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## Alcohol abuse and dependence:

### Psychopathology, medical management and dental implications

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### Abstract

**Background.**—The authors review the clinical features, epidemiology, pathophysiology, medical management, dental findings and dental treatment of patients with alcoholism.

**Literature Reviewed.**—The authors conducted a MEDLINE search for 1995 through 2001 using the key terms of alcoholism, epidemiology, pathophysiology, treatment and dentistry. Reports selected for further review included those published in English in peer-reviewed journals. The authors gave preference to articles reporting randomized, controlled trials.

**Conclusions.**—Alcoholism is a chronic and progressive psychiatric illness that afflicts more than 14 million Americans. It is characterized by a loss of control over the use of alcohol, resulting in impaired social functioning, and the consequent development of medical illnesses. The disease arises in genetically vulnerable people when they are overwhelmed by their cravings for the alcohol-associated euphoria that results from the actions of several neurotransmitter systems in the brain's pleasure center. New medications to counteract alcohol-induced neurotransmission imbalance may assist patients in reducing their craving.

**Clinical Implications.**—The prevalence of dental disease usually is extensive because of a disinterest in performing appropriate oral hygiene techniques and diminished salivary flow. Concurrent abuse of tobacco products worsens dental disease and heightens the risk of developing

oral cancer. Identification of the alcohol-abusing patient, a cancer-screening examination, preventive dental education, and use of saliva substitutes and anticaries agents are indicated. Special precautions must be taken when performing surgery and when prescribing or administering analgesics, antibiotics or sedative agents that are likely to have an adverse interaction with alcohol or psychiatric medications.

Alcohol (ethanol) is consumed in moderation by most people for its relaxant and euphoric effects. However, when an individual loses control over its use and this use results in impaired social or occupational functioning, legal difficulties or the development of associated medical diseases, it is construed to be a psychiatric illness. The American Psychiatric Association divides the illness into two main components: alcohol abuse and alcohol dependence.<sup>1</sup>

## DEFINITIONS

Alcohol abuse occurs early in the disease process and is distinguished by the harmful consequences of repeated use. Repeated episodes of alcohol intoxication that are severe enough to alter mood, impair judgment and slur speech invariably prevent the individual from fulfilling his or her obligations, such that school and job performance suffer, and child care and household responsibilities are neglected. While intoxicated, these people often participate in physically hazardous activities, such as driving an automobile, which often leads to an arrest and to legal problems. During episodes of intoxication, alcohol abusers also frequently act inappropriately in social situations, getting into arguments with friends and family and engaging in domestic violence.<sup>2</sup>

Alcohol dependence is an advanced stage of the disorder and is distinguished by physiological dependence, as evidenced by tolerance or symptoms of withdrawal. During this phase of the illness, the individual progressively increases the amount of alcohol imbibed (tolerance) to achieve the same level of intoxication, and may drink continuously so that blood concentration levels are high and withdrawal symptoms (such as insomnia, sweating, rapid pulse, anxiety, nausea, vomiting and xerostomia) are avoided. The compulsive use of alcohol also forces these people to curtail social and occupational activities. Despite their often expressed desire to quit drinking and the knowledge that it is causing them psychological and physical harm, they continue to drink. Severe intoxication leads to disinhibition and feelings of sadness, which may contribute to suicide attempts and completed suicides.

### **Low-grade hypertension.**

Long-term, high-dose alcohol use adversely affects nearly every organ system. One of the most common adverse effects is the development of low-grade hypertension, which, in combination with marked increases in levels of triglycerides and low-density lipoprotein, fosters an elevated risk of coronary artery and cerebrovascular diseases. Similarly, excessive alcohol ingestion may damage cardiac myofibrillar architecture and myocardial contractility and may promote congestive heart failure secondary to cardiomyopathy.

### **Gastrointestinal tract.**

Alcohol also is an irritant to the gastrointestinal tract. It may cause gastritis, stomach ulcers or duodenal ulcers (or any combination of the three) and, in about 20 percent of people with long-term consumption, leads to cirrhosis and pancreatitis.<sup>3</sup> People with long-term consumption also experience an increased rate of cancer of the oral cavity, pharynx, hypopharynx, larynx, esophagus and stomach. Central nervous system, or CNS, effects include neuronal cell death, with shrinkage or atrophy of several regions of the brain, most notably the frontal lobes, limbic structures, hippocampus and cerebellum. The exact mechanism underlying these anatomical changes remains unknown; however, they clinically correlate with deficits in judgment, decision-making, attention span and short-term memory; emotional lability; and ataxia (impaired coordination).<sup>4-6</sup>

In the Orofacial Findings and Dental Treatment sections below, we describe other adverse effects of long-term alcohol abuse, such as impairment of the liver's ability to produce coagulation factors and to metabolize medications, impairment of white blood cells' chemotactic abilities and impairment of bone marrow's production of platelets.

### **Poor nutrition.**

Alcohol abuse also may result in inadequate nutritional intake, because ethanol ingestion often accounts for half of the daily caloric intake, thereby displacing from the diet proteins, minerals, trace elements and such normal nutrients as folic acid, riboflavin (vitamin B<sub>2</sub>), pyridoxine (vitamin B<sub>6</sub>), and vitamin E.<sup>7,8</sup> To compound this problem, chronic alcoholic ingestion causes malabsorption of folate, thiamine (vitamin B<sub>1</sub>) and vitamins D and K, as well as the enhanced excretion of such trace elements as magnesium and zinc.<sup>9</sup>

### **Dual diagnosis.**

About half of all people who have received a diagnosis of alcohol abuse or alcohol dependence have additional psychiatric illnesses. This situation is termed "dual diagnosis." The most common comorbid conditions include abuse of, or dependence on, other substances (for example, cocaine, nicotine), antisocial personality disorder, anxiety disorder, bipolar mood disorder and major depressive disorder.<sup>10,11</sup> A significant number of patients receive psychiatric medications for these disorders; unfortunately, this group of alcoholics also is least likely to abstain from alcohol use and is at highest risk of experiencing alcohol-associated morbidity and mortality.<sup>12</sup>

## **EPIDEMIOLOGY**

Some 14 million Americans meet the diagnostic criteria for alcoholism, making it the nation's third most prevalent psychiatric disorder and costing more than \$165 billion each year in reduced productivity, premature death and direct treatment expenditures. It is an all-pervasive illness, with 10 percent of women and 20 percent of men meeting the diagnostic criteria for alcohol abuse during their lifetimes, and an additional 3 to 5 percent of women and 10 percent of men meeting the diagnostic criteria for alcohol dependence during their lifetimes.

Estimates of suicide among patients with the disorder range from 10 to 15 percent. Those at highest risk of committing suicide are men who have lost their spouses within the year. People aged 20 to 35 years consume the most alcohol; from approximately age 40 years and older, there is a gradually declining pattern of alcohol use. Among people aged 65 years and older, 7 percent of men and 2 percent of women are considered to be heavy drinkers.<sup>13,14</sup>

Women tend to start drinking heavily later in life than do men and to develop alcoholism later. However, once it develops, alcoholism progresses more rapidly because women develop higher blood alcohol concentrations than do men at a given dose of alcohol per kilogram. This is because of the lower percentage of body water and higher percentage of body fat in women. Furthermore, women have less of the gastric enzyme alcohol dehydrogenase, or ADH, to metabolize ethanol; with less alcohol broken down in the stomach, a proportionally larger amount enters the blood stream. Because of their higher alcohol levels, women are at greater risk of developing some of the health-related consequences of heavy alcohol intake (in particular, cirrhosis of the liver, cardiomyopathy and atrophy of the brain).<sup>15</sup>

## ETIOLOGY AND PATHOPHYSIOLOGY

Although the etiology of alcoholism remains ill-defined, researchers now presume that it arises from a complex interaction of genetic predisposition, neurochemical influence, anatomical variation and cultural influences. Alcoholism aggregates in families, with the risk in relatives of alcoholics being three to four times higher than it is in the general population. At least some of the transmission can be traced to genetic factors.<sup>16</sup>

Most studies have found a markedly higher risk of alcoholism in the monozygotic (identical) twin than in the dizygotic (fraternal) twin of an individual with alcoholism.<sup>17</sup> In addition, the children of parents who abuse alcohol often have behavioral and neurophysiological deficits that cannot be explained by their mother's prenatal use of alcohol. These children frequently exhibit impulsive behavior, abnormal brain waves, body sway and significant deficits in reading and math scores, all possibly caused by delays in maturation of neural circuitry.<sup>18-20</sup> Of greatest significance is the fact that as adolescents and young adults, these children of alcoholics often begin to drink alcohol and abuse other substances at an earlier age than do their peers.<sup>21</sup>

Alcoholism usually develops when an individual is overwhelmed by the craving and positive-reinforcing properties (that is, reward mechanisms) of ethanol that arise from the ability of the substance to influence the interactions of several neurotransmitter systems in the CNS. Ingestion of alcohol is associated with the release of endogenous opioid peptides (that is, endorphins and enkephalins) known to evoke a pleasurable sensation.<sup>22</sup> These peptides then stimulate release of dopamine in the nucleus accumbens, the brain's reward and reinforcement center.<sup>23-26</sup> Alcoholism also may develop if an individual is anxious or depressed and continually uses the substance to alleviate these feelings. This is because of ethanol's ability to interact with the  $\gamma$ -aminobutyric acid, *N*methyl-Daspartate and serotonergic (5-hydroxytryptamine) neurotransmitter systems.<sup>27</sup>

## TREATMENT

The main components of treatment consist of confrontation, detoxification and rehabilitation. Confrontation involves overcoming patients' denial, convincing them of the adverse consequences of continued drinking and motivating them into treatment. Detoxification consists of removing alcohol from the body and protecting the patient from the serious effects of alcohol withdrawal (particularly delirium tremens). This process usually takes about five days and involves replacing alcohol with CNS depressant medications such as lorazepam or diazepam in gradually reducing dosages to avoid withdrawal symptoms.

$\beta$ -adrenergic blocking agents (for example, atenolol) may be administered concurrently to reduce tremors and lower the patient's heart rate and blood pressure. Rest, adequate nutrition and the administration of multiple vitamins (especially those containing thiamine and magnesium) are vital to the process. Rehabilitation consists of continued efforts to increase and maintain high levels of motivation regarding abstinence and readjustment to a lifestyle free of alcohol. This aspect of treatment includes psychosocial interventions such as cognitive behavioral therapy, or CBT, and varied counseling approaches such as Alcoholics Anonymous, or AA.

### **Cognitive behavioral therapy.**

Psychosocial therapies are integral to preventing relapse; however, the efficacy of specific modes is controversial. CBT, a highly structured intervention, helps patients recognize that their beliefs and thinking styles are pathological and contribute to addictive behavior. CBT teaches people skills for coping with difficult and stressful situations, cravings and feelings that in the past would have forced them to drink alcohol.<sup>28</sup>

### **Alcoholics Anonymous.**

AA is a social network that supports abstinence by providing its adherents with a philosophy for living, and by encouraging them to develop relationships with people who are living without alcohol. The basic philosophical tenant of AA is to motivate the individual to admit that he or she lacks the power to control alcohol use, and through prayer to ask God to correct this character defect. Approximately 5 percent of Americans with an alcohol disorder have become members of AA; alcoholics who benefited most were those without supportive family or friends.<sup>29-31</sup> Historically, AA and clinicians treating alcoholism were at odds, but more recent collaborations involving patients who continued to receive medical care while attending AA meetings appear to have improved patient outcomes.<sup>32</sup>

### **Medications.**

During the rehabilitative phase of care, many people are at high risk of experiencing relapse because they are preoccupied with thoughts of drinking. Three medications are available to assist patients during this vulnerable period. Two of these drugs counteract the craving and positive-reinforcing properties of alcohol, and the third produces unpleasant sensations when the patient drinks alcohol.

**Naltrexone.**—Naltrexone, an opioid antagonist, inhibits an individual's desire to drink by blocking the rewarding/pleasant feelings (euphoria) associated with alcohol consumption.<sup>33</sup> It does so directly by binding to receptor sites in the ventral tegmental area and indirectly by blocking the release of dopamine in the nucleus accumbens (reward center), because of the rich neural connections between these two structures.<sup>34</sup>

Physicians prescribe naltrexone to patients who are abstinent, as well as to those who are attempting to control the amount they consume each day.<sup>35</sup> The medication regimen varies, with some patients taking the medication daily and others taking it only when they experience craving. Several controlled studies have shown that the drug, when combined with specific psychosocial-behavioral treatment programs, successfully reduces the desire to drink and the consumption of alcohol. The most common systemic adverse side effects of naltrexone therapy are nausea, abdominal pain, hypertension, palpitation and tachycardia.<sup>36-39</sup>

**Acamprosate.**—Acamprosate (calcium acetylhomotaurinate) has been used for many years in Europe and is expected to be approved by the U.S. Food and Drug Administration for marketing in the United States in 2003. The medication reduces an individual's craving for alcohol. The exact neurobiological mechanism remains unknown. Some investigators believe the drug, like naltrexone, blocks alcohol-associated euphoria by suppressing dopamine release in the nucleus accumbens; others postulate that it lowers the activity of glutaminergic neurons that have become hyperexcited because of long-term exposure to alcohol.<sup>40-42</sup>

Acamprosate is particularly effective in patients who have chosen abstinence rather than controlled drinking as their treatment objective and who are concurrently enrolled in a psychosocial support or behavioral treatment program. The most common adverse effects of this medication are diarrhea, pruritus and headache.<sup>43,44</sup>

**Disulfiram.**—Disulfiram is one of the oldest and most widely prescribed medications for treating patients with alcoholism. The drug inhibits the normal metabolic pathway of ethanol by blocking the action of the enzyme acetaldehyde dehydrogenase. This results in the accumulation of acetaldehyde, a toxic product that makes the patient ill (that is, negative reinforcement/aversion therapy).

Patients who drink alcohol while receiving disulfiram therapy experience facial flushing, throbbing headache, nausea, vomiting, chest pain, palpitation, tachycardia, weakness, dizziness, blurred vision and confusion. The medication is widely used, but has not proved to be effective in the best of controlled studies.<sup>45</sup> This may be because the medication has little effect on craving, and treatment maintenance requires a strong degree of self-motivation or external pressure.<sup>45</sup>

## OROFACIAL FINDINGS

Salivary gland enlargement, most often of the parotids, may occur in some patients who ingest large quantities of alcohol on a long-term basis, with resultant liver damage. The

condition is termed “sialosis” (sialadenosis) and is thought to result from an ethanol-produced peripheral autonomic neuropathy that causes disordered salivary metabolism and secretion.<sup>46,47</sup> Reduced parotid salivary flow and a reduction in saliva-buffering capacity (that is, the ability to neutralize acid), combined with a preference for drinking very sweet and cariogenic nonalcoholic beverages and neglect of oral hygiene, frequently give rise to extensive dental decay and advanced periodontal disease.<sup>48-51</sup>

The periodontal disease with eventual loss of teeth is exacerbated by the often-encountered concomitant cigarette smoking (nicotine addiction).<sup>52</sup> The residual dentition commonly exhibits signs of erosion secondary either to the alcohol content and acidity of the beverage or to subclinical regurgitation of acidic stomach contents (commonly referred to as gastroesophageal reflux disease).<sup>53-56</sup> The latter condition may develop because of alcohol’s direct relaxant effect on the lower esophageal sphincter.

### **Signs and symptoms.**

Oral signs and symptoms of alcohol-induced nutritional deficiencies include glossitis, angular cheilosis and gingivitis. In the early stages, the tongue is painful, with the fungiform papillae being swollen, flattened and mushroom-shaped. As the deficiency progresses, the tongue begins to burn and becomes intensely red, and the filiform and fungiform papillae atrophy. The cheilosis is typified by ulcerations at the corners of the mouth, and the gingivitis manifests itself with necrotic areas at the tips of the interdental papillae.<sup>57,58</sup>

### **Malignancies.**

Squamous-cell carcinoma of the oral cavity, especially of the tongue and floor of the mouth, develops in some people who concurrently abuse alcohol and tobacco products. These malignancies are thought to arise because the ethanol metabolite acetaldehyde promotes tobacco-initiated tumors by damaging DNA and altering oncogene expression in oral keratinocytes. The process is facilitated by the high and long-lasting concentrations of acetaldehyde that are present in the mouths of people who consume large amounts of alcohol and have poor oral hygiene.

After an individual drinks, ethanol is absorbed in the gut and is evenly distributed in the water phase of the body (that is, blood serum, tissue fluids [extracellular cellular-extravascular compartment]), such that its concentration in salivary gland secretions equals that in blood. The saliva then comes into contact with oral bacteria and yeast that contain ADH, which oxidizes the ethanol to acetaldehyde. Compounding the problem, lingual and gingival mucosal cells also contain ADH and produce intracellular acetaldehyde.<sup>59-63</sup> Epidermal growth factor, or EGF, which is produced by the salivary glands, has been shown to protect the oral mucosa from injury by acidic fluids. A reduction in salivary flow resulting from the adverse effects of alcohol on the glands may result in reduced EGF and an increased prevalence of oral ulcerations.<sup>64</sup>

### **Bleeding during surgical procedures.**

Excessive bleeding during oral surgical procedures may occur. This problem may arise because long-term alcohol ingestion suppresses megakaryocyte maturation, leading to

decreased numbers of platelets, and inhibits the release of thromboxanes A and B, which adversely affects platelet aggregation. These defects in hemostatic function are demonstrated by a prolonged bleeding time. Coagulation defects also may occur as the result of impaired vitamin K absorption, which affects the syntheses of clotting factors. This coagulopathy is worsened in patients with advanced liver disease, because a scarred, cirrhotic liver with relatively few hepatocytes is unable to synthesize fibrinogen, prothrombin and factors V, VII, IX and X. These coagulation defects are demonstrated by an alteration in the prothrombin time.<sup>65,66</sup>

### **Mandible fractures.**

Fractures of the mandible also are common because of falls or fights.<sup>67,68</sup> An open reduction of the fractured segments with rigid internal fixation (that is, use of a bone plate) often is preferable to a closed reduction (that is, use of arch bars and intermaxillary fixation) because of the danger of alcohol ingestion and associated vomiting and aspiration.<sup>69</sup> However, irrespective of the treatment modality, a fibrous union or an infection leading to osteomyelitis may develop.<sup>70,71</sup>

Poor wound healing, infection and osteomyelitis also may develop after routine dental extraction. These complications arise because people who abuse alcohol on a long-term basis are less able to accumulate protein and collagen at the surgical site and because ethanol suppresses activation and proliferation of T lymphocytes, as well as the mobilization and phagocytic capability of monocytes, macrophages and neutrophils.<sup>72,73</sup>

Patients beginning disulfiram therapy may complain to their dentist about a metallic or garlic-like aftertaste. Clinicians should reassure them that it is caused by the medication and that the problem will resolve itself within the first two weeks of therapy.<sup>74</sup> Patients receiving naltrexone treatment may complain of xerostomia, cold sores, pharyngitis, sinusitis, tinnitus and headache.<sup>75,76</sup>

We should make a special note of caution with respect to the use of acetaminophen by patients who abuse alcohol over a long period. Acetaminophen toxicity, resulting in severe, potentially fatal hepatocellular failure, may develop in patients who ingest large quantities of acetaminophen and who actively drink. Acetaminophen also is available as an active component of many over-the-counter cold and flu medications, as well as prescription medications such as acetaminophen and oxycodone (Percocet, Endo Pharmaceuticals, Chadds Ford, Pa.) and acetaminophen and hydrocodone (Vicodin, Abbott Laboratories, North Chicago, Ill., and Lorcet, Forest Pharmaceuticals, St. Louis). Because a significant percentage of patients who are longterm alcohol users also may take these medications for legitimate or illegitimate uses, the potential exists for severe liver abnormalities to develop (Table).

## **DENTAL TREATMENT**

Millions of Americans meet the diagnostic criteria for alcoholism, but given the stigma associated with past or present addiction, it is the rare patient who discloses this information to his or her dentist. Similarly, dentists may be reluctant to delve into the issue of a patient's



alcohol dependence because it may seem intrusive, or because they view the disorder as a moral shortcoming rather than a valid psychiatric disorder. However, this avoidance presents a danger because of alcohol's effects on oral and systemic health, and because of an enhanced risk of medication-related adverse events and drug interactions.

While obtaining a medical history, dentists may uncover indicators of patients who are at risk of developing alcoholism. Historical factors that may indicate a problem include insomnia, headache, seizures, dyspepsia, diarrhea, palpitations, sexual dysfunction, anxiety, irritability, depression, trauma, motor vehicle accidents, domestic violence and multiple visits to a hospital emergency department for any reason.

### **CAGE questionnaire.**

Dentists who believe that a patient may be currently (or in the past) abusing alcohol or may be dependent on it can screen for the illness using the CAGE questionnaire. This instrument is most effective when the questions are embedded in a benign component of the medical history, such as the health habits review (for example, diet and exercise).<sup>77</sup> Below are the questions from the CAGE questionnaire<sup>77</sup>: "Have you ever felt you should cut down on your drinking?"

"Have people annoyed you by criticizing your drinking?"

"Have you ever felt bad or guilty about your drinking?"

"Have you ever had a drink first thing in the morning to steady your nerves or get rid of a hangover (eye opener)?"

Each positive response receives one point. A score of 2 or more is considered probable for alcoholism.<sup>78</sup> In a typical outpatient population, the predictive value of two positive responses to questions is 30 to 60 percent; of three positive responses, 60 to 75 percent; and of four positive responses, higher than 90 percent.<sup>78</sup> Dentists should refer all patients with test results that suggest alcohol dependence to their primary care physician for a more in-depth evaluation.

### **Alcohol and CNS depressants.**

The safety and efficacy of many dental therapeutic agents are influenced by their concomitant ingestion with alcohol. Of greatest concern are the combined effects of alcohol and CNS depressants and the complex effects of alcohol on the liver's ability to metabolize medications (Table).<sup>79-81</sup> Acute ingestion of intoxicating amounts of ethanol (several drinks over several hours) depresses the respiratory center in the CNS in a dose-dependent fashion. Simultaneously, alcohol also inhibits the liver's ability to metabolize certain medications, which results in elevated blood concentrations of the drugs.

This presents a particular danger to patients who are being treated with medications that depress the CNS (for example, sedatives, hypnotics and opioid analgesics), because they may experience a supra-additive increase in the drugs' effects that can result in dizziness,

drowsiness, loss of coordination, impaired judgment or respiratory depression. In turn, these effects increase the risks of falls and other accidents.<sup>82-84</sup>

Long-term ingestion of alcohol by an individual who has yet to develop liver damage results in stimulation (induction) of the liver's ability to metabolize certain medications, resulting in depressed and sometimes subtherapeutic blood concentrations of the drugs. This increased rate of drug metabolism may persist even in recovering alcoholics and may require that meprobamate, pentobarbital or diazepam be prescribed in high dosages for them to be clinically effective.<sup>85</sup>

Long-term ingestion of large amounts of alcohol also may result in acetaminophen being rapidly converted into an intermediate metabolite that is highly toxic and can cause severe and sometimes fatal liver injury.<sup>86</sup> Some people also may develop cirrhosis of the liver, a disease associated with decreased rates of drug metabolism because of destruction of hepatocytes and impaired blood flow. Affected agents include local anesthetics, analgesics, sedatives and certain antibiotics. In view of the opposing effects of short- and long-term alcohol consumption, it is difficult to predict the net effect of concomitant alcohol and medication use in a given long-term alcohol consumer.

Clinicians must also consider the effects of other metabolic pathways involving medications used to treat dental conditions or alcoholism. Metronidazole should not be prescribed for patients unable to abstain from continued alcohol ingestion because metronidazole inhibits acetaldehyde dehydrogenase, thereby permitting the accumulation of acetaldehyde and resulting in a classic disulfiram-aversive reaction (such as facial flushing, throbbing headache, nausea).<sup>87,88</sup> Patients being treated with naltrexone who need elective dental procedures requiring opioids for sedation and analgesia should, in consultation with their physician, discontinue the naltrexone therapy for 48 hours before the procedure. This is because the therapeutic effectiveness of the opioids is diminished when naltrexone and opioids are administered concurrently.<sup>76</sup>

Patients receiving disulfiram treatment who require diazepam treatment should receive a lower dosage of the latter medication because disulfiram inhibits the oxidative biotransformation of diazepam, which permits excessive CNS depression.<sup>89</sup> In addition, clinicians should advise patients who receive disulfiram therapy to avoid using mouthwashes containing alcohol because of their potential adverse interactions.<sup>90</sup>

Alcohol also inhibits the absorption and enhances the breakdown of penicillins in the stomach for up to three hours after ethanol intake. Aspirin and other nonsteroidal antiinflammatory drugs promote gastric bleeding when combined with ethanol, and can cause gastric hemorrhage in alcoholics who suffer from alcoholic gastritis. Clinicians should administer low dosages only of acetaminophen alone or in combination with hydrocodone (Vicodin, Lorcet) or oxycodone (Percocet) because of the danger of hepatocellular failure (Table).

The American Society of Addiction Medicine recommends that clinicians avoid prescribing all potentially addicting (for example, opioid analgesics) and mood-altering medications (for example, benzodiazepines) for recovering alcoholics, unless a thorough evaluation of the

associated risks (such as development of addiction to a new substance) and benefits justifies their use in treating a specific medical condition.<sup>91</sup> In such cases, individualized dosages and cautious monitoring help prevent unwanted effects and reduce the risk of relapse.

### **Dental education.**

Preventive dental education and the maintenance of good oral health are paramount for alcoholics given the new research findings that suggest that oral microflora may contribute to the development of intraoral carcinoma.<sup>92,93</sup> Dentists should instruct patients in proper toothbrushing and flossing methods that maximize removal of dental plaque. They may prescribe artificial salivary products for patients with signs of xerostomia. This therapeutic approach is particularly germane to this patient population, because a paucity of saliva is associated with increased concentrations of bacteria able to produce acetaldehyde.<sup>94</sup>

Dental treatment should consist of subgingival scaling, root planing and curettage, caries control and restorative treatment. Profound local anesthesia is mandatory to perform potentially painful procedures in these often-anxious patients. Patients requiring extensive surgery who have a long history of alcohol abuse require a comprehensive medical evaluation that includes a complete blood cell count, coagulation profile, liver function studies and consultation with their treating physician.<sup>95,96</sup>

We recommend that dental professionals perform a clinical examination and oral prophylaxis at three-month follow-up visits, and apply a fluoride gel with a fluoride concentration of at least 1.0 percent. Dentists also should correct any defects in the natural dentition or prostheses during these recall visits. Patients may experience enhanced self-esteem as a result of dental treatment, which may contribute to the psychotherapeutic aspect of management.

## **CONCLUSION**

Alcoholism is best viewed as a disease without moral implications. Dentists, in concert with physicians, have much to offer patients with the disorder. Dentists who are familiar with the manifestations of the illness and the newer pharmacological interventions can confidently offer these patients the full range of dental treatment options.

## **Biography**



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TABLE

ADVERSE INTERACTIONS BETWEEN ALCOHOL OR RESULTANT ALCOHOLIC LIVER DISEASE AND MEDICATIONS USED IN DENTISTRY.

| MEDICATION   | DRUG-INTERACTION   | DENTIST'S ACTION  |
|--|--|---|
| <b>Analgesics</b><br>Acetaminophen                   | Hepatotoxicity may occur because of toxic acetaminophen metabolites and glutathione depletion  | Limit acetaminophen dosage to 2 grams per day; counsel patient about the risks of long-term alcohol use and acetaminophen toxicity                    |
| <b>Aspirin</b>                                       | Excessive bleeding may occur because of aspirin-induced prolongation of bleeding time  | Counsel patient to discontinue alcohol use during analgesic therapy   |
| <b>Ibuprofen</b>                                     | Increased risk of gastric mucosal ulceration and gastrointestinal hemorrhage. Renal toxicity has been reported in association with binge drinking  | Counsel patient to discontinue alcohol use during analgesic therapy   |
| <b>Antibiotics</b><br>Cephalosporins (some)          | A disulfiram effect may occur, permitting the accumulation of acetaldehyde, leading to facial flushing, headache, palpitation and nausea   | Avoid use of cefoperazone and cefotetan   |
| Erythromycin   | Decreased absorption of erythromycin, with a consequent decrease in effectiveness  | Counsel patient to discontinue alcohol use during erythromycin therapy  |
| Metronidazole  | A disulfiram effect may occur, permitting the accumulation of acetaldehyde, leading to facial flushing, headache, palpitation and nausea   | Counsel patient to discontinue alcohol use during metronidazole therapy   |
| Tetracycline   | Increased absorption and increased plasma concentration in healthy subjects after acute ingestion of ethanol; however, diminished effectiveness in long-term alcoholics because of induction of metabolizing enzymes | Counsel patient to discontinue alcohol use during tetracycline therapy  |
| <b>Antifungal Agent</b><br>Ketoconazole              | May increase risk of liver damage. A disulfiram effect may occur, permitting the accumulation of acetaldehyde, leading to facial flushing, headache, palpitation and nausea  | Counsel patient to discontinue alcohol use during ketoconazole therapy  |
| <b>Barbiturates</b><br>Pentobarbital<br>Secobarbital | Concurrent use may increase CNS* depressant effects. Diminished effectiveness in long-term alcoholics because of cellular tolerance to CNS depression, increased metabolism, or both                                 | Advise patients to never drink alcohol when taking barbiturates   |
| <b>Benzodiazepines</b><br>Diazepam<br>Lorazepam      | Concurrent use may increase CNS depressant effects. Diminished effectiveness in long-term alcoholics because of cellular tolerance to CNS depression, increased metabolism, or both                                  | Initially decrease the usual dosage of medication and observe patient for CNS depression. Counsel patient to discontinue alcohol use during treatment |
| <b>Chloral Hydrate</b>                               | Concurrent use may significantly increase CNS depressant effects   | Initially decrease the usual dosage of medication and observe patient for CNS depression. Counsel patient to discontinue alcohol use during treatment |
| <b>Opioids</b>                                       | Sedative side effects are markedly increased   | Initially decrease the usual dosage of medication and observe patient for CNS depression. Counsel patient to discontinue alcohol use during treatment |

\* CNS: Central nervous system.