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Back from the Drawing Board: Useful Ideas About Chronic Axial Lumbar Pain

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Clinical Vignette

M.M. is a 44-year-old man who was referred by his primary care physician due to chronic low back pain.

M.M. has a long history of episodic low back pain, dating back to 2013, with episodes initially spaced out by a couple of years. He worked with a physical therapist on body mechanics and remembers being told to be very careful. His pain episodes are usually triggered by physical actions such as bending and or twisting. Recently, these have been occurring every 18 to 24 months. When back pain occurs, he either spends time incapacitated at home or simply goes to the hospital. Usually, his symptoms improve over two to three weeks.

His current complaints following his most recent exacerbation three months ago include low back pain, posterior right thigh pain, and very occasional shooting left lower extremity pain. If M.M. stands or walks for longer than he can tolerate, he gets a burning pain in his bilateral ribs.

Magnetic resonance imaging (MRI) of the lumbar spine, obtained by his primary care physician, showed multi-level disc-height loss that appeared stable compared to the prior MRI from five years before. M.M. was seen in the musculoskeletal spine clinic hoping to get treatment to definitively address his chronic low back pain.

Definition, Etiology, and Epidemiology

Low back pain (LBP) is a symptom, not a disease, and can be associated with a variety of physiological processes and symptoms.[1,2] Strictly speaking, LBP refers to pain occurring between the 12th ribs and the iliac crest.[1,3] LBP is often "referred" to the buttocks and even the posterior lower extremities superiorly to the knees without any compression of neurologic structures.[3–5] For practical purposes in clinic, we consider such referred pain to be within the broad scope of "axial" LBP. By contrast, pain experienced along one or more dermatomes, associated with irritation or injury of one or more involved nerve roots, is termed radicular pain and is beyond the scope of this article.[5]

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LBP is often categorized based on the duration of symptoms; acute (less than six weeks), subacute (six to twelve weeks), and chronic subacute/chronic (greater than twelve weeks).[3] This is a bit of an oversimplification, however, as LBP recurs in a large proportion of people. Prior inception cohort studies have demonstrated one-year recurrence rates between 33% and 69%, with up to 41% of patients experiencing recurrences of activity-limiting LBP.[6] This often-episodic nature of LBP creates difficulty in quantifying the amount of people who develop chronic pain, although one systematic review reported a median value of 26%.[7] The overall burden of LBP mainly stems from recurrent and/or chronic LBP with these individuals more at risk for increased disability and decreased quality of life.[1]

The complications of chronic LBP can be severe and tend to relate to loss of function (e.g., professional disability, loss of social roles and activities), onset/exacerbation of behavioral-health issues (e.g., depression), consequences of decreased physical activity (e.g., cardiometabolic disease, frailty, falls), and/or iatrogenic harm (e.g., side effects of prescription medications, over-the-counter medications, or self-administered substances; complications of surgeries or other procedures).[1,2] Meanwhile, people who have a single self-limited episode of LBP may not experience any lasting ill effects. The purpose of this review article is therefore not to focus on acute, self-limited LBP, but rather to focus on those individuals who already experience or are at high risk of experiencing recurrent and/or chronic LBP. This review does not address people who experience buttock pain without LBP, e.g., due to a hamstring strain or pure sacroiliac-joint mediated pain, and it does not address people with radicular lower-extremity pain of even a mild degree (i.e., the symptom of sciatica). Moreover it does not address people with the clinical syndrome of lumbar spinal stenosis (i.e., worse pain in the buttock and/or lower extremity compared to the back; worse with standing, walking, or lumbar extension; with evidence on MRI or computerized tomography [CT] of narrowing of the lumbar spinal canal).[8] For the sake of simplicity we also do not discuss people who have had prior lumbar spine surgery.

The low back contains several tissues which can generate painful signals including muscles, fascia, ligaments, bones, intervertebral discs, and zygapophyseal (facet) joints. The nearby sacroiliac joints, though they are inferior to the iliac crest, are sometimes included among potential LBP "pain generators," though these are not within the scope of this paper.[1] Some writers classify LBP as "specific LBP" or "non-specific LBP" based on whether they think that one or more specific structures can be invoked as an anatomical cause of the patient's symptoms.[9] We do not find the term "non-specific LBP" to be useful, nor do we imagine that many patients would be satisfied with that diagnostic label.

Similarly, some writers like to label certain pain as nociplastic — deriving from central sensitization in the absence of any tissue damage[1] — in contrast to nociceptive pain resulting from tissue irritation or injury. In clinical practice we have not found the nociceptive/nociplastic dichotomy to be helpful to us or to our patients with chronic LBP. We think that making sense of chronic spine-related pain is rather more complex, analogously to other conditions that are familiar to all physiatrists.

The value of a medical diagnosis largely relates to its implications for prognosis and treatment. For some medical conditions, the prognosis may be tightly correlated to features of the underlying pathophysiology, but in rehabilitation there are often other factors that are very important. For example, two patients with a thoracic spinal cord injury at the same sensory level will have different prognoses if one of them is able with the support of loving family members to return to a fulfilling career and find a new social community of athletes with disabilities, while the other begins drinking in excess, gains fifty pounds, never checks their skin for pressure injuries, and isolates themselves socially while ruminating about the external circumstances that ruined their life.

In the patient with chronic LBP, to sort through the etiological and prognostic complexity, as well as to identify salient opportunities for improvement, we like to use the "4P" model. In this framework, the clinician builds their case formulation around predisposing factors, precipitating factors, perpetuating factors, and protective factors.[10] Which factors are most important varies from patient to patient. A personal history of repeated sexual abuse at the hands of one's stepfather, as a predisposing factor for chronic pain,[11] may be more relevant than the precipitating factor of a disc bulge that was identified on a lumbar MRI from five years ago.[12] In the patient with stage IV lung cancer, the precipitating factor of vertebral body metastasis may be more relevant than the predisposing factor of mildly sedentary lifestyle. For the patient with a scoliosis who sustained a traumatic compression fracture a couple years ago, and who has never been offered physical therapy, their prognosis may be favorable thanks to the protective factors of high self-efficacy, good social supports from a loving spouse, and a strong motivation to be able to take care of their grandchildren a few days a week for years to come.

The physiatrist must be skilled and comfortable with the evaluation and management of biomechanical factors[13,14] (Table 1), rare serious medical conditions that can present with LBP[1] (Table 2), biopsychosocial factors[1] (Table 3), and radiologic findings. Amidst this complexity, we try to be curious and open-minded about identifying whatever etiological factors seem most relevant in the case of each patient, honest and constructive with patients about our diagnostic impression within the limits of medical certainty, and pragmatic about identifying ways that patients might feel better.

Evaluation

The goal in evaluating LBP is to develop a diagnostic impression and a treatment plan. Any evaluation of LBP should consider disability and quality of life[3,6] as well as rare, life-threatening causes of LBP.[1]

History

The evaluation of LBP should start with a thorough history and physical exam.[9] These elements can assist in understanding why the patient hurts, what barriers may be standing in the way of clinical improvement, and how they may ultimately overcome some of these barriers on the road to recovery. The clinician should ascertain the location, onset (including a known trauma or inciting factor), duration, timing, quality, severity, and prior history of pain, patterns of radiating or referred pain, and known exacerbating and alleviating factors.[3]

A physiatric evaluation can sometimes shed light on possible biomechanical contributors to LBP, some of which are mentioned in Table 1.

Table 1: Biomechanical Contributors to Low Back Pain[14]

Non-neutral spinal postures in standing (e.g., hyperlordosis, flat back)
Chronic muscle guarding
Weakness of gluteal or lower-limb muscle-strength
Weakness of back-muscle strength
Proprioceptive deficits

The history should also incorporate evaluation of the so-called red and yellow flags.[1,9] As shown in Table 2, red flags indicate serious etiologies of LBP such as infection, malignancy, fracture, and cauda equina that may require urgent further medical work-up and intervention.[1,2,9,15] As with many factors in medicine, in practice each of these red flags exists on a spectrum. For example, it is less concerning if for years someone has been experiencing occasional nighttime pain when they roll over in bed, and then they are quickly able to find a position of comfort and return to sleep. It is more concerning if someone is being awoken in the middle of the night with severe pain that they have never experienced before, and they cannot find a position of comfort.

Table 2. Red Flags[1,2,9,15]

Fevers or recent infection
New bowel or bladder dysfunction
Significant and unexplained weight loss
Night sweats or pain that is worst at night
Gait dysfunction or new bilateral lower extremity weakness
Saddle anesthesia
History of intravenous drug use, malignancy, osteoporosis, or use of corticosteroid or other immunosuppressant
History of preceding significant trauma

Conceptually related to red flags are factors that point towards systemic diseases that can present with LBP, such as axial spondyloarthropathy (AS). This condition classically involves morning stiffness lasting longer than one hour and pain that is worse at night. It may involve peripheral joints, and pain improves with activity and nonsteroidal anti-inflammatory drugs (NSAIDs).[16] Family history in these cases may indicate a predisposition to autoimmune disease.

Yellow flags (displayed in Table 3) were first described by Kendall et al. and refer to psychosocial and socioenvironmental risk factors that have been associated with poorer outcomes.[1,9,15] Yellow flags have been associated with the transition to chronic and persistent pain, prolonged disability, and failure to return to work; investigating their presence is a critical part in the LBP evaluation.[15]

Table 3. Yellow Flags[1,2,9,15]

Pain catastrophizing
Fear-avoidance
Perception of lack of control over one's life
Poor job satisfaction or conflicts at work
Preference for passive rather than active treatment strategies
Overwhelming distress, low mood, and/or social withdrawal
Lack of social support system and/or presence of toxic relationships
Financial or compensation concerns

The history presents an important opportunity to build a therapeutic alliance with the patient. Prior literature has demonstrated the association between strong therapeutic alliances and positive health outcomes in medicine, such that the patient-clinician relationship might be considered a key part of any treatment plan.[17] More specifically, higher levels of therapeutic alliance between physical therapists and patients with LBP have been associated with greater improvements in function and perceived efficacy of treatment, in addition to reductions in both pain and disability.[17] We strive during the evaluation to create a non-judgmental environment, with an emphasis on active listening, empathy, curiosity, and candor.

Physical Examination

Any physical examination of LBP should assess for serious pathologies requiring urgent attention.[9] A detailed neurologic examination, including assessment of gait, strength, sensation, and deep tendon reflexes can help to identify neurologic compromise.[18] Special tests such as the slump test and straight leg raise are generally more useful to characterize radicular than axial pain. Significant focal tenderness to palpation over the vertebral column may indicate fracture, infection, or malignancy, though this finding should be interpreted in the context of other factors on history and exam (e.g., whether the patient is also tender diffusely over the paraspinal muscles and elsewhere in the body).[18] If AS is suspected, the exam should include an assessment for both peripheral joint inflammation (e.g., arthritis, enthesitis, or dactylitis) and non-musculoskeletal features (e.g., signs of psoriasis or ocular inflammation).[16] In some patients, an abdominal exam can be relevant (e.g., if it seems that the pain might actually be coming from the abdominal viscera, or if the patient has undergone extensive abdominal surgery that has compromised their core musculature).[3,18] In addition, every patient with LBP should undergo a focused assessment of each hip joint.

In our experience, the physical exam tends not to be very helpful in sorting out the extent to which specific spinal structures might be contributing to most patients' chronic axial LBP. Inspection of spinal alignment and range of motion are helpful in analyzing the biomechanics of the spine, in addition to providing information about the patient's current function and where there may be room for improvement.[13,14]

Screening Tools

One commonly used tool to screen for LBP is the Keele STarT (Subgroups for Targeted Treatment) Back instrument. STarT Back is a validated tool designed to assist in diagnosing patients with LBP who may be at risk for a worse prognosis.[19] This tool is a nine-question survey that incorporates physical and psychosocial elements to stratify patients into low, medium, or high risk for a worse prognosis. The resulting score can help the clinician to better understand the patient and to guide treatment accordingly. Patient Reported Outcomes Measurement Information System (PROMIS) refers to a system of instruments that have been validated in a variety of conditions, including LBP. Developed by the National Institutes of Health, the PROMIS questionnaires can provide useful information regarding patients' lives, including fatigue, emotional distress, sleep, physical function, and pain interference.[20] In our clinical practice, both the Keele STarT Back tool and PROMIS-16 questionnaire are part of the intake process.

Laboratory Tests

In most patients presenting with LBP, laboratory tests are not necessary. In rare cases where history and physical examination are concerning for vertebral osteomyelitis/discitis, ordering the inflammatory markers erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) and a complete blood count with differential is helpful, as ESR and CRP are highly sensitive for spine infections. The combination of ESR, CRP, and the HLA-B27 antigen test can be useful in the diagnosis of AS.[18] Laboratory evaluations also certainly have a role in the evaluation of possible malignancy, although this topic is beyond the scope of this article.

Imaging

Most clinical guidelines support the use of imaging if red flag symptoms are present or if concerns exist for specific diseases.[1,9,21] However, in the majority of cases, imaging is not typically recommended. In a review of twelve clinical guidelines with diagnostic recommendations for "non-specific" LBP, 100% recommended against routine imaging, and only two of the twelve recommended imaging if pain persists beyond four to six weeks.[9] A prior meta-analysis of imaging strategies for LBP without red flag symptoms was consistent with this review, documenting no significant difference in short- or long-term outcomes between those individuals who underwent immediate lumbar imaging and those who did not.[22]

Clinical recommendations dictating when to obtain imaging in chronic axial LBP in the absence of concern for serious underlying pathologies are less definitive. It is well known that radiographic evidence of spine degeneration is present in large proportions of asymptomatic individuals and increases in prevalence with age.[12] Thus, the presence of degenerative spine findings on imaging may not necessarily explain or correlate with patients' symptoms. In comparing individuals with clinical spinal stenosis versus mechanical LBP, Haig et al. concluded that MRI was not able to distinguish between the two.[23] Higher health care expenditures, increased radiation, overdiagnosis, and risk of unnecessary procedures can occur with the overuse of imaging.[21] As such, international guidelines now promote reserving imaging for those patients with LBP and either red flag symptoms or planned therapeutic interventions for which imaging is necessary.[24]

Nonetheless, imaging is still commonly used in clinical practice. Approximately one-quarter of patients presenting to primary care settings with acute LBP in the United States (US) and Australia undergo imaging.[2] These rates are even higher in emergency department settings, with one-third of patients receiving imaging in the US.[2] In fact, a systematic review examining over nineteen million cases of LBP from 1994 to 2015 demonstrated a 53% relative increase in CT, MRI, or nuclear bone scans.[21]

If patients have available imaging, whether ordered by us or by a prior clinician, we find it useful to show people their images while we discuss the relevant anatomy. There is an art to educating patients about the tenuousness of the relationship between imaging findings and current or future symptoms.[1,12] We strive to frame imaging findings in such a way that the patient feels informed and empowered to move toward a realistic and tangible improvement in their life, rather than disabled or doomed by their circumstances. We understand such efforts to be in line with paradigms such as cognitive behavioral therapy (CBT)[25] and acceptance and commitment therapy (ACT),[26] as well as the foundational medical-ethical principle of autonomy.[27]

Treatment Paradigm

The treatment of chronic axial LBP is not straightforward. When considering treatments for chronic axial LBP, it can be helpful to distinguish between patient-controlled and provider-controlled treatments. We define patient-controlled treatments as ones for which the patient is responsible, or for which the majority of agency lies with the patient. Conversely, we define provider-controlled treatments as interventions that a provider is responsible for (such as lumbar injections or spinal manipulation), where the patient's role is basically limited to scheduling, attending, and paying for the care (as in the case of procedures) or acquiring and applying the treatment (as in the case of medications and heating pads). There is arguably some gray area here: we would consider over-the-counter medications to be more patient-controlled than Drug Enforcement Administration-scheduled ones, and self-massage with a tennis ball to be more patient-controlled than chiropractic manipulation. We try to emphasize patient-controlled treatments, as they seem to work roughly as well as the alternatives (see below), and their chief side effects include beneficial ones like providing patients with ownership over their own rehabilitation. For most patients with chronic LBP that we see, our aim is to catalyze their eventual independence from us.

Provider-Controlled Treatments

Some provider-controlled treatments can be helpful, and varying levels of evidence support their use. In cases when we prescribe one or more of these treatments for chronic LBP, we try to frame them as potentially beneficial adjuncts, but not strictly necessary. Our concern is that, among other side effects, provider-controlled treatments can exacerbate patients' sense of powerlessness, which is counterproductive to the overall recovery process. In case this seems like a minor or abstract point, we will attempt to illustrate our perspective with the following hypothetical example:

There are two 55-year-old men with several years of chronic axial LBP. Both are neurologically intact, and their X-rays show disc endplate changes that are typical for their age. Patient 1 had a few visits with a physiatrist whose name he has forgotten, who prescribed a two-month course of weekly physical therapy. In physical therapy, there was some trial and error with regard to pain flareups, but pain improved after his therapist and physiatrist collaborated to adjust the exercise program. Patient 1 now maintains an average pain level of four by doing several simple home exercises a few times a week with occasional pain flares after pickleball that reliably calm down with stretching, hot showers, and over-the-counter naproxen. Patient 1 recalls being advised to switch his social exercise to something gentler, like Silver Sneakers classes, but is not yet ready for this.

Patient 2 self-discontinued physical therapy after the second session because pain worsened. His pain management physician, whom he adores, found a regimen that provides relief using tramadol, duloxetine, cyclobenzaprine, baclofen, and diclofenac, with annual radiofrequency ablations (RFA) of the bilateral facet joints at multiple levels. Pain is maintained at an average level of four by sleeping every night on a heating pad and by avoiding walks in his neighborhood, yardwork, and pickleball, all of which he used to enjoy. Unpredictable pain flares occur after seemingly minor "wrong" movements; the only thing that helps is a twelve-day oral taper of prednisone. Patient 2 worries what will happen when the pain management physician retires because his general practitioner, who periodically reminds him to watch out for symptoms of serotonin syndrome, has stated that he is unwilling to prescribe chronic tramadol. Patient 2 is also concerned that spine surgery will eventually be required or he will end up in a wheelchair. His pain management physician's partner, a spine surgeon, told him that these are indeed his only two options.

Passive Modalities

Passive modalities for chronic LBP can include acupuncture, heat, ice, massage, and spinal manipulation.[28] We will sometimes recommend deep-tissue massage (or soft-tissue manipulations by a physical therapist) to loosen tight muscles and thereby allow for better movement patterns, though we do not expect the massage itself to provide durable pain relief. Acupuncture does not seem to offer any long-term benefit in LBP.[1] Spinal manipulations have been reported to improve pain and quality of life compared to placebo,[1] but these spinal manipulations are not long-term solutions for postural abnormalities or kinetic chain dysfunction.

Oral Medications

Oral medications are commonly taken by patients with chronic LBP. NSAIDs are an effective class of medications, though we (and many patients) prefer to avoid lifelong use due to risks on the kidneys, gastrointestinal tract, and cardiovascular system.[29] Opioid and benzodiazepine (BDZ) use for non-terminal causes of chronic LBP has shown benefits in limited follow-up periods usually less than three months.[30,31] Acetaminophen, gabapentin, selective serotonin reuptake inhibitors (SSRI), tricyclic antidepressants (TCA), and non-benzodiazepine muscle relaxants have likewise shown minimal efficacy at short-term follow-up periods of less than three months.[31] However, there remains a lack of high-quality long-term data that either supports or refutes the use of these medications in chronic LBP.[32] See Table 4 on page 6 for a list of medications that may be utilized for LBP and their effects on pain intensity, disability, and function.

Non-Surgical Procedures

Non-surgical procedures are widely performed for chronic LBP and include trigger-point injections, epidural corticosteroid injections, intra-articular facet-joint injections, medial-branch RFA intradiscal electrothermal therapy, spinal cord stimulation (SCS), basivertebral nerve ablation, and medial branch stimulation.[31,37] In RFA, a probe is used to burn (ablate) the lumbar medial nerve branches that receive pain signals from the lumbar facet joints. Of note, RFA denervates the multifidus muscles that are innervated by the medial branches, which has been confirmed via MRI post-RFA.[38] We are unaware of any definitive evidence that multifidus denervation is clinically harmful; however, it is theoretically concerning given that optimal management of other arthritic joints involves strengthening as opposed to weakening musculature across joints. See Table 5 on page 7 for a list of non-surgical procedures for LBP. We once again note that the literature is limited by a dearth of long-term data.

Surgeries

Spinal fusion surgery is an option to address dangerous instability (e.g., in certain vertebral fractures and cases of vertebral osteomyelitis, among other indications). Spinal fusion is typically used in the subpopulation of LBP patients who fail to respond to reasonable conservative measures and whose pain is thought to be due to segmental instability.[47] In a systematic review on spinal fusion for chronic LBP related to degenerative disc disease, Phillips and colleagues analyzed six randomized controlled trials that compared fusion surgery (n=547) to non-surgical management (n=372). They found that the weighted average of improvement in back pain, on a scale of 100, was 22.8 (surgical group) versus 10.6 (nonsurgical group). The weighted average improvement in Oswestry Disability Index (ODI), a patient-reported-outcome questionnaire that measures back-pain-related functional disability on a scale of 100,[48] was 13.9 (surgical group) vs. 8.2 (nonsurgical group).[47] This unclear benefit of surgery must be weighed against the risk of surgical complications, including post-operative pain, paresthesias, epidural hematoma, spinal cord injury, blood loss, and infection.[49] One must also consider that in the overall population of people with chronic LBP who have not responded to conservative treatment, many individuals may be suffering related to yellow flag factors.

Unfortunately, just as yellow flags bode ill for LBP prognosis generally, they also predispose to poor operative outcomes.[50] In many others with chronic LBP, pain relates to underlying biomechanical factors which surgery will not address.

We believe that surgery has an important role in addressing certain anatomic pathologies, which is beyond the scope of this paper to define or limit. Yet, we dispute the common framing that surgery can be kept as a 'last-line' option for essentially any patient with chronic LBP if only they manage to 'fail' enough conservative therapies.

Table 4. **Medications for LBP**

Drug Class	Examples	Data
Acetaminophen	Paracetamol, acetaminophen	• No data analyzing long-term effectiveness in isolation versus placebo[33] • No increased risk of adverse event versus placebo[33]
NSAIDs	Celecoxib, ibuprofen, diclofenac, naproxen, meloxicam	• No compelling evidence that any NSAID is more efficacious than others[31] • No known data analyzing long-term effectiveness in isolation versus placebo[32] • 2.5 times increased risk of gastrointestinal adverse events versus placebo[34]
Muscle Relaxants	BDZ Non-BDZ: Methocarbamol, cyclobenzaprine	• Hazard of death was doubled in BZD use versus control[35]
Opioids	Tramadol, oxycodone, hydrocodone	• No known data analyzing long-term effectiveness in isolation versus placebo[32] • At 31 days of use, 43% risk of persistent use at 1 year[34] • Increased risk of nausea, constipation, headaches, and somnolence were 10-fold versus placebo[36]
Gabapentin-oids	Gabapentin, pregabalin	• Gabapentin is recommended by most organizations for neuropathic pain only, but lacks strong evidence[1] • No support for axial LBP [31]
Anti-depressants	SSRI, SNRI, TCA	• No known data analyzing long-term effectiveness in isolation versus placebo[32] • Can increase suicidal behavior, dry mouth, constipation, hypotension, and drowsiness[31] • No data to support use of SSRI or TCA specific to LBP[31]

BDZ = benzodiazepines; LBP = low back pain; Non-BDZ = non-benzodiazepines; NSAIDs = nonsteroidal anti-inflammatory drugs; SNRI = serotonin norepinephrine reuptake inhibitor; SSRI = selective serotonin reuptake inhibitor; TCA = tricyclic antidepressant; VAS = visual analog scale.

Patient-Controlled Treatments

Patient-controlled treatments can be further divided into psychosocial rehabilitation (which we define as enhancing appropriate insight, emotional tools, and coping mechanisms) and active rehabilitation (which we define as enhancing function through physical exercise). The psychosocial rehabilitation subgroup can be stratified into education and behavioral therapy, while the active rehabilitation subgroup can be divided into physical therapy and exercise. In clinical practice, these concepts are often closely related. In a single conversation, a physiatrist might explain that the patient's radiological findings are not as dire as previously thought, in an effort to reduce catastrophization and hopelessness, so that the patient is willing to try physical therapy, with a near-term goal of building a sustainable a home exercise program that helps them to build task-specific conditioning, so that they can return to their preferred activities with less pain interference. Despite the real-world overlap, because these concepts are often researched separately, in the following paragraphs we treat each as distinct.

Psychosocial Rehabilitation

Education

Previous studies have shown that pain education for patients with chronic pain leads to lower pain intensities and greater expectations of recovery.[51] The focus of this education is multifactorial and includes helping patients to understand why they are hurting and how to constructively frame their goals. Additionally, it can be helpful to acknowledge the complex interrelationship between their pain and other key components of a healthy lifestyle (e.g., sleep, movement, food, love, purpose), so that patients can envision ways that their life may get better even if their pain intensity does not. Helping patients to recognize classic red flag symptoms (see Table 2 on page 3) can aid in decision-making — such as when to seek urgent care. Fortunately, most patients with LBP do not have these conditions.[1]

Behavioral Therapy

Behavioral therapy has significant potential for helping patients with restoring values, goal-setting, and disengaging from avoidant behaviors.[25] Behavioral therapy aims to break the cycle of pain-avoidant behaviors by providing new information to patients that pain can be self-managed and does not require aggressive protection. One randomized controlled trial measured outcome variables in patients with chronic LBP: patients who received behavioral therapy scored better on 26 out of 33 measures, including fear-avoidant beliefs and pain-free days. These patients overall lowered their risk for long-term disability.[25] Behavioral therapy often includes exposure treatments to achieve this reduction in pain-related fears and decreased recovery time. One systematic review on ACT revealed medium-sized effects on improving function, anxiety, and depression.[1] Overall, restoring individual-valued goals is of utmost importance, and this is the foundation for constructing meaning and quality of life, despite having chronic LBP. This can be facilitated with CBT, ACT, or individualized mindfulness such as meditation, deep breathing, or journaling. Encouraging patients to incorporate one or more of these techniques can help them to regain ownership of their quality of life.

Active Rehabilitation

A foundational pillar in the patient-controlled treatment of LBP is active rehabilitation. As patients develop chronic LBP, their activity levels decrease due in part to pain-avoidant behaviors; a return toward activity is often part of healing. A 2020 systematic review and

Table 5. Non-surgical Procedures for LBP

Procedure	Description	Risks	Data
Trigger Point Injections	Target soft tissue superficial to the vertebral column with anesthetic, with or without steroid	Pain, paresthesia, infection, hematoma, headache, dizziness	• No significant reported self-improvement between lidocaine versus placebo[39] • Improvement with addition of steroid seems plausibly due to systemic steroid exposure[39]
Epidural CSI	Target epidural space surrounding nerve roots with steroid, with or without anesthetic	Pain, paresthesia, CSF leak, spinal cord injury, infection	• Can be helpful for radicular pain • No definitive studies have been conducted in axial LBP
Intra-articular Facet Joint Injection	Target facet joints with steroid, with or without anesthetic	Pain, paresthesia, infection	• Review of 9 RCTs demonstrated Level IV evidence and weak strength of recommendation to support lumbar facet intra-articular injections[40]
Facet RFA	Target facet joint-innervating medial branches with thermal energy to destroy the nerves	Pain, paresthesia, infection, radiculopathy	• RFA versus placebo sham RCT showed no significant improvement between groups (28% versus 29% respectively) at 3 months follow-up[41] • RFA versus exercise-only control RCT showed no clinically significant improvement in pain at 3 or 12 months follow-up[37]
Intradiscal Electrothermy	Target intervertebral disc with heat	Pain, vertebral osteonecrosis, cauda equina syndrome, transient radiculopathy[42]	• High quality RCT showed no significant differences in pain, disability, or quality of life measures between treatments[42]
SCS	Implant stimulator that targets dorsal columns of spinal cord with electrical leads	Pain, paresthesia, CSF leak, spinal cord injury, infection	• SCS revision rates in 31% of patients at 2 years[43] • No significant improvement in back or leg pain, function, or quality of life compared with placebo[43]
Basivertebral Nerve Ablation	Place probe within vertebral body and heat it up to ablate basivertebral nerve, which supplies vertebral endplate	Pain, paresthesia, radiculopathy, retroperitoneal hemorrhage, urinary retention infection[44]	• Mean difference between active and sham-control groups was less than 10 for ODI and less than 1 for VAS at 3, 6, and 12 months[45] • 11% of ablated patients were excluded from intent-to-treat analysis due to the anatomic target having reportedly been missed, in an astonishing mischaracterization of the term "intent-to-treat"[45]
Medial-branch Nerve Stimulation	Implant stimulator that targets medial branches, "to restore multifidus neuromuscular control"[46]	Pain, numbness, infection[46]	• Failed to meet primary efficacy endpoint in industry-funded sham-controlled trial[46] • Close analysis of the sham and active groups, both of which experienced dramatic initial benefit, suggests that slight between-group differences at 120 days may have been driven by a gradual waning of placebo effect in the former[46]

CSF = cerebrospinal fluid; CSI = corticosteroid injection; LBP = low back pain; ODI = Oswestry Disability Index; RCT = randomized controlled trial; RFA = radiofrequency ablation; SCS = spinal cord stimulation; VAS = visual analog scale

meta-analysis by Huang et al. assessed exercise, education, back belts, insoles, and ergonomic adjustments and demonstrated statistically significant improvements in LBP.[52]

Across the population of people with LBP, data do not support any one specific exercise as being superior to another.[31] As such, we try to steer patients towards a type of exercise that they are likely to enjoy within reasonable limits. For example, in people with pain related to ligamentous laxity, we recommend therapeutic exercises that involve stabilization rather than aggressive stretching. Our general principle for maximizing function with exercise is to begin increasing activity tolerance, range of motion, and confidence with small, incremental progressions back toward premorbid activity levels.

UPMC Program for Spine Health

UPMC and other centers have developed interdisciplinary programs to help patients who are chronically in pain.[53] Our Program for Spine Health incorporates nurse care managers, physical therapists who have undergone postgraduate training in spine care, a pain psychologist, a dietician, and a health coach. In a preliminary outcome analysis, most patients report clinical improvement in PROMIS scores relative to baseline, and rates of spine surgery are lower in our patients than in a matched comparator group (unpublished internal data). Anecdotally, patients and referring clinicians appear to appreciate the program's evidence-based and comprehensive approach to diagnosis and management of chronic LBP.

Case Vignette Outcome

Mr. M.M.'s STarT-Back Score was consistent with a medium risk profile, and his social history was notable for family stressors causing anxiety. We referred him to our physical therapist, health coach and dietician. At time of our initial visit, his tolerance of standing or walking was limited to 10 to 15 minutes. By the time of our second visit two months later, he was walking 30-40 minutes with less bothersome LBP symptom severity. He appeared excited to regain physical activities that he enjoyed, especially bicycling. He completed a total of 16 sessions with physical therapy, several with the health coach, and two with the dietician. By the time of his third physician visit, four months after the initial visit, he had lost 45 pounds and had been habitually walking four-to-five times a week and going to the gym multiple times a week. We congratulated him on his progress and advised him to follow up with us as needed.

References

Click here for References 1-53.

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ABOUT THE DEPARTMENT OF PHYSICAL MEDICINE AND REHABILITATION

- Our flagship location, UPMC Mercy, is nationally ranked #7 in rehabilitation by *U.S. News & World Report*.
- The Department of Physical Medicine and Rehabilitation is consistently a top recipient of NIH funding for rehabilitation-related research.
- The Spinal Cord Injury Program at UPMC is one of only 14 in the country selected by the National Institute on Disability, Independent Living, and Rehabilitation Research (NIDILRR) as a model for other rehab providers.
- The Department has 72 renowned faculty members, one of the largest residency programs in the nation, and offer four ACGME-accredited fellowships and one innovative spine care fellowship.
- Department clinicians lead UPMC's rehabilitation network, which includes 264 inpatient beds across 12 units, more than 80 outpatient locations, and five transitional rehab locations within skilled nursing facilities — one of the country's largest.

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