Failed back surgery pain syndrome: therapeutic approach descriptive study in 56 patients

Manoel Jacobsen Teixeira[1], Lin Tchia Yeng[2], Oliver Garcia Garcia[3], Erich Talamoni Fonoff[4], Wellingson Silva Paiva[5], Joaci O Araujo[6]

[1] Lecturer; Chair Professor in Neurosurgery, School of Medicine, Universidade de São Paulo (USP), São Paulo, SP, Brazil
[2] Ph.D. in Science; Pain Center Coordinator, Division of Physical Medicine, Clinical Hospital of the School of Medicine, USP, São Paulo, SP, Brazil
[3] M.D.; Residence in Neurosurgery; Medical Trainee, Pain Center, Clinical Hospital of the School of Medicine, USP, São Paulo, SP, Brazil
[4] Ph.D. in Neurology; Pain Center Coordinator, Department of Neurology, Clinical Hospital of the School of Medicine, USP, São Paulo, SP, Brazil
[5] Ph.D. Student in Neurology; Attending Physician, Pain Center and Functional Neurosurgery, Clinical Hospital of the School of Medicine, USP, São Paulo, SP, Brazil
[6] M.D.; Residence in Orthopedics; Musculoskeletal Outpatient Clinic Coordinator, Pain Center, Clinical Hospital of the School of Medicine, USP, São Paulo, SP, Brazil

Summary

Objective: The authors show the clinical evaluation and follow-up results in 56 patients diagnosed with a failed back surgery pain syndrome. **Methods:** Descriptive and prospective study conducted over a one-year period. In this study, 56 patients with a failed back surgery pain syndrome were assessed in our facility. The age ranged from 28 to 76 years (mean, 48.8 ± 13.9 years). The pain was assessed through a visual analog scale (VAS). **Results:** Postoperative pain was more severe (mean VAS score 8.3) than preoperative pain (7.2). Myofascial pain syndromes (MPS) were diagnosed in 85.7% of patients; neuropathic abnormalities associated or not with MPS were found in 73.3%. Drug therapy associated with physical medicine treatment provided ≥ 50% pain improvement in 57.2% of cases; trigger point injection in 60.1%, and epidural infusion of morphine with lidocaína in 69.3% of refractory cases. **Conclusion:** In patients with a post-laminectomy syndrome, postoperative pain was more severe than preoperative pain from a herniated disk. A miofascial component was found in most patients.

Keywords: Low back pain; intervertebral disk displacement; post-laminectomy syndrome.

Study conducted at the Pain Center, Department of Neurology, Clinical Hospital of USP School of Medicine, São Paulo, SP, Brazil

Submitted on: 3/21/2008
Approved on: 3/21/2011

Correspondence to:
Wellingson Silva Paiva
Divisão de Neurocirurgia Funcional e Centro de Dor
Hospital das Clínicas da Faculdade de Medicina da USP
R. Dr. Eneas Aguiar, 255
5º andar – Cerqueira César
São Paulo – SP
CEP: 05403-010
wellingsonpaiva@yahoo.com.br

Conflicts of interest: None.

Introduction

According to the International Association for the Study of Pain (IASP), the post-laminectomy syndrome is defined as a "low back pain of unknown origin, persisting at the same location as the original pain despite operative interventions or with a post-surgery onset. The low back pain may be associated with a referred or radiating pain"[1]. This definition applies to all surgeries designed to treat pain arising from the low back spine, including those aiming to treat a herniated disk. Surgical management applied to herniated disks is a hemilaminectomy with a flavectomy, nerve root dislocation and a hernia excision. The many clinical manifestations of the post-laminectomy pain often overlap and have a low back pain as a common expression. The term "unknown origin" in the definition should not be strictly used, as although the post-laminectomy syndrome is complex and the pain can arise from various nosological entities affecting varied anatomical elements in the spine or away from the spine or even being a result from systemic conditions, its origin can be found in many cases[2].

Low back pain causes are varied and differential diagnosis is wide. The structure causing the pain is identified in less than 20% of cases[3]. The herniated disks are the most common indication for laminectomy to treat low back pain. Over 300 thousand laminectomies are estimated to be carried out in the United States, with a failure rate higher than 40%[4]. Misinterpretation of pain origin as resulting from a herniated disk seen in imaging studies[5], misidentification of spine instability and other mechanical causes, including incomplete removal of the herniated disk[6], and operative complications are blamed for the poor surgical results[2]. Pain can also result from articular facet instability or from reduced intervertebral space due to structure abnormalities or intervertebral disk removal, with consequent change in the articular facet angle[7]. Among non-mechanical causes for the failed back surgery pain syndrome, disk infection, peridural "fibrosis", arachnoiditis and psychosocial factors should be mentioned[8]. In this study, we aimed to assess the clinical features and the non-surgical management outcome in patients with a failed back surgery pain syndrome seen in a pain center.

Methods

Fifty-six patients diagnosed with a failed low back surgery pain syndrome were prospectively followed over one year at the Neurological Clinic Pain Center of the Clinical Hospital of *Universidade de São Paulo* in a descriptive study. The design was approved by the Institutional Ethics Board (213/05). Every patient signed an informed consent form. Patients with a persisting low back pain or an early relapse within less than three months of a surgical herniated disk procedure were selected. The patients were admitted into a specific postoperative low back pain outpatient clinic according to a spontaneous demand in the unit. Patients with evidence of metabolic, inflammatory, and oncological diseases or a radiological segmental instability picture were excluded. No patients experienced a cognitive impairment. Out of 73 patients admitted into our unit, 14 were excluded by exclusion criteria and the follow-up was lost in three cases. We obtained data from history, general physical exam, neurological and physiatric evaluation in addition to pre- and postoperative imaging studies. Laboratory tests to rule out rheumatic or metabolic diseases (ANA, RF, ESR, CRP, blood cell count) were performed. The pain magnitude, characteristic, nature and location, as well as the radiating course in pre- (retrospectively) and postoperative periods. The pain intensity was assessed according to a visual analog scale (VAS) before and after treatment. The physiatric exam aimed specially to assess low back spine and paravertebral, low back, gluteal and lower limb muscle groups, consisting of muscle power assessment, miofascial trigger point presence, spasms, sensitivity, cutaneous changes, and trophism. Data collection was performed by using a standard protocol. The statistical analysis used Sigmastat 4 software.

For all patients, a drug therapy with a rehabilitation program using kinesiotherapy and muscle stretching was adopted and if a rehabilitation refractory myofascial component (MPS) was associated, these patients underwent needling with a 1% lidocaine injection. MPS was identified in 48 (85.7%) patients. Drug therapy consisted of amitriptyline 25 to 150 mg/day (mean, 64 mg/day), chlorpromazine 20 to 100 mg/day (mean, 48 mg/day), naproxen 1000 to 1500 mg/day (mean = 1150 mg/day), acetaminophen 2 to 4 g/day (mean = 2.4 g/day) and codeine phosphate 120 to 240 mg/day (mean = 184 mg/day) according to the requirements and tolerability in each case. All patients underwent physiatric follow-up.

In case there was not ≥ 50% original pain improvement (VAS) all over the follow-up, the patients were considered frankly refractory, undergoing a 2 mL infusion containing morphine 1 mg/mL and 2% lidocaine by catheter placement into the low back peridural compartment bid over two weeks.

Results

Overall, 37 (60.5%) patients were male and their ages ranged from 28 to 76 years (mean, 48.8 years ± 13.9 years). The patients' mean age at the original pain onset ranged from 22 to 66 years (mean = 37.2 years). The patients had undergone from one up to four low back laminectomies (mean = 1.5) to treat low back pain or lumbosciatic pain. The mean symptomatology length was 96 months.

The length of the low back pain or lumbosciatic pain complaints ranged from 8 to 168 months (mean = 36 months). Regarding the pain intensity, the patients with a radiculopathy persisting postoperatively were found with a higher pain score by the pain analog scale. Those

with a postoperative root pain diagnosed had a mean score of 8.7, compared with 6.6 for those with no radiculopathy (p = 0.001).

In 17 (30.3%) patients, the preoperative pain history was consistent with a root origin, in 9 (16.1%) with referred pain in musculoskeletal conditions in both lower limbs, in 22 (39,3%) with referred pain in one lower limb, in 2 (3.6%), the pain location was only the lower back region and in 6 (10.7%) the pain had polyneuropathy characteristics. Three (5.4%) patients who did not experience pain with root characteristics preoperatively had undergone a diskectomy and spinal fixation.

X-ray studies disclose a one- or two-lumbar-segment hemilaminectomy unilaterally (L4 or L5) in 53 (94.6%) patients and bilateral lumbar laminectomy in 3 (5.4%). The lumbar spine dynamic study did not show instability in any patient. The computed tomography (CT) scan or magnetic resonance (MR) imaging did reveal a periradicular scar at the operative site in 32 (57.1%) cases.

The pain intensity before the operations ranged from moderate to severe, according to the VAS, scoring from 5 to 10 (mean = 7.2); the pain intensity at the first attendance to the Pain Center, Clinical Hospital, *Universidade de São Paulo* was severe, scoring from 7 to 10 (mean = 8.3).

Thirty-six patients (64.9%) had undergone physical therapy, 53 (94.6%) had been on nonsteroidal anti-inflammatory drugs (NSAIDs), and 17 (30.4%) had been on corticosteroids alone or in combination with B complex vitamins, four (7.1%) had been on opiates, and five (8.9%) had been on tricyclic antidepressive agents before and after the surgery. All of the patients had stayed at rest and those exerting an occupational activity had been put away from work.

In 38 (67.9%) patients, uniradicular (53.7%) or multiradicular (14.3%) syndromes were shown. Ten (17.9%) patients had muscle pain and myofascial painful points in several body regions, sleep disturbances and depression, suggesting the fibromyalgia syndrome.

Trigger points characteristic of MPSs were identified in 48 (85.7%) patients. Out of 17 (30.3%) patients with preoperative nerve root pain, 15 (88.2%) had lumbar or gluteal MPSs (Table 1).

Satellite or secondary trigger points were found in 29 (51.8%) patients (Table 2).

The treatment outcome was rated as excellent (> 75% improvement over the original pain according to the VAS), good (50% to 75% improvement), fair (25% to < 50% improvement) and poor (< 25%).

Drug therapy combined with physical rehabilitation measures provided an excellent outcome in 5 (16.1%) patients, a good outcome in 23 (41.1%), a fair outcome in 16 (28.6%) and a poor outcome in 12 (21.4%).

In 48 patients, MPS was found and trigger point injections were performed by using 1% lidocaine 0.5 mL.

Table 1 – Patient disposition according to muscles affected more severely by the myofascial pain syndrome

Muscle	n	%
Lumbar quadrate	33	69
Gluteus medius	9	19
Gluteus minimum	2	4
Piriform	3	6
Total	48	88

n, absolute numbers; %, percentage.

Table 2 – Affected muscle distribution by MPS satellite or secondary trigger points

Muscle	N	%
Gluteus medius	17	35
Gluteus minimum	6	11
Lumbar quadrate	3	5,5
Piriform	3	5,5
Vastus lateralis	2	4
Vastus medialis	1	2
Gastrocnemius	1	2
Levator ani	1	2

MPS, myofascial pain syndrome

By comparing the pain improvement scores, patients with MPS had worse outcomes over post-laminectomy syndrome without MPS. The immediate results were excellent in 5 (10.4%), good in 17 (35.4%), fair in 18 (37.5%) and poor in 8 (17.8%). The results were satisfactory for 68.75% of patients with MPS *versus* 75% in patients without MPS, but the difference was not statistically significant (p = 0.2).

At the end of treatment, significant improvement (excellent and good outcomes) occurred in 34 (60.1%) patients, while 13 (23.2%) had a fair outcome. However, the outcome was considered poor in 8 (17.5%) patients. Regarding the pain intensity at the final follow-up, we found a reduced general mean in the visual analog scale from 7.2 to 4.7 (p = 0.01).

A peridural catheter for spinal infusion of a morphine and lidocaine solution was placed in thirteen patients; all of them had a MPS, being considered refractory after a rehabilitative treatment attempt. The outcome was excellent in 4 (30.8%) patients, good in 5 (38.5%), and poor in 4 (30.8%), meaning the outcome was excellent or good in 9 (69.2%) patients treated with a peridural infusion.

Discussion

Low back pain is present at some point in life and constitutes a serious public health problem in 40% to 85% of individuals[2]. Treatment cost, compensation, and lost productivity are high. The patients' mean age at pain onset was 37.2 years, with the individuals within this age group usually exercising their occupational activity to a very great extent.

In most cases, the course is favorable, even when no care measures are taken. However, low back pain becomes chronic in 15% to 20% of individuals[9]. In 13.8% of patients studied by Frymoyer[10], the pain lasted more than two weeks and in 22% it was severe. In 21.2% of Deyo and Tsiu's patients[9], the pain was mild; in 43.4%, it was moderate and in 35%, severe; in 40% of cases, the low back pain irradiated to lower limbs and in only 1% a true sciatic pain could be found. In our series, preoperative pain was severe, with a 7.2 score according to the VAS; 45.4% had a low back pain and referred pain to lower limbs history before the surgery. In only 30.3%, the history suggested a true sciatic pain, and these findings indicate selection criteria for the surgery were likely inappropriate in most cases.

According to Hanley *et al.*[11], the operative treatment outcome of herniated disks is poor in 14% of cases. The numbers of spine surgeries to relieve pain have steadily grown in the United States, with 170 thousand operations in 1974, 300,413 in 1994 reaching 392,948 in 2000[12], with 80 thousand cases of failed back surgery pain syndrome per year[13]. According to Deyo and Tsiu[9], the main reason for an increasing number of laminectomies is the growing number of surgeons operating the spine in each country. In different countries and different regions, the frequency of operation indications is variable, and this is not explained only by the different prevalence of low back pain or lumbosciatic pain; in 3% or 4% of individuals, herniated disk surgeries are indicated in the USA, but only in 1% of individuals in Sweden and Denmark.

Poor outcome of operative treatment might result from an incorrect diagnosis. Among the identified causes for low back pain, the following could be highlighted: rheumatic conditions, primary or secondary spine tumors, vascular conditions, hematological abnormalities, endocrine conditions, pelvic or abdominal viscus diseases (endometriosis, ovarian cyst torsion, pelvic inflammatory disease, prostatitis, cystitis, pancreatopathy, nefropathy, kidney disease, peptic ulcer, urinary tract, biliary or duodenal conditions), mechanical abnormalities (herniated intervertebral disk, articular facet injury, segmental instability or sacroiliac joint instability), systemic conditions (fibromyalgia, myositis, autoimmune or immune-allergic diseases), psychiatric diseases and other conditions (hip joint disease, trochanteric bursa injury, polyradiculoneuritis, meningeal irritation signs)[14]. Because of the great number of possibilities, the high surgical therapeutic failure rate is justifiable in care provided to these patients, but it also indicates there must be a more judicious semiologic evaluation.

Surgeries that do not meet the indication criteria to treat a herniated disk can result in maintenance or worsening of pain and preoperative deficits. A herniated disk misinterpreted as a cause for low back pain is the most common reason for indicating spine surgeries that progress to a post-laminectomy chronic pain syndrome with an early onset postoperatively. This is partly due to overvaluing the anatomical findings not related to the low back pain that are shown in imaging studies, but those usually do not warrant the pain and the surgical intervention[14]. In 35% of asymptomatic individuals studied by Hitselberg and Wihen[15], the x-ray imaging revealed abnormalities suggesting a herniated disk. In 35% of asymptomatic individuals studied by Wiesel *et al.*[16], a spine CT scan found abnormalities; in 20.2% of cases, there was a herniated disk evidence. Boden *et al.*[14] observed 60% of asymptomatic individuals had a herniated disk on magnetic resonance imaging. Therefore imaging studies can confirm a herniated disk clinical diagnosis, but they are not the main determinants for indicating a surgery, since asymptomatic herniated disks are so commonly seen[2].

Even in symptomatic conditions, there is a progressive absorption of the herniated disk fragment, a phenomenon accompanied by symptom improvement in most cases[17]. Hakelius[18] observed 38% of patients with a herniated disk not undergoing a surgery, but having been on medical treatment, were clinically improved within a month, 52% within two months, and 73% within three months. Saal and Sall[19] conducted a retrospective study involving 58 patients with a radiculopathy resulting from a herniated disk; 52 underwent a conservative treatment, resulting in improvement in over 90% of cases; only in three cases surgical ablation of extruded fragments was required. This means the indication criteria for diskectomy, represented by a cauda equina syndrome, marked acute or progressive motor deficit or lumbosciatic pain occurrence and evident radiculopathy, characterized by sensory, motor and deep tendon reflexes deficits over one or more nerve root territory, nerve root irritation evidence, translated as a positive straight leg raising maneuver and consistent imaging study findings[20] in patients achieving no improvement after symptomatic drug therapy with physical medicine measures during a period of over 6 to 12 weeks[20,21], are not always met. Only 64.9% of patients included in the current casuistry had undergone physical medicine treatment and only 8.9% had undergone a tricyclic antidepressant therapy before the operations, suggesting medical methods had not been adopted in most cases.

Mechanical causes are responsible for 90% of post-laminectomy pain cases[6]. Among them, residual or relapsed herniations, spinal instability, post-vertebral fixation pseudarthrosis, articular facet abnormalities, spinal canal stenosis, meningocele and pseudomeningocele are highlighted[22]. In no patient of ours a spinal instability or residual herniation was found. In addition to the diskectomy, a spinal fixation and fusion was proposed[23]. However, there is little evidence the spinal fusion is useful in patients with no actual spinal instability[7]. This occurred in 5.4% of study subjects. Postoperative imaging of a residual disk herniation does not imply this is necessarily the cause for a persisting pain, as postop-

erative imaging studies often show similar abnormalities in individuals whether they are symptomatic or not[24]. Peridural scar occurring after a laminectomy is a frequent postoperative finding. Newly formed tissue can involve, distort and/or compress the nerve root. However, epidural fibrosis is often shown on CT scan or MRI postoperatively in cases there is no pain[8]. In 57.1% of patients in this study, a periradicular scar was found.

The patients included in our study had undergone up to 4 surgical lumbar spine surgical procedures with no improvement; the mean was 1.5 operations per patient. Many patients undergoing further operations to treat persisting or residual pain get frustrated. The improvement rate in reoperations is low, around 30% after the second surgery, 15% after the third surgery and 5% after a fourth procedure with up to 20% of worsening[13].

Out of 56 patients analyzed in the present study, 85,7% had MPS not found previously on physical exam. There is evidence that MPS is involved in low back pain genesis or maintenance[23]. However, MPS diagnosis is frequently disregarded[25]. Many lumbar muscles affected by MPS and the operative injury would result in pain worsening. Although physiatrically speaking lumbar and gluteal muscle MPS is considered the most important cause for low back pain, bone, tendinous, nerve, disk and bursa conditions are still valued as symptom causes[26]. The muscle fiber injury is not necessarily a cause for pain, since in patients with primary degenerative conditions, as in Duchenne muscular dystrophy, there is a disruption in a large amount of myofibrils and the sarcoplasmic reticulum, but there is no pain, suggesting MPS symptoms result from nonstructural muscle fiber changes or dysfunctions[26]. The main electrophysiological abnormality seems to be a neuromuscular dysfunction in the motor endplate.

The energy crisis theory postulates there is an increased calcium concentration in the sarcoplasm due to a sarcoplasmic reticulum, sarcolemma and or muscle cell membrane disruption. The sarcoplasmic reticulum function is storing and releasing ionized calcium, which activates contractile elements and causes sarcomere shortening. Sustained sarcomere contraction results in increased metabolism, causes localized ischemia and generates a localized energy crisis. The combination of electrophysiological and histopathological theories generated the neuromuscular endplate multiple dysfunction concept. The potentials recorded as spontaneous activity or spikes in trigger points would result in abnormal acetylcholine release by the nervous ending. Acetylcholine release would accentuate depolarization and calcium release from the sarcoplasmic reticulum, causing sarcomere contraction and small-caliber vessel compression. Increased depolarization due to acetylcholine release and sarcomere contraction would cause increased energy demand, which, if associated with hypoxia resulting from reduced muscle blood flow, would then cause the energy crisis. This energy crisis generates metabolites which sensitize nociceptores and referred pain from trigger points[26]. The abnormalities in nerve fibers responsible for supplying the muscle could cause localized muscle contraction and MPS[27]. The referred pain from the trigger point is due to a sensory neuron sensitization in the spine cord posterior horn and may have a distribution similar to that in the radiculopathic pain. This referred pain associates with tingling and numbness[26]. In 88.2% of 17 patients with preoperative nerve root pain history, lumbar and gluteal MPS was identified.

The ages of patients included in the study when firstly seen at the Pain Center ranged from 28 to 76 years (mean age, 48.8 years). The mean symptom length was 96 months and the mean pain intensity was 8.3, showing the magnitude and the extended distress the patients went through. The postoperative pain was also shown more severe than the preoperative pain.

The chronic pain treatment should involve a multidisciplinary team and pharmacological, physiatric, psychotherapeutic, and neuroanesthesic procedures; functional neurosurgical procedures should be performed if required[28]. The treatment with analgesic drugs, whether they are anti-inflammatory drugs or not, psychotropic drugs and physical medicine provided > 50% original pain improvement in 57.2% of patients evaluated in this study. Myofascial trigger point treatment consists of using analgesic drugs, psychotherapeutic agents, muscle relaxant drugs, refrigerant vapor, dry needling, local anesthetic injection and stretching, as well as correction of causal or perpetuating factors[29].

In 69.4% of patients undergoing administration of a morphine and lidocaine solution via peridural route in our study, the original pain had > 50% improvement.

The pain in patients with failed back surgery pain syndrome is severe, affects individuals in the fullness of their activities, is often found as a lumbar and/or gluteal MPS and, less frequently, has a neuropathic pattern alone or associated with MPSs[29].

Conclusion

The failed back surgery pain syndrome evaluation and management is challenging for the medical team. Analgesic drugs and physical medicine provide major improvement in most cases. The pain intensity in post-laminectomy syndrome is worse than the herniated disk preoperative pain. The injection of myofascial trigger points and opiate infusion into the lumbar spine compartment can be required in refractory pain cases.

References

1. Merskey NB. Classification of chronic pain: descriptions of chronic pain syndromes and definitions of pain terms prepared by the International Association for the Study of Pain. 2nd ed. Seattle: IASP Press; 1994.

2. Follett, KA, Dirks BA. Etiology and evaluation of the failed back surgery outcome. Neurosurg Q. 1993;3:40-59.
3. Saal JS, Franson RC, Dobrow R, Saal JA, White AH, Goldthwaite N. High levels of inflammatory phospholipase A2 activity in lumbar disc herniations. Spine 1990;15:674-8.
4. Martin BI, Mirza SK, Comstock BA, Gray DT, Kreuter W, Deyo RA. Are lumbar spine reoperation rates falling with greater use of fusion surgery and new surgical technology? Spine 2007;32:2119-26.
5. Deyo RA. Back surgery-who needs it? N Engl J Med. 2007;356:2239-43.
6. Deyo RA. Back pain revisited newer thinking on diagnosis and therapy. Consultant 1993;33:88-100.
7. Goupille P. Cause des echecs de la chirurgie discale. Rev Rheumat 1996;63:255-60.
8. Willberger RE, Wettenberg RH. Prostaglandin release from lumbar disc and facet joint tissue. Spine 1994;19:2068-70.
9. Deyo RA, Tsiu WU. Descriptive epidemiology of low-back pain and its related medical care in the United States. Spine 1987;12:164-8.
10. Frymoyer JW. An international challenge to the diagnosis and treatment of disorders of the lumbar spine. Spine 1993;18:2147-52.
11. Hanley EN Jr, Shapiro DE. The development of low-back pain after excision of a lumbar disc. J Bone Joint Surg Am. 1989;71:719-21.
12. Hazard RG. Failed back surgery syndrome: surgical and nonsurgical approaches, Clin Orthop Relat Res. 2006;443:228-32.
13. Ragab A, Deshazo RD. Management of back pain in patients with previous back surgery. Am J Med. 2008;121:272-8.
14. Boden SD, McCowin PR, Davis DO, Dina TS, Mark AS, Wiesel S. Abnormal magnetic-resonance scans of the cervical spine in asymptomatic subjects. A prospective investigation. J Bone Joint Surg Am. 1990;72:1178
15. Hitselberger WE, Witten RM. Abnormal myelograms in asymptomatic patients. J Neurosurg. 1968;28:204-6.
16. Wiesel SW, Tsourmas N, Feffer HL, Citrin CM, Patronas N. A study of computer assisted tomography. The incidence of positive cat scans in an asymptomatic group of patients. Spine 1984;9:549-51
17. Weber H. Lumbar disc herniation. A controlled, prospective study with ten years of observation. Spine 1983;8:131-40.
18. Hakelius A. Prognosis in cciatica: a clinical follow-up of surgical and non surgical treatment. Acta Orthop Scand. 1970;129 (Suppl):1-76.
19. Saal JA, Saal JS. Non operative treatment of herniated lumbar intervertebral disk with radiculopathy: An outcome study. Spine 1989;14:431-7.
20. Hurme M, Alaranta H. Factors predicting the results of surgery for lumbar intervertebral disc herniation. Spine. 1987;12:933-8.
21. Gibson JN, Grant IC, Waddell G. Cochrane Database Syst Rev. 2000;(3):CD001350.
22. Finnegan WJ, Fenlin JM, Marvel J. Results of surgical intervention in the symptomatic multiply-operated patient: analysis of sixty seven cases followed three to seven years. J Bone Joint Surg. 1979;611:1077-82.
23. Licber RL. Low back pain. A scientific and clinical overview. Washinghton (DC): American Academy of Orthopaedics Surgeon Workshop; 1995.
24. Fritsch EW, Heisel J, Rupp S. The failed back surgery syndrome: reasons, intraoperative findings and long term results: a report of 182 operative treatments. Spine 1996;21:626-33.
25. Imamura ST, Fischer AA, Imamura M, Teixeira MJ. Pain management using myofascial approach when other treatment failed. Myofascial pain update in diagnosis and treatment. Phys Med Rehab Clin North Am. 1997;8:179-92.
26. Rachlin ES. Importance of trigger point management in orthopedic practice myofascial pain update in diagnosis and treatment. Phys Med Rehabil Clin North Am. 1997;8:171-7.
27. Cannon DE, Dillingham TR, Miao H, Andary MT, Pezzin LE. Musculoskeletal disorders in referrals for suspected lumbosacral radiculopathy. Am J Phys Med Rehabil. 2007;86:957-61.
28. Teixeira MJ. Princípios de tratamento da dor. In: Teixeira MJ, Figueiró JAB, editors. Dor, epidemiologia, fisiopatologia, avaliação, síndromes dolorosas e tratamento. São Paulo: Grupo Editorial Moreira Jr; 2001. p. 86-92.
29. Lin TY, Kazyiama HHS, Teixeira MJ. Síndrome dolorosa miofascial. Rev Medicina 2001;80(1):94-110.