alcohol withdrawal, dietary neglect and malnutrition, severe vitamin deficiencies, TBI, cerebrovascular events, and alcoholic liver disease, all of which can contribute to impairment in brain structure and function. Whether KS or not, abstinence and an enhanced nutritional focus are central to management. Alcohol-related cognitive deficits generally improve with abstinence.

A note about alcohol use as a risk factor for traumatic brain injury or recovery from TBI

Alcohol use and TBI are often closely related. Up to two-thirds of people with TBI have a history of alcohol abuse or risky drinking. Between 30% and 50% of people with TBI were injured while they were drunk and about one-third were under the influence of other drugs. Around half of those who have a TBI cut down on their drinking or stop altogether after injury, but some people with TBI continue to drink heavily, which increases their risk of having negative outcomes (Bjork and Grant, 2009).

In epidemiologic surveys, emergency room patients have reported elevated incidence of recent alcohol use (Cherpitel and Ye, 2008) and have also shown high incidence of actual presence of alcohol or other drugs at the time of injury (Vitale and van de Mheen, 2006). Moreover, alcohol abuse can exacerbate the effects of TBI. Jorge et al. (2005) reported that alcohol abuse or dependence following TBI impaired vocational outcome, possibly by exacerbating the neurologic sequelae of the injury itself. Similarly, Corrigan (1995) reported that substance abuse following TBI portended a poorer trajectory of rehabilitation following injury. In a sample of over 1600 TBI subjects, Horner et al. (2005) reported that a pre-existing substance abuse diagnosis at time of injury was predictive of heavy drinking following the injury. Therefore, screening and brief intervention may be especially important for TBI patients as well as education on TBI recovery for families.

CONCLUSION

In this chapter we reviewed the components of SBIRT as a way of targeting AUD in an ambulatory care setting, specifically the neurology clinic, although the information herein can be integrated into most primary care settings. We emphasize that AUDs exist on a continuum from normative use, to risky use, to a use disorder. Within the subset of pathologic use, the new *Diagnostic and Statistical Manual* further subdivides the spectrum of use disorders into mild, moderate, and severe, based on compulsive use, out-of-control use, and continued use despite consequences. Frequency and quantity of alcohol consumption should be considered the fifth vital sign, and an important starting point for a discussion about alcohol use within the ambulatory care setting. Data show that brief interventions for at-risk or pathologic use can be effectively implemented in 5–10 minutes and should involve exploring the patient's ambivalence about change, understanding the patient's motivation to change, and adjusting the intervention to meet each individual's needs. Patient and provider should agree on a goal and strive toward it, whether that goal is reducing alcohol consumption, reducing risky behaviors associated with alcohol consumption, or abstinence. Healthcare providers can effectively limit medical consequences of chronic heavy alcohol use by screening and intervening for the AUD itself.

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