Chapter

Management of Co-Occurring SUD and Chronic Pain

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Abstract

Though there has been a 44.4% decrease in the number of prescriptions written for opioid analgesics between the years 2011–2020 in the United States, drug overdose rates continue to climb sharply, reaching nearly 107,000 for a prior 12-months period as of early 2022, driven primarily by the use of illicit opioids. It is estimated that 80–90% of individuals with a substance use disorder (SUD) receive no treatment, and for those with opioid use disorder (OUD) who do find their way to treatment, less than half are offered potentially life-saving medication. Contemporaneously, chronic pain is one of the most common and most disabling health conditions, and frequently involves complex decision-making between the patient and the health care team regarding the treatment approach. Though prescribing trends have ebbed in recent years, opioids continue to be the most prescribed class of drug in the United States despite well-publicized associated harms. It is more critical than ever that stakeholders urgently work to facilitate and destigmatize evidence-based substance use disorder treatment, and promote safe, effective, and holistic care pathways for patients suffering from chronic pain.

Keywords: chronic pain, opioid use disorder, prescription opioids, substance use

1. Introduction

The "opioid crisis" as a major public health problem in the United States has been prominently recognized in medical literature and the press for at least a decade. Prompted by a variety of factors, the first of three defined "waves" of the crisis began with a dramatic increase in written prescriptions for opioid analgesics starting in the mid-1990's [1]. In lockstep with the surge in opioid prescribing, there was a coincident fourfold increase in overdose deaths and admissions to substance use disorder treatment programs. In 2015, more than 33,000 Americans died of opioid overdoses, and an estimated 2 million individuals suffered from substance use disorders related to prescription opioids [2].

There were multiple factors contributing to a threefold increase in the number of opioid prescriptions written in the United States between 1999 and 2011 [3]. The first relates to changing clinical practice norms, where indications for use of opioids for the treatment of chronic non-cancer pain widened significantly in response to escalating concerns about perceived widespread undertreatment of chronic pain. For example, at that time, the American Pain Society in its 1996 guidelines encouraged providers

to assess pain as "the fifth vital sign" at each clinical encounter to avoid failing to actively address patients' pain symptoms. At the same time, aggressive marketing of opioids by pharmaceutical manufacturers, particularly the makers of Oxycontin™ following its 1996 FDA approval, served to amplify and drive this shift in prescribing culture, with sophisticated methodologies to increase provider prescribing patterns, and with the dissemination of now-discredited scientific information alleging low risk of misuse or addiction related to prescription opioids [4]. Payers limited access to other evidence-based pain interventions such as multidisciplinary pain rehabilitation, bodywork treatment, and psychological counseling, and these modalities of treatment thus became more difficult to access, increasing patients' reliance on pharmacotherapy, and doctors' willingness to prescribe opioids. Taken together, these factors created a prescribing climate which normalized opioids as a common treatment for chronic non-cancer pain [3].

Alongside the challenge of containing the harms caused by overprescription of opioid analgesics exists the very high prevalence and burden of suffering from chronic pain. The debilitating effects of chronic pain on quality of life involve not only physical symptoms, but also emotional well-being, identity, and interpersonal relationships [3]. Among patients with chronic pain who are newly prescribed opioids for longer than 90 days, approximately 6% develop OUD, with the likelihood increasing dramatically with an escalation in dose and extended duration [5]. In 2016, the Centers for Disease Control and Prevention issued guidelines for prescribing opioids for chronic pain which recommended soft limitations on dose levels to lower than 90 mg morphine equivalents per day (MED), and implementation of clinical practices to assess and prevent harm to use and misuse, such as urine toxicology screening and use of state prescription monitoring program [6]. This document strongly influenced prescriber practices nationwide, contributing to a general decrease of 40–60% in aggregate opioid prescriptions across the US from peak levels in the mid-2000s. Unfortunately, not all consequences of this shift in prescribing patterns have been positive. Many patients who had been using opioids over the long term, especially at higher doses, were forced by their prescriber to undergo dose tapering or discontinuation despite evident clinical stability, due to misapplication of the CDC guidelines, with associated harmful adverse events and outcomes, including increased rates of illicit drug use, emergency department visits and hospitalizations, and overdose deaths [7]. There have been recent attempts to better understand the disruptive power these changing norms and policies have continued to have on patients' pain management experiences [8].

This change in prescribing practices and the resultant barrier to patients receiving prescription opioids legitimately for pain was exacerbated by abrupt closures of "pill mill" clinics by law enforcement. These clinics operated on a profit-based model and provided substandard care and monitoring while prescribing outsized quantities of opioids and other controlled substances. As a result of these factors, proactive networks of drug traffickers primarily from Mexico were able to capitalize on a ready-made source of demand for cheaper and more reliably available illicit opioids, ushering in the second wave of the opioid crisis by 2015: increased heroin use [1, 9]. Sources from Mexico accounted for 90% of the US heroin market share by 2016 [9, 10]. Within a few more years, fentanyl, a synthetic opioid that is much cheaper, more potent, and easier to manufacture and distribute than heroin, took over the illicit opioid market. Showing no signs of slowing, fentanyl-related overdose death rates have only worsened as the so-called third wave of the opioid crisis continues [1].

In this context, management of chronic pain in the US has undergone systematic re-examination to elucidate best practices for patient safety and treatment effectiveness [8]. Elsewhere in the world, countries are attempting to learn from the mistakes of the US and prevent or mitigate major prescription opioid-related public health problems [11]. As providers are regularly tasked to straddle the intersecting realms of chronic pain disorders, substance use and mental health disorders, treatment approaches must emphasize evidence-based, compassionate, inclusive, patient-centered, safe, and sustainable interventions. Effective solutions to the crisis will include not only optimization of care delivery, but also relevant non-clinical aspects such as the roles of law enforcement and drug marketing and regulation [12]. Some countries have already made a marked shift in policy toward harm reduction, such as Canada, which has provided safe consumption sites for drug users, and the Netherlands and Portugal, which have decriminalized all drug use in favor of approaches emphasizing safety and access to treatment [13]. Though these measures have improved opioid-related death rates, countries outside the US have seen use of prescription opioids grow substantially (47% increase in Europe between 2004 and 2016) even though it is illegal to market drugs directly to patients in Europe and other parts of the world [13].

In this chapter, we will review the complex intersection of SUD and chronic pain disorders in several subcategories: 1) understanding the scope of the problem and its neurobiological underpinnings, including risk factors, and biopsychosocial mechanisms of chronic pain and substance use disorders; 2) the controversial role of opioids in chronic pain care; 3) best practices in chronic pain and SUD treatment.

2. Key concepts in understanding Co-occurring chronic pain and SUD

2.1 Epidemiology and neurobiology

The prevalence of either regional or widespread chronic pain in adults has been estimated at 30% [14]. About 20% of US adults (50 million people) report a moderate to severe level of pain which affects their daily quality of life and activity, while in the United Kingdom, chronic pain is estimated to affect 20–50% of the adult population. Worldwide, chronic low back pain is the single leading cause of disability across all age ranges, genders, and demographics [15]. Several other chronic pain conditions including chronic headache and peripheral joint pain from arthritis are also in the top 10 causes of disability worldwide. Factors that are consistently associated with disability from chronic low back pain include older age, poor general health, increased psychological or psychosocial stress, worse baseline functional disability, sciatica, and the presence of compensation related to disability [5]. Social determinants of health that are known to predict poor outcomes related to disability from chronic low back pain include low socioeconomic status and/or low income, unemployment, and occupational factors such as lack of adequate support staff, manual lifting, and frequent overtime work hours [16].

The prevalence of opioid use disorder (OUD) in patients with chronic pain has been notoriously difficult to determine with specificity. Through the historical lens of the opioid crisis, it was dramatically underestimated in the years preceding and including the spike in opioid prescribing starting in the 1990s. In a letter that was published in 1980 in the New England Journal of Medicine, authors Porter and Jick described a crude study in which charts were pulled for patients in a hospitalized setting who had been given opioids for a variety of indications, with neither dosing

nor duration of treatment identified. The investigators concluded that only 4 of these 11,882 hospitalized patients that were treated with an opioid medication were subsequently diagnosed with an "addiction". Unfortunately, this study was widely cited as "proof" that there was only about a 1% risk that a patient treated with opioids for pain (even in an outpatient setting, even long-term) would develop opioid addiction. More recently, it has been recognized that the real prevalence of OUD is much higher, with various estimates ranging from 3.2 to 27%, with most of these estimates falling in the 20–25% range [17]. Though the level of prescription opioid use, and opioid-related deaths in most European countries and worldwide is still much lower than that of the US, as noted above Europe has seen a steady increase in prescription opioid use over the past 15 years, mainly due to increased tramadol, fentanyl, and oxycodone prescriptions; there are calls for proactive investigation into these trends and their potential subsequent harms [11].

Over the past several decades, significant progress has been made in understanding the neurobiology of pain and addiction. CNS receptor binding targets and associated neural circuitry has been elucidated to explain the rewarding effects of substances with known abuse potential. Moreover, we know that repeated drug exposure over time causes adaptations in the brain's reward pathways which are evident even on a gross structural level via neuroimaging [17]. Affected areas include the following:

- *the limbic system*: contains the brain's reward circuitry, leading to a drive to repeat behaviors that activate this pathway, such as using drugs

- *the brain stem*: controls basic functions critical to life such as heart rate and breathing

- *the cerebral cortex*: controls functioning via multiple sub-regions that govern thinking, feeling, sensory experience, motor coordination, planning, and decision-making.

Substances with potential for abuse that affect these areas of the brain, such as alcohol, opioids, cannabis, stimulants, sedatives, and nicotine, enhance specific brain neurochemical pathways in ways similar to that produced by other natural rewards such as food and sex, but in a sometimes more acutely intense and prolonged manner. Conversely, when long-term use of such substance is interrupted, a pronounced sense of dysphoria is typically experienced, which has been referred to as *hyperkatifeia* [18]. The threat of this unpleasant state produces a desire to avoid interruption in use, due to the behavioral negative reinforcement that the undesired state exerts over the individual, who otherwise recognizes the benefits of stopping the behavior and genuinely prefers and seeks to do so. In addition, the rewarding effect of use is itself diminished with prolonged use as tolerance is developed to the effects of the substance, and the ability of natural rewards to activate the reward pathways is likewise compromised. In this fashion, over time, these substances strongly influence the choices and behavior of the individual [19].

Chronic pain is best viewed as a distinct diagnosis and medical condition, with its own definition and taxonomy [14]. It has been associated with multiple physical, psychological, and social factors which affect its level of impact on a given individual [15]. The diagnosis of a chronic pain disorder can be made based on objective evaluation, as with an imaging test or a blood test, for example, with relatively clear etiology and a specifically identifiable pain generator. However, even in cases where the diagnosis

is clear, patients with very similar objective findings might have a completely different "pain experience" based on other less quantifiable factors. These include:

- their level of central nervous system-mediated pain sensitivity or pain tolerance, which itself may be influenced by both genetic and environmental factors;
- their current psycho-emotional state and prior mental health history;
- the effect of the medications or substances they are using on pain processing pathways;
- other factors which can influence pain signaling and processing in the central nervous system (CNS) such as the history of trauma or adverse childhood experience (ACE), and attitudes/beliefs about pain [6].

Moreover, many chronic pain disorders are not typically associated with specific identifiable anatomic pathology. In the case of chronic low back pain, 85–95% of patients presenting to primary care providers do not have a clearly identifiable etiology for their symptoms [5]. Some pain syndromes are inherently caused or defined by a CNS-mediated pain state, such as fibromyalgia (prevalence estimated at 2–8% of the population). Other examples of this include headache syndromes, irritable bowel syndrome, temporomandibular joint syndrome, and interstitial cystitis. It should be noted, however, that what may have started as a focal, well-defined pain condition, such as low back pain from degenerative disc disease or lumbar facet arthropathy, may become a chronic, CNS-mediated pain state, such as in the case of failed surgical back syndrome [20]. Thus, it is incumbent upon care providers to recognize the complex role of the CNS in all chronic pain states, and to utilize treatment approaches that address the patient as a whole person rather than just as a structural or anatomic abnormality [21].

Pain can generally be subdivided into three general types, including nociceptive, inflammatory, and neuropathic [12]. Nociceptive pain is our bodies' sensory response to an actual painful stimulus, divided further into visceral (such as gastrointestinal) and somatic (such as musculoskeletal) pain. Inflammatory pain is a biological response within the body to facilitate tissue repair due to injury and can be either acute or chronic in nature. Acute inflammatory pain is exemplified by a sprained ankle, whereas chronic inflammatory pain is exemplified by osteoarthritis of a peripheral joint such as the hip. Neuropathic pain is typically defined by nerve injury or impairment leading to central pain sensitization (defined below), which results in a persistent pain response without a stimulus and is generally pathologic or maladaptive because it does not serve a useful purpose. Both the CNS and the peripheral nervous system (PNS) are involved in all 3 types of pain. The PNS comprises nerves and ganglia outside the brain and spinal cord, which define the CNS.

An individual's response to pain signals can be viewed in a general sense as either adaptive or maladaptive, with their emotional state playing a key role in this determination. In a person with normal pain sensitivity, pain signals from a twisting back injury would be transmitted from the dorsal horn of the spinal cord through ascending spinal pathways to be received by the brain, and the signals would then be modulated by descending inhibitory interneuron signals which serve to dampen the severity of the excitatory pain signals, in accordance with the now-classic gate control theory of pain as first proposed by Melzack and Wall in 1965, still supported in concept by the International Association for the Study of Pain [5]. Facilitating less

distress and greater functional capacity, this normal sequence of pain processing is considered adaptive to the organism and species.

The same pain stimulus could be processed quite differently for a patient with chronic pain. Due to the emotional response caused by chronic pain signals, the inhibitory interneuron modulation of the descending pathway could be decreased, which leads to an increase in relative excitatory pain signaling input, with some relief coming from accompanying increase in endorphin release from the periaqueductal gray (PAG) and the dorsal horn of the spinal cord. Over time, via a process termed central sensitization, pain processing in the CNS recalibrates to adjusted thresholds for modulation of both ascending excitatory and descending inhibitory signals, resulting in the patient experiencing greater baseline pain sensitivity, and a higher level of distress with exacerbations of pain. The affective or emotional component, then, is recognized to have a significant influence on the ongoing physical experience of pain [22]. Moreover, this increased baseline pain sensitivity does not serve a functional purpose and would therefore be considered maladaptive [5].

When exogenous opioids are added longitudinally to this scenario, there is an accompanying decrease in the production and release of pain-relieving endogenous opioids from the PAG and dorsal horn, resulting in an increase in pain signaling along the ascending pathway. In addition to these effects on pain processing, prolonged opioids will also affect the pain experience by influencing the brain's limbic system (emotional circuitry, see below) and sleep patterns. These gradual changes in the CNS persist, even long after the opioid has been discontinued [19]. Such long-term CNS changes clearly have major implications in designing and implementing effective treatment approaches for both chronic pain and opioid use disorder/opioid physiologic dependence.

Despite the gains in knowledge and active investigation into the neurophysiology of pain and addiction, there is still a great deal that is incompletely understood. Much of our knowledge comes from laboratory and animal studies rather than from the actual patients who present for care. The influence of environmental factors such as trauma, for example, has been studied and shown to be quite impactful on the development of pain sensitivity, cognitive capability, memory, emotional resilience, and the likelihood of developing a SUD [17]. Also, very influential is the role of organic mental illness on the behaviors and brain function of a patient with chronic pain. In addition, issues such as genetics, social/housing instability, discriminatory disparities in accessing health care, and others, are known to greatly affect clinical outcomes for all patients experiencing chronic pain and addiction [6].

2.2 The duality of opioids: a brief review of general opioid pharmacology

Opioids have played a central role in the relief of human suffering for millennia; they are also a direct cause of great harm. They are a class of prescription drugs derived from the opium poppy plant, some directly and some via laboratory synthesis using similar chemical structures. They contain chemicals that can relieve pain and relax the body. They can also produce a euphoric effect which gives them potential for misuse. Further, they can induce depression of the respiratory drive, leading to an overdose death. For these reasons, prescription opioids are regulated by the Drug Enforcement Agency of the US government, and by analogous agencies elsewhere. Some common examples of prescription opioids are oxycodone, morphine, hydrocodone, codeine, tramadol, fentanyl, buprenorphine, and methadone. In individuals with co-occurring chronic pain and SUD, consideration of treatment with the use of

opioids or other controlled substances that have the potential to be misused and cause harm is a common clinical dilemma for health care providers, as well as a quality assurance and risk management issue for policymakers. In the years since the advent of the current opioid crisis, which as noted above began with exponential increases in the widespread use of prescription opioids to treat chronic non-cancer pain, there has been increased effort to define best practices for dispensing prescription opioids for appropriate indications, for appropriate patients, and for the appropriate length of time [23].

Opioids can be classified in various ways, such as by potency, half-life, opioid receptor activity, or specific opioid chemical class. It is useful to start with categorizing opioids based on the way they are synthesized:

- *endogenous opioids*: opioid peptides produced organically by the body itself; examples are endorphins, enkephalins, dynorphins.

- *exogenous non-synthetic opioid agonists*: opioids derived from the opium poppy plant; examples are codeine, morphine, thebaine, diacetylmorphine (heroin).

- *exogenous semi-synthetic opioid agonists*: opioids derived from the poppy plant that have been chemically altered in the laboratory; examples are oxycodone, hydrocodone, hydromorphone.

- *exogenous synthetic opioid agonists*: opioids that are synthesized entirely in a laboratory; examples are methadone, fentanyl, buprenorphine.

Opioid pharmacodynamics, or the activity of the drug at the opioid receptor and the resultant physiologic and clinically relevant response, is at the heart of the question of how these drugs can be safely and effectively administered by clinicians [24]. Potency is certainly a key factor in this equation, and it is important that medical providers have a solid understanding of the relative potencies and basic pharmacologic properties of various opioid medications. Ranging from opioid partial agonists such as tramadol to high potency full agonists such as fentanyl, these potencies are classified by the World Health Organization on their "Analgesic Ladder" [11]. Of note, opioids with the highest potency also have the greatest addiction liability and highest risk of overdose death. The recent emergence of illicit synthetic analogs of fentanyl on the street has exposed users to levels of potency not seen before in clinical medicine; carfentanil, one such analog, is 100 times more potent than prescription fentanyl, which is itself 100 times more potent than morphine [25]. It is therefore not surprising that even users who are very experienced with other less potent opioids are highly susceptible to overdose when using substances containing illicit fentanyl.

Other important factors influencing the clinical effect of an opioid besides potency include half-life and route of administration. Typically, short-acting opioids have a more rapid onset and decay, which can be useful in treating acute pain, but can frequently create a pattern of unstable levels of drug when dosed repeatedly, resulting in both positive and negative reinforcement driving continued use. Moreover, use of short-acting opioids leads to more rapid neuroadaptation, or CNS sensitization/homeostasis, whereby the stronger the opioid and the more rapidly it reaches the brain, the greater the neuroadaptive response [26]. This neuroadaptation is what leads to development of tolerance and physiologic dependence, which tend to occur much earlier with short-acting opioids than with longer-acting opioids.

Longer-acting opioids are typically used only in chronic rather than acute pain. Many such drugs are simply "extended-release" versions of short-acting opioids with an insoluble substance matrix that delays intestinal absorption and metabolism. Others, such as methadone and buprenorphine, are long-acting by design, primarily due to their high affinity for the opioid receptor. Though long-acting opioids may not be quite as reinforcing as short-acting opioids, clinicians should not assume that they are safer or less addictive than short-acting opioids. Importantly, long-acting opioids such as the prior version of Oxycontin™ were commonly tampered with by misusers to instantly produce high levels of the short-acting form of the drug, oxycodone. Abuse-deterrent extended-release formulations have helped to curb this widespread misuse technique (Oxycontin was reformulated in 2010), but not all available long-acting opioids have this technology. Notably, long-acting formulations have been associated with higher rates of overdose, in part due to the above, as well as the fact that combining a long-acting opioid with a CNS depressant such as alcohol or a prescription sedative may be more likely to produce a lengthier and therefore deadlier respiratory depression effect. Even without a second substance, accumulated dosing of long-acting opioids taken at higher levels of frequency than prescribed presents elevated overdose risk, particularly at the initiation stage of use. It typically takes roughly 5 half-lives of a long-acting drug to achieve steady-state in the circulation, which amounts to five full days with methadone, and patients who are impatient to achieve either pain relief or euphoric effect may increase dosing frequency before steady state is reached and before protective tolerance to the drug has begun [27].

2.3 The role of opioids in the treatment of chronic pain

Regarding the controversial therapeutic value of opioids in the treatment of chronic non-cancer pain, there is very little high-quality research evidence. In a widely publicized 2018 JAMA study known as the SPACE trial, done in a Minnesota Veterans population, opioid-naïve patients with chronic back pain, or hip or knee osteoarthritis, were placed on a 12-months period of medication management and randomized to either opioid or non-opioid medications. The primary outcome was pain-related function, measured via Brief Pain Inventory scale; secondary outcomes were pain intensity and medication-related symptoms. The trial results showed no difference between the two groups in pain-related function, lower pain intensity in the non-opioid group, and more common adverse medication-related symptoms in the opioid group. The authors concluded that treatment with opioids was not superior to treatment with non-opioid medications for improving pain-related function over 12 months for moderate to severe chronic back pain or hip or knee osteoarthritis pain [28].

There are other views in the literature more favorable to the appropriate use of prescription opioids for chronic non-cancer pain, particularly in the current context of the ongoing escalation of overdose deaths driven primarily by the use of illicit opioids rather than prescription opioids. In a 2021 analytic review of evidence, Nadeau argued that restricting physicians from prescribing opioids for reasonable indications is a "failed strategy", opining that it was "pill mill" clinics rather than the average medical provider that was responsible for flooding much of the country with large supplies of prescription opioids, prior to a widespread law enforcement crackdown [25]. These clinics not only provided voluminous quantities of opioids, but they also delivered substandard care without appropriate support and supervision, which exacerbated the risk of misuse, diversion, and development of substance use disorders. These effects were prevalent not only in places with large local pill

mill distribution networks, but also in more remote areas that were perhaps more susceptible to the lure of opioids because of poverty, mental illness, hopelessness, and other psychosocial factors [29]. According to Nadeau, well-designed studies have demonstrated that the annual case fatality rate attributable to prescribed opioids >100 mg daily morphine-equivalent dose (MED) is in the vicinity of 0.25% per year, which is similar to the risk of death from anticoagulation for stroke prophylaxis for a patient with atrial fibrillation. The twin crisis of high-impact chronic pain, which as noted affects approximately 20 million US adults and an estimated 30% of adults worldwide, necessitates a balanced approach. It remains unclear, however, whether future studies of the effectiveness of long-term use of opioids to ameliorate pain and improve functioning will support or refute the arguments of those who advocate for loosening current restrictions on the prescription of opioids for chronic pain.

Prescription opioid misuse is defined as use of the opioid "in any way other than how the provider directed the use, including greater amount, greater frequency, greater duration, using it for an effect other than intended, using someone else's medication, or using via unauthorized route of administration" [30]. In a 2017 "review of reviews" on chronic pain and opioid misuse, the prevalence of chronic non-cancer pain in individuals known to be misusing prescription opioids is estimated at 48–60%, which is substantially higher than the prevalence of chronic pain in the general population (11–19%) [31]. This finding highlights chronic pain as a major driver of opioid misuse. Reviews were noted to be commonly compromised by limitations including inconsistencies, imprecision, and lack of standardized assessment instruments and definitions of SUD, misuse, addiction, and abuse. They cited an overall lack of high-quality evidence on prevalence, risk factors, optimal clinical assessment, and treatment approaches related to co-occurring chronic pain and substance misuse.

2.4 Opioid use disorder vs. prescription opioid dependence

Development of physiologic dependence on a substance should be viewed as distinct from the development of a substance use disorder (SUD) as defined by DSM-5. Tolerance to a substance is typically expected with prolonged use, and withdrawal symptoms upon cessation of the substance are also part and parcel of physiologic dependence, even without aberrant drug-taking behavior, misuse, craving, or other behavioral components more consistent with substance use disorder [32].

Patients who fall into the category of longtime users of prescription opioids for pain who have developed associated tolerance, physiologic dependence, and fear of physical and emotional distress and withdrawal symptoms when presented with the idea of tapering, have been classified as having a variant of dependence to opioids called complex persistent opioid dependence [33]. This is a distinct phenomenon from opioid use disorder. It is also distinct from simple persistent opioid dependence, in which the patient may have developed tolerance and physiologic dependence but does not approach the idea of tapering or dose adjustment with the degree of fear and/or resistance as does the patient with complex persistent opioid dependence. Given the sheer volume of patients classifiable in one of these two categories, providers in the era of the opioid crisis have been faced with the challenge of how to safely and humanely help them reduce their opioid usage, if/when indeed that is an appropriate and patient-centered goal to pursue. As noted previously, there are significant known harms that can come to patients for whom tapers have been non-consensual, overly rapid, and/or unskillfully executed [7].

3. Screening chronic pain patients for risk of opioid misuse and other substance use

3.1 Factors that contribute to opioid misuse and examples of validated screening tools

Given the need to balance the risks and benefits of use of opioids for chronic pain, how do we select who should receive an opioid prescription in any given clinical scenario? One key aspect of the answer lies in the determination of relative risk of misuse of the prescribed drug, which is dependent on multiple factors: drug factors, provider factors, and patient factors [34].

3.1.1 Drug factors

The relative degree of euphoric or reinforcing effect of a drug depends primarily on the rapidity of its onset of action, and secondarily on both its potency and its half-life. The more quickly a substance reaches the brain and causes dopamine release, the more abuse potential and street value it tends to have.

3.1.2 Provider factors

If the first wave of the opioid crisis taught us anything, it is that most health care providers receive inadequate training in the appropriate prescribing of controlled substances, particularly in scenarios involving acute or chronic pain, anxiety and depression, insomnia, and substance use disorders [23]. The American Medical Association (AMA) has classified these practitioner-based inadequacies as to the "4 D's" (to which 2 additional have subsequently been added):

- **Dated practitioners** whose knowledge of best patient care practices is out of date, leading to inappropriate prescribing choices.
- **Deceived or Duped practitioners** are easily misled by drug-seeking patients who report symptoms or conditions indicating an accepted indication for a controlled substance.
- **Disabled practitioners** have their own psychiatric or medical issues, which may include a substance use disorder, and impair their judgment regarding appropriate prescribing and monitoring.
- **Dishonest practitioners** are typically motivated to prescribe controlled substances by the money they will obtain with this practice; this group is thought to be quite rare in the overall current provider population but during the heyday of "pill mill" clinics, numbers were likely greater.
- **Defiant practitioners** believe they have greater knowledge or expertise than others in a specific practice area and practice in ways that are not supported by evidence on the topic.
- **Distracted practitioners** are overwhelmed by patient care, documentation, and administrative duties and are inadequately attentive to providing safe care and monitoring for patients whom they are treating with controlled substances.

3.1.3 Patient factors

There will always be a proportion of patients who misuse prescription opioids and other controlled drugs for non-medical purposes or will divert them to others for intended profit or for non-medical use. However, it is incumbent upon the modern practitioner to use validated screening tools to try to mitigate and avoid contributing to these potential behaviors when considering initiating the prescription of a controlled substance, and at regular intervals such as every 1–2 years for those using them in a stable and responsible fashion. Specific validated tools and questionnaires are easy to implement in clinical practice and can be filled out by the patient as they wait in the exam room for the practitioner to start the visit. It should be noted that these questionnaires rely on honest self-reporting by the patient, an inherent limitation on reliability. Commonly used validated tools include the following:

- **Pain Medication Questionnaire**: 26-item survey that predicts future opioid misuse and stratifies patients into low, medium, and high risk. It has been validated with good sensitivity and specificity [34].

- **Screener and Opioid Assessment for Patients with Pain-Revised** (SOAPP-R) and **Common Opioid Misuse Measure** (COMM) were cited in a large systematic review to be helpful in the treatment of chronic pain for patients who are either already on long-term opioid therapy and being assessed for recent behavior (COMM), or who are being considered for potential initiation of opioid therapy (SOAPP-R) [34].

- **Opioid Risk Tool**: designed to predict which patients who are prescribed opioids will develop aberrant drug-taking behaviors. Developed in 2005, a 2013 study did not validate its predictive capability [35].

As the COVID-19 pandemic ushered in a sudden and widespread adoption of telemedicine by necessity, the care of patients being prescribed controlled substances was immediately transformed. The use of these screening tools may assist providers with determination of which patients are appropriate for continued support with telemedicine, and which require assessment in clinic with appropriate thorough risk assessment including physical exam, urine toxicology screening, pill count, or other measures not accessible via telehealth [36].

3.2 Assessment of overdose risk and use of naloxone

It is prudent to consider any patient using prescription drugs with the potential to cause overdose to be at some risk of such an outcome, even when that risk is relatively low. For patients considered to be at greater than low risk, proactive prescription of naloxone to mitigate the risk of overdose is the current standard of care and is endorsed by the AMA, CDC, and many other influential public health organizations [37].

To better assess overdose risk, Zedler and colleagues developed a new tool first presented in 2015, called the Risk Index for Overdose or Severe Opioid-induced Respiratory Depression, or RIOSORD, which has been validated in a veteran population of almost two million patients. It contains 17 questions, with a maximum score of 115. Included in the assessment are questions regarding history of psychiatric disorder, presence of pulmonary or liver disease, use of an extended-release opioid,

concurrent use of a benzodiazepine or antidepressant, daily morphine equivalent dose (MED), and any recent hospitalizations or emergency department visits [38].

Naloxone is an opioid receptor antagonist and will reverse the respiratory depressant effect of opioids [9]. There are multiple formulations of naloxone which vary by route of administration (intranasal, intramuscular, or intravenous), cost, and shelf life. All are highly effective, and many states offer access to naloxone without a prescription.

As previously noted, screening tools are imperfect and will fail to identify some individuals who will develop aberrant drug-related behaviors. These tools are not a substitute for common sense and good clinical judgment over time. However, they do contribute to the enhancement of patient safety, patient-provider trust, and risk mitigation for patients prescribed or being considered for opioid therapy for treatment of chronic pain.

4. Diagnosis and treatment of opioid use disorder or other SUD in the setting of co-occurring chronic pain

4.1 Approach to the patient

Patients presenting for care who are suffering from chronic pain and/or SUD must be evaluated with thoroughness, compassion, empathy, and respect. A successful clinical approach typically involves reflective listening, acknowledgment and validation of the patient's feelings, absence of judgment, evident expertise, and use of gentle persuasion for the patient to choose to pursue collaborative and volitional change. Motivational interviewing is one evidence-based technique in which patients are encouraged to consider change without confrontation or power struggle, where the provider will "roll with resistance" expressed by the patient, and instead focus on the patient's strengths while projecting optimism regarding their ability to change with proper support [39, 40].

As noted in the previous section, screening tools can be useful in identifying SUD or potential prescription drug misuse, investigating potential mental health comorbidities, and providing a periodic ongoing reassessment of the plan of care. These ideally can be reviewed along with other relevant patient data either before or during the first consultation visit. A thorough patient history is essential for accurate diagnosis and development of a safe and appropriate treatment plan [41, 42]. Ideally, the practitioner will obtain a good understanding of the patient's subjective symptoms and they are established underlying or co-occurring diagnoses, and a detailed understanding of past and current treatment, including medication management, non-pharmacologic interventions, and self-care practices. As above, the clinical approach to gather history should be non-judgmental and empathetic.

The physical exam is a compulsory, standard-of-care component of a medical evaluation for any new patient, and patients presenting with chronic pain and/or SUD histories are no exception. Naturally, a problem-focused exam of the area(s) of the body where pain symptoms are localized will contribute to the diagnosis, and assessment of symptom severity and functional limitations. For patients with concern for SUD, examination of the skin for evidence of injection sites, both old and more recent, can be illuminating, though, in the era of illicit fentanyl, drug smoking has replaced intravenous use in some locales. Evidence of advanced liver disease may manifest as icteric sclerae, jaundiced skin, or abdominal ascites. Poor dentition can be

a marker of a variety of SUD-related factors but is particularly common with regular use of methamphetamine. The presence of lymphadenopathy in a patient with SUD may be indicative of an immunocompromised state due to HIV, tuberculosis, or other infectious diseases. A thorough neurologic and mental status examination is also critical to establish neurocognitive and behavioral status, motor and/or sensory deficits, and cranial nerve functioning.

Laboratory tests can be helpful in assessing the presence and severity of organ diseases such as liver and kidney functioning, along with evaluation of relevant metabolic, infectious, hemodynamic, hormonal, and other data. Urine toxicology screening is a standard method of evaluating adherence to expectations around the use of prescribed medications and confirming absence of use of unauthorized substances. Imaging tests are often useful in working up musculoskeletal, gastrointestinal, or CNS complaints. Other useful data can typically be found in the electronic health record, such as other practitioners' assessments, as well as easy access to review of the state prescription monitoring program database, which provides evidence for use of prescribed medications and any detection of unreported use of other controlled prescription medications.

4.2 Reaching a diagnosis and formulating a treatment plan

Substance use disorders are diagnosed using the standardized definition provided in the Diagnostic and Statistical Manual of Mental Disorders, 5th edition (DSM-5), which contains 11 criteria [26]. Diagnosis is classified as mild with a score of 2–3, moderate with a score of 4–5, and severe with a score of 6 or more. It is important to clarify whether the patient's substance use has the potential to have reached a state of physiologic dependence and/or withdrawal with repeated use and attempted cessation, so that management of withdrawal can be prioritized in the earliest stages of treatment.

There is no single treatment or approach that is appropriate for all patients in all scenarios. Treatment planning should always involve a "menu of options" that is considered in a collaborative fashion in consultation with the patient. Ideally, treatment choices will be informed and supported by other stakeholders such as the patient's loved ones and other members of their care team.

For patients with chronic pain who may have developed OUD, treatment options include the following:

- Tapering of medication without other treatment. This course of care will not only fail to provide any therapeutic benefit, but it could also result in the patient seeking relief from drugs obtained from unauthorized sources which, in the era of ubiquitous illicit fentanyl, is acutely life-threatening.

- Tapering followed by treatment with opioid antagonist (naltrexone).

- Treatment with buprenorphine in the outpatient clinic.

- Referral to an opioid treatment program (OTP) for opioid agonist treatment with methadone or buprenorphine.

- Referral to short- or longer-term residential SUD treatment, with the potential to initiate medication for opioid use disorder (MOUD).

Regardless of the specifics of the treatment approach, all patients who have been identified as misusing prescribed medications or otherwise engaging in unhealthy substance use are deserving of the medical provider's concern and attention. The clinical circumstances should be viewed as a therapeutic opportunity to intervene in an impactful and potentially life-saving way.

4.3 Medications for opioid use disorder (MOUD) and their potential utility in the patient with co-occurring chronic pain

There have been many studies confirming the effectiveness and first-line status of MOUD in the treatment of OUD [24, 43]. Interactions between patients with OUD and the medical system, whether in an emergency department, hospital unit, primary care office, or specialty clinic should whenever possibly include prescription of MOUD coupled with timely, logistically feasible follow-up.

4.3.1 Buprenorphine

A semi-synthetic derivative of thebaine developed in the 1980s as an analgesic, buprenorphine is a complex and commonly misunderstood opioid. In the United States, the sublingual formulation was approved by the Food and Drug Administration (FDA) in 2002 for the indication of treatment of opioid dependence (DSM-4 equivalent of opioid use disorder). Its use was limited by restrictions placed on potential prescribers, who were mandated to undergo formal education and training along with application and official authorization by the Drug Enforcement Agency (DEA) to legally prescribe buprenorphine for OUD. These restrictions, which were intended to prevent diversion of the drug, have resulted in a persistently low percentage of available prescribers relative to candidates for use, and a resultant catastrophic unmet need. Further, the implications of these restrictions have led many practitioners to falsely conclude that learning to prescribe buprenorphine is cumbersome, difficult, and better left to others, such as addiction specialists. Tragically, as noted above, at least 80% of individuals with OUD receive no treatment. Ironically, buprenorphine is an extremely safe opioid, indeed safer than not only all other reasonably potent opioids but also many non-opioid medications that all practitioners who treat pain commonly prescribe. Currently, as of the writing of this chapter, there is legislation pending in the US Congress that would remove restrictions on the prescription of buprenorphine for the treatment of OUD.

Buprenorphine's pharmacology includes highly potent mu-opioid receptor (MOR) activity, at least 50 times the analgesic potency of morphine, along with kappa-opioid receptor antagonist activity and delta-opioid receptor agonist activity. Buprenorphine's antagonism at the kappa opioid receptor contributes to an anti-depressant effect, as well as contributing to its ability to diminish and resolve tolerance and hyperalgesia resulting from prolonged use of other opioids. Its delta agonism may contribute to additional analgesia. Its primary metabolite, norbuprenorphine, is unable to cross the blood-brain barrier, which therefore mitigates overdose risk, as brain stem opioid receptors are spared; the opioid receptors in the reward circuitry/limbic system are also spared, which is why buprenorphine can be given to patients with OUD and there is no reinforcing effect. Due to this combination of high potency, excellent safety profile, and lack of psychoactive effect, buprenorphine evolved from its origins as an analgesic to become a first-line treatment for opioid use disorder.

The utility of buprenorphine in the treatment of chronic pain is even more underappreciated and misunderstood. Due to the above-described legal restrictions, the marketing of the sublingual formulation as a treatment for OUD, and knowledge gaps around its pharmacology, many practitioners are unaware of the advantages of buprenorphine as a medication for patients with chronic pain who require or benefit from a long-acting opioid [44]. There are multiple formulations of buprenorphine that can be used effectively as an analgesic, including transdermal buprenorphine, which was approved in 2010; buccal buprenorphine (brand name Belbuca), approved in 2015; and the sublingual formulation, described above and still approved for the treatment of OUD and not pain, even though buprenorphine's analgesic effects are dose-dependent, and the sublingual formulation is the most potent of any of them. One of the common misconceptions around buprenorphine's pharmacology is that there is a "ceiling effect" for analgesia. In fact, the existence of a ceiling effect pertains specifically and exclusively to respiratory depression, but not to analgesia, which is dose-dependent [45].

4.3.2 Methadone

Methadone is an older synthetic opioid analgesic developed in Germany and introduced in the US in 1947 [46]. It was first described in the treatment of opioid (heroin) addiction in 1965 [47]. It has complex pharmacology, particularly with respect to its long and variable half-life (about 24 hours) and pharmacokinetic properties which, combined with its high potency, pose a significant risk of harm when prescribed by providers less well-versed in best practices around its use, or when it is misused. Due to the delicate nature of initiation of methadone and conversion to stable dosing following transition from other opioids, it has been suggested that only health care providers experienced with this process should undergo this task [48].

For treatment of OUD, since its inception in this context, methadone has been federally regulated and required to be dispensed in a licensed opioid treatment program (OTP), rather than in a typical medical office setting. This is one major practical distinguishing feature from buprenorphine. Many OTP facilities provide not only the medication, which is dispensed daily on-site in liquid formulation, but also support services such as counseling, medical care, and complementary treatment such as acupuncture. For these reasons, methadone's utility in current OUD treatment remains strong, particularly for individuals who benefit from the more rigid structure and robust support of the OTP setting.

Methadone poses significant and unique risk factors within the opioid class, however. It exhibits large inter-individual variation in both bioavailability and elimination half-life. Further, there is a major disconnect between its analgesic effect of only about 6–12 hours, contrasted with half-life of up to 59 hours. Patients will sometimes make the dangerous mistake of taking more than instructed as they are trying to get stabilized during initiation. Each dose's respiratory depressant effect can last up to several days and is cumulative over regular dosing, so too high a starting dose, or overuse of the prescribed dose, is particularly problematic during this early phase before the drug has reached a steady state, which normally takes about five days. Methadone is particularly risky for patients co-prescribed or otherwise using benzodiazepines or other CNS depressants, conferring additional overdose risk. Lastly, particularly at higher doses, methadone can prolong the QT interval on an electrocardiogram (ECG) which is a sign of potential cardiac arrest. It is prudent to check a baseline ECG before methadone is initiated. Some have suggested checking again at 50 mg, 100 mg, and any incremental increase of 20 mg thereafter, though there is no clear consensus [48].

Methadone does offer some attractive pharmacologic advantages as a potent long-acting opioid analgesic compared to other options. It is an N-methyl-d-aspartate (NMDA) receptor antagonist as well as a serotonin-norepinephrine reuptake inhibitor, which together may contribute to its anti-hyperalgesic effect after transitioning from other opioids, as well as its reputation for effectiveness in neuropathic pain syndromes [48].

4.3.3 Naltrexone

Developed in the 1970s, naltrexone is a long-acting competitive antagonist at opioid receptors with the capacity to block the subjective and objective effects of opioids [49]. It is an effective antagonist at mu- and kappa-opioid receptors, less so at the delta subtype. It is thought to block glutamate and may contribute to reduction in craving for and protracted withdrawal from alcohol through that mechanism.

Oral naltrexone was approved by the FDA in 1984 for "blockade of the effects of exogenously administered opioids" [49]. It is also approved for alcohol use disorder. Its onset is rapid, reaching peak plasma level within 1 hour, and has a relatively brief half-life of 4 hours. It is metabolized by the liver and severe liver impairment may be an obstacle to use.

An extended-release, injectable formulation of naltrexone was approved in 2006 first for treatment of alcohol use disorder, with opioid use disorder following in 2010. It is administered intramuscularly in the gluteal region, and is typically well-tolerated, particularly with a period of several days of use of oral naltrexone prior to injection [49]. The most common adverse effects are injection site pain, nausea, other gastrointestinal upset, or flu-like symptoms. The formulation contains 380 mg of naltrexone, releasing levels of 1 ng/mL or above for a period of 4–5 weeks, without the need to adjust the dose for weight, age, health status, or other factors.

Studies comparing the effectiveness of ER-naltrexone (XR-NTX) and buprenorphine-naloxone (BUP-NX) for the treatment of opioid use disorder have been performed. In the "X:BOT" study, a multicenter, open-label randomized controlled trial published in 2018 and funded by the National Institutes of Drug Abuse (NIDA) Clinical Trials Network, the 2 medications were found to be equally safe and effective once initiated [50]. Some patients had more difficulty with initiation of XR-NTX compared to BUP-NX, which was in part due to a lack of formal optimization and uniformity of induction and withdrawal protocols between testing sites, of which there were 8 around the country. Authors concluded that the research community should prioritize improving XR-NTX induction and retention strategies. In a more recent study published in JAMA in 2022, however, Xu and colleagues importantly found that buprenorphine outperformed naltrexone in the category of decreasing overdose risk [51].

Naltrexone in compounded oral formulation has been used in the context of chronic pain in the form of "low dose naltrexone" for treatment of CNS-mediated generalized pain syndromes such as fibromyalgia, inflammatory bowel conditions, or multiple sclerosis. Dosing is generally in the range of 4–5 mg daily, whereas daily dose for treatment of OUD is 50 mg. It is thought to work as a modulator of glial cells and inflammatory chemicals in the CNS. It was systematically reviewed in 2020 with favorable findings suggesting a need for further investigation and increased clinical use [52].

Patients may find it difficult to know which of the 3 above-described medications approved for OUD to choose, particularly in the context of chronic pain. Although individual cases will vary, generally it would not be advisable to choose naltrexone for patients with the following circumstances:

- At high risk for overdose
- Have not tolerated extended periods of opioid abstinence in past attempts
- Have tended to experience protracted and severe withdrawal symptoms following cessation of opioids
- Have unstable psychiatric symptoms
- Level of chronic pain requires or is currently being treated with opioids
- Have advanced liver disease, impending liver failure, or acute hepatitis

Whichever of these evidence-based treatment options is ultimately pursued, it is critical that patients are carefully matched to the best MOUD treatment for their specific circumstances and needs, then monitored and supported to optimize stability in the patient-provider relationship to foster best outcomes.

4.4 Other medications useful in co-occurring chronic pain and SUD

In the management of chronic pain, typically a combination of agents is prescribed to provide analgesia using different neurochemical pathways [17]. Opioids should be used only when other non-opioid medications, and non-pharmacologic pain management strategies, have failed to adequately address pain severity and associated functional impairment [6]. Patients with active SUD, and/or a personal or family history thereof, should even more pointedly be recommended to avoid controlled substances which may trigger unhealthy substance use in a manner that is unsafe and counterproductive to the goals of treatment. Examples of commonly used non-opioid medications include the following [17]:

- **Non-steroidal anti-inflammatory drugs (NSAIDs):** these are the most widely used analgesics, indicated for mid to moderate somatic pain, and used often in combination with opioids for severe pain. They work by inhibiting prostaglandin production, thereby reducing the sensitization of peripheral nerves, and curbing the inflammatory response. Most common adverse effects include gastrointestinal symptoms, renal toxicity, and inhibition of platelet functioning leading to an increased risk of bleeding.

- **Antidepressants:** include older agents such as tricyclic antidepressants, and newer selective serotonin and norepinephrine reuptake inhibitors (SNRIs). Tricyclic agents are considered first-line treatment for neuropathic pain such as diabetic neuropathy, with the greatest potency in amitriptyline, imipramine, and doxepin, and less effective but also fewer anticholinergic adverse effects with nortriptyline and desipramine. SNRIs include duloxetine, which is approved by the FDA for fibromyalgia and is considered a first-line drug for patients with chronic low back pain by the American College of Physicians, along with venlafaxine.

- **Anticonvulsants**: this class has been used widely in the treatment of neuropathic pain and other dysesthesia pain syndromes characterized by burning or lancinating pain. Gabapentin has been commonly prescribed, along with pregabalin,

both of which are approved for diabetic peripheral neuropathy and post-herpetic neuralgia, the latter also approved for fibromyalgia. Carbamazepine, valproic acid, and other agents have been used effectively for paroxysmal facial pain and headache syndromes. All medications in this category have potential for significant adverse effects and tolerability issues.

- **Muscle relaxants**: this category includes several classes of medications with a variety of mechanisms of action, some of which are poorly understood. All tend to have significant sedating effects due to primarily CNS-mediated activity, which may limit daytime use. Use at bedtime may assist with insomnia along with overnight pain control in appropriate patients.

- **Topical agents**: a variety of agents are available which can be applied locally to painful areas when symptoms are focal. These include topical diclofenac, an NSAID; lidocaine transdermal patches or lidocaine-based ointment, gel or cream, a local anesthetic; capsaicin, a naturally-occurring extract from cayenne pepper which inhibits substance P and is useful for neuropathic pain after initial worsening of burning symptoms; products now available in many states containing formulations of cannabinoid-derived salves, creams, gels and ointments; and other options available over-the-counter in pharmacies such as camphor-based and menthol-based counterirritants.

5. Non-pharmacologic pain management modalities

In approaching a patient with co-occurring chronic pain and SUD, it is important for the clinician to recognize the multiple functional domains affected: body, brain (CNS), mind, and spirit. Structural and functional changes in the brain have been well-documented and contribute to reorganization of neural networks involved in behavior, emotional regulation, identity formation, and capacity for enjoyment of life. Thinking may be distorted due to chronically focused attention on pain and distress signals, frequently attached to drug-related cues, which biases thoughts and attitudes toward negative, maladaptive patterns of fear, avoidance, catastrophizing, and disengagement. Treatment modalities aimed at improving self-efficacy and coping skills are critical in achieving good outcomes. Below are some categories and examples of such approaches.

Special mention is given to the existence and strong evidence in favor of structured interdisciplinary pain rehabilitation programs [53]. Though not commonly found, these programs provide holistic care to a cohort of patients and typically include medical management, psychological counseling, movement-based therapy, and relaxation training. They are often managed by a nurse or allied health provider. The team of specialists communicates about each patient in the program in a longitudinal and coordinated fashion to optimize individual patient progress and outcomes, emphasizing training the patient in sustainable self-care techniques.

5.1 Movement-based therapies

Ref. [54] At a basic level, exercise and movement are helpful and adaptive for patients with chronic pain, many of whom have developed kinesiophobia, or fear of movement. Physical therapy is often a helpful first step in addressing that fear and

gradually improving activity tolerance. Occupational therapy assesses and intervenes to maintain or re-establish meaningful activities or occupations specific to a patient's circumstances, such as working in an office environment or providing care to small children. Tai chi is an evidence-based form of ancient Chinese movement, breathing, and harnessing of energy (chi) that has been used widely in chronic pain treatment, especially in interdisciplinary chronic pain rehabilitation programs. Yoga is another validated technique for combining anaerobic exercise, breathing, mindfulness, and energy movement, and has become very popular both in chronic pain treatment and as an approach for general wellness.

5.2 Bodywork treatments

Ref. [54] Acupuncture is an ancient Chinese technique using specific needle placement, with available modern innovations such as the use of percutaneous electrical nerve stimulation, which has been shown to be effective for a variety of chronic pain conditions, including headache syndromes, chronic low back pain, and other common structural and functional pain syndromes. Chiropractic treatment aims to realign structural abnormalities via manual manipulation of the spine, with evidence most positive for low back pain and less so for neck and upper back pain. Osteopathic manipulation therapy (OMT) similarly uses structural manipulation techniques but in contrast to chiropractic care, OMT is not exclusively focused on the spine as a site for application of gentle pressure on body tissues. Massage therapy is performed by trained, licensed providers using various styles ranging from very gentle to deep tissue techniques, with the goal of reduction of muscle tension as well as general relaxation of body and mind. It has been shown in a host of studies to be useful in chronic pain [55].

5.3 Psychosocial treatment for chronic pain

Ref. [54] Enhancing insight into the role of a patient's thoughts, emotions, and behaviors on functional capacity and subjective level of pain is of paramount importance and may be approached with specific counseling techniques, which are often provided by mental health specialists but may also be used by medical providers such as pain or addiction medicine specialists, or primary care providers [56]. Motivational interviewing is a patient-centered approach that encourages the patient to undergo behavior change in a manner consistent with their own choice and empowerment. Cognitive restructuring aims to increase a patient's awareness of maladaptive thoughts and behavioral patterns and encourages replacing those with more positive, adaptive ones. Cognitive-behavioral therapy (CBT) is a broad category of a well-studied treatment that, like cognitive restructuring, emphasizes the pursuit of change in maladaptive thoughts and behaviors. Emphasis is on improvement of coping skills, reducing fear of movement and activity, development of techniques for relaxation and enjoyment, and establishing adaptive routines. Acceptance-Commitment Therapy (ACT) is a modification of CBT that emphasizes cognitive flexibility and encourages non-judgmental detachment from the experience of pain, with the goal to engage in meaningful and rewarding activities despite the presence of pain.

5.4 Self-directed non-pharmacologic management of chronic pain

Self-management of chronic pain is a vital component of a successful pain management approach. It is often impractical at best to access the full gamut of

evidence-based professional treatment, due to both availability and cost; moreover, self-care tends to enhance the patient's sense of autonomy, self-reliance, and self-efficacy while incorporating activities and techniques that are consistent with the patient's belief system. The following are examples of self-care approaches:

- **Mindfulness-based Stress Reduction** (MBSR): developed by Jon Kabat-Zinn in his 1990 book Full Catastrophe Living, MBSR is an 8-week evidence-based course offered in hundreds of medical settings and teaches a Buddhist-influenced style of mindfulness meditation practice, along with body scanning and certain yoga postures. It promotes a non-judgmental uncoupling of the sensory aspects of pain from its emotional evaluative dimensions. It has been studied extensively in the context of chronic pain and has been shown to be equally effective and more cost-effective than CBT provided professionally [57].

- **Spirituality**: engagement in prayer and faith-based activity has been shown to facilitate well-being and contribute positively to the management of chronic pain [58].

- **Exercise**: an essential component of a comprehensive approach to pain management, exercise may be part of a prescribed movement treatment such as physical therapy, or may involve less structured, patient-directed activity. It is acknowledged that while individualized exercise therapy generally enhances functional outcomes, there are limitations to the level of intensity and duration of activity that a given patient attempt if exacerbations in pain and both physical and psychological setbacks are to be avoided. In general, exercise should be approached gradually and increased incrementally. Aquatic therapy, such as walking in a warm pool, can be a good first step [59].

- **Sleep hygiene**: sleep disorders are among the most common comorbidities for those experiencing chronic pain [60]. Sleep medications are commonly prescribed, and though these may be of benefit, it is appropriate for practitioners to emphasize sleep hygiene improvement for more effective and sustainable results. Examples of recommended aspects of sleep hygiene include sticking with a sleep/wake schedule, not eating too close to bedtime and avoiding evening foods that are difficult to digest or cause reflux, creating a "bedtime ritual", limiting daytime napping, and engaging in routine daily physical activity.

- **Nutrition** is both a "mainstream" and complementary/integrative component of a well-rounded chronic pain management approach. Of course, eating a healthy, balanced diet with avoidance of overeating or restrictive eating patterns is advisable. The use of dietary supplements in pain management has not been supported by evidence, according to the National Center for Complementary and Integrative Health (NCCIH, an agency of the National Institutes of Health). Obesity is a known risk factor for spinal degenerative disc disease, and osteoarthritis of the lower back, hips, and knees [61].

- **Peer support groups:** there are available online and in-person meetings for patients suffering from a range of chronic pain conditions such as fibromyalgia, lupus, irritable bowel syndrome, sickle cell disease, and many others [62]. In addition, there are 12-step "pain recovery" books that focus on the intersection

of pain and substance use disorders, using the model and framework that has been so critical and well-trod by individuals who have participated in Alcoholics Anonymous or Narcotics Anonymous programs.

6. Interventional procedures used in chronic pain management

A variety of procedural management options are available for patients with chronic pain, with a range of techniques and associated therapeutic effects [63].

- **Trigger point injections**: local injection of anesthetic into focally tender areas of muscle and soft tissue known as "trigger points" can produce short-term relief for both acute and chronic myofascial pain conditions. Controversy exists as to what agent should be injected and how often. Dry needling may concurrently or alternatively be employed, and both acupuncture and physical therapy may be useful adjunctive approaches. Techniques for this approach were described in depth by Dr. Janet Travell, physician to President John F Kennedy, in her seminal 1983 text on myofascial pain [64].

- **Local neural blockade**: can be used for both diagnostic and therapeutic purposes. Commonly used on the medial branch nerves at spinal facet joints, if pain relief is greater than 80%, patients may be candidates for radiofrequency nerve ablation (neurotomy) at that level, with much longer-lasting relief. Recently, peripheral nerves have also been targeted [65].

- **Spinal epidural steroid injections**: used in the treatment of mechanical back and neck pain, with best results typically seen when radiculopathy is present, and when symptoms have been present less than 6 months or are acutely exacerbated [66]; facet blocks are typically used in cases without radicular signs.

- **Sympathetic blockade**: indicated for pain involving the sympathetic nervous system and viscera. Nociceptive input from the upper extremities, head, and neck can be blocked via the stellate ganglion; abdominal visceral pain can be approached by blocking the celiac ganglion, while urogenital visceral is approached via the hypogastric plexus. Lumbar sympathetic ganglia mediate pain in the lower extremities, and blockade may be useful in neuropathic lower extremity pain from failed back surgery syndrome or complex regional pain syndrome of the lower limb [67].

- **Spinal cord stimulation** was first introduced in 1967, but there have been innovations in recent years that have significantly improved the effectiveness of this intervention, in particular for failed back surgery syndrome and peripheral neuropathy. Because it involves surgical implantation, it is generally reserved for patients who have failed more conservative interventional therapies [68].

7. Conclusion

Patients who are living with chronic pain and a history of SUD and are attempting to improve their quality of life and avoid unhealthy substance use are to be credited

for their resilience, courage, and perseverance in staying "within the system" and entrusting their care to a team of providers. Often, these patients are the victims of stigma and judgment, experiencing negative interactions with individual providers and with the health care system as a whole. Today's unprecedented levels of fatal drug overdoses necessitate a shift toward harm reduction and toward a willingness to partner with patients whose choices do not always perfectly align with prescribed recommendations, so that we may help them avoid making their last dangerous choice. With the right approach, these patients can be offered safe, effective, holistic care that aims to address the various domains of their suffering, including physical symptoms, emotional distress and trauma, and disengagement from meaningful activity. Best results are generally found when a team of professionals provides coordinated interdisciplinary care. When such care is impractical or unavailable, individual components of evidence-based interventions and approaches can still be pursued with excellent patient-centered results, and with fulfilling, gratifying long-term relationships between patients and providers.

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