Chronic Pain: Fundamental Scientific Considerations, Specifically for Legal Claims

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Introduction

Chronic pain is common, affecting almost half of adults in the United States per a 2011 Gallup poll (Brown). More specifically, the survey revealed:

- 31% of US adults have chronic neck or back pain
- 26% have chronic knee or leg pain
- 18% have some other chronic pain
- 47% have at least 1 of these chronic pain problems

The high prevalence of chronic pain in the population is often overlooked in workers’ compensation and personal injury claims, where ongoing pain is often misinterpreted as not only an indication that injury or illness occurred, but that it has also left permanent residuals manifested in the pain. General medical clinicians (those not specializing in psychological issues) are regularly asked to evaluate chronic pain in the context of a claim, but this often compounds the misattribution, given the definition of pain (Merskey & Bogduk), which states:

- Pain is “always a psychological state.”
- “Many people report pain in the absence of tissue damage or any likely pathophysiological cause; usually this happens for psychological reasons.”

Scientific findings, examples of which are provided below, have indicated that psychological and social factors are the driving forces behind most chronic benign pain presentations, especially in a claim context. Because general medical clinicians are frequently asked to evaluate chronic benign pain, and are often asked to render opinions regarding its cause, this article summarizes scientific evidence regarding the etiology thereof.

The scientific findings listed in this article are of relevance to at least 3 of the AMA Guides:

- AMA Guides to Evaluation of Disease and Injury Causation (Melhorn & Ackerman): For example, these findings are directly relevant to the fourth step of the causation-analysis protocol, which calls for “determining if other risk factors provide a better explanation for the clinical presentation than that which is provided by the claimed cause” (Barth 2012).
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• AMA Guides to Work Ability and Return to Work (Talmage 2011): These findings provide direction for determining which scientifically established risk factors for disability are of relevance to the individual case, so that an individualized rehabilitation/treatment plan can be formulated.

• AMA Guides to the Evaluation of Permanent Impairment (Rondinelli et al): These findings provide direction for determining the factors that are driving the claims of impairment.

The Dominant Role of Financial Compensation

Scientific findings have indicated that eligibility for compensation is the dominant factor for chronic pain claims.

Prospective Research

Prospective research designs provide the most credible and reliable scientific information (Melhorn & Ackerman). Because of the unique value of prospective research, this article begins the review of scientific findings by focusing on Carragee’s one-of-a-kind prospective project. The results of that low-back-pain (LBP) project were first presented at the 2005 Annual Meeting of the North American Spine Society (NASS) and then published in 2006, with discussions thereof in both Spine and The Spine Journal. The researchers recruited 200 participants who denied any history of significant LBP and proceeded as follows:

• The researchers gathered detailed baseline medical data, including findings on physical examination and spine imaging.
• They also obtained minimal, but significant, baseline psychological data: the Modified Somatic Perception Questionnaire (which addresses somatoform phenomena) and the Zung Depression Inventory. The data set, while limited, is significant because these 2 questionnaires have been scientifically identified as predictive of the development of low back pain and insensitive to any effects from LBP (Mannion). The relationship between abnormalities on these questionnaires and development of pain is unidirectional: abnormal responses predict pain onset, but the development of pain does not change the response pattern on the questionnaires.
• The researchers then followed the participants for 5 years, checking the status of each every 6 months.
• The experience of physical trauma was monitored over the course of the project. The project’s definition of physical trauma included lifting (as well as falls, traffic accidents, sports/exercise injuries, and an “other” category). These phenomena were considered traumatic if they were associated with the definitions of “serious low back pain,” “minor trauma,” or “major injury,” provided below.

The researchers defined “serious low back pain” as “pain intensity . . . >6/10 for at least 1 week.” “Minor trauma” was defined as “any perceived injury to the low back area with a back-pain intensity >2/10 for at least 48 hours but not meeting the major injury definition.” The “major injury” definition was LBP “associated with high energy trauma resulting in serious visceral injury, proximal long bone, or pelvic or spinal fracture or dislocation.”

Details of the study were published in Spine and The Spine Journal in 2006, but for the current discussion, Dr. Carragee’s simplest summary of the findings, presented at the 2005 NASS meeting (as documented in The Back Letter, Volume 20, No. 11, November 2005), will suffice. He explained, “Minor trauma was only associated with serious low back pain in a compensation setting.” None of the
participants who were not eligible for compensation developed serious LBP after minor trauma.

The findings also included a general lack of association between minor trauma and adverse low back pain events. The risk of developing serious LBP was similar for those who experienced minor trauma and those who did not. Contrary to claims of “cumulative trauma,” the study revealed no significant increase in serious LBP in people who experienced more than 4 minor traumatic events compared to those having none.

The researchers further reported, “Serious low-back-pain episodes were most frequently seen arising spontaneously or with usual daily activities rather than involving trauma of any sort.”

For motor vehicle accidents, the risk of serious LBP was significantly greater when the subject perceived others were at fault (19%) versus his or her own or no one’s fault (2.1%, and none of these instances of serious low back pain were associated with disability). When the participant did perceive the accident to be his or her own or no one’s fault, the episodes of serious LBP were limited to relatively high-speed collisions (30 to 35 mph). By contrast, for those who reported serious LBP following a collision perceived to be caused by someone else, only 1 occurred at a speed over 30 mph, the majority being between 20 and 30 mph, several at less than 20 mph, and 2 at less than 10 mph. Among the results published in Spine, the researchers noted:

• “Serious low-back-pain events were more likely at low speed when others were perceived as responsible for the accident (p = 0.001).”
• “It is interesting that traumatic episodes associated with the least relative forces described were highly correlated with compensation claims or the perception of others being at fault for an accident.”

In terms of anatomical findings, the researchers’ summary statements included:

• “Subjects with advanced structural findings were not more likely to become symptomatic with minor trauma events than with spontaneously evolving low-back-pain episodes.”
• “Follow-up magnetic resonance imaging evaluating new serious low-back-pain illness rarely revealed new clinically significant findings.” Only 3% of the cases of new back pain produced new imaging findings that were clinically relevant.

For subjects whose disability lasted more than 1 month, only 14% had new findings on spine MRI (1 had new spondylolisthesis, progression of end-plate changes, and advanced stenosis; 1 an extruded disc herniation with root compression; and 1 worsening of degenerative disc disease from grade 1 to grade 3–4). The most important of these findings (the new spondylolisthesis and severe stenosis and the new disk extrusion) occurred without trauma.

In Spine, Dr. Carragee et al explained that even though subjects with compensation claims were more likely to have a new MRI performed after minor trauma, they were less likely to have new or progressive findings. In The Spine Journal, the researchers further stated: “No patient with a compensation claim had a clear new finding of significant pathology.” The researchers further expressed concern in regard to the finding that factors that indicate a lack of need for spine imaging (the filing of a medical-legal claim, prepain abnormal responding to psychological questionnaires, pre-existing chronic pain, a history of smoking) were all actually predictive of a higher likelihood of spine imaging being conducted (researchers reference previous scientific findings that indicate that such unjustified use of imaging is predictive of a lesser sense of well-being for the patients).

The researchers reported the following additional anatomical findings:

• “Serious low-back-pain events were not significantly more common in subjects with disc degeneration or annular fissures, whether the subjects had a minor trauma or not.”
• 21% of subjects with no disc degeneration had a disability event during the study, compared to 22% with degeneration. Severe loss of disc height was not significantly associated with back pain.
• “There was no increased disability in subjects with end-plate changes compared to those without.” Moderate to severe end-plate changes were not significantly associated with back pain.
• Moderate to severe spinal canal stenosis was not significantly associated with low back pain.

In the discussion of results that was published in The Spine Journal, the researchers additionally emphasized the common nature of several spine findings for individuals who are free from pain. They noted that such spine findings are often mistakenly interpreted as an explanation for pain complaints and even as evidence of injury. They warned that the common nature of these findings among people who do not have any pain causes the premise that there is an association between these findings and specific symptoms or events to become “untenable.” They offered the following examples of spine-imaging findings that are common for people who are free from pain:

• disc protrusion or extrusion (present in 50% of their sample of pain-free individuals)
• annular fissures (nearly 30% of their sample of pain-free individuals)
• potential root irritation (22% of their sample of pain-free individuals)
Chronic Pain (continued)

In Spine, the researchers reviewed previous literature that similarly documented the lack of relationship between spine-imaging findings and back pain. In The Spine Journal, they included a more extensive literature review and noted that all of the following have been hypothesized as causes of LBP, but those hypotheses have been thwarted by scientific findings that all of these issues can be found in subjects with no back pain or only minor problems:

• acute annular tear extending into the innervated outer annulus
• existing annular fissure that becomes inflamed and appears bright on MRI
• minor end-plate failures causing rapid structural failure of the disc
• disc herniation and distention of the annulus with compression of neural elements

In The Spine Journal, the researchers also reported that the most common imaging finding (progressive loss of disc signal intensity) has been shown, in prior scientific investigations, to be primarily an aging phenomenon that is not well correlated with symptoms. The second most common finding (progressive facet arthrosis) is a slowly evolving process unlikely to be causally related to trauma or any other recent event. The researchers concluded:

• There is not a causative relationship between structural changes in the spine and serious low back pain.
• There is not a credible basis for an “injury model” for low back pain.

While minor trauma was not predictive of serious low back pain, baseline factors that did predict serious LBP were:

• previous history of chronic pain complaints in another part of the body
• history of smoking
• abnormal responding to the psychological questionnaires at baseline
• previous history of filing medical-legal claims

A model using the above four factors correctly predicted 80% of the serious LBP events. A model using only abnormal baseline psychological questionnaire responses and a previous history of medical-legal claims correctly predicted 93% of the disability attributed to LBP.

In Spine, the researchers provided a review of previous studies, which found that preexisting psychological factors were significant predictors of back pain and work incapacity, while spine imaging was not predictive. In The Spine Journal, they summarized scientific findings indicating that, “Progression of subclinical common backache or acute back pain to serious disabling low-back-pain illness appears to be associated with various nonstructural issues such as emotional distress, poor coping strategies, compensation disputes, and other chronic pain problems.” Dr. Carragee et al also reiterated that prior scientific research has repeatedly found psychological and social issues were better predictors of significant LBP than general medical findings.

The dominant role of financial, psychological, and other nongeneral medical factors in disabling LBP is especially noteworthy in view of other features of the research design:

• The researchers recruited participants at high risk to develop significant LBP based on previous chronic pain in other body parts, past medical-legal claims, and abnormal responding on the somatoform and depression questionnaires.
• They also specifically recruited participants with degenerative spinal disease to address the unvalidated premise that spinal degeneration is a significant risk factor for development of significant LBP.
• Trauma was specifically analyzed as a potential risk factor for the development of serious low back pain.

Because of these design features, this study was especially well-suited to reveal the predictive strength of general medical factors. It can be said the deck was stacked in favor of validating these as risk factors for chronic pain. Despite this, eligibility for compensation still emerged as a necessary condition for development of chronic pain following minor trauma. Hence, compensation must be considered a, if not the, primary risk factor for chronic pain in a claims context.

Meta-Analyses

Rohling et al, 1995

In 1995, Rohling et al published a meta-analysis focused on the relationship between financial compensation and chronic pain. They found 157 relevant published studies, but only 32 contained quantifiable data sufficient for meta-analysis. Those 32 articles led to a sample of 3802 compensated and 3849 noncompensated chronic pain patients.

Their simplest/broadest finding was that “patients who received compensation also reported greater experience of pain” (effect size 0.60, p less than 0.0002). The researchers additionally discovered: “receiving financial compensation is associated with . . . reduced treatment efficacy.”

Because back pain dominates scientific research on chronic pain, they next investigated the possibility that the significant effect of compensation on chronic pain presentations is limited to back pain. When back-pain projects (effect size 0.62) were considered separately from those focused on other chronic pain (effect size 0.50), the difference in effect sizes was not statistically significant.
They researchers utilized both liberal and conservative methods of calculation to rule out “publication bias” (the risk that the effect size is exaggerated by the project’s focus on published research, because of the tendency of journals to reject studies with nonsignificant findings). The overall effect size was in the range of 0.50 to 0.60 regardless of which calculation method was utilized. They explained, “... it is unlikely that there are a sufficient number of unpublished or unidentified studies in existence to diminish the finding to non-significance.”

The researchers conducted several subsequent analyses to address the following questions: “What is the most likely understanding of this association? Does compensation result in increased pain, does increased pain result in compensation, or is a third factor controlling the obtained association?”

Their analyses produced the following conclusion: “The most likely interpretation of this association is that compensation results in an increase in pain perception and a reduction in the ability to benefit from medical and psychological treatment.” Reasons for this conclusion included:

- All treatment-outcome studies included in this meta-analysis used a control group of noncompensated chronic pain patients who were either matched to the compensated group on pain and general medical variables or considered sufficiently similar to be satisfactory controls.
- The researchers conducted a new analysis to address the issue of whether “compensated patients were more physically injured, which would justify their need for compensation.” This was limited to research that adjusted or matched patients for the extent of physical injury. This analysis included treatment-outcome studies (as was discussed above) and studies that simply contrasted compensated and noncompensated patients with respect to their pain experience. The resulting effect size of 0.57 was almost identical to the original 0.60.

Because being away from work is reliably detrimental for pain they additionally analyzed the data to determine whether the association between compensation and pain was an artifact of employment status. That analysis did not eliminate the significance of the compensation effect or indicate that employment status had a stronger effect on chronic pain than compensation status.

Additional analyses corroborating the researchers’ conclusions included:

- The effect size did not significantly differ when treatment-outcome studies were compared to those that simply contrasted compensated and noncompensated patients with respect to their pain experience.
- An analysis focused on the duration of the pain complaints did not reduce the significance of the effect size.

- An analysis focused on quality of the research design (3 levels of quality were used) for the included studies did not reveal a significant difference in effect size.
- A comparison of objective (eg, number of days missed from work due to pain) and subjective (eg, pain severity) measures did not reveal a difference in effect size. (There was actually a trend for objective measures to be more strongly influenced by compensation.)
- The comparison of clinician ratings to patient ratings did not reveal a significant difference in the effect size (although there was a trend toward clinicians’ ratings being more strongly affected by compensation than patients’ ratings).
- A comparison of participants who were simply granted benefits without an adversarial process, versus those who engaged in an adversarial process in an attempt to gain compensation, also failed to significantly affect the effect sizes.

The researchers found that noncompensated chronic pain patients have experiences that average, across different outcome measures, 24% better than those of compensated patients. This could mean 24% less pain, 24% fewer lost days of work, a 24% lower rate of complete withdrawal from work, a 24% better chance of benefiting from treatment, etc.

However, they warned that the averaging on which this 24% figure is based can be misleading. For example, referring to a published typology that distinguishes between different chronic pain scenarios, they explained:

- The subtype of chronic pain patients who are found to have objective explanations for their pain, and who are competently coping with their discomfort, may experience no change in their pain experience with elimination of financial compensation.
- By contrast, chronic pain patients who do not have objective explanations for their pain, and who are coping poorly, would balance the average by experiencing a 48% improvement with elimination of compensation.

Harris et al, 2005

In 2005, Harris et al published a meta-analysis of the association between compensation and surgery outcomes. They considered a variety of outcome measures, specifically including (but not limited to) pain ratings. They did not separately report the effect of compensation on pain. Indicative of the relative lack of attention this important issue has received, the only meta-analyses that Harris et al referenced as relevant predecessors were Rohling’s on pain (discussed above) and recovery from brain injury.

For outcome variables as a whole, Harris et al reported that the summary odds ratio for an unsatisfactory outcome
in compensated patients was 3.79. This was based on 129 studies involving 7244 compensated and 13,254 noncompensated patients. All but 5 of the studies had individually produced results indicating that compensation leads to worse outcomes. In a larger review of 211 studies, including those that could not be in the meta-analysis, 175 reported that compensation leads to worse outcomes, 30 reported no difference between the groups, and 1 reported a better outcome for compensated patients.

The researchers noted that studies that allowed for comparison of compensation to other potential factors influencing outcomes (including demographic, diagnostic, and treatment variables) found that “compensation status was the most significant predictor of outcome.”

Most of the surgeries were orthopedic, plastic, or spine. The odds ratios were similar for the 5 most common surgeries (shoulder acromioplasty, carpal tunnel release, lumbar discectomy, lumbar spine fusion, and lumbar intradiscal injection of chymopapain). Revision surgeries were more strongly affected by compensation than primary surgeries.

There was no difference between studies that included only workers’ compensation (WC) versus those with both WC and personal-injury claimants/plaintiffs. No studies were found that looked at personal-injury plaintiffs exclusively.

The researchers provided a review of other projects, which revealed:

- There is a dose-response relationship between level of compensation and health outcomes (more compensation is associated with worse outcomes).
- Legal systems that discourage compensation for pain produce better health outcomes. This has been demonstrated within systems that changed the nature of their compensation rules, as well as in comparison of different systems.

Societal Experimentation on a Grand Scale

In 2000, Cassidy et al reported the results of a large-scale societal experiment, made possible by a radical revision in 1995 of the compensation system for motor vehicle accidents in Saskatchewan, Canada. Among other changes, payments for “pain and suffering” were eliminated. The researchers compared whiplash claims from the era when pain could be compensated to the postrevision era.

Findings included all of the following:

- The 6-month cumulative incidence of claims dropped from 417 per 100,000 persons in the last 6 months of the pain-compensated era to 302 in the first 6 months of the noncompensated era and 296 in the second 6 months of the noncompensated era. This 28% decrease in injury claims occurred despite an increase in accidents during the noncompensated time frame.
- The median time from the date of accident to closure of a claim decreased from 433 days prerevision to 194 and 203 days for the first and second 6 months of the noncompensated era, respectively, a 54% decrease in claim length. The researchers conducted analyses demonstrating that time to case closure was an indication of pain severity, and recovery from pain was indeed more rapid in the noncompensated era.

Cassidy et al concluded:

- “Our findings confirm that providing compensation for pain and suffering after a whiplash injury increases the frequency of claims for compensation and delays the closure of claims and recovery.”
- “. . . the type of insurance system has a profound effect on the frequency and duration of whiplash claims and . . . claimants recover faster if compensation for pain and suffering is not available.”

Additional findings from this study included:

- Hiring a lawyer led to worse outcomes, and this factor was as important as any other in determining outcome. The researchers referenced previous studies that produced similar results. Another review of relevant science that also indicated the detrimental effect of hiring a lawyer has been provided in the Occupational Medicine Practice Guidelines of the American College of Occupational and Environmental Medicine (Caruso).
- Working with a chiropractor or physical therapist led to worse outcomes (even after controlling for pain severity), and this factor was also as important as any other in determining outcome.
- Minimizing health care in the acute period after the accident produced better outcomes, consistent with randomized trials referenced by the researchers.

“Outside the Medical-Legal Context”

Lithuania’s judicial and compensation systems provide “minimal possibilities for economic gain” following injury (Miceviciene et al). Consequently, Lithuania has provided a natural laboratory to compare and contrast injury outcomes with the US and other societies that provide ample opportunities for gaining compensation by complaining of chronic pain following compensable trauma.

Scientific study of Lithuanian accident and injury survivors revealed the following scenarios simply do not exist in circumstances that, as phrased by Obelieniene et al, are “outside the medical-legal context”:

- chronic injury-related neck pain (“whiplash”) (Obelieniene et al; Schrader et al)
- chronic injury-related temporomandibular pain (Ferrari et al)
persistent posttraumatic headache (Miccioni et al; Schrader et al)
persistent postconcussion syndrome (Miccioni et al)

Disability Data from the Official Disability Guidelines

The Official Disability Guidelines (ODG; http://www.disabilitydurations.com) allow for a comparison of disability duration datasets of indemnity claimants only, versus all absence data including nonclaimants. In almost every instance involving pain, the harmful effect of compensation is evident in the ODG data. Examples of relevant midrange data include:

- headache (ICD-9 784.0): 15 days for claimants, versus 1 day for the entire data set
- cervicalgia (723.1): 19 days, versus 12 days
- neck sprain (847.0): 25 days, versus 5 days
- lumbar sprains and strains (847.2): 17 days, versus 10 days
- sprains and strains of shoulder and upper arm (840): 19 days, versus 10 days
- ankle sprains (845.0): 22 days, versus 7 days
- carpal tunnel syndrome (9354.0): 42 days, versus 24 days
- tear of medial cartilage or meniscus of knee (836.0): 37 days, versus 21 days
- lumbago (724.2): 17 days, versus 9 days
- myalgia and myositis, unspecified (729.1): 21 days, versus 14 days
- pain in or around eye (379.91): 14 days, versus 3 days
- otogenic pain (388.71): 13 days, versus 2 days

Carpal Tunnel Syndrome

In 2008, Sperka et al published the results of a study of the impact of workers’ compensation on carpal tunnel syndrome outcomes. Claimants had worse outcomes than nonclaimants, with an odds ratio of 5.1, despite receiving more treatment (surgery and/or physical therapy). The researchers conducted a follow-up analysis that controlled for treatment. This actually increased the odds ratio to 9.6. Sperka et al concluded, “...the results are suggestive of poorer outcomes among claimants despite greater use of treatment and comparable severity of disease.”

Complex Regional Pain Syndrome (CRPS)

CRPS, and the failed concept of reflex sympathetic dystrophy (RSD) that preceded one type of CRPS, provide especially strong examples of the association between compensation incentives and chronic pain:

- In a large-scale epidemiological study of CRPS, Allen et al discovered that 71% of cases involved a workers’ compensation claim or personal-injury lawsuit.
- Similarly, Verdugo and Ochoa found that 81% of those diagnosed with CRPS had workers’ compensation claims.
- In a study of reflex sympathetic dystrophy (a diagnosis now replaced by CRPS type 1), Nelson discovered that 67% of those with the diagnosis were receiving workers’ compensation benefits.
- Less than 10% of all injuries are work-related, and yet scientific findings indicate the overwhelmingly majority of cases of CRPS involve claims of work-related injury (Talmage et al, 2013).

These findings are noteworthy because the diagnosis of CRPS was actually created in a manner that causes it to be inherently noninjury-related (Barth RJ. Guides Newsletter, November/December, 2009). Despite this, cases of CRPS are dominated by legal claims, a disparity that highlights the dominant role of compensation in such presentations.

“Posttraumatic” Headache

A review of scientific findings relevant to claims of persistent posttraumatic headache (Barth, May/June 2009) explained:

- Posttraumatic headaches have an excellent prognosis, typically resolving quickly. When they do not, the most well-established predictor of persistence is compensation incentives. Aspects of the head trauma are not predictive of chronicity.
- When compensation is unavailable, there is no dose-response gradient between trauma (either severity or frequency) and headache.
- When posttraumatic headache was studied “outside the medical-legal context” (see the relevant discussion above), the longest duration for any such complaint was 20 days.
- “The scientific findings . . . indicate that the phenomenon of permanent (or even persistent) ‘posttraumatic’ headache is best predicted by compensation/litigation incentives, and does not apply to people who are free from those incentives.”

Rotator Cuff Studies

Studies of outcomes following rotator cuff surgery have repeatedly demonstrated that compensation leads to worse outcomes. This issue has even received coverage in the popular press. An article in Time magazine (Haig) used rotator cuff tears as an example of the generalized phenomenon of compensation leading to more severe pain and worse health outcomes.
Misamore et al reported the following discrepancies, even though the compensation and noncompensation groups were comparable in age, gender, size of the rotator cuff tear, and preoperative strength, pain, and active range of motion of the shoulder:

- 92% of the noncompensation patients had good/excellent outcomes, compared to only 54% of the compensation patients
- 94% of the noncompensation patients returned to full activity versus only 42% of the compensation patients

Henn et al controlled for age, sex, comorbidities, smoking, marital status, education, duration of symptoms, work demands, expectations, and tear size. Despite the multiple controls, their results indicated that compensation was predictive of worse pain and other outcome measures.

**Additional Findings of Relevance to Back Pain**

In a systematic review published by the International Association for the Study of Pain, Sanders reported: “The vast majority of evidence supports the notion that receiving compensation for low back pain or being unemployed is predictive of developing a chronic disability.”

In a long-term study focused on sciatica associated with a herniated lumbar disc, Atlas et al (2000, 2006) reported workers’-compensation claimants were more likely to be receiving disability benefits (odds ratio 3.5) and less likely to report symptom relief and improvement in quality of life at 4-year follow-up when compared to patients not receiving workers’ compensation at baseline. The effect of compensation was significant even after controlling for treatment history and other clinical factors.

In another study focused on radicular symptoms in patients who underwent lumbar discectomy, Hanley reported disabling low back pain postsurgery was predicted by workers’-compensation coverage (p less than 0.00001), more than 15 pack-years of cigarette smoking (p less than 0.01) and age over 40 years (p less than 0.05).

In 2002, Suter published findings from a study that followed 200 chronic-back-pain patients for 2 years. He concluded that involvement in litigation is associated with increased pain and disability and is a risk factor for chronicity thereof. However, the litigation effect is reversible. Litigants’ pain severity, disability, etc “. . . decreased after the settlement of litigation.” On all measures, litigants returned to “much the same level as nonlitigants . . . once litigation was concluded.”

Suter’s findings are particularly noteworthy because various factors specific to the nonlitigating participants (such as older age and longer prestudy duration of pain) would be expected to result in more severe pain and worse outcomes. Hence, the effect of litigation/compensation is so strong that it overwhelms other risk factors for chronic pain.

Suter’s research also revealed that returning to/staying at work had beneficial effects on pain. Litigation and being away from work are also risk factors for higher levels of claimed depression.

Rainville et al conducted a prospective controlled long-term study and found that chronic-back-pain patients who were being compensated reported more pain, depression, and disability and less treatment benefit (compared to those who were not compensated). At 12-month follow-up, pain scores were improved for the noncompensation group but not for those in the compensation group.

**Additional Research Findings Focused on Chronic Pain of Any Form**

In a sample of more than 1000 chronic pain patients, Chibnall and Tait discovered compensation/litigation was associated with more severe claimed disability (relative to chronic pain patients without any claim).

**“Nonorganic” Findings on Physical Examination**

In the aforementioned meta-analysis by Rohling et al, the researchers conducted further analysis after discovering two studies involving 357 participants that looked at the relationship between compensation and “nonorganic” findings on physical examination (such as nondermatomal sensory loss). The effect size for compensation from these two studies was 0.50, meaning there is “some support” for the premise that compensation is a risk factor for “nonorganic” signs.

That meta-analysis did not consider Hayes’s study of the relationship between compensation and Waddell signs. Hayes’s project revealed that Waddell signs were 90% accurate in discriminating between patients who were and were not anticipating compensation. The researchers explained, “Almost all non-AFC [not anticipating financial compensation] subjects scored ‘0’ on nonorganics, whereas 83% of AFC [anticipating financial compensation] subjects scored ‘2’ or higher.” A number of AFC patients demonstrated a set of 5 Waddell signs versus none of the non-AFC patients. Hence, if all 5 Waddell signs are positive during a physical examination, the examiner should conclude the presentation is compensation-driven.

**Controlling for Psychopathology Fails to Eliminate the Detrimental Effect of Compensation**

Since psychopathology is a risk factor for both chronic pain and for filing a medical-legal claim (Barth, 2013), there could be concern that the effect of compensation is simply an artifact of mental health problems. At least two scientific projects have addressed this concern and found an effect for compensation even after somewhat controlling for psychopathology.
Talo et al attempted to control for both psychological and general medical pathology and found workers’ compensation claimants failed to experience treatment benefits that were strongly demonstrated by nonclaimants, specifically in response to a multidisciplinary treatment program.

Rainville et al similarly compared the effects of compensation on outcome of a chronic pain rehabilitation program involving aggressive physical conditioning/exercise and attempted to control for baseline levels of depression, pain, and disability. The compensation group reported worse treatment outcomes, including more depression, greater disability, and a complete lack of benefit in terms of pain.

Personality Disorders

The research discussed above indicates that compensation is the primary risk factor for chronic pain in a claim context. Of course, compensation is not an injury or illness. Among health issues, the most important risk factor for chronic pain in a medical-legal claim appears to be a personality disorder.

Personality disorders are a pervasive form of mental illness (American Psychiatric Association) first manifested in adolescence or early adulthood at the latest. Hence, when present, personality disorders preexist virtually all medical-legal claims by adults. They lead to distress and/or impairment whether the individual was injured or not.

The American Psychiatric Association’s diagnostic manual recognizes 10 personality disorders and is open-minded regarding the potential existence of others. Those recognized are:

- Obsessive-Compulsive Personality Disorder (characterized by perfectionism and preoccupation with orderliness/control)
- Paranoid Personality Disorder (characterized by distrust and suspiciousness)
- Antisocial Personality Disorder (characterized by disregard for and violation of the rights of others)
- Borderline Personality Disorder (characterized by instability in interpersonal relationships, self-image, emotionality, and behavior)
- Histrionic Personality Disorder (characterized by excessive emotionality and attention seeking)
- Avoidant Personality Disorder (characterized by social inhibition, feelings of inadequacy, and hypersensitivity to negative evaluation)
- Dependent Personality Disorder (characterized by subservience, clinging behavior, and an excessive desire to be taken care of)
- Schizoid Personality Disorder (characterized by detachment from social relationships and restricted emotional range)
- Schizotypal Personality Disorder (characterized by acute discomfort in close relationships, cognitive/perceptual distortions, and eccentric behavior)
- Narcissistic Personality Disorder (characterized by grandiosity, need for admiration, and lack of empathy)

When chronic pain populations have been credibly studied to determine the extent personality disorders are risk factors for the pain, personality disorders have dwarfed all other risk factors except compensation. For example:

- Dersh et al evaluated a population of workers’ compensation claimants allegedly disabled by chronic back pain and found 73% had personality disorders (compared to 10%–13% for the general population; Hales).
- Monti et al found a 60% rate of personality disorders among persons diagnosed with CRPS type 1 and a 64% rate in “patients with chronic low back pain from disc pathology.”
- For fibromyalgia, Martinez et al reported a 63.8% rate of personality disorders, and Rose et al reported a 46.7% rate. An elevated rate was also found by Frankenburg & Zanarini, even though their research was limited to a single personality disorder.
- For temporomandibular pain, Gatchel et al (1996) reported a 42% rate of personality disorders. An elevated rate was also found by Frankenburg & Zanarini, even though their research was limited to a single personality disorder.
- For 283 consecutive admissions to a chronic pain specialty clinic, Fishbain found a 58% rate of personality disorders.
- A review of research regarding personality disorders among chronic pain patients of all types (published prior to some of the above information) reported rates of 31%–64% (Gatchel et al, 2000).
- Gross et al discovered a 76% rate of personality disorders among chronic pelvic pain patients.
- Prospective research revealed personality disorders to be one of the best predictors of who will develop claims of vocational disability attributed to chronic back pain (Gatchel et al, 1995).
- Based on a review of relevant scientific findings, First and Tasman reported that approximately 75% of cases that present for medical help with complaints of pain will not lead to any significant or explanatory general medical findings, and at least half of those cases will involve “major personality problems.” Of note, their review was not limited to chronic pain.

Given the prominence of personality disorders as a risk factor for chronic pain, it is noteworthy (and distressing) that workers’ compensation claimants are almost never evaluated for same, even when examined by a mental health specialist (Melhorn).
Narcotics

Misuse of narcotics prescribed for chronic benign pain has become an epidemic in the United States, one that pertains to both personal-injury and workers’ compensation claims (Barth, AMA Guides Newsletter, 2011). Components of the increasingly severe problem with narcotics include excessive prescription, overuse, abuse, diversion, and death. However, most pertinent to the topic of this article is that narcotic analgesics are, ironically, a risk factor for chronic pain.

Relevant findings from the Barth 2011 AMA review include:

• Long-term narcotic usage reliably causes worsening of pain, and often more harm than good, for chronic-benign-pain patients.
  • Did I find evidence of the diagnosis that is completely independent of what the examinee told me (in contrast to allowing the examinee to self-diagnose, eg, “the accident caused me to have migraine headaches,” “the accident injured my back,” etc)?
  • Scientific findings have indicated that narcotics reliably cause abnormal sensitivity to pain, termed hyperalgesia. For example, in a prospective study of the effect of narcotics on pain sensitivity in chronic-low-back-pain patients, all participants developed increased vulnerability to pain after just 1 month of prescription narcotics. Pain thresholds and tolerance dropped by an average of 16% and 24%, respectively (Chu et al, 2006).
  • The same researchers subsequently warned that narcotic-induced hyperalgesia might be responsible for some of the common perplexing complaints of chronic pain patients, including pain that is unexplained, discrepant from previous complaints, diffuse, and reported following a normally nonpainful stimulus (allodynia) (Chu et al, 2008).
  • In a separately published “qualitative systematic review,” two members of the same research team warned that narcotic-induced hyperalgesia will worsen the pain for which the narcotics were originally prescribed (Angst and Clark).
  • In a large-scale study involving almost 2000 subjects reporting pain, those utilizing narcotics were more likely to have severe pain, perceive their health as poor, be unemployed, utilize health care more extensively, and have a worse quality of life. The researchers noted that narcotics do not seem to have even a superficial beneficial effect on any of the key goals of pain treatment—pain reduction, increased quality of life, or improvement of function (Eriksen).
  • Other studies have produced similar results, indicating that prescription of narcotics leads to dramatically higher rates of disability (Webster, Volinn, Franklin).
  • The harmful effects of narcotics do not appear to be permanent. For example, a recent review reported that pain improves subsequent to narcotic discontinuation. The benefit from eliminating narcotic prescription also appears reliable. In 1 sample, 21 of 23 chronic pain patients reported a significant decrease in pain after they were detoxified from narcotics (Bannister, Baron).
  • Since narcotic intake creates an artificially severe pain level, and perhaps resultant impairment, a patient receiving narcotics cannot credibly be considered to have reached maximum medical improvement (MMI) or be ratable, because any impairment may not be permanent. The individual’s true pain level and impairment (if any) cannot be known until after narcotic detoxification.

The review of scientific findings regarding headache discussed above (Barth, May/June, 2009) also highlighted the detrimental effects of narcotics and other analgesics. Relevant findings include:

• The vast majority of a large sample of chronic refractory headache patients overutilized the medications prescribed to treat their headaches. The researchers concluded that most persistent headache sufferers were experiencing medication-induced headaches (Mathew).
  • A study directed at the medication-induced headache hypothesis in patients who were not overutilizing analgesics found that discontinuation of even appropriately used drugs led to headache relief for the vast majority of the sample (Warner).
  • Such scientific findings have led to conclusions in published reviews that medications (narcotics, ergotamine derivatives, NSAIDs, etc) are the necessary and sufficient cause of chronic daily headache complaints, and treatment will not be successful unless the drugs are discontinued (Levenson).

In addition to the aforementioned harmful effects of narcotics, publications from the American Medical Association and American Academy of Orthopaedic Surgeons have highlighted scientific findings that even a short trial of narcotics is capable of producing a CRPS-like presentation (see the Barth and Haralson references in regard to CRPS).

Malingering

There is little published data specifically for chronic pain regarding empirically established base rates of malingering. The best evidence is the review of malingering for all types of claims by Larrabee. This revealed “base rates of malingering that approach or exceed 50%.” The reader is also referred to a recently published review of diagnostic and scientific approaches to malingering (Patterson).

A Focus on 1 Painful Body Part Will Usually Be Misdirected

In medical-legal claims involving chronic pain, the reported discomfort commonly involves a single body part (eg, head, neck, back, upper extremity, or lower extremity). Von Baer et al speculated that this is an artifact of the narrow scope
of certain medical specialties (eg, neurologists may focus on headache; gastroenterologists on abdominal pain; gynecologists on pelvic pain; orthopedists on discomfort in the neck, back, and extremities). Carnes et al reported their suspicion that patients tend to prioritize their complaints and present that which fits the specialty of the physician they happen to be seeing. For example, if consulting a hand specialist, only hand symptoms are reported, even if headaches are more problematic. This prioritization may occur because patients are aware of the physician’s specialty and limited expertise outside it and are cognizant of the limited amount of time clinicians have to spend per visit. However, focusing on anatomically isolated pain is usually short sighted, because chronic pain is seldom limited to one body part.

Relevant scientific findings include:

- Carnes et al discovered that 73% of chronic pain patients had pain in multiple sites. For example, only 13% of chronic-low-back-pain sufferers were free from chronic pain in other body parts.
- Von Korff et al found that 68.6% of individuals with chronic “spine” pain were simultaneously experiencing some other type of chronic pain. The comorbid conditions accounted for one-third of the disability reported by such individuals.
- Saunders et al discovered that chronic migraine patients had an elevated rate of other chronic pain complaints (odds ratio 3.3), as did patients with other types of chronic headache (odds ratio 3.5). Comorbid conditions accounted for 65% of the disability reported by chronic migraine sufferers, and all of that reported by patients with other types of chronic headache.
- Raspe et al discovered that more than 70% of chronic-back-pain claimants acknowledged simultaneous chronic extremity pain.
- In a general population sample of more than 2,000 children and adolescents, Kroner-Herwig et al discovered that a majority (54%) reported recurrent pain in at least 2 anatomical sites. The pain reports were stable when reassessed a year later.
- Walker et al (2010) did follow-up research with adults who had experienced functional abdominal pain during childhood. Of those with continued abdominal pain, 48% were also experiencing chronic nonabdominal pain (compared to 13% in the control group, p less than 0.01). Even the adults who recovered from their childhood abdominal pain reported an elevated rate of chronic nonabdominal pain (24.7%). The researchers’ literature review also reported a high comorbidity of functional gastrointestinal disorders with other chronic-pain presentations, including fibromyalgia, headache, and back pain.
- Peterlin et al discovered that migraine was a risk factor for complex regional pain syndrome, including earlier onset and development of more widespread complaints.

- Kamaleri et al (Pain, 2009) conducted a 14-year prospective population-based study and found that the extent to which any person reported multiple painful sites at any given moment was a relatively stable phenomenon. Only 13.2% of the participants reported being pain-free at the beginning of the project. Only 5.4% who reported pain at the beginning of the study reported being pain-free 14 years later. Of the 1644 participants, 599 (36%) who were available for the full length of the project reported pain in 5 or more anatomical sites at the beginning. Of those, 68.8% were still reporting pain in 5 or more sites 14 years later. Similarly, of those participants who reported pain in fewer than 5 sites at the outset, 75% still reported pain in fewer than 5 sites 14 years later. Participants who reported no pain at initiation also demonstrated a stable pattern over the 14-year course. Eighty percent of the variance in the number of pain sites reported by an individual at the end of the study was accounted for by the number of sites he or she reported 14 years earlier. The researchers concluded that a relatively stable pattern of pain is established early in life, and the tendency to experience pain, including pain in multiple sites, was a reliable individual characteristic (rather than an indication that something external to the individual, such as injury, caused the pain).
- In a separate publication, Kamaleri et al (European Journal of Pain, 2009) reported that the number of pain sites an employed individual reported at the beginning of their project was predictive of whether that person would claim disability 14 years later (participants who reached retirement age by the end of the project were excluded). Their analysis revealed a “strong dose-response relationship between number of pain sites [14 years ago] and [current claims of] disability with a 10-fold increase from 0 to 9–10 pain sites.”
- A 12-year prospective study by Andersson found the development of chronic pain was best predicted by the number of pain complaints a participant had prior to onset of the chronic pain (odds ratio 15.8).
- Tschudi-Madsen et al discovered that pains in various musculoskeletal sites were not only associated with one another, but individuals experiencing such pains were also more likely to endorse nonmusculoskeletal complaints such as palpitations, breathing difficulties, diarrhea, constipation, eczema, tiredness, dizziness, etc. They concluded that the strong associations among this wide variety of complaints indicated they could have a common etiology.
- Hestbaek et al (2006a) discovered that adult-onset chronic back pain was predicted by a history of significant headache, back pain, or asthma in adolescence. In a discussion of their research (Hestbaek et al, 2006c), they concluded that there was such a strong relationship between adolescent and adult back pain that research on prevention of the latter should focus on adolescent years.
- Verne et al discovered that irritable-bowel-syndrome patients demonstrated elevated rates of alldynia/hyperalgesia for the hands and feet.
Chronic Pain (continued)

• Carragee (Spine, 2006) references other studies that similarly indicated it is typical for people with a history of chronic pain to develop chronic pain in another body part (reported rates of such comorbidity being 60%–70%).

In response to such findings, Carnes et al pointed out that it will often be inappropriate to target health care for, or scientific investigation of, chronic pain on single anatomical sites. For example, since only 13% of chronic-back-pain sufferers were free from chronic pain in other body parts, it does not make sense to focus on the back as the pain generator and target of treatment. Patients would more likely benefit from scientifically validated treatment approaches that are not anatomically specific, such as exercise or cognitive-behavior psychotherapy.

Von Baeyer et al portrayed functional pain syndromes not as separate disorders, but as manifestations of an underlying propensity to respond to stressors with the experience and reports of pain. Their review of scientific findings and theory from diverse sources pointed to the possibility that multiple pains cluster together in a patient because of an underlying susceptibility to pain (pain vulnerability or sensitivity).

Other Forms of Mental Illness

As noted, personality disorders are strong risk factors for chronic pain. However, as is discussed more extensively in the Barth references below, other categories of mental illness have also been scientifically established as significant risk factors for the development of chronic pain, including mood disorders, anxiety disorders, substance-related disorders, psychotic disorders, somatoform disorders, and factitious disorders.

A common issue is the “chicken or the egg” question. Did the mental illness cause, in whole or part, the pain, or vice versa? Scientific findings have repeatedly indicated that mental illness is far more likely to manifest prior to the onset of fibromyalgia in 80% of comorbid cases.

In a prospective study, McBeth et al (2001) discovered preexisting somatoform tendencies were the necessary and sufficient risk factor for development of fibromyalgia-like presentations.

Dersh et al (as discussed above) found a 73% rate of preexisting personality disorders among workers’ compensation claimants reporting chronic disabling back pain.

Polatin et al discovered that among chronic-back-pain patients with a positive lifetime history for mental illness, 54% of those with a depressive mental illness, 94% of those with substance abuse, and 95% of those with anxiety disorders acknowledged that the mental illness preceded onset of the pain. The overwhelming probability that an anxiety disorder preexisted rather than followed chronic pain is especially notable given scientific findings indicating anxiety accounts for 54% of the variance in pain severity and associated claims of disability (McCraken et al). While the Polatin results indicate it is probable a depressive disorder will precede the chronic pain in any 1 case, the magnitude of this probability is likely to be substantially larger than the 54% reported because of the strong tendency for depressive illness to present with only physical complaints (as demonstrated by 95% of patients who satisfied diagnostic criteria for major depressive disorder in at least 1 sample) and the tendency for depressed individuals to actually deny any emotional symptoms (Simon, et al).

Mykletun et al found that preexisting levels of depression and anxiety were predictive of reporting whiplash injury and associated disability pensions 2 years later.

Prospective research has highlighted trait anxiety as being predictive of who will develop CRPS-like presentations (Harden et al), rather than vice-versa.

The relationship between mental illness and development of chronic pain is perhaps most easily understood in light of scientific findings indicating that mental illness is inherently painful. For example, King discovered that 87% of psychiatric patients reported current physical pain when asked, and 58% reported their pain was of greater than 2 years duration. Consequently, it is clear that pain is a normal and expected manifestation of mental illness. In fact, the inherently painful nature of mental illness is reflected in its formal definition by the American Psychiatric Association, which repeatedly incorporates pain as a defining issue.

Detailed Discussion of the Psychodynamics of Chronic Pain

This section summarizes material from the most recent attempt by the International Association for the Study of Pain (IASP) to publish a comprehensive book on chronic pain (Flor and Turk). The text has many attributes, but also some significant limitations including:

• It takes the discussion of psychodynamics to a depth that will not be helpful for most general medical practitioners.

• It fails to address the dominant role of eligibility for compensation in workers’ compensation or personal-injury claims involving chronic pain. In fact, it appears to avoid claims altogether. None of the following words are listed in the index: litigation, compensation, financial, claim, workers’ compensation, legal, lawsuit, tort, attorney, or lawyer.

Consistent with the scientific findings discussed above, this IASP book emphasizes:

• “Pain is a multidimensional experience based on psychosocial as well as physiological processes.”
• Scientific findings “demonstrate that psychological variables predict disability, doctor visits, and other pain related behaviors of chronic pain patients to a much larger extent than do physiological variables.”

The book focuses on psychodynamics in the development and maintenance of legitimate chronic-benign-pain presentations. Following is a brief, simplified summary of the extensive discussions in the full text:

• The etiology of chronic pain begins with a predisposition to pain. This premise is consistent with the above discussions of personality disorders and other forms of mental illness as risk factors for chronic pain. The book emphasizes genetic predispositions, which is consistent with risk factors for mental illness given scientific findings indicating psychopathology is primarily genetic (First & Tasman). The book also emphasizes prior learning, such as parental modeling during childhood that pain is to be paid attention to and responded to in maladaptive ways.

• The next reported step in the development of chronic pain involves some precipitating stimuli for acute pain. This may or may not be an experience that causes pain in most people. It could be a stressor that is nonpainful for most people (eg, a disagreement with a family member) but causes pain for an individual with a relevant predisposition. The precipitating stimulus can be completely internal to the individual—eg, a thought, emotion, increased muscle tension, etc.

• The acute pain can be an unconditioned stimulus that plays a role in a learning experience that leads to chronic pain. Alternatively, depending on previous learning, such acute pain could already be a conditioned stimulus—ie, pain that is already a consequence of conditioning/learning can begin a new round of conditioning/learning.

• Pain has many components, including behavioral, cognitive, and physiological. The person who becomes a chronic pain patient develops maladaptive responses in 1 or more components. The maladaptive response can involve involuntary physiological mechanisms (such as Pavlov’s dogs having no voluntary control of salivation yet demonstrating learned/conditioned salivation). The maladaptive response is adopted in an attempt to modulate the impact of aversive environmental or internal stimuli and is reinforced if it succeeds in doing so.

• An especially important role is played by cognitive components of the pain experience, such as preoccupation with and overinterpretation of physical sensations.

• Learning processes contribute significantly to the development and maintenance of the pain.
  • The learning can occur through classical conditioning (eg, the person learns to experience pain in response to circumstances not originally associated with pain). A common example of this is fear of activity and consequent excessive disuse/inactivity, the latter contributing to the pain.
  • The learning can also occur through operant conditioning. In this case, the experience of pain and demonstration of pain behaviors are reinforced by various rewards (eg, attention from caregivers, prescription of narcotics, relief from occupational and avocational responsibilities, financial compensation, familial support, etc).
  • The learning can occur through observation (eg, of pain behavior modeled by other people with resultant rewards).
  • These learning processes contribute to the formation of powerful pain memories “on all levels of the nervous system” that, for chronic pain sufferers, maintain pain in the absence of peripheral nociceptive input.

All of this psychodynamic learning leads to central nervous system “sensitization.” The text classifies such “sensitization” as “a behavioral learning factor” and defines it as “an increase in the intensity of a response when an identical stimulus is presented multiple times over an extended period of time.” “Sensitization” is a learned phenomenon. As such, it can be unlearned. The treatment discussion from the book is focused on such unlearning (for example, through appropriate utilization of cognitive-behavior psychotherapy).

The book offers direction for both general medical and psychological evaluation and treatment of chronic pain but emphasizes psychological treatment in accordance with the primarily psychological nature of chronic benign pain.

**Smoking**

Shiri et al (2010a) discovered through meta-analysis that smoking is a risk factor for chronic back pain and associated disability. Active smokers were at greatest risk, but former smokers were also at higher risk than participants who never smoked.

Hestbaek et al (2006b) discovered through a longitudinal study that the relationship between smoking and onset of low back pain was significant even when the smoking occurred during adolescence and back pain began during adulthood. Their findings satisfied several Bradford Hill criteria for causation analysis, including temporality (smoking preceding the pain).

Viikari-Juntura et al discovered, through systematic review, that smoking was associated with chronic shoulder pain in workers.

In a review paper, Shi et al summarized scientific findings indicating that smoking is a risk factor for chronic pain (including facial, back, arm, and knee pain, as well as fibromyalgia); greater intensity of chronic pain; increased number of painful sites in any one patient; more severe functional disability; greater depression; longer duration of pain; and poorer outcomes in general.
In an analysis of more than 6000 cases from a community health registry, Mitchell et al found that smoking was associated with a wide variety of chronic pain presentations, and they also discovered a dose-response gradient for that relationship. Daily smokers demonstrated the highest odds ratio, occasional active smokers a medium odds ratio, and former smokers a lower odds ratio (but still elevated compared to individuals who had never smoked).

The relationship between smoking and chronic pain has apparently produced an artifact that might lead to the erroneous conclusion that heavy physical labor is a risk factor for chronic pain. Specifically, a relationship between heavy physical labor and chronic pain was discovered in a preliminary analysis, but it disappeared when the effect of smoking was considered (McBeth & Jones, 2007).

Obesity

Shiri et al (2010b) discovered, through meta-analysis, that obesity is a risk factor for chronic back pain. They also found a dose-response relationship between body mass index and chronic back pain. This effect was still significant even when meta-analysis was limited to studies that attempted to control for confounders.

Obesity has also been scientifically established as a risk factor for chronic shoulder pain (Rechardt et al, 2010; Viikari-Juntura et al).

Research findings from Heuch et al indicated that the relationship between obesity and pain is unidirectional (obesity predicts the development of pain, but pain does not predict the development of obesity).

However, the relationship between obesity and pain may be an artifact of the relationship between mental illness and obesity. For example, Bruffaerts et al discovered a relationship between mental illness and obesity and that obesity by itself did not predict lost work days, but mental illness did.

Childhood Abuse and Neglect

A great deal of research has focused on an association between childhood abuse or neglect and adult-onset chronic pain. Much of it was summarized in Davis et al’s 2005 meta-analysis, which demonstrated a reliable association and a dose-response gradient for childhood abuse or neglect as a risk factor for adult-onset chronic pain.

Arrow’s 2004 review also summarized such findings, including the strong nature of the relationship and the dose-response gradient for not only chronic pain, but also other forms of mental illness in adult life. He additionally commented on the manner in which the effects of childhood abuse/neglect on chronic pain and other forms of mental illness overlap. This raises the possibility that the relationship between childhood abuse/neglect and chronic pain is simply an artifact of the role mental illness plays in chronic pain—ie, childhood abuse/neglect leads to psychopathology, and this in turn leads to chronic pain. Supporting this possibility is limited research indicating that childhood neglect/abuse is 9 times more likely to lead to adult mental illness that involves a variety of pain and other physical complaints than mental illness involving episodes of significant depression (Spitzer et al). Similarly, fibromyalgia-like presentations are strongly associated with severe maltreatment in childhood (Imberowicz & Egle; Winfield; Walker et al, 1997); and prospective research has indicated that preexisting somatoform tendencies are the necessary and sufficient risk factors for such presentations (McBeth et al, 2001).

Recommendations for Evaluators/Reviewers to Consider in Any Individual Case

The scientific knowledge base, such as the findings discussed above, indicates that general medical (ie, nonpsychological) evaluation will seldom be able to identify an adequate explanation for chronic benign pain. Consistent with this, Frist and Tasman explained (in their textbook review of the subject) that the process of attempting to find a general medical explanation for chronic pain is “exasperating.”

The recommendations provided below are limited to such cases in which general medical investigation fails to provide a credible explanation for the chronic pain. Since most cases of chronic benign pain (especially the types of cases that become the focus of legal claims) will not involve explanatory general medical findings, these recommendations will be almost universally applicable.

- The evaluator should clearly specify whether general medical findings provide a comprehensive explanation for the chronic pain.
- Given the low-probability relationships between general medical findings and chronic pain, any conclusion that the general medical findings actually provide a comprehensive explanation for the pain should be referenced with scientific findings that can be reviewed in order to find independent confirmation of that conclusion. Additionally, any such conclusion should only be offered if it has arisen through application of the protocol from the AMA’s Guides to the Evaluation of Disease and Injury Causation (Melhorn et al 2012).
- In the typical case where general medical findings do not provide an explanation for the chronic pain, the evaluator/reviewer should consider explaining that this is not a surprising result in that the scientific knowledge base indicates psychological and social factors play a more significant role in the development of chronic pain, while general medical factors are generally not significant. This can help to prevent an iatrogenic search for general medical issues that are unlikely to be found.
The evaluator/reviewer should consider recommending education for the examinee about the potential health benefits of extricating himself/herself from the claim/lawsuit as soon as possible.

The evaluator/reviewer should consider recommending that the patient/examinee be educated about the primarily social and psychological nature of chronic pain and encouraged to seek out credible psychological evaluation. That evaluation should focus on the scientifically established risk factors for chronic pain (as have been discussed above), so a relevant treatment plan can be developed for whatever findings emerge. To ensure a quality evaluation, guidance can be found in the AMA's Guides to the Evaluation of Disease and Injury Causation (Melhorn et al) (specifically the mental illness chapter). Because any mental illness is unlikely to be injury- or work-related, and because involvement in workers’ compensation or personal-injury claims is reliably detrimental for health outcomes (Caruso), the psychological evaluation and treatment should take place outside of a workers’ compensation or any other claims context.

The patient/examinee should also be educated regarding other scientifically established risk factors for chronic pain, such as smoking, obesity, etc.

The patient/examinee should be educated about scientifically validated treatments for chronic pain, which have a high probability of success regardless of the risk factors. Examples include the activity paradigm (responding to pain by increasing, instead of withdrawing from, activity) and cognitive-behavior psychotherapy.

If someone else has claimed there is a general medical explanation for the clinical presentation (eg, injury-relatedness, work-relatedness, etc), the evaluator/reviewer should scrutinize such claims by applying the protocol from the AMA's Guides to the Evaluation of Disease and Injury Causation (Melhorn et al). If application of that protocol is beyond the scope of the evaluator/reviewer's referral issues, then consideration should be given to recommending that any such seemingly untenable causation claims should be given a full review in accordance with that protocol.

Note: If the evaluator/reviewer is specifically aware of sciatica due to a lumbar disc herniation, consider recommending a thorough investigation of the examinee’s history, records from his or her entire life should be reviewed to determine whether this case follows the typical pattern of multiple complaints developing over time.

Readers are additionally referred to Cornerstones of Disability Prevention and Management (Caruso) for a long list of additional relevant recommendations (eg, avoiding “aggressive, extensive, or prolonged medical treatment of benign conditions such as nonspecific low back pain because it increases the risk of iatrogenic and advocagenic impairment and work disability”).

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Chronic Pain (continued)

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Q &A: Catastrophic Foot Injury

**Question:** My question relates to how to rate a patient with a catastrophic foot injury resulting in multiple metatarsal amputations. The patient is a 55-year-old man. Approximately 15 years ago, a long, heavy pipe fell and crushed the distal lateral aspect of his left foot. His multiple surgeries have included a partial ray amputation of his fourth and fifth metatarsals and neuroma excisions. Approximately 8 months ago, he had a partial third ray amputation, and thus a new impairment rating is needed. He has had significant problems with chronic foot pain and is on chronic opioids. He does not use a gait aid. His Pain Disability Questionnaire is 75. An amputation of the second toe or a transmetatarsal amputation has been suggested; however, the patient prefers not to have surgery. Examination at this time reveals a slight limp, with healed amputations of the third, fourth, and fifth metatarsals. His left second toe deviates laterally from the first toe. There are no neurological deficits.

In 1999, two impairment ratings were performed by the treating physician, using the Fourth Edition. The first rendered an impairment rating based on two metatarsal amputations, resulting in 10% lower extremity impairment (LEI). A subsequent rating was based on amputations of the first metatarsal (20% lower extremity) and second metatarsal (5% lower extremity) and nerve deficits (5% lower extremity); resultant impairment was 30% lower extremity or 12% whole-person permanent impairment (WPI). (It is unclear why the rating was based on amputation of metatarsals that were not amputated, why impairment for nerve deficit was given, and why the values from amputation and nerve deficit were added rather than combined.) In 2001, an additional impairment of 3% WPI was given for pain, resulting in 15% WPI.

How should this case be rated?

**Answer:** Your quandary in rating this tough case is understandable. Transmetatarsal amputation (TMA) is a relatively uncommon operation. It was used originally for trench foot. Now it is used in treating patients with significant infection of the forefoot, necrosis, gangrene, and diabetic neuropathy. The goal is to remove nonviable tissue and to maintain limb functionality by preserving the midfoot. With the loss of each metatarsal there is significantly greater loss of function. It is probable in this case that the deviation of the second toe will progress and require further surgery. Ultimately, it is probable that the patient will have additional transmetatarsal amputations, or more proximal amputation, and chronic pain.

The Fourth, Fifth, and Sixth Editions of the Guides define impairment for the loss of each metatarsal, assigning 20% lower extremity (28% foot) for the first metatarsal and 5% lower extremity (7% foot) for each of the other metatarsals. The impairments are additive; however, functionally with the loss of each metatarsal there is increasing loss of function, which is typically more additive. However, the goal of the Guides is to provide a consistent approach, so ratings are consistent among examiners.

First, it should be noted that the prior rating of 15% whole-person permanent impairment was incorrect. You state that the treating physician based the impairment on amputations of the first and second metatarsals; however, it should have been based on the amputation of the fourth and fifth metatarsals, each resulting in impairment of 5% lower extremity or 7% foot. Therefore, on the basis of amputation, there was an impairment of 10% lower extremity. There is no neurological deficit, as the rating for amputation includes the loss of distal nerve function. You suggest that, in 2001, the treating physician had access to the then-recently-published AMA Guides, Fifth Edition, which for the first time had a rating system for pain, and he added 3% WPI. This may or may not be appropriate, as amputees frequently have chronic pain, and Chapter 18 (page 571) indicates that pain-related impairment must be greater than expected for the condition being rated. If appropriate, the 3% WPI for pain would be combined with the 10% LEI, or 4% WPI, to yield a 7% WPI using the AMA Guides, Fifth Edition.

In the Guides Casebook, Case 16-3, Osteomyelitis Secondary to Frostbite, illustrates the process of rating multiple toe...
amputations. With the Fourth, Fifth, and Sixth Editions of the Guides, each toe amputation is added.

In the Sixth Edition, Section 16.6, Amputation Impairment (6th ed, 542), provides default impairment values that are consistent with the impairment values in prior editions. It is not possible to decrease impairments below the value specified; however, proximal problems may increase the impairment and may result in assignment to grade D or grade E. The default values in Table 16-16, Amputation Impairment (6th ed, 542), are identical to those provided in the Fifth Edition in Table 17-32, Impairment Estimates for Amputations (5th ed, 545), and in the Fourth Edition in Table 63, Impairment Estimates for Amputations (4th ed, 83). In the Fourth, Fifth, and Sixth Editions, the value for amputation of the first metatarsal is 20% lower extremity or 28% foot impairment, and for the other metatarsals, 5% lower extremity (or 7% foot). Transmetatarsal amputation results in 40% lower extremity impairment; this is the same as the additive values of the individual amputations—20% for the first metatarsal and 5% for each of the additional 4 metatarsals (ie, 20%), resulting in 40% lower extremity. Transmetatarsal amputation is 57% foot impairment, which is close to the 56% foot value based on the first metatarsal (28% foot) and 4 other metatarsals (total of 28% foot, based on 7% for each metatarsal). Values for the metatarsals are added, rather than combined, in a manner similar to the hand.

Therefore, in keeping with the philosophy used in the Sixth Edition, there would be a default impairment of 5% lower extremity for each of the metatarsals, a class 1 rating. The patient has significant complaints of pain; however, pain is not rated separately with the Sixth Edition. There is no information about more proximal diagnoses that would affect the rating. For functional adjustments, assuming an “antalgic limp with asymmetric shortened stance, corrects with footwear modifications and/or orthotics,” the assignment would be grade modifier 1 per Table 16-6, Functional History Adjustment—Lower Extremities.

The question does not describe his standing and walking tolerances. If his function is worse than the “slight limp” suggests, grade modifier 2, moderate deficit, could be considered. This is defined as “antalgic limp (in the presence of objectively defined significant pathology) with asymmetric shortened stance phase; stable with use of external orthotic device (eg, ankle-foot orthosis), routine use of single gait aid (cane or crutch), or positive Trendelenburg test.” Thus, use of an external gait aid is required, and this man does not use one. Physical examination was used for placement, and clinical studies are not relevant.

If he is rated properly using a functional history grade modifier 1, there is no adjustment, and the impairment value remains at the default, grade C, for an impairment value of 5% lower extremity for each metatarsal. Three metatarsals were amputated; therefore, the impairment would total 15% lower extremity, which equates to 6% whole-person permanent impairment. If he is hypothetically rated using a functional history grade modifier 2, there is an adjustment of +1, and the assignment is grade D, at 6% lower extremity for an impairment value of 6% lower extremity for each metatarsal. The additive impairment is 18% lower extremity, which equates to 7% whole-person permanent impairment.

The long-term survival rate for the remaining great toe and second toe is questionable, and therefore it is possible that the patient will ultimately have an impairment value equal to that of a transmetatarsal class 3 impairment with a default of 40% lower extremity. Ratings are based on current findings, not the potential of future impairment. Therefore, it is possible that this case will be reopened with a finding of greater impairment in the future.

Typically, the functional impact of multiple metatarsal amputations is significant, and in future editions there may be additional amputation values assigned for this situation. Lacking these changes, it would be appropriate to give the examinee the correct value and rate for a mild functional deficit—ie, assign 5% for each toe/metatarsal for a total impairment of 15% lower extremity impairment (6% whole person).

The current impairment is greater than the correctly performed 1999 Fourth Edition rating of 10% LEI; however, it is less than the prior incorrectly performed rating of 15% WPI. The permanent impairment difference is related to the errors in the prior ratings (ie, basing the impairment on amputation of the first and second metatarsal, not the fourth and fifth), the inappropriate additional rating for nonexistent proximal neurologic deficit, and the questionable inclusion of a rating for pain.

It is not unusual to encounter a case where a prior rating was incorrect but judicially accepted. With treatment, the goal is to decrease impairment; however, in some circumstances, such as amputation, this would increase anatomic impairment. Many patients with posttraumatic injuries to a foot have chronic pain, and although amputation results in greater anatomic impairment, their pain and function are much better after appropriate amputation and prosthetic fitting.

Christopher R. Brigham, MD, James B. Talmage, MD, Glenn Pfeffer, MD, Naomi N. Shields, MD
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### Calendar of Events

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For further information about training, contact:

- **AADEP** American Academy of Disability Evaluating Physicians ([www.aadep.org](http://www.aadep.org)) 800-456-6095
- **ABIME** American Board of Independent Medical Examiners ([www.abime.org](http://www.abime.org)) 304-733-0095