Addiction *in the* Older Patient

Edited by
MARIA A. SULLIVAN • FRANCES R. LEVIN

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CHAPTER 1

Introduction

Maria A. Sullivan and Frances R. Levin

Scope of the Problem

Alcohol and drug use among older or elderly patients has received relatively little attention, either as a clinical focus, or as a research initiative, to date. This apparent neglect of a critical cohort of affected individuals may be partly explained by prevailing cultural biases among both family members and practitioners, which serve to minimize the perceived extent of the scope of the problem among older adults. Ageism contributes to a pattern of underdiagnosis, in that behavior considered a problem in younger adults does not engender the same urgency for care in older adults (SAMHSA 1998). Primary care physicians as well as specialists do not routinely address or screen older adults for substance use disorders (SUDs) (Rothrauff et al. 2011). But it is also important to recall that evidence for best treatment practices in this older population is lacking, since most clinical research trials specifically exclude older participants. The vast majority of studies exclude individuals older than 65, and indeed, many trials exclude anyone age 60 or older. Thus, our ability even to identify SUDs in the older population relies on extrapolation from younger cohorts. At present, the field of addiction treatment is lacking in data on the clinical presentation and course of illness in older individuals. In particular, we are in need of both sensitive diagnostic instruments and specific prevention and treatment strategies focused on the characteristics of older adults with SUD.

The problem of limited clinical knowledge regarding the phenomenology of alcohol and substance use disorders in later life is rendered more acute by the changing demographics of the U.S. population. Over the next decade, the cohort of aging adults will continue to grow at a rapid rate—and this will pose a significant challenge for the field of addiction treatment. It is estimated that the number of individuals age 65 or older will grow from 40 million in 2010 to 55 million in 2020 (U.S. Census Bureau 2009). And by 2030, there will be

about 72.1 million older adults (representing 19.3% of the total population). This considerable increase in older adults will constitute almost a doubling since 2008 (United States Census Bureau 2008). Moreover, the cohort of aging adults is continuing to use alcohol and psychoactive prescription medications at a higher rate than previous generations did (Blow and Barry 2012). For instance, about 50% of adults aged 65 and older and about 25% of individuals over 85 years old drink alcohol (Caputo et al. 2012). Thus, a considerable proportion of the aged 65+ population are at potential risk for the development of alcohol use disorder (AUD). This risk is heightened by the increased effects of ethanol on the central nervous system in the elderly, for whom reduced activity of gastric and liver antidiuretic hormone (ADH) leads to the elevation of blood alcohol levels by up to 25% (Lieber 2005).

Along with alcohol, prescription drugs are the most frequently abused substance by older Americans (Weintraub et al. 2002). Approximately one-third of all prescription drugs in the United States are used by older (age 65+) adults (National Institute on Drug Abuse [NIDA] 2005). Polypharmacy is also common among older adults, who tend to have multiple underlying medical disorders (Ballentine 2008). It is estimated that a rising proportion of older adults will experience prescription SUD because of the aging cohort, and increased accessibility of prescription drugs (Dowling et al. 2008).

Varying definitions of "older adult" appear in the literature, ranging from 50+ years to 65+ years old. The broader age-based definition of older adults reflects the fact that, among individuals with SUDs, there is an accelerated rate of biological aging to a higher medical burden. Furthermore, up to 30% of older patients hospitalized medically and up to 50% of those hospitalized psychiatrically present with AUDs (Blazer and Wu 2009). Yet, for many older adults, a diagnosis of addiction is missed. Accurate assessment of the prevalence of AUD and SUD among older adults in the community is, in fact, hampered by a number of factors.

Challenges to Identifying Addictive Disorders in Older Adults

The misuse of alcohol or other substances is often a hidden phenomenon, because family members, friends, and employers are not available to notice the types of changes in behavior or personality by which AUD or SUD is frequently identified in younger persons (Johnson 1989). Retirement effectively eliminates the observational aspects of relationships with co-workers and supervisors (Boeri et al. 2008). Alcohol or substance use disorders in the older adult may also be difficult to identify because many screening instruments

measure the presence of legal, social, and work-related problems (Zimberg 1984, Johnson 1989). In addition, older adults are often reluctant to seek help for addiction because of the perception of its stigma and shame, which is stronger among older individuals than among younger persons (Oslin et al. 2005).

Another factor that often confounds clinicians is the co-occurrence of multiple medical problems in older patients. Concurrent neurological, cardiac, or gastrointestinal disorders can mimic or mask the acute effects of alcohol or other substances, as well as the withdrawal syndromes associated with alcohol, benzodiazepines, stimulants, or opioids. The 2005–2007 National Survey on Drug Use and Health (NSDUH) found that, among 10,015 respondents aged 50–64 years and 6,289 respondents older than 65 years, "diagnostic orphans" among middle-aged and elderly community adults showed an elevated rate for both binge drinking and the non-medical use of prescription drugs, requiring attention from healthcare providers (Blazer and Wu 2011). Thus, alcohol or prescription misuse in older adults is often missed when strict diagnostic (*Diagnostic and Statistical Manual of Mental Disorders*, 5th Ed. [DSM-5]) criteria for alcohol or substance use disorders are invoked. This text will highlight some of the risk factors and signs of addictive disorders in older individuals that are frequently overlooked.

Risk Factors in Older Age, and Projected Trends

The National Survey on Drug Use and Health (NSDUH 2004) found that, of individuals aged 50 or older, 12.2% were heavy drinkers, 3.2% were binge drinkers, and 1.8% used illicit drugs. The 2005–2006 NSDUH revealed a relatively high level of binge drinking in men (14%) and women (3%) over the age of 65 years (Blow 1998). Historically, the use of illicit drugs has been relatively rare in older adults. Based on the 2004 NSDUH, among individuals aged 50+ years, 1.8% used illicit drugs (Office on Applied Statistics [OAS] 2005, Huang 2006, Colliver et al. 2006). But by 2020, use of any illicit drug by this cohort is estimated to increase from 2.2% (1.6 million) to 3.1% (3.5 million), and nonmedical use of prescription drugs (opioids, sedatives, tranquilizers, and stimulants) is projected to increase from 1.2% (911,000) to 2.4% (2.7 million). Analysis of the Baby Boom generation in the National Health and Nutrition Examination Survey (NHANES 2012) data found that this cohort is continuing to maintain a higher level of alcohol consumption than did previous older-age cohorts. Moreover, the NSDUH (2012) found that past-month illicit drug use in the aged 55–59 group increased by 50%, from 4.1% (2010) to 6.0% (2011) (Substance Abuse and Mental Health Services Administration [SAMHSA] 2012). In addition to lifetime marijuana use, recent use has

continued to rise over the last decade (National Institue on Alcohol Abuse and Alcoholism [NIAAA] 2015). It is expected that the problems of morbidity and mortality related to illicit SUDs will also continue to increase.

Several studies have identified social isolation or loneliness as a risk factor for psychotropic drug misuse among older persons (Jinks et al. 1990, Nubukpo and Clement 2013). Other risk factors for the development of psychoactive medication misuse include: (1) being female, (2) having a history of a psychiatric disorder or a prior SUD, (3) higher levels of psychosocial distress, and (4) poorer functioning (Simoni-Wastila and Yang 2006, Jinks et al. 1990). Additionally, symptoms of pain, anxiety, or sleep disturbance (Patterson et al. 1999, Schonfeld et al. 2009) increase the likelihood of psychoactive prescription misuse.

Treatment Considerations

There is a substantial body of evidence that motivational brief interventions, delivered by a variety of healthcare providers, can reduce at-risk drinking, alcohol misuse or alcohol consumption among both younger (Babor et al. 1992, Fleming et al. 1997, Wallace et al. 1988) and older adults (Fleming et al. 1999, Lin et al. 2010, Moore et al. 2011). Lin and colleagues found that early reductions in at-risk alcohol use were associated with the following: older adults' concerns about risks, reading educational materials, and the perception of physicians providing advice to reduce drinking. Two large-scale effectiveness studies have examined the implementation of such brief intervention trials in older adults: the Primary Care Research in Substance Abuse and Mental Health for the Elderly study (PRISM-E; Oslin et al. 2006) and the Brief Intervention and Treatment for Elders (Florida BRITE) project (Schonfeld et al. 2010). These two trials identified several barriers to implementation of these brief interventions: (1) stigma from the perspective of older patients; (2) lack of training for healthcare professionals in screening and brief interventions; (3) chronic medical conditions in older adults, which may make it more difficult to identify the role of AUD or SUD in decreased functioning or quality of life; and absent or low reimbursement for providing this service (Blow and Barry 2012).

Certain modifications to a standard pharmacotherapy regimen may need to be made when treating older adults. For instance, detoxification from alcohol or drugs may need to be carried out over a longer period of time (Johnson 1989), and the incidence of medical (e.g., myocardial ischemia, arrhythmias, orthostatic hypotension) and neurological (e.g., delirium tremens, dizziness, seizures) problems is higher in elderly patients than in their younger

counterparts (Letizia and Reinbolz 2005). Unfortunately, insurance carriers, including Medicare, do not always recognize that physiological needs of aging patients may require that they remain hospitalized for a longer period of time (Douglass 1984). In addition, detoxification regimens may need to be adjusted to avoid overmedicating-withdrawal symptoms. And disulfiram (Antabuse) is not recommended in older adults, many of whom have cardiac arrhythmias or pulmonary disease, because of the risk of adverse effects (Lamy 1988, Council on Scientific Affairs 1996). On the other hand, difficulties with memory may recommend the use of a monthly long-acting injectable formulation of naltrexone (Vivitrol), in preference to the oral form often used in younger individuals seeking treatment for AUD (Caputo et al. 2012).

And finally, special services for older adults are largely unavailable at present. Using nationally representative cross-sectional data from 346 private substance treatment centers, Rothrauff et al. (2011) found that only 18% provided age-specific services. In both inpatient and outpatient settings, older adults can be expected to relate more readily to groups of their peers who have experienced similar challenges, such as loss of spouse, contraction of social network, loss of physical vigor, and declining cognitive and sexual function. An expert panel commissioned by SAMHSA (1998) offered specific SUD treatment approaches with older adults. These include: engaging in nonconfrontational treatment; focusing on (re)building self-esteem; teaching skills to cope with depression, loneliness, and loss; focusing on (re)building social networks; tailoring content and pace of presentation toward older adults; hiring staff who are interested in and experienced with working with older adults; and providing linkages with medical services and community-based services. Rothrauff et al. (2011) also recommend wraparound services to address older adults' special needs, such as inadequate availability of primary care, housing assistance, and transportation to and from treatment.

Conclusions

The incidence of alcohol and substance use disorders in older adults is fairly high and certainly underestimated. Addictive disorders in middle-aged and older patients are often undetected in the clinical setting and rarely examined from a research perspective. Symptoms of AUD or SUD overlap with those of other medical conditions prevalent in this population, and the kinds of distress engendered by SUDs in later life frequently evade detection on standardized assessments of work or social functioning. Older patients themselves are often reluctant to reveal SUDs because of the shame and stigma associated with addiction, which is heightened in this generational cohort.

The field of addiction treatment has for too long overlooked the needs of older adults struggling with addictive disorders. An emerging literature on tailored treatments for this population suggests the benefits of brief interventions, cognitive-behavioral therapy, and age-specific treatment services in both the inpatient and outpatient settings. Given the opportunity to benefit from these treatments, older patients have the capacity to demonstrate outcomes at least as successful as those in younger individuals.

The goal of this book is to review the evidence-based literature on addiction in the older patient. We will examine each of the major classes of substances, both prescribed and illicit, and offer guidelines for the accurate assessment and effective treatment of alcohol and substance use disorders in this vulnerable and rapidly growing population. We will consider diagnostic challenges that arise from cultural or practitioner bias against older patients, as well as medically related or atypical presentations of alcohol or substance use disorders in older adults, including unique challenges faced by older women challenged by addiction. And we will explore the role for both traditional and technology-based screening and brief interventions, as well as the importance of developing and implementing effective treatment strategies tailored to the needs of an older population. It is our hope that clinicians will find this text useful in helping them identify individuals at risk for, or actively engaged in, addictive disorders, and that the recommendations set forth here may help frame and guide clinical interactions with older patients who could benefit from timely treatment interventions in this domain. Above all, this book is offered as a practical handbook of useful clinical information to aid clinicians in increasing their awareness of, attention to, and skills in assessing and treating, addiction in our older patients.

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CHAPTER 2

Recognizing Addiction in Older Patients

Anna Levesque and Edward V. Nunes

Introduction

Substance use disorder (SUD) is often perceived as a problem affecting mainly adolescent or younger adults. However, its increasing prevalence in the geriatric population represents a significant public health concern (Simoni-Wastila et al. 2006, Han et al. 2009). Various factors, such as widowhood, divorce, isolation, poor health status, depression, and anxiety, may predispose older adults to initiate or to persist in using psychoactive substances (Taylor et al. 2012, Aira et al. 2008, Jinks et al. 1990, St. John et al. 2009, Brennan et al. 1990, Fink et al. 1996). According to the 2012 National Survey on Drug Use and Health (NSDUH), 2% of people 65 and older reported alcohol dependence or abuse (SAMHSA 2013a). In addition, nearly 22% of American older adults report using at least one psychoactive medication, and it is estimated that there will be a 100% increase in medication misuse between 2001 and 2020 in this population (1.2–2.4%) (Simoni-Wastila et al. 2005, Colliver et al. 2006). An increasing number of older patients report addiction treatment, and emergency department visits related to SUDs, further indicative of this growing problem (SAMHSA 2010). Although alcohol and psychoactive medication are the substances most frequently used among the elderly, the prevalence of illegal drug use is increasing with the aging of the baby-boomer generation, who are more likely than previous generations to bear the risk factor of having experimented with illicit drugs in their youth (Koechl et al. 2012).

Recognizing the signs of SUDs in elderly is not always easy, as they may manifest in subtle and confusing ways. Clinical indicators interpreted as warning signs of substance use in younger patients (for example, cognitive impairments, unsteadiness of gait, insomnia, or social isolation) are symptoms

that may be indicative of other common medical or psychiatric problems frequently encountered among aging adults, making diagnosis more challenging (Mulinga 1999, Lang et al. 2007). Social stereotypes, such as the false assumption that older adults do not suffer from SUDs, may also contribute to misidentification of such conditions by decreasing suspicion by families and of healthcare providers (Naik et al. 1994). As a result, the problem often remains unrecognized by doctors and family members, preventing patients from receiving appropriate treatment. In addition, most clinical screening and diagnosis tools were designed and validated for younger populations and are not always adapted to the elderly, which also limits problem recognition (Aalto et al. 2011, .O'Connell et al. 2004, Graham 1986).

With age, metabolism of alcohol and other substances is slower, leading to higher blood levels and stronger effects. Pharmacodynamics of alcohol and other substances may also change with age. Misrecognition of SUDs in the elderly is especially concerning, given that older individuals are more vulnerable to suffering serious complications from their use. Concerns of family and friends are the most common causes for elderly patients seeking consultation for SUDs (Finlayson et al. 1988). Primary care providers also play a key role in the prevention, screening, and treatment of such disorders. Increasing families' and healthcare providers' awareness of the different ways SUDs can present in older adults could significantly improve health outcomes in this population.

DIAGNOSIS OF SUBSTANCE USE DISORDER

The Diagnostic and Statistical Manual of Mental Disorders (DSM) includes the criteria most widely used in North America to diagnose SUDs. In the fifth edition (DSM-5), "Substance Use Disorder" is defined as a problematic pattern of substance use leading to clinically significant impairment or distress, as manifested by two or more of the 11 criteria listed in Box 2.1, within a 12-month period (APA 2013). SUD is classified as mild, moderate, or severe according to the number of fulfilled criteria.

These criteria have lower sensitivity in geriatric populations, as some of the diagnostic criteria do not apply well to older patients (Blow et al. 2014). For example, metabolic changes associated with aging result in higher sensitivity to the effects of substances such as alcohol, benzodiazepine, and opioid following lower consumption amounts (Vogel-Sprott et al. 1984, Vestal et al. 1977, Atkinson 1990, Greenblatt et al. 1991, Greenblatt et al. 1982, Moore et al. 2007, Blow 1998). Hence, an older patient with a problematic pattern of substance consumption may not necessarily spend a great deal of time in activities related to substance use and may not develop tolerance or withdrawal, as

Box 2.1 **Diagnostic Criteria for Substance Use Disorder According to the 5th Edition of the DSM (APA 2013)**

1. The substance is often taken in larger amounts or over a longer period than was intended.
2. There is a persistent desire or unsuccessful efforts to cut down or control substance use.
3. A great deal of time is spent in activities necessary to obtain the substance, use the substance, or recover from its effects.
4. There is a craving, or a strong desire or urge, to use the substance.
5. Recurrent substance use results in a failure to fulfill major role obligations at work, school, or home.
6. Substance use is continued despite persistent or recurrent social or interpersonal problems caused or exacerbated by the effects of the substance.
7. Important social, occupational, or recreational activities are given up or reduced because of substance use.
8. There is recurrent substance use in situations in which it is physically hazardous.
9. There is continued substance use despite patient's knowledge of having a persistent or recurrent physical or psychological problem that is likely to have been caused or exacerbated by the substance.
10. Tolerance develops, as shown by a diminished effect when using the "usual" amount, or a need for increasing amounts to achieve the desired effect.
11. Withdrawal symptoms follow efforts to quit.

Mild = Two to three symptoms; **Moderate** = Four to five symptoms; **Severe** = Six or more symptoms

suggested in the DSM-5 criteria. For example, an older patient might present with a pattern of drinking that has not changed over the years, and that was not a problem when he was younger. Thus, there do not appear to be escalation of use, tolerance, withdrawal, or loss-of-control features. However, the patient may have substantial alcohol-related impairments (e.g., insomnia, gastrointestinal [GI] problems, or interpersonal problems) that might erroneously be attributed to other medical or psychiatric conditions. Also, the elderly may show fewer of the behavioral disturbances or social "red flags" typically found in younger adults with addictions (Graham 1986). Therefore, criteria regarding

reduced activities and failure to fulfill obligations may not apply to retired patients with a decreased baseline level of daily activities. Alternative definitions, such as hazardous drinking and medication misuse, may be more helpful in identifying patients with less severe problematic patterns of substance use that do not fulfill SUD diagnostic criteria (Fink et al. 1986, WHO 1992, SAMHSA 2013b). Or, a patient may simply present with a medical condition (e.g., GI distress, insomnia) that is being caused or exacerbated by alcohol or another substance without a use disorder per se (e.g., if the patient is able to cut down or quit upon advice from the physician without difficulty).

Alcohol

NORMAL ALCOHOL METABOLISM

There are multiple pathways involved in alcohol metabolism, each of which creates metabolites that contribute to its toxicity. Alcohol is first metabolized into acetaldehyde, mainly by the enzyme alcohol dehydrogenase (ADH) located in the stomach and in the liver (Auty et al. 1977, Kaplowitz et al. 2007). To a lower extent in non-chronic users, enzymes from the cytochrome P4502E1 located in the liver are also involved in this reaction (Lieber 2004). The acetaldehyde generated from this first reaction is then metabolized into acetate through the action of acetaldehyde dehydrogenase (ALDH), an enzyme located in the liver.

Following its ingestion, alcohol metabolism starts in the stomach, where there is a significant amount of ADH (Auty et al. 1977). Remaining alcohol is slowly absorbed from the stomach, and then, more rapidly, from the small intestine. Once it enters the bloodstream, alcohol is directed throughout the portal vein to the liver, where the majority of the metabolism takes place. The first phase of alcohol metabolism that follows ingestion and absorption from the digestive system is called the *first-pass metabolism*, which eliminates about 10% of the absorbed alcohol (Weathermon et al. 1999). Residual alcohol from the first-pass metabolism is redistributed into the body and eventually returns to the liver, where metabolism is subsequently repeated. First-pass metabolism is less efficient in women, who have lower amounts of ADH in the stomach than men do, leading to higher levels of circulating alcohol following the ingestion of an equivalent amount (Frezza et al. 1990, Thomasson 1995).

ALCOHOL METABOLISM IN THE ELDERLY

Aging modifies the metabolism and distribution of alcohol through different mechanisms, in addition to increasing the sensitivity of certain organs to its

toxicity. Those physiological changes are clinically meaningful because they have an impact on clinical presentation, screening, diagnosis, and treatment of alcohol use disorder (AUD) in this population.

The amount of ADH enzymes in the stomach naturally decreases with aging (Seitz et al. 1993). Common medical conditions found among the elderly, such as atrophic gastritis and *Helicobacter pylori*, also reduce the amount of gastric ADH, as these conditions are associated with the reduction in the number of mucosal cells containing the enzymes (Thuluvath et al. 1994). The quantity of ADH is further compromised in older women, who already have a lower baseline gastric level of ADH than men (Frezza et al. 1990, Thomasson 1995). This decline in the available number of metabolic enzymes reduces first-pass metabolism efficacy, which in turn leads to higher blood alcohol levels compared to those in younger adults following an equivalent consumption. Reduced hepatic blood flow has been observed in older adults, but it is unclear whether or not it contributes to the decline of first-pass metabolism (Moore et al. 2007, Seitz et al. 2007, Durnas et al. 1990). Aging also causes an increase in the proportion of body fat and a decrease in the proportion of body water (Vogel-Sprott et al. 1984, Vestal et al. 1977). Given that alcohol is mostly distributed in the aqueous space, alcohol volume of distribution decreases with aging, hence increasing blood concentration (Vogel-Sprott et al. 1984, Vestal et al. 1977). Furthermore, increased central nervous system sensitivity to the effect of alcohol has been described among older adults (Moore et al. 2007). The cumulative effect of those changes may lead older adults to experience marked intoxication symptoms following the ingestion of amounts of alcohol that would be judged safe among younger adults, making the screening and the diagnosis of problematic alcohol use more challenging.

CLINICAL PRESENTATION

Recognizing AUD in the elderly is particularly challenging because many of the associated symptoms are similar to those of other medical conditions frequently encountered among older adults. In addition, alcohol intake may exacerbate preexisting health problems or contribute to the onset of common diseases in the elderly population (Moore et al. 2007). Hence, it is essential for clinicians to maintain a high level of suspicion when assessing older adults, as symptoms evocative of AUD can easily be confused with common geriatric conditions. Screening for AUD should be performed when older patients present with deterioration of a chronic disease, new onset of a condition potentially associated with alcohol use, deterioration or new onset of a cognitive or a psychiatric disorder, or decreased effectiveness of a pharmacological

treatment (Moore et al. 2007, Caputo et al. 2012). In the following paragraphs, different possible clinical presentations of AUD among the elderly will be discussed.

Heavy alcohol intake has toxic effects on different organs of the digestive system. It is estimated that about 90–100% of chronic heavy drinkers eventually develop alcoholic fatty liver, 30% develop alcoholic steatohepatitis, and 10–20% ultimately develop cirrhosis (Meier et al. 2008). Given certain modifications in alcohol distribution and metabolism, older adults are more likely to suffer from alcoholic liver disease (ALD) and to experience complications such as portal hypertension, ascites, and esophageal varices (Seitz et al. 2007, Potter et al. 1987). Although the clinical presentation of ALD is similar to that encountered in a younger population, older adults more frequently report general malaise and anorexia (Woodhouse et al. 1985). The risk of upper GI bleeding is higher among older alcohol users, given the lower number of parietal cells associated with aging, a change that predisposes them to gastritis and ulcers (Menninger 2002). Furthermore, AUD is a common cause of acute and chronic pancreatitis, chronic diarrhea, and electrolyte imbalances (Bode et al. 2003, Kristiansen et al. 2008).

Consumption of large quantities of alcohol can lead to malnutrition, including primarily depletion of folic acid, vitamin B-6 (pyridoxine) and vitamin B-1 (thiamine), due to poor dietary intake and decreased nutrient absorption (Bode et al. 2003, Cabre et al. 2001, Fonda et al. 1989, Vech et al. 1975). Folic acid deficiency is found among 60–80% of alcoholics and can cause macrocytic anemia and intestinal malabsorption (Markowitz et al. 2000). There are rare complications of pyridoxine depletion, including peripheral neuropathy, stomatitis, glossitis, cheilosis, irritability, confusion, and depression (Markowitz et al. 2000, Cook et al. 1997). Deficiencies in thiamine occur in 30–80% of chronic drinkers and can lead to peripheral neuropathies, cardiomyopathies, and Wernicke-Korsakoff syndrome (Markowitz et al. 2000, Thomson et al. 1987). A triad of oculomotor abnormalities, ataxia, and delirium is characteristic of Wernicke's encephalopathy. If it remains untreated, this condition can progress to Korsakoff's syndrome, a dementia that is characterized by anterograde and retrograde amnesia and confabulation. Data on the role of alcohol in the development of dementia are mixed (Moriyama et al. 2006). The prevalence of dementia is estimated to be five times higher among the elderly with chronic alcohol use than among non-drinkers (Caputo et al. 2012). Also, about 25% of people with dementia have a comorbid AUD (Oslin et al. 1998). A direct neurotoxic effect of alcohol is thought to be associated with brain atrophy and overall cognitive impairments, although this concept remains under debate (Moriyama

et al. 2006). Aging reduces the number of brain cells in the basal ganglia, the neocortex, the reticular activating system, and the hippocampus—leading to higher risk of delirium during intoxication or withdrawal from alcohol (Menninger 2002). In the clinical context of acute confusion in the elderly, it is important to consider delirium tremens among the differential diagnosis, as it is a potentially life-threatening condition.

Although light alcohol intake has been shown to have some cardiovascular benefits (Ronksley et al. 2011), chronic heavy drinking can deleteriously impact various risk factors of cardiovascular disease, increasing risk for hypertension, glucose metabolism abnormalities, and truncal obesity with increased waist circumference (Caputo et al. 2012). Older adults suffering from AUD are also at higher risk of stroke and of dilated cardiomyopathy, which may lead to ventricular dysfunction and heart failure (Piano 2002).

Alcohol use also impacts the endocrine system, notably through alterations in the hypothalamic-pituitary axis (Caputo et al. 2012). High cortisol levels have been described among chronic alcohol users, causing pseudo-Cushing syndrome, whose clinical presentation is hardly distinguishable from that of the primary form of Cushing (Newell-Price et al. 2006). Alcohol also inhibits antidiuretic hormone, which leads to increased diuresis that can exacerbate or cause incontinence problems (Menninger 2002). In addition, AUD is associated with poorer control of diabetes, and it may also increase risk of hypoglycemia due to an inhibition of gluconeogenesis (Moore et al. 2007, O'Keefe et al. 1997, Yki-Jarvinen et al. 1988).

AUD is a significant risk factor for accidents, falls, and bone fractures in the geriatric population (Caputo et al. 2012). Consequences of drinking such as confusion, ataxia, balance problems, orthostatic hypotension, neuropathies, and myopathies predispose patients to accidents and falls (Moore et al. 2007, Blow 1998). In addition, lower bone density is common among older patients with AUD, especially when it is combined with other risk factors for osteoporosis (i.e., tobacco smoking), leading to increased risks of fractures (Bikle et al. 1985, Israel et al. 1990).

Alcohol is a dose-dependent risk factor for the development of multiple tumors, including those of the oropharynx, larynx, esophagus, liver, colon, rectum, prostate, and breast (Thun et al. 1997, Zhu et al. 2014, Gong et al. 2009). Chronic alcohol consumption also significantly reduces the level of T and B lymphocytes, leading to an increased susceptibility to infectious diseases (Girard et al. 1987). Bacterial pneumonia or reactivation of latent tuberculosis can be consequences of a decline in the immune system. Finally, alcohol consumption in the elderly often leads to suboptimal control of chronic diseases such as hypertension, diabetes, gout, or epilepsy because of poor

compliance with treatment and directly deleterious effects of alcohol on these diseases (Moore et al. 2007, Blow 1998, Kerr et al. 1990).

PSYCHIATRIC COMORBIDITIES

Older adults with any SUD have high rates of comorbid psychiatric disorders, estimated between 21% and 66% (Blow et al. 2014). Predominant psychiatric diagnoses in the elderly are depressive disorders, generalized anxiety disorder, alcohol dependence, dementia, and bipolar disorders (Seby et al. 2011). SUD and mental health disorders have a dynamic effect on one another, as substance use increases the risk of experiencing symptoms of mental illness, which in turn can lead to substance use as a form of self-medication (USDHHS 2010). In addition, both SUD and other mental illnesses can be caused by overlapping factors such as genetic vulnerabilities and early exposure to trauma (USDHHS 2010).

Many seniors report drinking in response to psychosocial triggers such as loneliness or depressed mood (Schonfeld et al. 1991). Depression is not a normal consequence of aging, and it is a frequently missed diagnosis in the geriatric population, given atypical presentations that are often confused with comorbid health problems such as cognitive impairment (Steffens et al. 2000, Koenig et al. 1992). Risk factors for depression in the elderly include female gender, social isolation, widowhood, divorce, low socioeconomic status, medical comorbidities, chronic pain, functional impairment, and cognitive impairment (Cole et al. 2003). Chronic drinking negatively affects mood, and depressed patients have been found to improve their mood after they stop drinking, compared to patients who continue to drink (Caputo et al. 2012). In addition, studies in adults demonstrated that AUD increases the risk of suicide (Hall et al. 1999). When combined, alcohol, depression, and anxiety disorders are responsible for about 70% of cases of suicide in the elderly (Caputo et al. 2012).

Sleep disorders may also lead to the development of late-onset AUD and may cause relapse in abstinent former drinkers (Blow 1998). Alcohol can temporarily facilitate falling asleep, but this effect rapidly fades with chronic use (Roehrs et al. 2001). Hence, long-term alcohol intake may cause difficulty falling asleep and decrease the ability to remain asleep (Blow 1998). Sleep disorder that is related to alcohol is a common clinical presentation. The sedative effects of alcohol facilitate falling asleep, but as the alcohol blood level decreases, there is a rebound of arousal and anxiety, resulting in waking in the middle of the night (middle insomnia). In this setting, cessation of alcohol can significantly improve sleep.

COMBINATION OF ALCOHOL WITH MEDICATION

It is estimated that 19% of the Americans aged 65 or older occasionally combine medication and alcohol, which can potentially lead to dangerous interactions and suboptimal treatment of medical conditions (Substance Abuse and Mental Health Services Administration [SAMHSA] 2013). The metabolism of alcohol as well as multiple prescription drugs involves a microsomal enzyme from the cytochrome P4502E1 (CYP2E1) located in the liver. CYP2E1 contributes to the metabolism of barbiturates, warfarin, phenytoin, some narcotics, some benzodiazepines, propranolol, acetaminophen, isoniazid, phenylbutazone, methotrexate, tolbutamide, isoniazid, and HAART drugs (Moore et al. 2007). Among occasional drinkers, concomitant use of alcohol with these drugs creates a competition for metabolism, which slows down their elimination. The subsequent rise in the blood concentration of the medication can lead to adverse reactions and drug toxicity. In contrast, chronic alcohol consumption increases the amount of CYP2E1 enzyme in the liver, which then accelerates the metabolism of those drugs (Seitz et al. 2007). This enhanced metabolism often reduces the therapeutic efficacy of the drugs. It can also lead to hepatotoxicity through the accumulation of harmful metabolites. This is the case with acetaminophen, isoniazid, phenylbutazone, and methotrexate, which should be prescribed very cautiously in patients with AUD (Moore et al. 2007).

Moreover, alcohol can exacerbate adverse effects from certain pharmacological treatments. For example, combining alcohol intake with ASA, nonsteroidal anti-inflammatory drugs (NSAIDs), or clopidogrel (an inhibitor of platelet receptors used to prevent clotting) increases the risk of GI bleeding (DeSchepper et al. 1978, Deykin et al. 1982). Symptomatic hypotensive episodes can be a consequence of combining alcohol with anti-hypertensive medication (Lieber 1991). Also, sedation and confusion can occur when combining alcohol with psychoactive medications such as benzodiazepines, antihistamines, sedatives, antidepressants, anticonvulsants, muscle relaxants, or barbiturates (Moore et al. 2007, Linnoila et al. 1990, Adams 1995). Finally, patients using alcohol with certain antibiotics inhibiting the aldehyde dehydrogenase (ALDH) (aldehyde dehydrogenase) enzyme in the liver (e.g., certain cephalosporins), may experience a reaction of flushing, nausea, and vomiting secondary to a toxic accumulation of acetaldehyde (Kitson 1987).

LABORATORY FINDINGS

Medical assessments of older adults often include a thorough physical examination, laboratory testing, and other forms of diagnostic studies. Different incidental findings can be suggestive of chronic alcohol consumption and may

help orient the diagnosis toward AUD. In addition, certain laboratory testing can be used as adjunctive screening tools for AUD, although the primary screening tool remains self-report (Babor et al. 1989, Maisto et al. 1985).

Several anomalies can be found in the complete blood count (CBC) of chronic alcohol users. Macrocytosis, an increase in the mean corpuscular volume (MCV) of the red blood cells, can develop after a sustained intake of 80 grams of alcohol per day (Bode et al. 2003, Girard et al. 1987, Savage et al. 1986). Before concluding that macrocytosis is due to alcohol intake, it is important to rule out other possible causes, such as vitamin B_{12} and folate deficiencies, liver problems, hypothyroidism, myelodysplastic syndrome, and the use of certain medications. However, the presence of liver disease and of low B_{12} and folate levels does not exclude a concomitant AUD, as these are common complications of chronic alcohol use. Anemia is another common consequence of chronic alcohol use and can result from direct alcohol toxicity or from alcohol-related complications, such as liver disease, GI bleeding, and vitamin B_{12} and folate deficiencies (Girard et al. 1987, Savage et al. 1986).Moreover, alcohol is the most common cause of thrombocytopenia (decreased platelets count), which is found in up to 80% of alcoholic patients (Girard et al. 1987(Girard et al. 1987). Thrombocytopenia can be a consequence of direct alcohol toxicity or of complications from chronic alcohol use such as hypersplenism caused by liver cirrhosis. Alcohol-induced thrombocytopenia rarely reaches platelet levels lower than 10,000/microL. Leukopenia is observed in about 8% of hospitalized alcoholic patients (Girard et al. 1987).

Certain findings on liver function tests may also be suggestive of AUD. Aspartate aminotransferase (AST) and alanine aminotransferase (ALT) elevation may indicate hepatocellular destruction from alcohol toxicity. As for gamma-glutamyl transferase (GGT), it may reach levels up to 300–1000 IU/L in chronic drinkers (Cohen 1988). Albumin deficiency, increased bilirubin, and elevated international normalized ratio (INR), a measure of clotting time, can result from liver dysfunction, reflecting a more advanced stage of the disease. The prevalence of albumin depletion increases with aging and is found in about 17% of the elderly with alcohol dependence, compared to 3% of younger adults with alcohol dependence (Caputo et al. 2012).

Electrolyte disorders are also common findings among patients suffering from AUD. Loss of appetite, vomiting, reduced water intake, and decreased water and sodium absorption in the intestine predispose drinkers to hyponatremia and dehydration (Bode et al. 2003). Furthermore, deficiencies in calcium, magnesium, and phosphorus may result from decreased intake and malabsorption (Knochel 1977, Leevy et al. 2005).

AST, MCV, GGT and carbohydrate-deficient transferrin (CDT) have been proposed as useful biomarkers for AUD screening (Table 2.1). Due to its low specificity, AST is a poor screening tool when used alone (Allen et al. 2003). However, it can be meaningful when combined with ALT, since a ratio of AST/ALT greater than 2 is rarely found in other pathologies than AUD, hence strongly suggesting this diagnosis. AST quickly returns to normal levels following sobriety, which makes it an interesting tool to assess relapse in chronic drinkers. Alcohol-related AST elevation increases with aging, with abnormal levels found in 56% of older chronic drinkers compared to 42% of those under 65 (Caputo et al. 2012).

MCV increases with chronic heavy alcohol intake. MCV is moderately useful in AUD screening as it has a rather low sensitivity. Given the long lifespan of a red blood cell, MCV can remain elevated up to four months after initiation of sobriety, which makes it a poor tool to detect relapse (Mundle et al. 1999). Increased MCV levels are found in about 44% of older adults with

Table 2.1 **Characteristics of Different Biomarkers Used to Screen for Alcohol Use Disorders**

*Biomarker**	*Return to normal levels in abstinence*	*Advantages*	*Disadvantages*	*Effect of aging*
AST	7 days	Good indicator of relapse Easily available	Low specificity (improved when combined to ALT)	Increases the prevalence of abnormal results
MCV	4 months	Easily available	Low sensitivity	Increases the prevalence of abnormal results
GGT	2–6 weeks	High sensitivity Easily available	Low specificity	Not affected
CDT	2–4 weeks	High specificity	Poor availability	Not affected

Modified from Allen et al. 2004.

*AST = aspartate transferase; MCV = mean corpuscular volume; GGT = gamma-glutamyl transferase; CDT = carbohydrate-deficient transferrin.

alcohol dependence, compared to a prevalence of 17% among their younger counterparts (Caputo et al. 2012).

Among all tests, GGT has the best sensitivity to detect AUD. Increased levels of GGT are found in approximately 75% of persons diagnosed with alcohol dependence, and it can remain elevated for two to six weeks after a return to sobriety (Allen et al. 2003). GGT has a low specificity for AUD, and elevated levels can be found in a wide variety of medical conditions, including obstructive liver disease, pancreatic disease, chronic obstructive pulmonary disease, and diabetes. The use of certain medications such as phenytoin and barbiturates can also lead to increased GGT levels.

When used as a single marker, CDT is the most informative laboratory screening tool, with a sensitivity of 60–70% and a specificity of 80–90% (Allen et al. 2003, Mundle et al. 1999). Abnormal levels of CDT develop following consumption of approximately 60 g of alcohol per day for two to three weeks and typically return to normal after two to four weeks of abstinence (Allen et al. 2003). Combining CDT with GGT offers an optimal screening capacity with a sensitivity of 90% and a specificity of 80–90% (Mundle et al. 1999). Neither CDT nor GGT is influenced by aging (Mundle et al. 1999). Unfortunately, CDT is still not usually available in routine clinical practice, although it has been used as a research tool to provide an objective measure of alcohol-use outcome to complement self-report. It is important to remember that, although biomarkers can be helpful to orient a diagnosis, they cannot be used to rule out an alcohol problem, as a substantial proportion of patients with AUD do not present these findings.

RECOMMENDATIONS AND DIAGNOSIS

Older adults are more vulnerable to deleterious consequences of alcohol consumption. Thus, the National Institute on Alcohol Abuse and Alcoholism (NIAAA) proposed specific drinking guidelines for adults over 65 years old, recommending a maximum of seven drinks per week, with no more than three drinks on any single occasion (NIAAA 1995, NIAAA 2014). A Consensus Panel endorsed the limit of seven drinks per week and recommended a stricter maximum of two drinks on any single occasion (Blow 1998). These amounts should possibly be further reduced for women and in the case of concomitant medication use (Blow 1998). Given that 60–78% of older adults are estimated to take one or more prescription or non-prescription medications, the safe quantity of alcohol consumption for older adults should be determined on a case-by-case basis (Chrischilles et al. 1992).

Some of the diagnostic criteria proposed by the DSM-5 (see Box 2.1) are not applicable to older adults, who are less likely to experience some of the

biological, psychological, and social consequences frequently encountered in younger adults with AUD (Blow et al. 2014, Atkinson 1990, Blow 1998). This lack of relevance of some diagnostic criteria to the geriatric population represents a barrier to appropriate diagnosis of certain individuals who do not meet a diagnostic threshold, despite potentially harmful drinking practices. The concept of *hazardous drinking*, defined as a potentially harmful pattern of drinking that can precipitate or exacerbate medical conditions, complicate treatments, and cause adverse reactions to drugs, may be more appropriate in older adults (Fink et al. 1996, SAMHSA 2013, Fink et al. 2002). Hence, when screening for alcohol problems in the elderly, it is important that clinicians not limit their diagnostic consideration to AUD as defined by the DSM-5, but also keep in mind the possibility of less severe types of problematic drinking patterns that also require interventions, such as hazardous drinking.

SCREENING

Screening for AUD should be part of older adults' annual medical examination. Since problematic drinking can emerge between annual assessments, it is also recommended to screen elders reporting any symptoms suggestive of problematic drinking, in addition to those facing significant life transitions such as a bereavement or separation (Blow 1998). The screening process should begin with a few brief pre-screening questions to quickly rule out individuals for whom further assessment is not required. Pre-screening can start by asking the following question: "Do you drink alcohol, including beer, wine, or distilled spirits?" Patients answering affirmatively should be asked the three following questions:

1. On average, how many days per week do you drink alcohol?
2. On a typical day when you drink, how many drinks do you have?
3. What is the maximum number of drinks you had on any given occasion during the last month? (NIAAA 1995)

Individuals whose alcohol intake exceeds NIAAA or experts panel recommendations (i.e., more than seven drinks per week or more than two or three drinks on any single occasion), those taking medications that can potentially interact with alcohol, and those with medical or psychiatric comorbidities that may be exacerbated by alcohol intake should undergo further screening.

Several self-reported tools have been developed to screen patients for possible AUD. Although most screening tools were initially tested among younger adults, some were validated specifically in geriatric populations. The CAGE (4-item questionnaire; an acronym for these questions, as detailed

below), the Alcohol Use Disorders Identification Test (AUDIT), the Michigan Alcoholism Screening Test–Geriatric (MAST-G) and its shortened version, the SMAST-G, are the four main instruments used among older patients (Ewing 1984, Blow et al. 1992, Saunders et al. 1993). Results from psychometric studies assessing the performance of those screening instruments in older populations vary widely, according to factors such as clinical setting, cultural elements, patient characteristics, and the prevalence of AUD in study populations (O'Connell et al. 2004).

The CAGE questionnaire is the screening tool most frequently used in adults, with the advantages of being simple to administer and easy to memorize as an acronym for the questionnaire is based on the content of the following four questions:

1. Have you felt the need to **C**ut down on your drinking?
2. Have people **A**nnoyed you by criticizing your drinking?
3. Have you ever felt **G**uilty about drinking?
4. Have you ever felt you needed a drink first thing in the morning (**E**ye-opener) to steady your nerves or to get rid of a hangover? (Ewing 1984)

The full questionnaire can be administered in less than a minute, and it can be formulated either to detect lifetime or recent and current AUD (Ewing 1984). In younger adults, a positive response to two or more questions is suggestive of problematic alcohol consumption and requires a more in-depth assessment. The CAGE is less sensitive in the geriatric population, and using a cut-off of one positive answer increases the instrument's sensitivity, at the expense of decreasing its specificity (Conigliaro et al. 2000). Median sensitivity and specificities of 66.5% and 89% were drawn from validation studies conducted in different geriatric populations and using a cut-off of two yesses (O'Connell et al. 2004). Buchsbaum and colleagues have demonstrated that decreasing the cut-off to one affirmative response yielded a sensitivity of 86% and specificity of 78% (Buchsbaum et al. 1992).

The MAST-G is a 24-item questionnaire following a yes/no format that can be administered in approximately five minutes. It was developed to detect AUD specifically in elderly patients (Blow et al. 1992). Positive answers to five questions or more leads to AUD detection with high estimated sensitivity (50–95%) and specificity (78–96%) (O'Connell et al. 2004, Blow et al. 2014). The SMAST-G is a shorten version containing ten questions that can be administered in approximately three minutes, with a score of 2 or more suggesting AUD (Blow et al. 1998). Both versions were found to be robust screening instruments in the elderly population (O'Connell et al. 2004).