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CONTINUING EDUCATION ACTIVITY

Hope for Hypermobility: Part 1—An Integrative Approach to Treating Symptomatic Joint Hypermobility

Victoria Daylor, BFA, Cortney Gensemer, PhD, Russell A. Norris, PhD, and Linda Bluestein, MD

Learning Objectives: After participating in this continuing professional development activity, the provider should be better able to:

1. Describe symptomatic joint hypermobility and associated comorbid conditions.
2. Explain the physiological basis of pain associated with joint hypermobility.
3. Examine the range of diagnostic testing available to identify the source of pain in individuals with joint hypermobility.

Key Words: Comorbid conditions, Ehlers-Danlos syndrome, Hypermobility, Pain types

In This Issue

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The authors, faculty, and staff have no relevant financial relationship with any ineligible organizations regarding this educational activity.

The authors have disclosed there are no FDA-approved treatments for symptomatic joint hypermobility as discussed in this article.

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This article is the first of 2 parts. In the first part of this series on hypermobility, the authors introduce symptomatic joint hypermobility (SJH) with a discussion of the many types of pain associated with hypermobility syndromes. In part 2, the authors will outline specific management according to a defined protocol.

Joint hypermobility has been an underappreciated consideration when treating patients with chronic pain and frequent injury. Clinicians encounter a multitude of patients seeking[1] pain treatment, and it is estimated that 11% to 40% of adults in the United States experience chronic pain. The statistic increases to 90% when observing people with SJH syndromes.[2,3] Pain is one of the most common and debilitating symptoms of joint hypermobility syndromes, which includes Ehlers-Danlos syndromes (EDS), a group of genetic connective tissue disorders that cause generalized joint hypermobility and tissue fragility.

EDS currently consists of 14 subtypes with phenotypic and genetic heterogeneity, including the more common hypermobile (hEDS), vascular (vEDS), and classical (cEDS), and other exceedingly rare types.[4] Each subtype of EDS has a known genetic cause, except for the most common type, hEDS, which accounts for 80% to 90% of all EDS cases, highlighting the urgency for research to meet population demands by determining the underlying genetic and molecular etiology of hEDS.[5] A 2019 study demonstrated a combined prevalence of 1 in 500 for EDS and "joint hypermobility syndrome," revealing them to be common conditions that health care workers should expect to see in their practice and manage in pain clinics.[6]

The continuing education activity in *Topics in Pain Management* is intended for clinical and academic physicians from the specialties of anesthesiology, neurology, psychiatry, physical and rehabilitative medicine, and neurosurgery, as well as residents in those fields and other practitioners interested in pain management.

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Generalized Joint Hypermobility and Related Conditions

Joint Hypermobility

Joint hypermobility is defined as an unusually large range of joint motion. Common sites of joint hypermobility include weight-bearing joints such as knees, ankles, hips, and the temporomandibular joint.[7] Current nosology considers joint hypermobility as a spectrum that includes asymptomatic generalized joint hypermobility, asymptomatic peripheral joint hypermobility, asymptomatic localized joint hypermobility, generalized hypermobility spectrum disorder, peripheral hypermobility spectrum disorder, localized hypermobility spectrum disorder, historical hypermobility spectrum disorder, and hypermobile EDS (Figure 1).

hEDS falls under the category of heritable connective tissue disorders, which can phenotypically overlap with hypermobility spectrum disorder (HSD). The correlation of EDS/HSD is not directly indicative of severity.[5] Both conditions are poorly recognized, complex, and vary significantly in presentation. HSD includes patients ranging from those with frequent injury to those with disabling joint instability. However, they neither meet the 2017 criteria for EDS, as indicated by the EDS International Consortium, nor have another disorder to explain their symptoms.[8]

The International EDS Consortium proposed a revised EDS classification, recognizing 13 subtypes in 2017.[8] This classification outlines the clinical features of each subtype and the genetic etiology that were known to date, before the discovery of the 14th subtype. For hEDS, a clinical diagnostic checklist is provided for evaluating a patient suspected to be affected. Due to the vast genetic heterogeneity and phenotypic overlap of the EDS subtypes, the definitive diagnosis of all EDS subtypes, except for the hypermobile type, depends on molecular confirmation with the identification of causative genetic variants. This is recommended to be done through a connective tissue panel, as SJH may also be present in other heritable disorders (eg, Marfan syndrome and Stickler syndrome). Genetic testing should always be used to rule out other potential hypermobility-related conditions that require routine screening and unique treatment approaches. Despite the 2017 revised EDS classifications, a diagnosis of hEDS can be difficult to make. Future revision of these criteria, and genetic and molecular studies, may allow for better distinction between hypermobility syndromes. Because the distinction between hEDS and HSD can be challenging to make clinically and may not be clear until underlying genetics are uncovered, in this review, they have been referred to together as SJH because of similar molecular and phenotypic presentation.[9,10]

Comorbid Conditions

Although pain and joint dysfunction are key features of hEDS and HSD, associated comorbidities substantially interfere with patients' daily lives. Individuals typically experience a multitude of systemic manifestations that require care by an interdisciplinary team. Immunologic disorders, such as mast cell activation disorder (MCAD), have been reported in patients with EDS/hEDS. Further research is needed to fully understand the relationship between connective tissue and mast cells.[11,12] Cardiovascular comorbidities include mitral valve prolapse and aortic root dilatation. Neurologic manifestations of hEDS include Chiari malformation type I, cerebrospinal fluid leak, craniocervical instability, atlantoaxial instability, spondylolisthesis, thoracic instability, scoliosis, small fiber neuropathy (SFN), headaches, migraines, and tethered cord syndrome.[4,13-17]

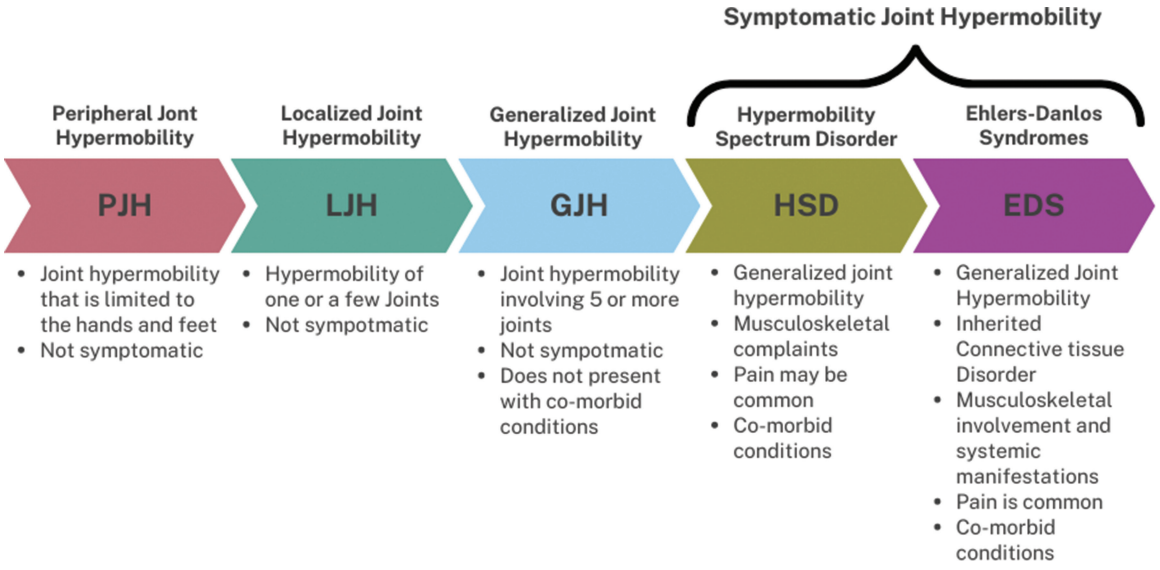

Figure 1. Diagram representation of the joint hypermobility spectrum. The 6 categories span a broad range, from asymptomatic to highly symptomatic with associated comorbid conditions. The degree of hypermobility does not necessarily correspond to the severity of symptoms. The presence of comorbid conditions can contribute to severity.

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The overlap between cardiovascular and autonomic dysfunction symptoms remains unclear, but may present as vasovagal syncope/neurocardiogenic syncope, orthostatic hypotension/delayed orthostatic hypotension, orthostatic intolerance, and postural orthostatic tachycardia syndrome (POTS).[18] Gastrointestinal manifestations have been reported to occur before a clinical diagnosis in 74.4% of patients with EDS (with hEDS accounting for 80.6% enrolled) in the largest study on EDS and digestion.[19] This study also reported that 48% of patients with EDS were also diagnosed with irritable bowel syndrome, 36% with functional constipation, and 79% with gastroesophageal reflux disease.[19]

Musculoskeletal problems may range from joint subluxations (partial dislocation), joint dislocations, muscle stiffness, muscle spasm, sprains, ligament tears, tendinitis, tendon rupture, chronic joint pain, and osteoarthritis.[4,8,20,21] Other manifestations include, but are not limited to, fragile skin, easy bruising, abnormal wound healing, sleep disturbances, psychologic disorders, anxiety, depression, chronic fatigue syndrome, Raynaud's phenomenon, recurrent hernias, and neurodivergence (eg, attention deficit hyperactivity disorder, autism spectrum disorder, social anxiety, dyslexia, and dyspraxia, among other conditions).[4,18,22-32]

Joint Hypermobility and Types of Pain

Those living with SJH often report pain varying from diffuse pain in load-bearing joints to muscle tension causing chronic myofascial pain. Pain is grouped into 3 categories: nociceptive, neuropathic, and nociplastic. Nociceptive pain is secondary to the original stimulus and is caused by activity in neural pathways.[33] Nociceptive pain can be categorized as either visceral or somatic in sensation. Somatic pain tends to be described as localized, sharp, aching, or throbbing, and visceral pain is often described as deep aching, vaguely distributed, or spasm-related.[34] Nociceptive tissue damage accounts for the majority of chronic pain and encompasses most forms of spinal pain, such as arthritis. Patients with SJH may experience nociceptive pain due to joint subluxation, dislocation, tendinopathy, and postoperative pain.[33,35-38]

> Patients with SJH often report pain varying from diffuse pain in load-bearing joints to muscle tension causing chronic myofascial pain.

Neuropathic pain is described by the International Association for the Study of Pain as "pain initiated or caused by a primary lesion or dysfunction in the nervous system" or disease affecting the nervous system.[39] Neuropathic pain is usually caused by nerve compression, inflammation, trauma, toxins, or metabolic diseases.[33] Patients with SJH may present with nerve root compression, complex regional pain syndrome (CRPS), brachial plexopathy, axonal polyneuropathy, and SFN.[40-48] Neuropathic pain may be observed when a hypermobile person overstretches beyond an appropriate range of motion.[41] Overstretching can occur with forced and/or congenital hypermobility, such as overstretching a joint without intent or due to hEDS. Chronic overstretching can cause nerve entrapment that does not always return to normal upon decompression.

> Often, more than one type of pain is present in patients with SJH.

Nociplastic pain is thought to arise from altered pain modulation without clear pathologic evidence of tissue or nerve damage.[49] It accounts for widespread pain and other symptoms such as fatigue, disordered mood, and memory problems. Examples of nociplastic pain include fibromyalgia, irritable bowel syndrome, and nonspecific back pain.[49] Distinguishing the type of pain is important to provide effective treatment. A mixture of pain types, such as lower back pain caused by neuropathic and nociceptive pain, can be less responsive to anti-inflammatory drugs or injections.[49] Often, more than one type of pain is present in patients with SJH[50] (Figure 2).

Pain States

Under normal physiological circumstances, pain is a response of the nervous system as a protective mechanism against harmful stimuli. The sensation of pain is the body's learning tool to prevent continued tissue damage. When pain persists, it is no longer a signaling system but becomes a disease itself.[51] Acute pain is typically caused by circumstances, either an injury or trauma, and is often described as a sharp or stabbing sensation. For those with SJH, acute pain can occur with everyday tasks, such as reaching for an item and then experiencing joint subluxation or dislocation. Acute episodic pain can occur rapidly, severely, and at irregular intervals. Informally known as a flare-up, this type of pain can be difficult to predict, making it disruptive to daily life. Pain that persists beyond the "typical" healing process, 3 months or longer, is considered chronic pain.[52] In this patient population, it is important for both patients and providers to be aware of the possibility of acute on chronic pain. It is possible to have an acute injury or flare-up in a region where a patient also experiences chronic pain. For example, suppose a patient has a prior diagnosis of chronic shoulder pain and is being

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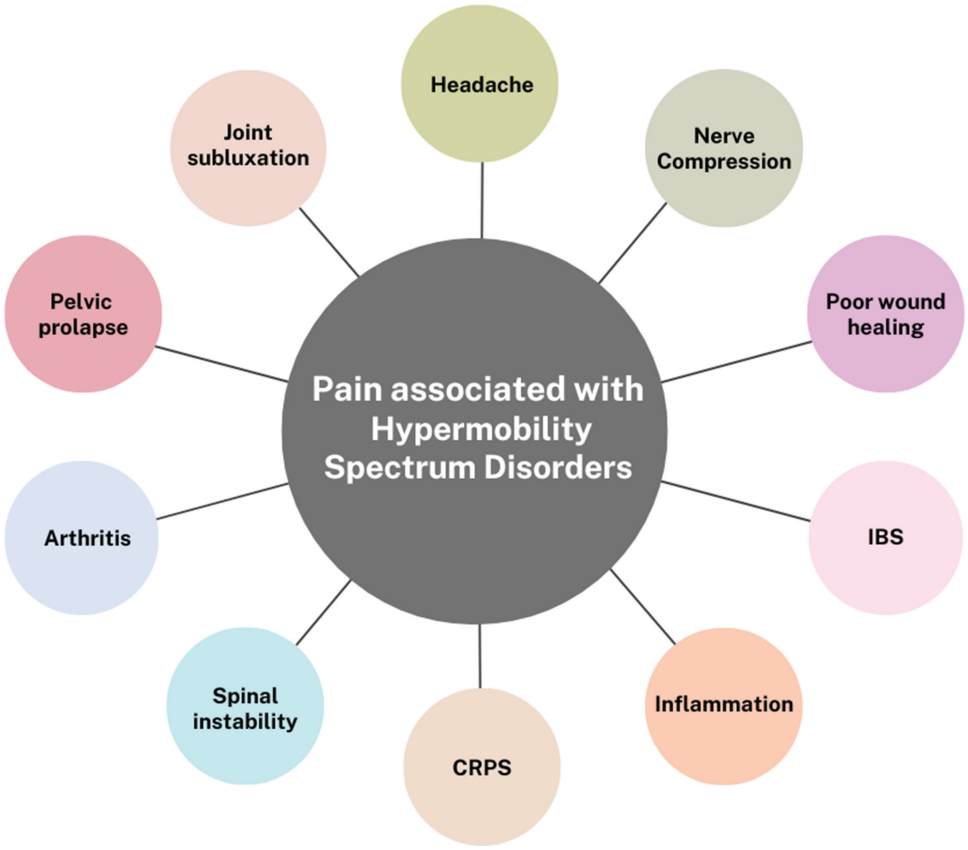

Figure 2. Examples of pain associated with SJH. Patients with SJH often experience a variety of pain, and the factors shown demonstrate a myriad of possible pain sources.

evaluated emergently. In that case, they should be evaluated for acute injuries such as dislocation or other musculoskeletal pathologies.

Intractable pain differs from chronic pain in that it is incurable, with definitions varying from state to state. For example, Minnesota defines intractable pain as "a pain state in which the cause of the pain cannot be removed or otherwise treated with the consent of the patient and in which, in the generally accepted course of medical practice, no relief or cure of the cause of the pain is possible, or none has been found after reasonable efforts."[53] People may experience restricted mobility, social isolation, depression, and often constant pain that interferes with sleep and sexual function.[54] Pain management can be incredibly difficult for patients and providers. This article aims to provide insight into treatment options to safely prevent and minimize acute, chronic, and intractable pain in people living with SJH.

Diagnosing Pain

Complex cases of chronic pain prove challenging to classify, and they overlap with various chronic pain syndromes, such as fibromyalgia, myofascial pain, rheumatoid arthritis, and irritable bowel syndrome.[55] Due to the overlap in presentation, SJH can be misdiagnosed as the previously mentioned disorders. Pain contributors to consider include duration, source, and intensity. The intensity of pain can be tracked using a variety of approaches, including categorical (mild to severe), numerical (1-10), and visual (image of faces in pain). Populations with more severe pain are reported to have a worse health status, including an increase in bed disability days, health-related inability to work, and difficulty walking or climbing stairs.[56] Those with chronic pain may struggle to provide values or categories for their pain, which can be crucial in creating a treatment plan. Further research is needed into the reliability of various methods to discern pain intensity.

Inflammation is a recognized contributing source of chronic pain and includes disease states in peripheral tissues, such as the skin, muscles, and internal organs. The production of inflammatory mediators leads to vasodilation, causing swelling, redness, heat, and pain.[57] Acute pain due to continuous peripheral inflammation can subsequently lead to chronic pain. Neurogenic inflammation also includes peripheral tissues, notably the skin, which activate nociceptors at a speed even more rapid than immune cell infiltration.[58] SJH patients may experience neurogenic inflammation in the form of migraine and CRPS.[58] Neuroinflammation occurs in the peripheral and central nervous systems, including the nerves, spinal cord, and brain.[57,58] Neuroinflammatory processes play a role in the maintenance of chronic pain, and are associated with fibromyalgia, musculoskeletal pain, sleep disturbance, and fatigue.[57,59]

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Hypersensitivity to Pain

Central sensitization is caused by increased nociceptive inflammation or injury, leading to lasting changes in the central nervous system, and has been indicated as a cause of pain in SJH.[60-62] Central sensitization contributes to inflammatory pain and involves heightened sensitivity to pain, categorized as allodynia, hyperalgesia, and secondary hyperalgesia.[63] Patients with allodynia experience pain from stimuli that are not usually painful; hyperalgesia patients are especially sensitive to pain, which can evoke an extreme response. In secondary hyperalgesia, pain sensitivity is delivered beyond the region of injury.[64] Central sensitization provides a framework for understanding extreme pain sensitivity. Although similar, central and peripheral sensitization differ mechanically and phenotypically. Central sensitization is attributed to an increase in responsiveness in the central nervous system, whereas peripheral sensitization refers to an increase in the responsiveness of nerves supplying inflamed tissues.[64] Nerve-blocking agents have shown varied success depending on the identification of the source of neuropathic pain.[65] Diagnosis of pain sensitization is essential for effective targeted medical treatment.

Imaging

Identifying the source of pain for a patient with SJH proves challenging due to insufficient clinical research and the complex overlap of comorbidities. Typical imaging and laboratory tests are recommended for patients with SJH as injury or symptoms manifest. Testing includes but is not limited to MRI for spinal injuries, upright or dynamic imaging for upper cervical instability, x-rays for dislocations/subluxations, ultrasound to diagnose inflammation, and a tilt table test to diagnose POTS.

Other recommended imaging techniques include dynamic ultrasound and imaging during weight-bearing.[7] Upon initial hEDS diagnosis, providers may recommend an echocardiogram to rule out cardiac comorbidities. Unfortunately, there are cases when a known injury or source of pain cannot be diagnosed through laboratory testing and imaging, which can be frustrating for both providers and patients. A normal test result related to SJH also does not indicate that a patient does not require treatment and/or pain management, and it is recommended that the patient receive care based on clinical assessment. A diagnosis of hEDS or HSD can provide guidance toward the proper tools necessary to diagnose comorbidities related to hypermobility and instability.

Although the research landscape of HSD is advancing toward the demand of its patient population, there are no FDA-approved treatments for SJH.

Treatment Approaches

Although the research landscape of HSD is advancing toward the demand of its patient population, there are currently no FDA-approved treatments for SJH. Without a treatment targeting the cause, patient care teams thus far treat individual symptoms of injuries and comorbidities. Extrapolating from related conditions with ample research can be constructive. Treatment options for other conditions can overlap with SJH, such as nociplastic pain in fibromyalgia patients or management of migraine, and managing comorbidities like MCAD and POTS. To effectively treat the many facets of SJH, obtaining a highly detailed patient history is the cornerstone of effective patient care. During an initial appointment, it is advised to note a history of treatment and medications that have and have not been effective in the past and why.

Acquiring these details takes time, but will contribute to a clearer path forward and potentially save time with more succinct understanding during future discussions.[66] Strategizing a treatment plan should include risk/benefit analysis, using medications with minimal side effects, relatively tolerable, low risk, and often over-the-counter options when possible. When treating pain, one must target the cause, location, and specific type of pain, which often coincides with working to stabilize the injured and surrounding joints.[4,67] Should lower-risk options become exhausted, surgery may be required with caution due to the potential of poor wound healing, slow recovery progress, and remissions.[68,69]

Iterative Approach

When treating the complex symptomology in HSD, methods must continue to adapt throughout the course of treatment. Currently, clinical research for HSD is still in its infancy, leaving clinicians little guidance when faced with patients in need of help.[70] Because of the high interpatient variability, including throughout life, and the lack of clinical data, an iterative approach to treatment becomes critical. This method requires trying methods and medications to see what works and what does not. There are always new options and the possibility of circling back to prior treatments with new circumstances. This iterative approach can also have positive psychological benefits by listening to the patient and offering direct actions of treatment.[66,71]

Multiple Discipline Involvement

Patients with SJH typically require a multidisciplinary care team to manage the different features of the disorder(s). Patients may utilize a primary care physician or specialist as the center point, to oversee and coordinate the entirety of the treatment plans. Depending on the patient's complaints, a wide range of specialists may be required, including but not limited to a gastroenterologist, immunologist, cardiologist, neurologist, ophthalmologist, psychologist, psychiatrist,

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orthopedist, and pain management specialist. Referrals to specialists familiar with these conditions can often be helpful, as many of these patients have experienced medical trauma and dismissal by health care providers.[72] There are 2 general categories of treatment approaches that both aim to relieve chronic and acute issues. Interventional treatment methods focus on medical treatments and medications, whereas integrative treatment approaches include complementary and alternative approaches. When working with complex disorders like HSD, offering treatment options from both approaches increases the potential of finding a method that improves the patient's quality of life.

Conclusion

The authors ask you to continue this continuing education activity with part 2 in next month's issue of *Topics in Pain Management*. In part 2, we will cover specific treatment modalities, in particular, a treatment method developed by one of the authors (L.B.) and referred to as "MENS PMMS." The acronym "MENS PMMS" stands for movement, education, nutrition, sleep, psychosocial, modalities, medications, and supplements, and was created after documenting the greatest degree of treatment success in those patients receiving a multimodal, comprehensive treatment approach.

Pain management in patients with SJH requires a multimodal approach.

To summarize part 1 of this series, SJH is a frequently overlooked cause of pain in patients seeking care and pain management. When joint hypermobility is present in a patient, HSD should be considered as a source of related symptoms. SJH cannot be detected by imaging or laboratory testing, except when a suspected genetic cause may be present. However, by obtaining a detailed medical history, assessing joint hypermobility using the Beighton score and other hypermobility assessment tools, examining tissue fragility and skin manifestations, and using the 2017 hEDS diagnostic checklist, physicians can screen for SJH and its contributions to patients' symptoms. Pain management in patients with SJH requires a multimodal approach, such as the MENS PMMS method, to maintain lower pain levels, promote function, and facilitate abundant lives. To understand MENS PMMS, continue to next month's issue, which will go into detail with each of the components of that approach.

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Formulating Pain Agreement Plans With Patients for Improved Care With Fewer Side Effects

Elizabeth A.M. Frost, MD

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More than 100 million Americans are affected by chronic noncancer pain, accounting for an estimated $635 billion in health care costs and lost productivity annually.[1] Once the mainstay of chronic pain treatment, opioids have many serious side effects, including addiction, and often their use leads to failed pain relief in chronic situations. Thus, the standard of care has been to move away from opioid prescribing.

With this shift from long-term opioid care has come increased patient safety, but that has been offset for many by withdrawal and negative health consequences including hospitalization, overdose, and suicide.[2] Despite clinical guidelines, health care workers and patients often remain uncertain about how to formulate individualized pain management plans. Moreover, the Centers for Disease Control and Prevention did not include pain guidelines in their updated 2022 recommendations.[3] Instead, they focus now on best practices such as shared decision-making, overdose prevention, and patient education about the risks of long-term opioid use. Nevertheless, tools are necessary to standardize, guide, and achieve these aims.

Key Elements	Sample Language
Clear purpose	"The purpose of this agreement is to help patients and their health care teams create a plan to effectively treat pain and improve quality of life. This agreement will describe the risks and benefits of opioid medications. It will also present alternative treatment options. It will describe steps that you and your healthcare team will take to safely treat your pain."
Chronic pain assessment	"Describe your pain. Please list 3 activities that are important to you that pain currently limits you from doing." Include PEG Scale.
Informed consent/ shared decision-making	"The use of opioids to treat chronic pain is controversial. Research studies comparing opioids to other types of pain medications have shown that they do not work any better long-term. There are some substantial risks associated with long-term opioid use." Include list of known risks of chronic opioid therapy (eg, hypogonadism).
Emphasis on multimodal pain management	"Current evidence shows that there is not a single magic treatment for chronic pain. Instead, building a 'tool kit' of different types of pain treatment is most effective." Include a framework for multimodal chronic pain treatment providing examples of behavioral, pharmacologic, physical, and complementary categories.
Prompts to assess understanding and elicit feedback	"What surprised you about the information stated previously? What questions do you have about the risks and benefits of long-term opioid treatment? What would successful treatment look like for you? What would unacceptable risk or treatment failure look like for you?"
Overdose prevention education	"Most patients who take long-term opioids should have the medication naloxone at home. This medication can immediately reverse slowed breathing and sedation from opioid overdose. Here is a video tutorial on how to use naloxone in an opioid overdose emergency: https://www.ama-assn.org/delivering- care/overdose-epidemic/how-administer-naloxone."
Clear expectations for patient and practitioner	"There are some important responsibilities that must be agreed upon by you and your health care team if you decide to take opioid medications for chronic pain. This is in order to stay safe and make sure the medication is doing more good than harm."
Clear contingency planning for aberrant behaviors	"As your clinician, I will check for signs of addiction and offer appropriate treatment if this occurs. I will work with you to slowly change your medications if you experience safety issues, the medication is not helping you meet your goals, or if this agreement is violated. I will NOT change or stop your medications without talking with you first unless there are extreme problems such as threatening/violent actions."

Figure 1. Key elements of a patient-centered pain agreement.

Michael A. Incze, MD, MSEd, who published an article in February on redesigning opioid pain agreements, makes arguments for drawing up just such a plan.[4] Although it may not be applicable in all settings, it does represent guidelines to help patients and clinicians by sharing responsibilities and offering substantial education. The author hopes that with implementation, these guidelines can lead to a stronger therapeutic alliance, greater efficiency for practitioners, improved patient experience, and safer, more evidence-based care.

The pain agreement begins with a template for a baseline pain assessment and elicits a set of patient values and functional goals. Current evidence and knowledge gaps pertaining to benefits and harms of long-term opioid treatment are emphasized, facilitating discussion between clinician and patient, and allowing a shared understanding of what successful treatment could be expected. The agreement outlines treatment options (including ongoing opioid treatment) and a monitoring plan to ensure safety and efficacy.

A possible universal pain agreement template that promotes patient-centered care and integrates current chronic opioid-prescribing guidelines is shown in Figure 1.

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1. **The percentage of patients with symptomatic joint hypermobility who experience chronic pain ranges from**
   A. 0% to 25%.
   B. 26% to 50%.
   C. 51% to 75%.
   D. 76% to 100%.

2. **On the basis of the current classification system, Ehlers-Danlos syndromes consist of up to _____ different subtypes with phenotypic and genetic heterogeneity.**
   A. 4
   B. 10
   C. 14
   D. 20

3. **The most prevalent subtype of Ehlers-Danlos syndrome is**
   A. vascular.
   B. hypermobile.
   C. classical.
   D. myopathic.

4. **Gastrointestinal problems associated with Ehlers-Danlos syndromes include**
   A. irritable bowel syndrome.
   B. diverticulitis.
   C. pancreatitis.
   D. Crohn's disease.

5. **Pain caused when a hypermobile person overstretches beyond an appropriate range of motion is most likely to be**
   A. nociceptive.
   B. neuropathic.
   C. nociplastic.
   D. somatic.

6. **Patients living with symptomatic joint hypermobility may experience neurogenic inflammation in the form of**
   A. arthritis.
   B. joint subluxation.
   C. dislocation.
   D. migraine.

7. **A person with symptomatic joint hypermobility who experiences pain from stimuli that are not ordinarily painful is most likely to be diagnosed with**
   A. allodynia.
   B. hyperalgesia.
   C. secondary hyperalgesia.
   D. hypoesthesia.

8. **After receiving a normal test or imaging result for a patient with symptomatic joint hypermobility, the clinician should assume that**
   A. the patient is currently healthy and does not require pain treatment.
   B. there is no injury identified, so pain treatment is not needed.
   C. pain should be treated based on the clinical assessment.
   D. the patient is a hypochondriac and should not be assessed for pain.

9. **Strategizing a treatment plan for the patient with symptomatic joint hypermobility should include**
   A. a risk/benefit analysis.
   B. use of a medication that has been FDA-approved for treatment of symptomatic joint hypermobility.
   C. avoiding over-the-counter medications.
   D. aggressive dosing escalations to achieve control of pain.

10. **To relieve chronic and acute issues, 2 general categories of treatment approaches that often are used for patients with symptomatic joint hypermobility are**
    A. pharmacologic and nonpharmacologic.
    B. surgical and nonsurgical.
    C. complementary and alternative.
    D. interventional and integrative.

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Notes from recent studies, trends, and thought leadership in pain management, compiled by Elizabeth A. M. Frost, MD

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The Cost of Opioid Misuse on the Health Care Systems and Emergency Rooms

Treatment of opioid use disorder (OUD) is now estimated to cost hospitals more than $95 billion a year, according to new data from Premier Inc AI Applied Sciences. That figure approximates 7.86% of all hospital expenditures (data, released first to Axios).

The opioid epidemic has had an enormous human toll, and solving the crisis is critical for patients and for society, but there is economic incentive, as well: Fixing the epidemic can also improve hospital finances.

Although Medicaid and private insurance cover most of the costs for nonelderly adults who are treated for OUD (medication-assisted treatment, counseling, and support services), health systems and hospitals still manage the cost of treating the uninsured and homeless. In addition, many payers are slow to reimburse virtual OUD care at parity with in-person visits.

Although opioid settlements from manufacturers, distributors, and retailers now exceed $50 billion, that sum still represents just over half of hospitals' estimated annual costs for emergency department care for overdoses, among other issues.

Moreover, the addiction crisis has been worsened by the addition and the proliferation of the synthetic opioid fentanyl, which is attracting even more younger people.

Statistics indicate that patients with an OUD diagnosis average 32.5% higher cost per emergency department visit. Overdose patients are at a high risk for multiple organ failure, hospitalization, increased costs due to intensive care unit stays and unplanned readmissions after discharge.

The Premier analysis, which compared data from 2017 and 2022, showed OUD patients were younger than other emergency department patients, were more likely to be male, and Native American or White. The analysis concluded that "Collectively, healthcare needs to address [social determinants], OUD and myriad inequities experienced among patient populations to improve health and outcomes, and positively impact hospital and health system margin."

Further review of the analysis showed an uneven distribution of OUD emergency department caseload across states. Nationwide, about 2.28% of emergency department inpatient stays were associated with OUD diagnoses. However, New Mexico was highest by 7.09%, followed by New Jersey with 5.54%, while Arkansas and Iowa had the lowest percentage.

In the meantime, federal health officials have laid out guidelines for prescribing opioids for chronic pain, emphasizing that they are not first-line therapy. The Premier analysis points to studies that show that 87% of people with OUD do not receive evidence-based treatment, and there are significant demographic disparities in pain management and development and management of OUD.

The better news is that the frequency of patients discharged from emergency department visits with a prescription for an opioid decreased in 2020, compared with 2017-2018, according to recent Centers for Disease Control and Prevention data. However, in the fall of 2022, Joint Economic Committee Democrats released a report estimating the opioid epidemic cost the United States nearly $1.5 trillion in 2020—up 37% from 2017.

Although the human toll takes precedence, the crisis-induced economic shocks to the health care system must be addressed. (*See* Goldman M. How opioid misuse is costing health systems. Axios Pro: Health Care Policy January 24, 2023. Axios.com/Health.)

Adapted from *ASA Monitor*, January 25, 2023.

Coming Soon:

- Hope for Hypermobility: Part 2—An Integrative Approach to Treating Symptomatic Joint Hypermobility

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