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INTRODUCTION

I became interested in treating addictions as a medical student, and eventually, after doing a residency in internal medicine, completed a fellowship in addiction medicine. I found that in treating substance abuse, I could make a remarkable and rapid difference in people's lives. Over the years, as we've developed new and more effective treatments, managing addiction is no longer the province of addiction specialists—nor should it be. General psychiatrists and primary care doctors can learn the basics of an effective approach to treatment, and can gain the immense satisfaction that comes with helping people make dramatic changes in their lives.

I've divided the book into two sections. In the first section, I teach you general skills applicable to any patient with a substance use problem. I share tips on efficiently screening for the use of different substances in the initial interview, as well as ascertaining the severity of the disorder. I teach you about the appropriate use of drug screens, including how to talk to your patients about problematic results. In addition, I give you some psychotherapy pointers that are especially applicable to a busy MD who may be able to budget no more than 20–30 minutes for patient follow-ups. I devote a whole chapter to 12-step programs because they are often misunderstood by both clinicians and patients. In the comorbidity chapter, I cover the common problem of patients who present with a non–substance use issue, but in whom you realize that an addiction may be contributing to the problem.

The second section is organized by specific substances of abuse. For each substance, I cover the essentials of what it is, how it works in the brain, and what sort of withdrawal symptoms are likely to occur. Then I discuss practical approaches to assessing the problem, followed by how to treat the patient. Treatments vary depending on the substance; sometimes they will be primarily psychosocial, other times mainly medication-based.

Finally, I've added various useful tools in the Appendix and have provided links to access these materials in the electronic version of this book.

CHAPTER 1

How to Diagnose Substance Use Disorder

"I used to think a drug addict was someone who lived on the far edges of society. Wild-eyed, shaven-headed and living in a filthy squat. That was until I became one . . ."

Cathryn Kemp, *Painkiller Addict: From Wreckage to Redemption—My True Story*

TAKE HOME POINTS

- Dopamine desensitization underlies most substance abuse.
- During your first interview, ascertain what substances are being used, how they are negatively affecting the patient's life, and how motivated the patient is to change.
- Use normalization to decrease stigma.
- When asking about specific drugs, start with the most socially acceptable substances and end with the most stigmatizing.
- Use the DSM-5 mnemonic: Tempted With Cocaine, Scotch, Rum.

CASE VIGNETTE: *G is a 45-year-old woman who presents for treatment of anxiety. She was prescribed alprazolam (Xanax) by her family practitioner to take once or twice daily for severe anxiety symptoms, but she has been taking it closer to 3 times every day. She also has 2 drinks of gin before bedtime most nights, and has done this for several years. She tells me that occasionally she will get "shaky" during the day, and an alprazolam or a drink will steady her nerves.*

WHAT IS SUBSTANCE USE DISORDER?

Before diving into the skills of diagnosing substance use disorders, it's fair to take a step back and ask what a substance use disorder actually is. Is it a brain disease? Is it self-medication? Is it a life choice?

The answer can be any of the above, or a combination, depending on the person and the substance. For the opioid addict with overwhelming cravings who is stealing money from her friends to buy her next fix, it is primarily a brain disease involving opiate receptors. For the college student taking Adderall a couple of times a week—borrowed from friends—to study for exams and write papers, it may be a lifestyle choice (though it can devolve into neurochemical dependency if the habit becomes a daily one). For the man with social anxiety disorder who downs a few shots of vodka before going to a social event, it may be a form of self-medication.

Like most disorders in medicine and psychiatry, substance use is multifactorial, and for this same reason, it can be treated in different ways.

Neurobiology of addiction

While our knowledge of the neurobiology of addiction is limited, researchers are beginning to work out some of the mechanisms. One particular neurotransmitter, dopamine (DA), seems to play a central role for most addictions.

Most psychiatrists are familiar with DA in the setting of psychosis. All antipsychotics block DA receptors, which implies that excessive DA can be a bad thing, as it may be one of the chemicals that can cause psychosis.

However, there is another side of DA—it's the primary neurotransmitter for the brain's reward system. Our brain releases high levels of DA during joyful events, like graduating from high school, winning a race, or enjoying a Thanksgiving dinner. Another experience that can cause a kind of "joy" is abusing drugs. Cocaine and methamphetamine cause the most DA release, leading the user to feel intensely exhilarated and powerful.

While a large release of DA can indeed produce positive emotions, the brain quickly institutes measures to maintain a stable internal environment, or homeostasis. One measure is to quickly clear the DA away, which the brain does by breaking the DA down with enzymes or recycling it. But when someone is consistently using drugs, there's too much DA for this process to work. Therefore, the brain alters itself to make the neurons a little

less receptive to DA. This process is called "desensitization," and it occurs in various ways biochemically, such as decreasing the number of DA receptors or slowing down receptor activation.

As the brain desensitizes to DA, the drug user experiences this as tolerance, meaning the person does not experience the same high from a given dose. If the dose is increased to compensate, the user will get high, but the brain will go through its homeostasis process again, forming tolerance to the higher dose. This is a simplistic neurobiological explanation of tolerance.

What about withdrawal—why does that happen? When there's no external stimulation causing the brain to release DA, the user must depend on the old-fashioned process of the brain releasing DA as it normally would: that is, in response to the prosaic pleasurable events of life, like having a snack or watching a ball game. But a brain that has gotten used to relying on high levels of DA has fewer DA receptors, and those receptors are less sensitive. Therefore, the normal amount of DA doesn't produce much, if any, pleasure compared to what the addict experiences when getting a "fix." When an addict's drug of choice is taken away, a DA deficiency results. This is one reason withdrawal is so unpleasant, and why stimulant withdrawal causes depression. With a damaged reward circuit, it becomes very hard for a user to experience normal healthy behaviors as motivating. The temptation to use drugs is extreme, because the user now feels the drugs are needed simply to feel normal. (For a review of the dopamine theory of addiction, see Nutt DJ et al, *Nature Reviews Neuroscience* 2015;16:305–312.)

Genetics of addiction

Drug addiction often runs in families, though the strength of the development of addiction varies between substances. Familial transmission of substance abuse does not necessarily imply genetic involvement; however, there is in fact a large amount of evidence that genes play a role.

One piece of evidence comes from studies of identical and fraternal twins. The most interesting of these studies compares these two types of twins when they have been separated at birth and put up for adoption. If addiction had nothing to do with genes, but everything to do with upbringing, one might expect that the diagnostic concordance rate of identical and fraternal twins would be the same—but in fact the identical twin concordance rate is higher. Using this kind of data, studies have estimated that the heritability of addiction to alcohol and drugs in general is 60%. This

CHAPTER 5

12-Step Programs

"To this day, I am amazed at how many of my problems—most of which had nothing to do with drinking, I believed—have become manageable or have simply disappeared since I quit drinking."
Alcoholics Anonymous

TAKE HOME POINTS

- Alcoholics Anonymous (AA) and Narcotics Anonymous (NA) are mutual help organizations based on the Twelve Steps.
- Advise patients to try some meetings; attending 2 to 3 meetings per week is associated with the most benefit.
- Although you don't need to memorize the Twelve Steps, becoming familiar with the basics of AA/NA can help you help your patients.
- Encourage patients to do more than just attend meetings—they should also get a phone list and find a sponsor.
- Advise patients to try several different meetings before giving up on 12-step programs.
- Alternative self-help groups are also available.

CASE VIGNETTE: *E is a 28-year-old woman with a long history of severe alcohol and cannabis use. She began drinking in earnest during college, and has had only brief periods of sobriety since then. She was recently placed on medical leave from her job in order to be admitted to a detox and rehab facility. It's clear that this is her last chance to get sober before she is fired. Her previous psychiatrist had prescribed acamprosate to reduce her cravings and recommends that she attend AA meetings. E responds, "I can't do AA. I tried it, and I can't deal with the religious stuff."*

How would you respond to E's reluctance to attend AA? How valuable is it for severe alcohol users to attend 12-step meetings? What are the steps?

INTRODUCTION: AA'S HISTORY

12-step organizations (AA, NA, and others) are the most commonly sought sources of help for substance-related problems in the U.S. All are considered mutual help organizations (MHOs) and have certain features in common: They are self-supporting, open to anyone with a desire to stop substance use, and do not have professional facilitators.

AA, founded in 1935, is the original and by far the most popular MHO, with more than 50,000 meetings a week nationwide. AA grew out of a Christian organization called the Oxford Group, which was founded in 1931 by Frank Buchman, an American Lutheran minister. Buchman had a religious conversion experience on a trip to England, and in 1921, during a visit to Oxford University, he formed a religious fellowship called A First Century Christian Fellowship. By 1931, its name had changed to the Oxford Group. This group had elements that would later be adopted by AA, including a rejection of hierarchies, an emphasis on beliefs rather than religion, and a specific series of stages to follow in order to improve one's life (mirrored later in AA's Twelve Steps).

The eventual co-founder of AA, William Griffith Wilson, was born in 1895 in East Dorset, Vermont, where his parents ran an inn and tavern. At some point, both of his parents abandoned him, and Wilson was raised by his grandparents. Not surprisingly, he suffered episodes of depression from an early age, and also had significant social anxiety. He discovered alcohol in 1917, and it quickly became his constant companion. Though he went to law school, he did not graduate because he was too drunk to pick up his diploma. A subsequent career as a stock broker also ended in shambles.

When an old drinking buddy, Ebby Thacher, visited him in 1934, Wilson was astonished to find that Thacher had been sober for several weeks. Thacher credited the Oxford Group, which had opened branches in New York City and Akron, Ohio, with helping him gain sobriety through Christian fellowship. With his encouragement, Wilson attended a meeting, but continued drinking and soon thereafter was admitted to a detox hospital, where he went into delirium tremens. During that hospitalization, he had a life-changing religious experience in which he saw a flash of light and felt the presence of God.

Wilson never drank again. The following year, in 1935, he met another alcoholic member of the Oxford Group, a physician named Bob Smith,

FUN FACT: AA and the Bronx Cocktail

Bill Wilson had his first drink during military training in Massachusetts. He went to a dinner party and drank some Bronx cocktails (which are classic gin martinis with a splash of orange juice). According to *Time* magazine, he recalled that the drinks liberated him from his shyness: "I had found the elixir of life" (Cheever, 1999).

and the two of them formed a sub-group within the Oxford Group that eventually split off to become AA. In 1939, Wilson published *Alcoholics Anonymous*, eventually known as "The Big Book" because of its heft. Still the basic textbook for AA, it is one of the best-selling books of all time, having sold over 30 million copies.

NA was formed as an offshoot of AA in the 1950s and follows the same principles.

Does AA work?

AA has been a fixture of alcoholism treatment for decades. But does it actually work? The question is hard to answer, in part because it's difficult to do a randomized controlled trial of AA. Such a study would have to track whether patients assigned to AA treatment are attending meetings—a difficult proposition since AA, by definition, is anonymous. Another challenge is that AA groups vary in size, content, and focus, making it hard to define the treatment under study.

This doesn't mean that AA can't be studied—just that doing so requires creative methods. One such method was Project MATCH. This was a large randomized trial in which 1,726 alcoholics were randomly assigned to one of three treatments: cognitive behavioral therapy (CBT), motivational enhancement therapy (MET), and a third treatment that was a stand-in for AA: a psychotherapy, based on encouraging AA attendance, called twelve

CHAPTER 10

Cannabis

"So many writers make dope glamorous; a form of romantic transgression, or world-weariness, or poetic sensitivity, or hipness. Mainly it's the stuff of ritualistic communion among inarticulate bores."
Leonard Michaels

TAKE HOME POINTS

- The psychoactive component of cannabis is THC, and THC content varies widely.
- Long-term use can cause chronic cannabis syndrome, impacting school and career performance and potentially aggravating anxiety, depression, and psychosis.
- Evidence-based medical uses for cannabis are few: intractable nausea, AIDS wasting syndrome, and pain due to neurological spasticity.
- Withdrawal from heavy use can cause nausea, irritability, and insomnia.
- There is no effective medication to treat marijuana use disorder.

CASE VIGNETTE: *J, a 23-year-old male, is referred to me by another psychiatrist. He had started using marijuana at age 13, and began using regularly (several times a week) when he was 18. He had enrolled at a state university, but dropped out after two years. He had suffered anxiety problems, including panic attacks, since high school, and the anxiety had worsened over the past year. J sought treatment for the anxiety through a local mental health clinic, and once his long history of cannabis use was disclosed, the treating psychiatrist referred J to me to help with reducing his use and to evaluate whether it was causing the anxiety.*

ESSENTIALS OF CANNABIS

Cannabis derives from the cannabis sativa plant, a variety of hemp that has been cultivated for much of human history. The leaves can be smoked, though the flowering tops and buds are more potent; a resin from the plant can also be concentrated into hash or hash oil. The main psychoactive component is delta-9-tetrahydrocannabinol (THC), which is one of many cannabinoids in the plant.

THC activates cannabinoid receptors in the brain. Two subtypes have been identified: Type 1 is predominant in the brain and is responsible for most of cannabis' psychoactive properties; type 2 is in the periphery, especially in white blood cells, and has effects on the immune system. There are also some endogenous cannabinoids (called endocannabinoids), such as anandamide, which is named after the Sanskrit word "ananda," meaning bliss. It's not clear why we have this endocannabinoid system, but it is ubiquitous throughout the central nervous system and appears to be part of the normal regulation of various functions, including memory, pain sensation, mood, and appetite (Ligresti et al, 2016).

Immediate effects

Cannabis' immediate effects are euphoria, distortion of one's sense of time, and a feeling of enhanced perception of things like colors and music. Some people experience hallucinations and anxiety, and drowsiness is common. The ability to form new memories is impaired during intoxication, though the ability to recall old memories is not affected. Immediate physiological effects include peripheral vasodilation (responsible for users' bloodshot eyes, which is caused by swelling of blood vessels in the sclera, or conjunctival injection) and elevated heart rate (to some degree, this is also caused by peripheral vasodilation).

Overdoses lead to panic attacks, psychotic symptoms, palpitations and tachycardia, and occasionally shortness of breath and chest pain.

Long-term effects

Chronic cannabis syndrome, formerly known as amotivational syndrome, results from long-term regular use of cannabis—especially use during adolescence, which is a vulnerable time for the developing brain. When the immature endocannabinoid system is repeatedly exposed to THC, there

FUN FACT: An Anti-Munchies Drug

It's well known that cannabis causes increased appetite, otherwise known as the munchies. Pharmaceutical companies have taken advantage of this property. For example, the synthetic THC agent dronabinol (Marinol) is FDA-approved for appetite restoration in AIDS wasting syndrome. Scientists have also developed cannabinoid receptor antagonists as potential weight loss agents, and in 2006, Sanofi Aventis launched a specific CB-1 antagonist called rimonabant (brand name Acomplia) (Pi-Sunyer et al, 2006). Acomplia was first sold in Great Britain, with plans for eventual FDA approval, but reports of depression and suicide as apparent side effects caused it to be withdrawn from all markets.

can be long-term subtle effects on learning and adaptation. This may be caused by interference with the normal process of neural pruning during adolescence (Lubman et al, 2015).

Chronic cannabis syndrome has two components: reduction in the ability to process and remember new information and skills, and lessened motivation for achievement in general. This can hinder educational and career trajectories, causing a user's IQ to not meet that of age-matched peers.

In addition to these long-term cognitive effects, cannabis can worsen a range of mental psychiatric symptoms. These include anxiety, with an increased frequency and intensity of panic attacks, as well as depression. Most alarmingly, there is an association between early cannabis use and development of schizophrenia and other psychotic disorders. The risk for psychosis increases with younger age of initiation and a family history of any psychotic disorder or major mental health disorder. A recent review estimated that chronic cannabis use is associated with a twofold increase in risk of developing schizophrenia; however, the causal link is not established (Gage et al, 2016).

Physical effects

Lungs. Inhaling smoke from any source exposes the lungs to potentially toxic material, such as particulate matter or carcinogens. It can worsen asthma and potentially cause chronic obstructive pulmonary disease. Unlike tobacco cigarettes, cannabis joints do not have filters, allowing more contaminants to enter the lungs. On the other hand, people generally

smoke fewer joints than tobacco cigarettes, somewhat mitigating this problem.

Heart. Regardless of how it is ingested, cannabis can raise the risk of heart attacks in people with preexisting ischemic heart disease. The mechanism for this risk is cannabis-induced tachycardia, which raises myocardial oxygen demand. This risk is greatest within the first 20 minutes of starting use.

Immune system. Regular cannabis use can reduce immune function, which is most problematic in those who are immunosuppressed, such as people with HIV. Patients who use cannabis during or immediately after receiving chemotherapy for cancer are at risk. Not only can cannabis further reduce immune function, but processing of cannabis is not regulated by the FDA—unlike dronabinol, which is FDA-approved for nausea due to chemotherapy—so cannabis products may contain fungi (especially Aspergillis) and other microorganisms that can lead to opportunistic infections during periods of immunosuppression.

Fertility. Cannabis can reduce fertility in both men and women. It lowers sperm counts in men, and long-term or heavy use can lead to irregular menstrual cycles in women.

Withdrawal

Cannabis withdrawal syndrome is well established and is more psychological than physiological. Symptoms include depression, irritability, appetite suppression, and headaches. In heavy users, there may be diarrhea and other intestinal discomfort, including nausea and vomiting. Cannabis withdrawal symptoms may last 3–7 days, depending on the amount of prior use.

A GUIDE TO CANNABIS PRODUCTS

As cannabis has become legal in many parts of the country, the cannabis industry has grown and the products available have multiplied. As a practitioner, you should be familiar with the major types of products, because they vary significantly in potency, duration of action, and safety. Here is a quick primer of the current state of cannabis products (see Table 10-1 for a quick reference).

Smoked products. The most common and familiar form of cannabis products are smokable joints or blunts, although water pipes and vaporizers are

TABLE 10-1. Available Cannabis Products

Product	Description	Notes
Smoked products (aka joint)	Cannabis flowers, leaves, and/or buds rolled in thin paper and smoked like a tobacco cigarette	Simplest and most common way to use cannabis
Blunt	Cannabis rolled with tobacco in a cigar	
Skunk	Cannabis plant bred with higher THC concentration	British slang term
Water pipe (aka bong or hookah)	Device to filter smoke through water during the smoking process	Paraphernalia for smoking; illegal in some states
Vaporizer	Type of device that uses a battery to heat and vaporize cannabis for inhalation	Paraphernalia for smoking; illegal in some states (an e-cigarette is a small, portable vaporizer)
Hash oil	Cannabis plant matter soaked in a chemical solvent to extract concentrated THC resin	Can be smoked, vaped, or ingested orally
Edibles	Wide variety of food products infused with hash oil and consumed orally	Baked goods and candies; may have 5 mg–100 mg of THC per product
Shatter (aka butane honey oil, dab, or wax)	THC resin extracted from cannabis plant with butane as the solvent	Flammable; may explode during manufacturing or storage

also used. In the 1960s and 1970s, the THC content in cultivated cannabis plants was in the low single digits. However, newer strains have THC levels in the 10%–20% range. There are also other varieties called "skunk," which are much more potent.

Hash oils. These are typically ingested using electronic vapor delivery

CHAPTER 11

Opioids

"I'd felt the pop of the needle sliding into my veins, like a fang into flesh. I'd been enveloped in the golden haze where nothing is wrong even when everything is falling apart. A dance with a hypodermic fiend, my hands in the claws of a vulture."
Taylor Rhodes, *Sixteenth Notes: The Breaking of the Rose-Colored Glasses*

TAKE HOME POINTS

- Assess the severity of the opioid problem.
- Use outpatient tapering for patients prescribed opioid analgesics for a specific pain diagnosis.
- Use intensive detox for patients with more severe addictions and those with comorbid medical or psychiatric disorders.
- Use methadone for patients with limited recovery resources, multiple previous relapses, no health insurance, or a need for close monitoring due to history of overdose or diversion.
- Use buprenorphine for patients with access to recovery resources, such as a therapist or Narcotics Anonymous groups, as well as health insurance or adequate financial support from a job or family.

CASE VIGNETTE: *F is a 23-year-old female, self-referred. She says her parents pressured her to make an appointment and are threatening to kick her out of the house because they found her stealing her father's Percocet tablets. F dropped out of college after completing her sophomore year and has been living with her parents ever since. She has had several jobs, but has been repeatedly fired for various reasons. She says her parents think*

she has a drug problem, but she disagrees. What are your initial guesses as to the extent of F's opiate problem? How would you proceed with your diagnostic assessment?

ESSENTIALS OF OPIOIDS

First, let's clarify the terminology. What is the difference between opioid, opiates, and opium? In modern parlance, "opioid" is a comprehensive term that includes every single substance that stimulates opioid receptors in the brain. This includes both natural substances (eg, derived directly from the opium poppy, such as morphine) and synthetic substances (eg, created in a lab, such as hydrocodone). "Opiate" is often used interchangeably with opioid, though many use the word opiate specifically to refer to natural substances derived from opium—as I do in this chapter. "Opium" is the specific sticky residue that can be extracted from the opium poppy. Opium has been in use since as early as 4000 BC.

I've created a table to allow you to quickly compare any of the commonly used types of opioids (see Table 11-1). For now, here's an overview.

Morphine and codeine are natural components of the opium poppy, and both were discovered in the early 1800s. Morphine was widely used as an analgesic during the U.S. Civil War. Most subsequent opioids (including heroin, hydrocodone, oxycodone, and buprenorphine) are termed "semisynthetics," meaning they were all originally derived from one of opium's components. Finally, there are more modern opioids synthesized from basic chemicals in labs, such as methadone, fentanyl, and tramadol. Generally, synthetic opioids have been developed with higher potency or longer duration of action.

Regardless of the origin, opioids are full mu-opioid receptor agonists with very similar effects (analgesia, euphoria, sedation) and side effects (nausea, constipation, itching). Differences between these substances are due to lipid solubility (which affects the rate at which a substance crosses the blood-brain barrier to produce the main opioid effects) and metabolism (which affects a substance's duration of action).

The only illegal opioid is heroin (DEA Schedule I). Other opioids are found in schedules II, III, or IV, depending on how widespread an opioid is, its potential for abuse, and other factors. Drugs are sometimes switched to different schedules as conditions change. For example, hydrocodone

was reclassified from Schedule III to Schedule II based on epidemiologic data indicating that it was more likely to be abused when less restricted, and tramadol was initially unscheduled, but after long-term post-marketing surveillance, it was placed on Schedule IV in 2015.

The popularity of heroin as a drug of abuse peaked in the 1960s, but declined somewhat during the 1970s and 1980s due to greater awareness of the risks of overdose and the increased popularity of cocaine. Throughout the 1990s and 2000s, liberal prescription of opioid analgesics for acute and chronic pain led to diversion for illicit use. Heroin use has again become a growing epidemic, because authorities have taken measures to clamp down on prescription opioid abuse, and heroin is cheaper and readily available on the streets.

Over 2 million people in the U.S. have abused prescription opioids, and the trend had been increasing throughout the 2000s, although the rate of use is starting to plateau. Around half a million people currently use heroin, but these numbers are increasing along with the numbers presenting for treatment of opioid use disorder.

CLINICAL PEARL: Side Effects to Motivate Patients

Many users don't realize that opioid use causes irregular periods or erectile dysfunction. By pointing out these effects, you can often generate more motivation to quit: "You may not realize it, but your irregular periods have a specific cause, which is your opioid use."

Immediate effects

Psychological effects: euphoria, tranquility, and a mild sleepiness. Opioid-induced drowsiness is referred to as "the nod" because users easily nod off to sleep with their chin lowering to their chest, then startle awake—like drowsy students listening to a boring lecture.