

# CHRONIC MECHANICAL LOWER BACK PAIN

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**OBJECTIVE:** To determine the significance of facet joint arthropathy (FJA) in chronic mechanical lower back pain (CMLBP) and the role of discopathy in CMLBP.

**METHODS:** This is a retrospective study of 732 cases of CMLBP who were treated from 1997 through 2007. Each patient had injection of Methylprednisolone and Bupivacaine into the facet joints of L3-L4, L4-L5 and L5-S1. None had tumor, infection or major trauma causing fracture of the spine, disruption of the major ligaments of the lumbosacral spine or cauda equine syndrome. Each patient was evaluated in the Recovery Room before discharge, and one week after the procedure and until the pain recurred. The charts were reviewed to evaluate the efficacy of facet joint injection (FJI) and the relevancy of the CMLBP to FJA. The magnetic resonance imaging (MRI) of each patient was reviewed to

determine the role of discopathy in the outcomes of the patients who had had FJI.

**RESULTS:** The overall success rate was 75.1% with a mean efficacy of 77 days. The charts of the patients with suboptimal results were reviewed, and after excluding the patients with psychological, social and economic factors for failure, the success rate was up to 95% with a mean efficacy of 95 days.

**CONCLUSION:** This study is suggesting that FJA has a major role in triggering CMLBP. Furthermore, this study questions the legitimacy of the Murphy and the Marshall theories in pathogenesis of LBP, a concept that has already been accepted in the literature as a frequent cause of CMLBP (40%).

Key Words: Low Back Pain, Discopathy, Facet Joint Arthropathy

## <sup>1</sup>INTRODUCTION

Given the incidence of CMLBP due to benign disorders, it is important that an accurate diagnosis be made and appropriate therapy applied.[1]

CMLBP is only a description of a symptom complex. One of the most common causes of LBP is the frozen back syndrome, which may occur with or without surgery due to muscular contractures for immobilization of the injured lumbar spine to protect spinal cord and nerves, soft tissue structures, facet joints and capsules and ligaments. Despite the development of numerous highly sophisticated diagnostic technologies, it is often difficult to diagnose the source of LBP because it is often complicated by psychological, social and economic factors. The assumption that the basic problem is degeneration of the lumbar disks is an unproven hypothesis. The source of acute LBP is often secondary to irritation or injury of musculoligamentous soft tissue structures

innervated by the posterior primary ramus of the exiting spinal nerve, with pain being referred to the ipsilateral extremity. Many patients with acute LBP have a history of trauma and complain of LBP with diffuse, nonspecific hip, groin, and leg pain radiation. Although the leg pain often follows the proximal course of the sciatic nerve, unlike with true sciatica, its termination and associated symptoms, such as appropriate sensory loss and paresthesias, are generally vague, histrionic and nondermatomal. Moreover, objective neurological findings are typically absent.[1]

The symptoms of CMLBP are usually worsened by activity and improved partially by rest. Physical activity, particularly bending, extending, twisting and lifting, commonly aggravates the symptoms, whereas restriction of pain-producing activities results in improvement at least temporarily. Typical physical findings are nonspecific, including restricted range of motion of the spine, tight hamstring muscles, paravertebral muscle spasms, muscular trigger points, tenderness and aggravation of symptoms on flexion or extension and straight leg raising tests.

The diagnosis of CMLBP is solely clinical; however, imaging studies may show degenerative spondylosis. Like asymptomatic individuals with lumbar disk herniation and spinal stenosis on imaging studies, there are

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individuals with imaging abnormalities consistent with excessive motion in dynamic flexion/extension who do not have clinical symptoms referable to those abnormalities. At present, the surgical treatment of CMLBP is arthrodesis of symptomatic vertebral motion segments in well selected patients. However, a premature decision for surgical therapy inflicts additional soft tissue injury, aggravating the primary condition and subjects the patient to unnecessary complication. [1, 2]

**MATERIALS and METHODS**

The 732 cases with the diagnosis of CMLBP were treated by the senior author from 1997 to 2007 [Table 1].

Table1.Patient demographic

Characteristic	Number
Total Number of patients	732
Mean age (years)	51
Range (years)	19-89
Sex	
Male	256 (35%)
Female	476 (65%)
Time Period for Data collection	1997-2007
Range of Preexisting Back Pain Length:	3 mo to 43 yrs
Range of pain free length after FJI till the pain recur (years)	0 to 5

All patients had MRI of the lumbosacral spine. Each patient had FJI at L3-L4, L4-L5 and L5-S1 bilaterally. If there was imaging evidence of facet arthropathy at higher levels, those levels were also included. In patients who had previous fusion, the FJI was carried out at the levels above and below the fusion. There was no clinical presentation or screening test that could pinpoint the level of symptomatic FJA. However, if fusion at certain levels was contemplated, individual facet blocks could be done in different sessions. Such a multiple session protocol was not practical or necessary for outpatient pain management. The subjective pain assessment is done and documented in each chart on the day of discharge, one week, four weeks, and until the pain recurred, based on Odom’s criteria. The fellow, unfamiliar with the patients, reviewed the charts independently. Psychological, social and economic factors that potentially could affect

unfavorable outcomes for the failed group were studied. Each chart was checked to make sure that our patient selection criteria and protocol had been followed with no deviation.

**Inclusion Criteria:**

1. Each patient had the diagnosis of mechanical lower back pain (frozen back syndrome) for more than three months and failed to respond to maximum medical treatment. (Three months has been proposed as a point of division between acute and chronic pain by the subcommittee on taxonomy of the international association for the study of pain. [3]
2. The lower back pain was disabling to the degree that the patient could not perform his or her routine job without restrictions and his or her lifestyle had been affected.
3. The CMLBP was the chief complaint with or without leg pain.
4. The patients had MRI with more or less evidence of degenerative lumbar spondylosis.

**Exclusion Criteria:**

1. Patients with tumor, infection and major trauma to the spine causing fracture or disruption of the major ligaments: supraspinous, interspinous or longitudinal, were excluded from the treatment.
2. Patients with cauda equina syndrome who needed urgent surgery were excluded.

**TECHNIQUE:**

The patient is placed on the fluoroscopic compatible operating table in prone position. Using conscious sedation, the skin is prepped and draped in sterile fashion. Using the imaging intensifier the facet joints bilaterally are identified from L3 to S1. The skin is marked with a marking pen. Skin wheels of 1% Lidocaine are raised at the site of each facet joint block. Under fluoroscopic guidance, the 20-gauge 3.5" needle is aimed towards the inferior aspect of each facet joint. After bony contact is made at each facet joint, the stylet is removed. Then 30 cc of preservative free 0.25% Bupivacaine and 160 mg of Methylprednisolone are mixed. Then 5 cc of the mixture is injected very slowly in each facet joint. All of the needles are removed. The patient is then transferred to the Recovery Room.

**RESULTS**

We used Odom’s criteria for subjective rating of pain relief. The overall success rate was 75.1% that lasted 0-5 years (mean of 77 days). The results were divided into four groups:

Excellent (182 patients); Good (367 patients); Fair (128 patients) and Poor (55 patients). The first two groups were considered successful FJI and the last two groups were considered failed FJI [Table 2]

Table 2. Outcome of FJI according to Odom’s Criteria

Odom’s Classification	n	Percentage, %
I (excellent)	182	25
II (good)	367	50.1
III (fair)	128	17.5
IV (poor)	55	7.4
I+II (success rate)	549	75.1

Odom’s Criteria:  
 Excellent: all preoperative symptoms relieved, abnormal finding improved  
 Good: minimal persistence of preoperative symptoms, abnormal finding unchanged or improved  
 Fair: definite relief of some preoperative symptoms, others unchanged or slightly improved  
 Poor: symptoms and signs unchanged or exacerbated

To analyze the reason for suboptimal subjective outcomes in the third and fourth group, the coauthor reviewed their charts further and analyzed the psychological, social and economic factors. In the failed group, we identified and excluded patients with workmen’s compensation claims, motor vehicle accident claims, patients who had a pending application for Social Security Disability, patients with the diagnosis of reflex sympathetic dystrophy (RSD), patients dependent upon narcotic pain killers and patients with psychiatric problems (under the care of a psychiatrist or psychologist). Out of total number of patients who had injection with Fair and Poor result (183 patients) 155 patients had the psychological, social and economic factors, the total number of the failed group was down to 28 and the overall success rate was up to 95% and the efficacy of the treatment improved to 15 days to 5 years (mean of 95 days). [Tables 3, 4, 5]

Table 3. Comparison of success and failure rate following FJI after exclusion of the psychological, social and economic factors according to Odom’s Criteria

Success Rate		Failure Rate	
Excellent	Good	Fair	Poor
182 (n)	366 (n)	22 (n)	6 (n)
31.5%	63.5%	4%	1%
95%		5%	

Table 4. Comparison of success and failure rate following FJI before and after exclusion of the psychological, social and economic factors

Outcome	Before	After
Success Rate	75.1%	95%
Failure Rate	24.9%	5%
Treatment Efficacy	77 days	95 days

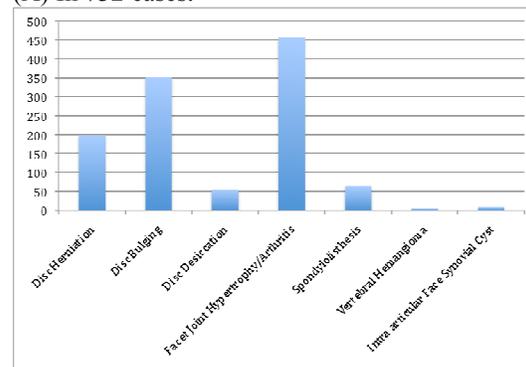
Table 5. Comparison of success and failure rate between male and female following FJI Before and after exclusion of the psychological, social and economic factors

Sex	Before		After	
	Success Rate	Failure Rate	Success Rate	Failure Rate
Male	68.7%	31.2%	96.8%	3.1%
Female	75.4%	24.5%	92%	7.1%

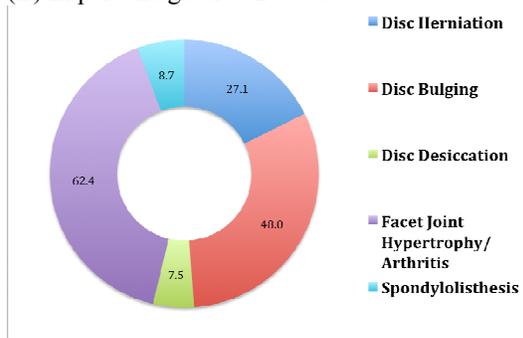
MRI findings read by our neuro-radiologists: 686 patients had discopathy, a common condition in asymptomatic population. 457 cases had imaging of facet arthropathy and 275 did not, indicating that the imaging findings are not necessarily diagnostic of CMLBP. [Graph 1 A&B]

Graph 1 (A&B): MRI Findings of Lumbar Spine. Some patients had multiple findings.

(A) In 732 cases.



(B) In percentage of 732 cases.



The 28 of patients in the second group who did not respond to FJI were reviewed. All had severe Lumbar stenosis compressing the cauda equina. Of those, 19 had decompressive laminectomy with or without fusion depending on stability of lumbar spine with good or excellent outcomes. 4 patients declined surgery and 5 patients were high risk for complex surgery and operation was not offered to them.

**DISCUSSION**

The rationale for discogenic LBP is based on innervation of the outer third of the annulus fibrosis, a notion that was first exposed by Inman and Saunders in 1947 and was confirmed by Malinsky in 1959. [4] In 1981 microdissection studies showed the source of nerve fibers in the posterior annulus fibrosis and posterior longitudinal to be sinuvertebral nerves. [5] Contemporary immunofluorescence techniques later confirmed the neuropeptides within them that are typical of nociceptive axon.[6]

The tears and fissures in the annulus have been seen in damaged lumbar discs and have been attributed to LBP.[7] Evidence that disc can hurt comes from clinical observations during operations performed under local anesthesia, probing the back of a disc reproduces pain.[8-9] Two theories have been postulated in the literature as pathogenesis of discogenic LBP:

1. Chemical Theory: In 1973 Marshall et al. postulated that the nucleus pulposus releases a glycoprotein that “hypothetically is a pain-generator” into the nociceptors of damaged annulus and nerve roots causes LBP and radiculitis.[10] In 1992 Saal et al. postulated that the nucleus pulposus releases phospholipase A2 with hypothetical inflammatory potential into the

2. Mechanical Theory: In 1967 Francis Murphey hypothesized that LBP is due to stretching of the innervated annulus and posterior longitudinal ligament secondary to increase in the intradiscal pressure, the condition that is seen in bulging and herniated discs. He presented his view in this regard in the congress of Neurosurgical Surgeons in San Francisco.[12]

The following observations are evidence to question the legitimacy of the Murphey and the Marshall theories but not to deny the existence of discogenic LBP.

1. Bilateral discectomy with removal of the posterior annulus and posterior longitudinal ligament (PLL) in patients with LBP often fails to relieve the LBP, evidence that the stretched annulus and PLL are not the source of the LBP (evidence against the mechanical theory and chemical theory).

2. The majority of patients who undergo microdiscectomy for free fragment disc herniation awaken from anesthesia with complete relief of radicular pain with minimal LBP despite a large hole in the annulus that allows the release of hypothetical toxic pain generator substance from the residual disc (evidence against the Marshall ‘s theory).[1]

3. Injection of Methylprednisolone and Bupivacaine in the lumbar epidural space often relieves the radicular pain and confirms that the source of the radicular pain is the nerve root; however, Simmons and associates in a prospective randomized double-blind study demonstrated no benefit of intradiscal steroid injection in relief of LBP.[13] this is evidence against Marshall’s theory.[10]

4. Intradiscal electrothermal therapy (IDET): The rationale for IDET is denervation of the posterior annulus by heat to relieve discogenic LBP. The advocates of the technique currently admit that the procedure is not effective in the short-term without giving any explanation for short term failure. The long-term efficacy of the procedure has not been proven either (evidence against both the mechanical and chemical theories). [14-18]

5. Several Intradiscal procedures have been designed by different investigators to reduce intradiscal volume and pressure, to treat CMLBP based upon the mechanical theory of the

damaged nucleus, irritating nociceptors of the annulus and generates LL

CMLBP.[19] None of them have been effective in relieving CMLBP (evidence against the Murphey theory).[20] A) Chymopapain – in a double-blind study Schwetschenau found no benefit from the Chymopapain in the treatment of herniated lumbar disc.[20] however, in an open-label double-blind study conducted in 3000 cases, success rate was 88% for radicular pain only. Similar results have not been achieved for CMLBP only (evidence against the Murphey theory).[21] B) Automated percutaneous lumbar discectomy (APLD) – There has been no prospective randomized, controlled study to suggest the APLD's efficacy in CMLBP; however, such a study in patients with radicular symptoms has been reported successful (evidence against the Murphey theory).[22] C) Intradiscal laser discectomy D) Nucleoplasty.[23-25]

6. Intradiscal injection of Methylprednisolone and Bupivacaine has failed to relieve the LBP.[26] in a prospective randomized study, Khot concluded that the treatment is not effective in chronic LBP.[27]

7. Provocative discography for the diagnosis of discogenic LBP has remained controversial for almost 50 years because the results are subjective, unreliable and unpredictable.[28-29] this is further evidence that the innervated outer third of the annulus is not the source of the lower back pain. In fact, it would make sense for Test to be highly sensitive and confirmatory if the innervated annulus was the source of the CMLBP (evidence against the mechanical theory of Murphey).

8. In our series of 732 patients, 539 responded to FJI, yet 493 had pathologic disc, an incidental finding of a condition that is very common in the asymptomatic general population (argues against discogenic LBP).

9. The rationale for arthroplasty is based on the theory of removal of the pain generating disc in CMLBP and preservation of the facet joint motion.[30] the efficacy of the procedure has remained the subject of debate among spine surgeons. As Resnick and associates opined: “the population of patients deemed ideal for disc arthroplasty is a population who are often well treated without surgery”.[31]

In 1911 Goldwaith described FJA as a cause of LBP. Twenty-two years later, in 1933, Ghormley reported in the literature FJA as an

etiology of LBP. The facet joints are true synovial joints with joint space, hyaline cartilage surfaces, a synovial membrane, menisci and capsule. Two medial branches of the dorsal rami innervate the facet joints. The lower pole of each facet joint is supplied by the exiting nerve root, and the upper pole by a branch of the exiting nerve root one level higher. In facet arthropathy, a high level of prostaglandins, that are pain mediators, have been measured and implicated as the cause of back pain. The incidence of CMLBP secondary to FJA has been a subject of controversy.[32-33] The current literature indicates the incidence of CMLBP due to FJA is 15% - 45%.[34]

The following observations are evidence that facet joint arthropathic lower back pain does exist:

1. Immobilization of the facet joints by bed rest, traction and bracing is effective in most patients with CMLBP.

2. Anti-inflammatory drugs are more effective than regular analgesics in the treatment of CMLBP because facet joints are true synovial joints in contrast to intervertebral body joints.[35]

3. Lumbar facet joint denervation has been effective temporarily in patients with CMLBP who had responded to FJI.

4. The arthrodesis, with or without instrumentation, by eliminating the inflamed facet joint motion, without removal of the posterior annulus and posterior longitudinal ligament has been found effective in the treatment of CMLBP in patients with severe degenerative disc disease.

5. In our experience 75.1% of the patients responded favorably to FJI in the short term. The success rate was 95% when we excluded patients who were considered red-flagged because of the psychological, social and economic factors.

While discogenic pain due to nerve root compression causing radiculopathy and cauda equina compression due to a large disc herniation causing severe lumbar stenosis and as a result LBP and neurogenic claudication are not debatable, we are questioning the legitimacy of LBP based on the Marshall and the Murphey theories.

Our success rate in FJI is higher than what has been reported previously in the literature (15%-45%).[34] Furthermore discussion regarding denervation of facet joints and the surgical treatment of MLBP including laminectomy and arthrodesis is beyond the scope of this article.[1,2]

## CONCLUSION

This is a retrospective non-controlled study and therefore does not prove that discogenic LBP does not exist; however, it questions the legitimacy of the Murphey and the Marshall theories in pathogenesis of LBP. Our study suggests that it is reasonable in the treatment of patients with CMLBP the treating physicians first address the abnormal facet joints rather than pathologic discs seen on MRI. Our experience suggests that the FJI is often a palliative rather than a curative treatment.

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