PREVENTION AND MANAGEMENT OF LOW BACK PAIN IN REHABILITATIOIN PRACTICE

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Introduction

- Chronic low back pain (CLBP) is a chronic pain syndrome in the lower back region, lasting for at least 3 months.
- CLBP represents the second leading cause of disability worldwide being a major welfare and economic problem.
- The prevalence of CLBP in adults has increased more than 100% in the last decade and continues to increase dramatically in the aging population, affecting both men and women in all ethnic groups, with a significant impact on functional capacity and occupational activities.
- It can also be influenced by psychological factors, such as stress, depression and/or anxiety.

“Pain generators” in low back pain

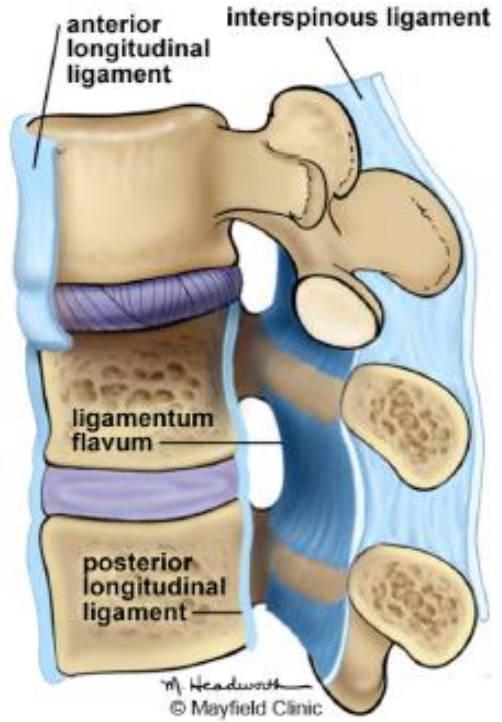
- Traditionally, the notion that the etiology of 80% to 90% of LBP cases is unknown has been mistaken perpetuated across decades.
- In most cases, low back pain can be attributed to **specific pain generator,** with its own characteristics and with different therapeutical opportunity.
- Diagnostic evaluation ---very challenging and requires complex clinical decision-making.

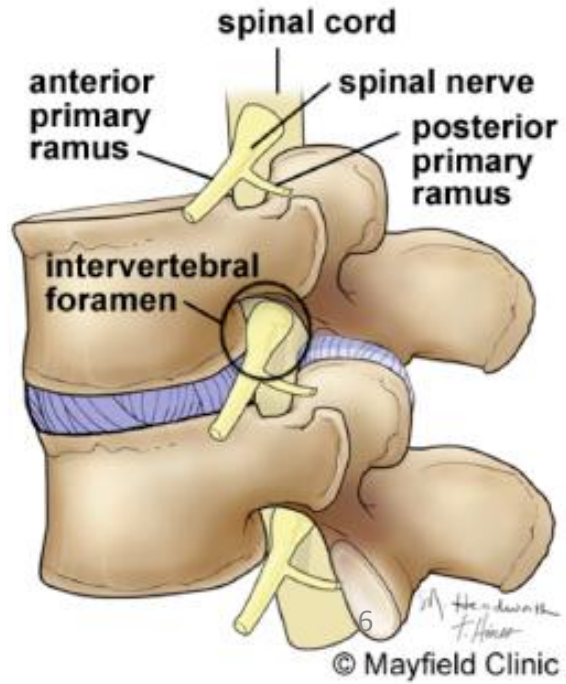
- Question ---“what is the pain generator” among the several structures potentially involved in CLBP is a key factor in the management of these patients, since a mis-diagnosis can generate therapeutical mistakes.

Pain Generators

- LBP symptoms can derive from many potential anatomic sources, such as nerve roots, muscle, fascial structures, bones, joints, intervertebral discs (IVDs), and organs within the abdominal cavity.

- Intervertebral discs
  - Keep the bone from rubbing together
  - Shock absorption
- Vertebral arch and spinal canal, containing spinal cord
- Facet joints - allow back motion
- Ligaments - stabilize the spine and protect he discs
  - ALL and PLL – prevent excessive movement of the vertebral bones.
- Spinal cord
- Spinal nerves
  - 31 pairs of spinal nerves branch off the spinel cord
  - innervate specific areas and form a dermatomes
- Covering or membrances – pia, arachnoid and dura mater (e.g. Epidural Steroid injections)





(Ref:/ Anatomy of the spine, Mayfield Brain and Spine, MayfieldClinic.com)

- the identification of the source of the pain is of fundamental importance in determining the therapeutic approach .
- Furthermore, during the clinical evaluation, a clinician has to consider that LBP can also be influenced by psychological factors, such as stress, depression, and/or anxiety.
- History should also include substance use exposure, detailed health history, work, habits, and psychosocial factors.
- As chronic LBP could have simultaneous multiple pain generators, a multi-disciplinary diagnosis and multimodal treatment is necessary.

MRI

- MRI should be considered only in the presence of clinical elements that are not definitely clear or in the presence of neurological deficits or other medical conditions.
- The recommendation of the American College of Radiology is not to do imaging for LBP within the first 6 weeks unless *red flags* are present. These are
    - Recent substantial trauma or milder trauma in those over 50 years old
    - weight loss or fever with no known cause
    - immunosuppression
    - a previous cancer diagnosis
    - intravenous drug use
    - sustained corticosteroids use or osteoporosis
    - being over 70 years old
    - focal neurologic deficit with progressive or disabling symptoms

Pathophysiology of spinal pain

- Pain is mediated by nociceptors, specialized peripheral sensory neurons that alert us to potentially damaging stimuli at the skin by transducing these stimuli into electrical signals that are relayed to higher brain centers.
- Nociceptors are pseudo-unipolar primary somatosensory neurons with their neuronal body located in the DRG.
- They are bifurcate axons: the peripheral branch innervates the skin and the central branches synapse on second-order neurons in the dorsal horn of the spinal cord.
- The second-order neurons project to the mesencephalon and thalamus, which in turn connect to somatosensory and anterior cingulate cortices in order to guide sensory-discriminative and affective-cognitive features of pain, respectively.
- The spinal dorsal horn is a major site of integration of somatosensory information and is composed of several interneuron populations forming descending inhibitory and facilitatory pathways, able to modulate the transmission of nociceptive signals.

- If the noxious stimulus persists, processes of peripheral and central sensitization can occur, converting pain from acute to chronic.
- Central sensitization is characterized by the increase in the excitability of neurons within the central nervous system, so that normal inputs begin to produce abnormal responses.
- It is responsible for tactile allodynia, that is pain evoked by light brushing of the skin, and for the spread of pain hypersensitivity beyond an area of tissue damage.
- Central sensitization occurs in a number of chronic pain disorders, such as temporomandibular disorders, LBP, osteoarthritis, fibromyalgia, headache, and lateral epicondylalgia.
- Despite improved knowledge of the processes leading to central sensitization, it is still difficult to treat.

- Peripheral and central sensitization have a key role in LBP chronification.
- In fact, minimal changes in posture could easily drive long-lasting inflammation in the joints, ligaments, and muscles involved in the stability of the low back column, contributing to both peripheral and central sensitization.
- Furthermore, joints, discs, and bone are richly innervated by A delta fibers whose continuous stimulation could easily contribute to central sensitization.

Type of spinal pain according to pain generator

- Mostly, LBP is considered to be nonspecific, and the mistaken idea that the cause of 80 to 90% of LBP cases is unknown has persisted for decades.
- Muscle tension and spasm are among the most common reasons for LBP, for example, in patients with fibromyalgia. In other cases, LBP can be attributed to different pain generators, with specific characteristics, such as radicular, facet joint, sacro-iliac, and discogenic pain, as well as spinal stenosis.

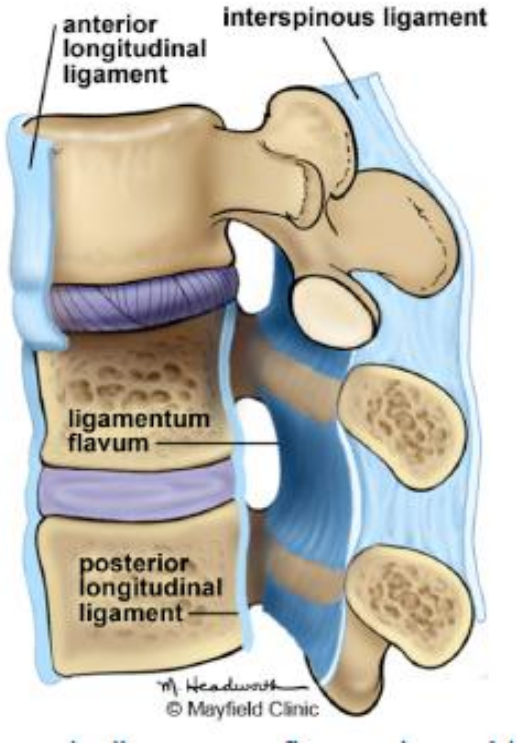
Radicular pain

- Radicular pain is pain evoked by ectopic discharges emanating from an inflamed or lesioned dorsal root or its ganglion; generally, the pain radiates from the back and buttock into the leg in a dermatomal distribution.
- Disc herniation is the most common cause, and inflammation of the affected nerve rather than its compression is the most common pathophysiological process.
- Radicular pain is pain irradiated along the nerve root without neurological impairment. Even though it is nociceptive pain, it is distinguished from usual nociception because in radicular pain the axons are not stimulated along their course or in their peripheral terminals but from the perinevrium.
- Radicular pain differs from radiculopathy in several aspects. Radiculopathy impairs conduction down a spinal nerve or its roots. The impairment of sensory fibers causes numbness (dermatomally distributed); however, blockade of motor fibers causes weakness (myotomal). Sensory or motor block may result in diminished reflexes.

- If a patient's history and physical examination findings indicate lumbar disc herniation with radiculopathy, the most suitable noninvasive test to confirm this could be an MRI.
- This is particularly important if it is necessary to proceed with an invasive treatment or to better define the neurological impairment.
- The next most appropriate test to evaluate the presence of lumbar disc herniation is computed tomography (CT) or CT myelography, which would be suitable for those individuals unable to have an MRI because it is contraindicated or those for whom MRI is inconclusive.
- Also, diagnosis of nerve root compression may be achieved by electrodiagnostic studies, although they are not able to distinguish between lumbar disc herniation and other causes of nerve root compression.
- Unfortunately, we have to remark that radiculopathy could be present without radicular pain and vice versa.
- For these reasons, electrodiagnostic tests are not recommended as a first-line approach but only as a second-line one in order to define if there is a concomitant presence of peripheral neuropathy or neuralgia or to follow up the impairment of the lesioned nerve.

Facet joint syndrome

- The lumbar zygapophyseal joints are the posterior articular process of the lumbar column. They are formed from the inferior process of upper vertebra and the superior articular process of lower vertebra.
- They are supplied by the medial branches of the dorsal rami (MBN). These joints have a large amount of free and encapsulated nerve endings that activate nociceptive afferents and that are also modulated by sympathetic efferent fibers.
- Lumbar zygapophyseal or "facet" joint pain has been estimated to account for up to 30% of CLBP cases, with nociception originating in the synovial membrane, hyaline cartilage, bone, or fibrous capsule of the facet joint.



- Diagnosis of facet joint syndrome is often difficult and requires a careful clinical assessment and an accurate analysis of radiological exams.

Symptoms

- LBP with or without somatic referral to the legs terminating above the knee, often radiating to the thigh or to the groin.
- There is no radicular pattern.
- Back pain tends to be off-center
- the pain intensity is worse than the leg pain
- pain increases with hyperextension, rotation, lateral bending, and walking uphill.
- It is exacerbated when waking up from bed or trying to stand after prolonged sitting.
- Finally, patients often complain of back stiffness, which is typically more evident in the morning.

- It is difficult to diagnose lumbar facet syndrome using radiology as there are no pathognomonic findings to look for.
- With MRI, we can find non-specific signs of arthrosis, osteophytes, and hypertrophy of flaval ligaments.
- However, if we want to better study arthrosis problems, CT is the preferred imaging method, even if radiation exposure should be kept in mind.
- One of the most important exams is provided by X-rays, especially dynamic projections, that can show column instability (listhesis that could be increased with flexion and extension of the low back column) with a clear overload of these joints.
- In conclusion, despite the contribution from neuroimaging, history and clinical examination remain fundamental steps in the diagnosis of facet joint syndromes.

Sacroiliac joint pain

- Sacroiliac joints (SIJs) are dedicated to providing stable but flexible support for the upper body.
- SIJs are involved in sacral movement, which additionally directly influences the discs and almost certainly the higher lumbar joints. Its innervation is still not well known but has been reported to be by branches from the ventral lumbopelvic rami ; however, this has not yet been confirmed.
- On the other hand, several authors have reported innervation of the SIJ by small branches from the posterior rami.
- In a 2012 study by Patel et al., the authors demonstrated that SIJ pain was successfully attenuated using neurotomy of the L5 dorsal primary ramus and lateral branches of the dorsal sacral rami from S1 to S3.
- Hence, there is sufficient evidence that this procedure has an important value for establishing diagnosis and prognosis. The SIJ is well recognized as a source of pain in many patients who present with CLBP.
- It is thought that pain could be generated by ligamentous or capsular tension, extraneous compression or shear forces, hypermobility or hypomobility, altered joint mechanics, and myofascial or kinetic chain dysfunction causing inflammation.

- Intra-articular sources of SIJ pain include osteoarthritis; extra-articular sources include enthesis/ligamentous sprain and primary enthesopathy.
- In addition, ligamentous, tendinous, or fascial attachment and other cumulative soft tissue injuries that may occur posterior to the dorsal aspect of the SIJ may be a source of discomfort.
- In physical examination, it is important to examine the movement of the joint, for example with a stress test, consisting of pressing down on the iliac crest (pelvis) or upper thigh, which may reproduce the patient's pain.

- SIJ pain is often underdiagnosed. It has to be considered in every situation in which the patient complains of postural LBP that worsens in a sitting position and with postural changes.
- Furthermore, it is possible that SIJ pain is often strictly related to facet joint syndromes as both are related to postural problems.
- Finally, it is important to consider that SIJ pain could also be a sign of rheumatic disease. MRI findings of articular effusion and inflammation (especially if bilateral) can alert the clinician to consider this condition.

Lumbar spinal stenosis

- Lumbar spinal stenosis (LSS) can be congenital or acquired (or both).
- It could be determined by inflammatory/scar tissue after spine surgery or, even in absence of previous surgery, by disc herniation, thickening of the ligaments, or hypertrophy of the articular processes.
- The majority of cases of LSS are degenerative, related to changes in the spine with aging.
- LSS is determined by a progressive narrowing of the central spinal canal and the lateral recesses and consequent compression of neurovascular structures.
- Usually, the diameter of the normal lumbar spinal canal varies from 15 to 27 mm.
- We can define lumbar stenosis as a spinal canal diameter of less than 10 mm, even though a stenosis with diameter of 12 mm or less in some patients can be symptomatic.

- The normal foraminal height varies from 20 to 23 mm, with the indicator of potential foraminal stenosis as 15 mm or less.
- Degenerative LSS is the most common indication for spinal surgery in people older than 65 years of age.
- The most frequent symptoms of lumbar stenosis are midline back pain, radiculopathy with neurologic claudication, motor weakness, paresthesia, and impairment of sensory nerves.
- Symptoms may have a different distribution depending on the type of LSS.
- If the LSS is central, there may be involvement of the area between the facet joints, and pain may be bilateral in a non-dermatomal distribution.
- With lateral recess stenosis, symptoms are usually found dermatomally because specific nerves are compressed, resembling unilateral radiculopathy.
- Trunk flexion, sitting, stooping, or lying can all ease the discomfort, while prolonged standing or lumbar extension can aggravate the pain.
- Sitting or lying down become less effective in alleviating pain as the condition progresses, and rest pain or a neurogenic bladder can develop in severe cases.
- Neurogenic claudication pain is the classical symptom of LSS, caused by venous congestion and hypertension around nerve roots. Pain is exacerbated by standing erect and by downhill ambulation but alleviated with lying supine more than prone, sitting, squatting, and lumbar flexion.

- LSS is generally diagnosed based on a combination of history, physical examination, and imaging.
- The most useful findings from the history are age, radiating leg pain that is exacerbated by standing up or walking, and the absence of pain when seated.
- The gait and posture after walking may reveal a positive “stoop test”, performed by asking the patient to walk briskly. As the pain intensifies, patients may complain of sensory symptoms followed by motor symptoms, and if they assume a stooped posture, symptoms may improve. If patients sit in a chair bent forward, they may have the same relief.

- The recommended method for confirming the diagnosis of LSS is MRI, which facilitates the assessment of the spinal canal and the anatomic relationship between spinal and neural elements.
- The natural course of untreated LSS is unclear.
- The North American Spine Society (NASS) clinical guidelines concluded that the natural course is favorable in a third to a half of patients with clinically mild to moderate LSS.
- Other reviews suggest that the condition may deteriorate in some patients and improve in about a third of others, with most patients remaining unchanged for up to 8 years of follow-up.

Discogenic pain

- Disc degeneration (DD) has been estimated as the source of CLBP in 39% of cases.
- Its symptoms are aspecific, axial, and without radicular radiation and they occur in the absence of spinal deformity or instability.
- DD is often a diagnosis of exclusion among other types of CLBP. Pathologically, it is characterized by the degradation, within the disc, of the NP matrix with accompanying radial and/or concentric fissures in the AF.
- Despite numerous recent advances, the main issue is how inflammation is initiated and sustained to lead to CLBP. A possible explanation could involve the growth of nerves capable of signaling pain deep into the annular structures.
- Another hypothesis involves a class of molecules, called damage-associated molecular patterns (DAMPs), including hyaluronic acid and fibronectin fragments, able to stimulate sterile inflammation of the disc through the action of pro-inflammatory cytokines (IL-1beta, IL-6, and IL-8) and matrix degrading enzymes (MMP-1, MMP-3, and MMP-13). Also, subclinical anaerobic bacterial infection, encouraged by hypoxic conditions, could have a role in the development of discogenic pain.

- Imaging MRI can detect changes in the endplates and in the vertebral bone marrow, such as edema in the vertebral bodies (Modic type 1).
- Clinical trials have demonstrated that some patients suffering from LBP have improvement following amoxicillin-clavulanate.
- Moreover, diabetes increases the risk of developing painful DD because advanced glycation end products (AGEs) induce catabolism and promote inflammation.

- MRI cannot definitively demonstrate whether a disc is painful.
- Provocation discography aims at reproducing patients' pain through contrast injection during live fluoroscopy plus CT imaging for clarifying associated morphological abnormalities of the disc.
- The clinical utility of discography and its diagnostic accuracy is, however, a matter of controversy because of poor specificity. Beyond the reported complications as discitis, neurologic injury, visceral injury, and dye reactions, it's been demonstrated that the needle puncture of the lumbar disc may lead to accelerated MRI-documented DD.
- The mechanism is likely multifactorial: structural damage caused by the needle, pressurization, and toxicity of the contrast media.

Causes of low back pain

- **Mechanical**

Strain and sprain

Herniated / ruptured intervertebral disc

Interverteral disc degeneration

Radiculopathy

Spinal stenosis

Spondylolisthesis

Traumatic injury

Skeletal irregularities

Infectious

Tuberculosis
Osteomyelitis
Discitis
Pyogenic sacroiliitis
Herpes zoster

Neoplastic / Infiltrative

Spine tumors
Osteoid osteoma
Osteoblastoma
Osteochondroma
Spinal metastasis
Ca lung, breast,prostate etc

- **Rheumatologic**
  - Osteoarthritis
  - Ankylosing spondylitis
  - Reiter's syndrome
  - Psoriatic arthritis
  - Enteropathic arthritis
  - Diffuse idiopathic skeletal hyperostosis
  - Fibromyalgia
  - Polymyalgia rheumatica

Endocrinologic

- Osteoporosis
- Osteomalacia
- Parathyroid disease

Referred pain

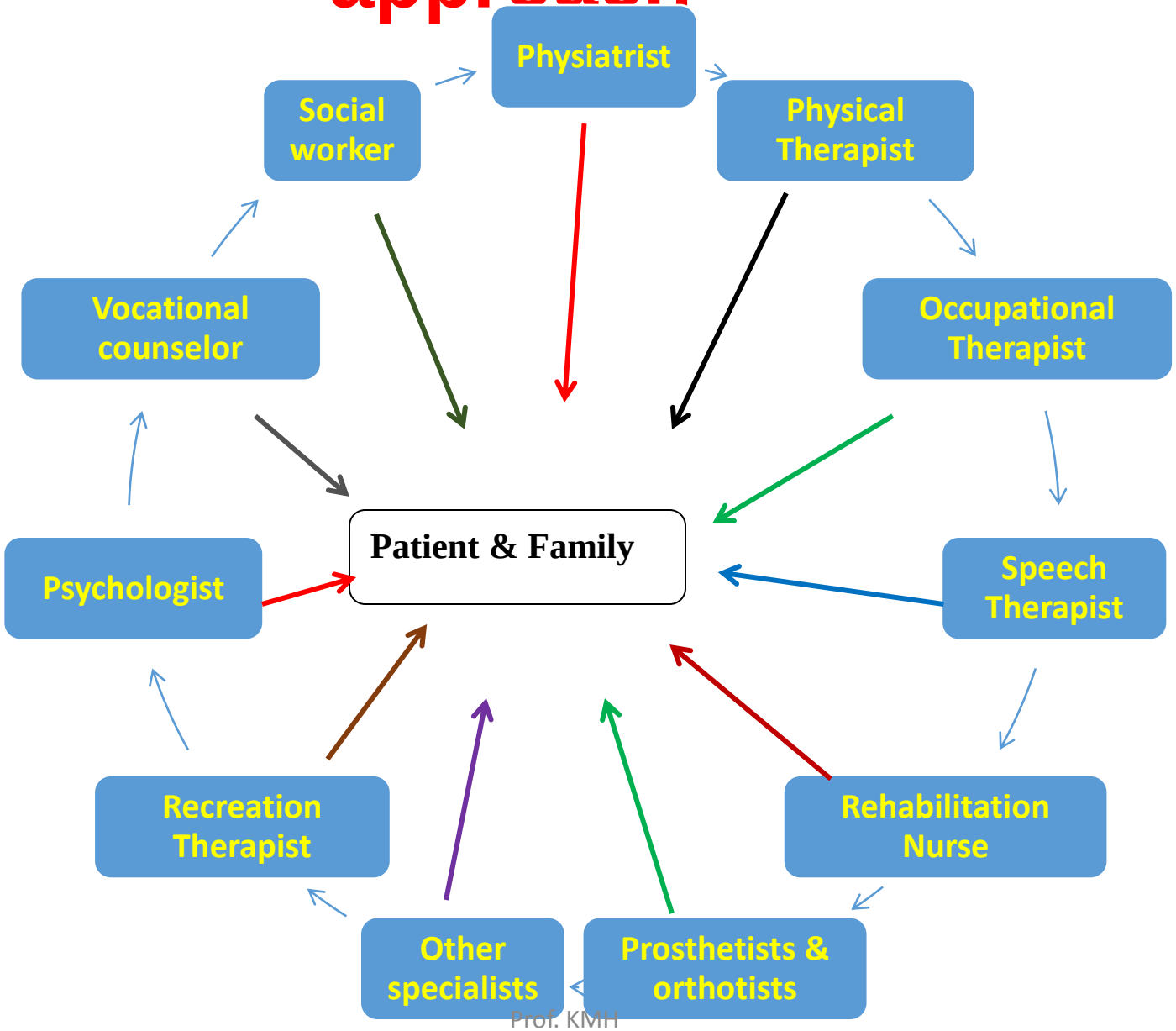
- Abdominal aortic aneurysm
- Pancreatitis
- Renal stones
- Endometriosis

Approach

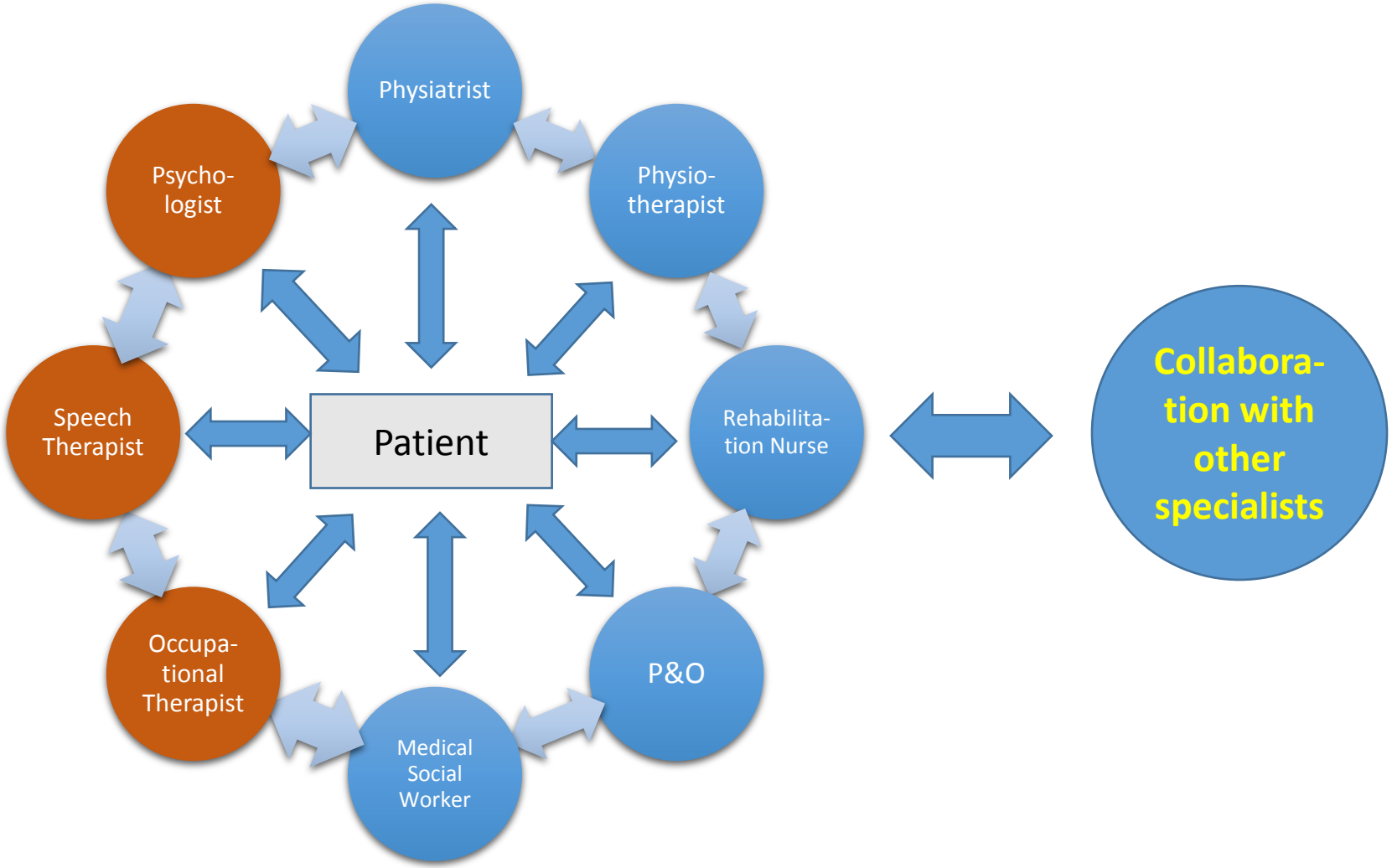
- To treat the whole patient (Holistic and comprehensive health care) with multidisciplinary team approach
  - not just the specific injury or condition
  - in order to improve overall recovery
  - prevent recurrence of back pain or other source of dysfunction.
  - needs to focus on Functional, vocational, and socioeconomic and psychological status
- Goals of treatment
  - to restore the patient's normal function and
  - improve quality of life for patients from a physical, emotional, psychosocial and vocational perspective.

Ref: Delisa, Physical Medicine and Rehabilitation Practice, 5th ed

Multi & Interdisciplinary team approach



Rehabilitation Team



Red flags(Risk Factors for Secondary LBP Due to Important Pathologies)

- <18 yr or onset >55 yr
- History of violent trauma
- Mild trauma in an aged patient
- Constant progressive pain at night
- History of cancer
- Systemic steroids use
- Drug abuse, HIV
- Weight loss
- Systemic illness
- Persisting severe restriction of motion
- Intense pain in minimal motion
- Structural deformity
- Difficulty with micturition
- Loss of anal sphincter tone or fecal incontinence; saddle anesthesia
- Widespread progressive motor weakness or gait disturbance
- Inflammatory disorders (AS) suspected
- Gradual onset <40 y
- Marked morning stiffness
- Persisting limitation of motion
- Peripheral joint involvement
- Iritis, skin rushes, colitis, urethral discharge
- Family history

Ref: Delisa, Physical Medicine and Rehabilitation Practice, 5th ed

Yellow Flags(Risk Factors for Chronicization of Acute and SALBP)

- Female gender
- Minor ethnicity
- Low income
- Low education
- Medical High BMI
- Previous surgery
- Impairment
- Neurological deficit
- Radicular impingement (SLR, Wassermann tests)
- Pain related Duration
- Intensity
- Leg pain
- Pain in lateral flexion and/or in flexion-extension
- Difficulties in sitting
- Impairment
- High referred impairment
- High functional limitation at 4 wk
- High disability
- Perceived risk of not recovering
- Psychosocial Not appropriate signs and symptoms
- Avoidance behavior
- Psychological burden
- Vital energy reduction
- Reduced emotional confronting capacity
- Social isolation
- Depression
- Somatization
- Reduced coping strategies

Ref: Delisa, Physical Medicine and Rehabilitation Practice, 5th ed

Management of acute LBP

- Prognosis is good
- auto- resolving in most of the cases, less than a month
- main objective and therapy in ALBP is, in fact, to reassure the patient and provide accurate preventive information and education more appropriate because LBP is less than a month and does not present any disability at this stage
- The key recommendation is to remain as active as possible and to avoid bed rest.
- two important factors related to prevention simultaneously: avoiding chronicity and regaining any fitness lost during the period of acute pain which may open the way to frequent relapses.
- transformation of acute to chronic depends on a complex series of not always identifiable psychosocial factors, “yellow flags” that must be carefully taken into consideration to prevent the transformation into a chronic disorder

Ref: Delisa, Physical Medicine and Rehabilitation Practice, 5th ed

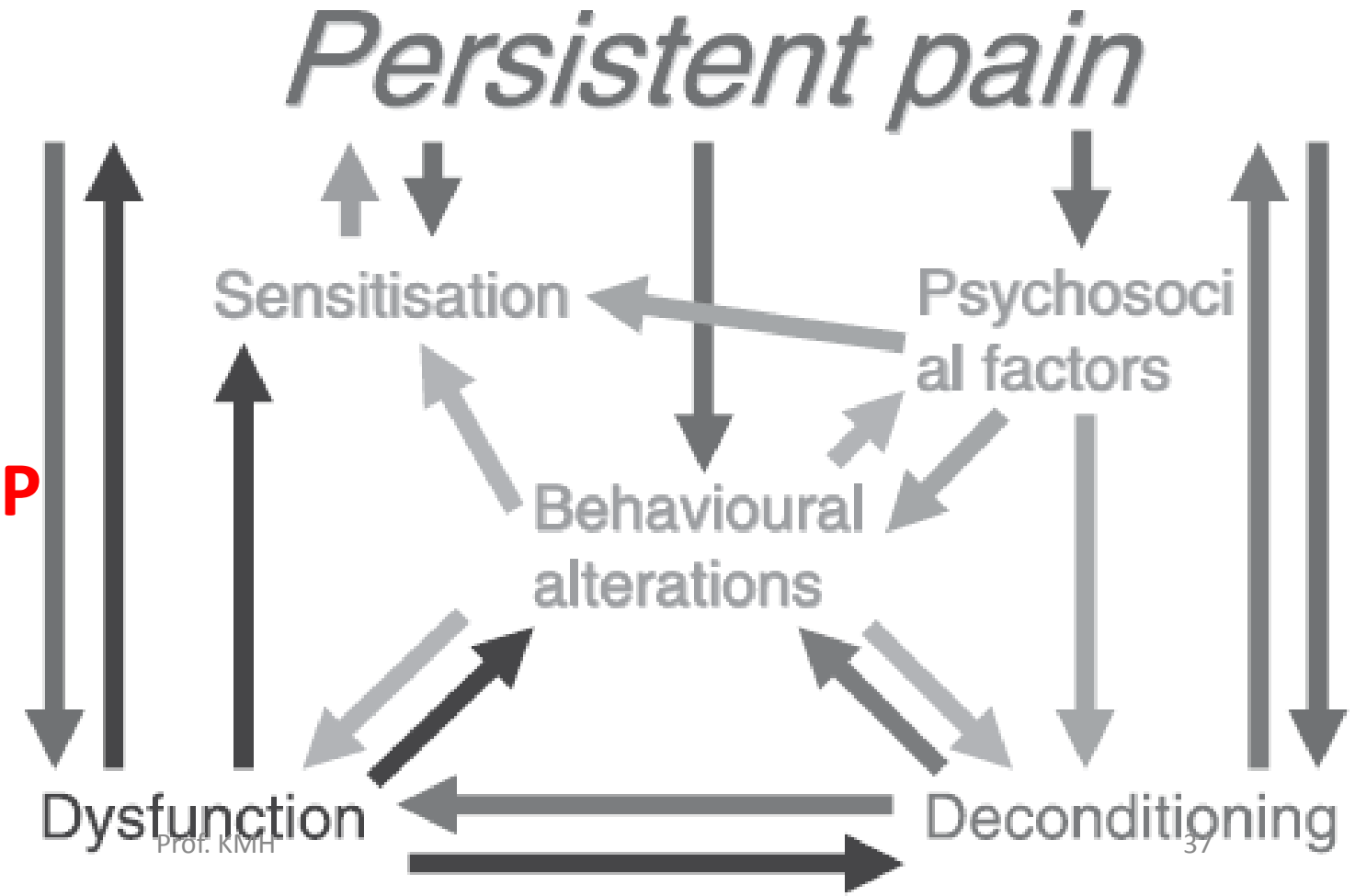
Management of Sub Acute LBP

- Subacute LBP - real intermediate stage between acute and chronic.
- In this period, apparently there is something that impedes the spontaneous resolution of ALBP, maintains the previous situation, makes it more complex and gradually drives the patient, through a series of vicious cycles, to CLBP
- These factors recognized as the chronicization risk factors
- Chronic LBP also leads to psychological and/or social disturbances

Ref: Delisa, Physical Medicine and Rehabilitation Practice, 5th ed

The patient is enveloped in a series of vicious cycles that emerge from Sub Acute LBP through a full development of the chronicity risk factors for LBP

Management of Chronic LBP



Ref: Delisa, Physical Medicine and Rehabilitation Practice, 5th ed

Risk factors for chronicity

- Previous history of low back pain
- Nerve root involvement
- Poor physical fitness
- Self rated health poor
- Heavy smoking
- Psychological distress and depressive symptoms
- Disproportionate illness behaviour
- Low job satisfaction
- Personal problems eg marital, financial

Ref: Delisa, Physical Medicine and Rehabilitation Practice, 5th ed

Treatment options

- Pharmacological- Analgesic, NSAIDs, muscle relaxants, anti- epileptics, tranquilizers, etc.
- Non Pharmacological
  - Health education
  - Postural care
  - Physical Modalities
  - Physical activity and therapeutic exercise
  - Bio-psychosocial approach
  - prevention of recurrence
  - Social integration and return back to work
- Surgery

Ref: Delisa, Physical Medicine and Rehabilitation Practice, 5th ed

Non-pharmacological management

- Reassurance
- Rest
- Education
- Physiotherapy
- Therapeutic exercises
- Back care program
- Alternative Medicine- Massage, Acupuncture

Ref: Delisa, Physical Medicine and Rehabilitation Practice, 5th ed

Non Pharmacological Management

Physical Modalities

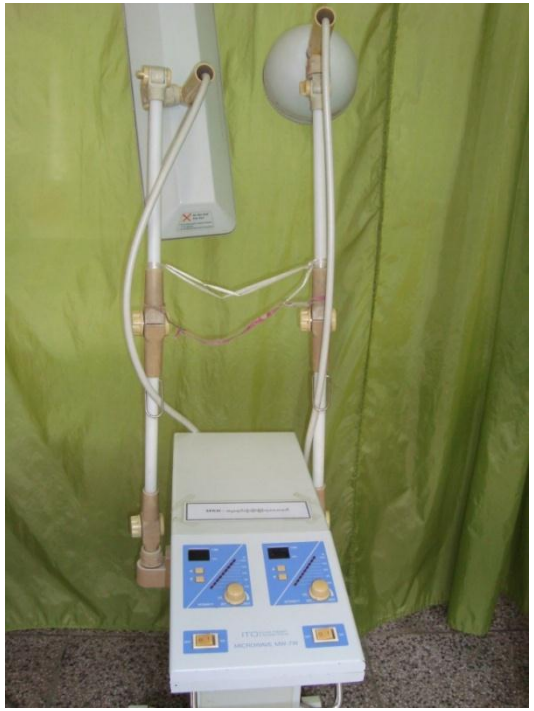
- Cold therapy
- Thermotherapy
  - Superficial heating
  - Deep Heat
- Electrotherapy
  - Transcutaneous electrical nerve stimulation (TENS)

Manipulation
Traction
Massage
Physical therapy and exercises
Acupuncture
Corsets and braces

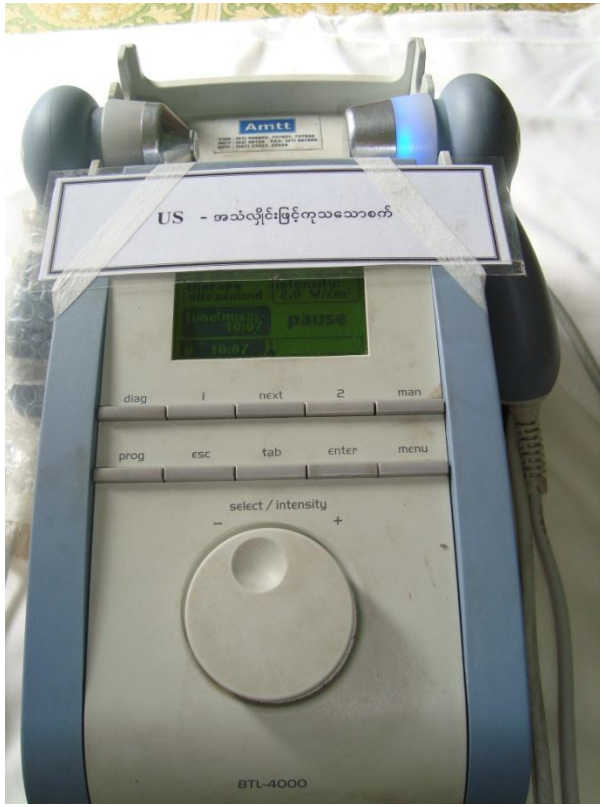
Ref: Delisa, Physical Medicine and Rehabilitation Practice, 5th ed

Superficial Heat

DEEP HEAT



Microwave therapy

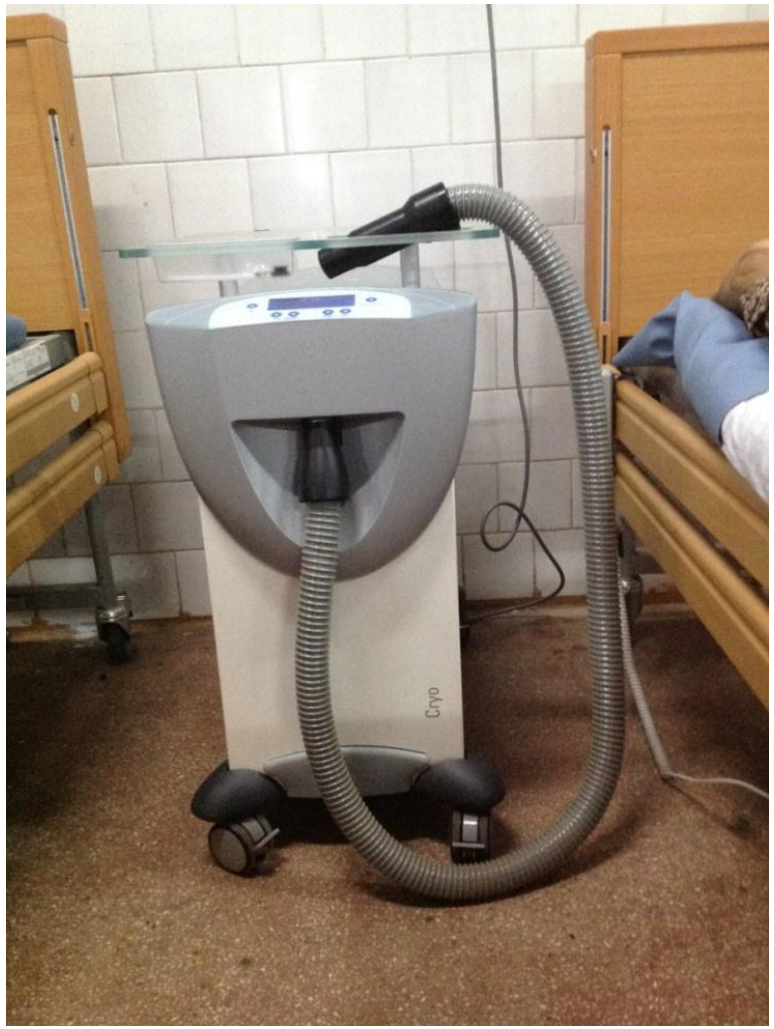


Ultrasound Therapy

Short wave Therapy

Cryotherapy

- Cold packs
- Ice massage
- Vapocoolant spray
- Cryotherapy Unit



Ref: Electrotherapy explained, 2nd Ed

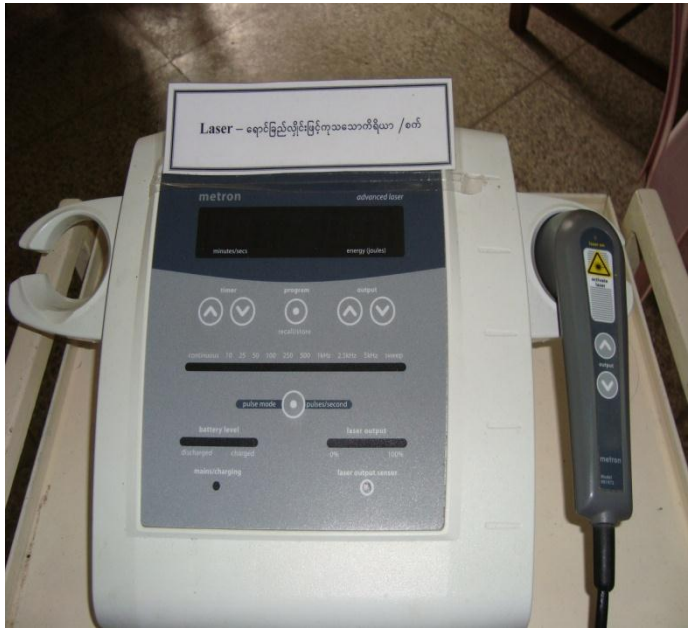
TENS (Transcutaneous Nerve Stimulator (TENS)

- This is a small battery operated instrument. It has a simple theory "gate control" i.e.
- Pain can be blocked by non destructive means.
- A gentle application of electrical stimulation (with special rate, width & voltage control) helps to close the gate, thereby blocking the pain message to the brain at the spinal cord level giving immediate pain relief in certain conditions.

Ref: Electrotherapy explained, 2nd Ed

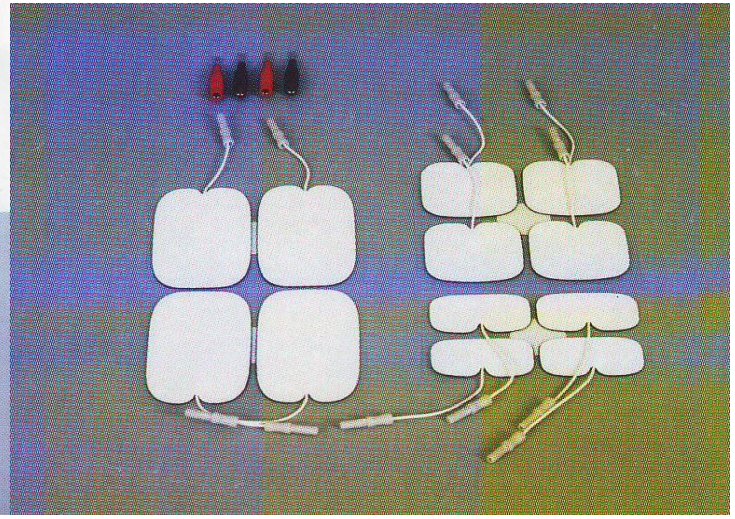
Extracorporeal Shock Wave Therapy

LASER Therapy



Functional and Motivating Back Training

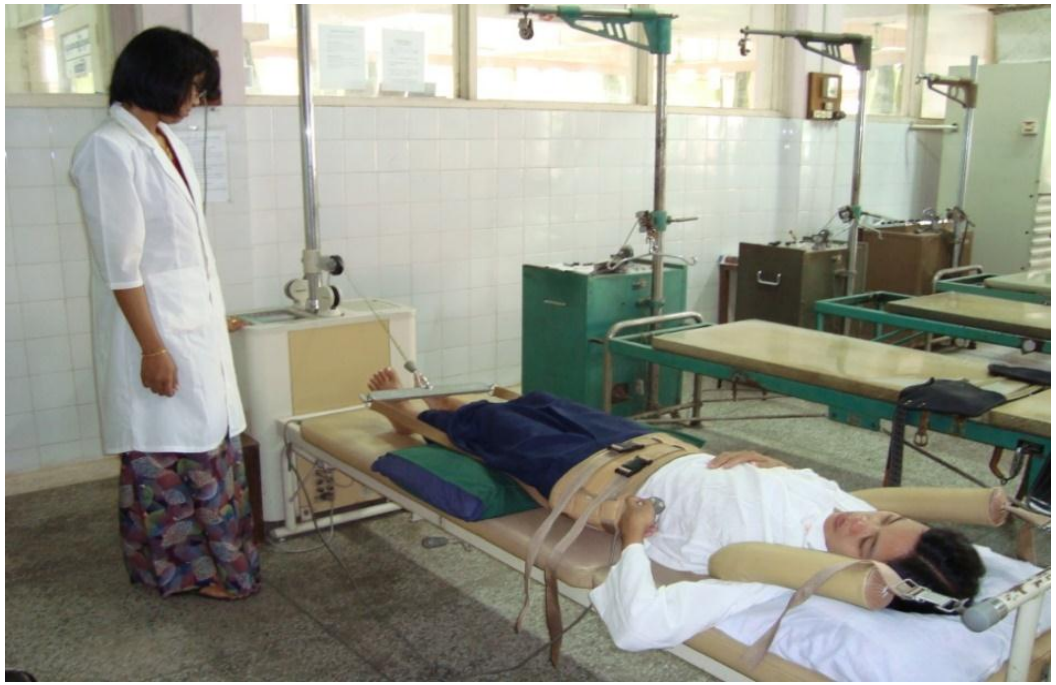
Myofeedback



Biofeedback

used to measure the electrical activity of back muscles and to train patients to reduce muscle activity and tension.

Traction



Manual therapy

Includes techniques that use a hands-on approach such as massage (stroking, friction, kneading) manipulation and mobilization.

Stroking massage decreases edema and produce relaxation of muscles, while friction and kneading massage break down intramuscular adhesions and prepare the muscles and soft tissue for stretching

Ref: Electrotherapy explained, 2nd Ed

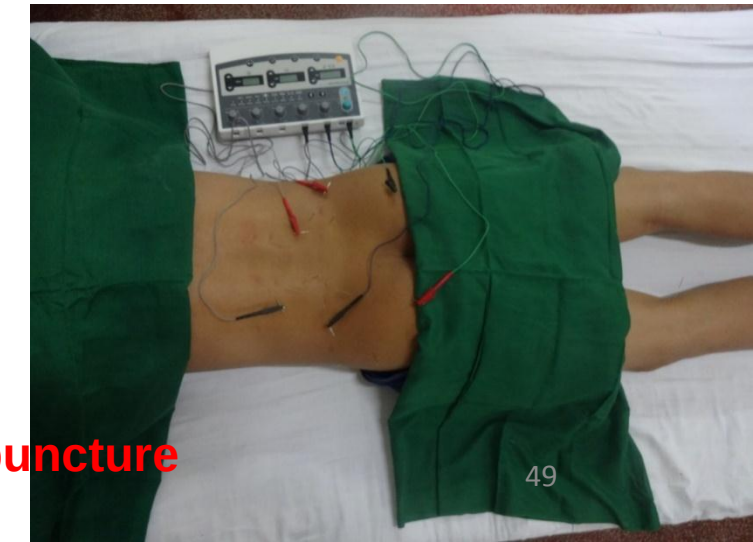
- **Shiatsu** (massage with finger pressures)

  warm and relax back muscles and improve blood flow.

Acupuncture

- Originated in China. It breaks up the pain –spasm cycle and stimulates endogenous opoids. However risk of infection is high if proper aseptic technique and disposable needles are not used.

Acupuncture



Electro-Acupuncture

Back corsets and belts

Results are controversial.

They helps prevent the patients from excessive movements and rotations and support the back.

Ref: Delisa, Physical Medicine and Rehabilitation Practice, 5th ed

Therapeutic exercises

- Most important to help people with low back pain to regain flexibility, to built up muscle strength and stamina, and to improve general fitness.
- **Three main group of exercises**

  Stretching exercises

  Strength, stamina, and stabilizing exercises

  Cardiovascular aerobic conditioning

  followed by exercises for general fitness: swimming, walking, running, cycling exercises

Ref: Delisa, Physical Medicine and Rehabilitation Practice, 5th ed

Health Education

- Accurate information
- Remaining active
- Reassure the ALBP patients
- Stretching, Strengthening and Flexibility exercises for back pain
- CLBP – useful advice on how to identify any behavior that could delay healing

Ref: Delisa, Physical Medicine and Rehabilitation Practice, 5th ed

Indications for surgery

- Intractable pain
- Nerve root compression with motor deficit
- Not improve with conservative treatment
- Cauda equina syndrome

  Inability to pass urine

  Full bladder with leaking

  Loss of bowel continence

  Loss of feeling or power in both legs

  Loss of feeling in the saddle area

Ref: Delisa, Physical Medicine and Rehabilitation Practice, 5th ed

PREVENTION:

- Once the severity of pain has decreased, a rehabilitation program to strengthen your hip, abdominal and back muscles can help prevent recurrences.
- Good Posture in which neutral spine, not slumped or over-arched.
- Proper lifting and body mechanics.

(Ref:University Health Services (Tang Center) (Physical Therapy))

Proper Sitting, Standing, and Lifting

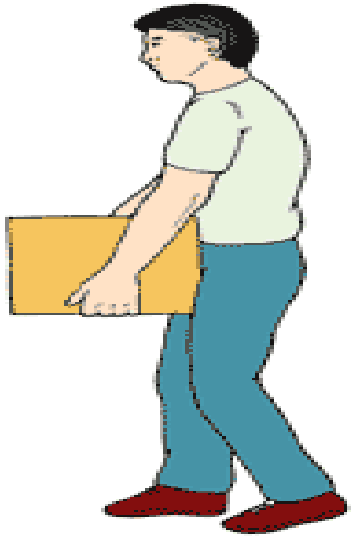
Sitting

Standing

Lifting

1. Bend your knees and squat down to a comfortable level.

2. Lift the object and bring it close to your body.



3. Return to an upright position by pushing up with your legs and buttocks.

Cognitive Behavioral Approach to Physical Exercise (CLBP) according to biopsychosocial approach

- a cognitive behavioral approach based on specific exercises, since it deals with the psychosocial and physical aspects of CLBP
- exercises are a very good and accepted way to reach a psychological recover
- fear-avoidance behavior must be faced through a reassuring and courageous challenge to what is considered the main problem: movement.
- CLBP needs both on the physical (exercises) and psychosocial aspects (counseling).
- It is not recommended when cognitive, behavioral, or motivational factors are present or when the patient does not believe in the possibility of a solution

Ref: Delisa, Physical Medicine and Rehabilitation Practice, 5th ed

OPEN ACCESS

Low-level laser therapy for chronic non-specific low back pain: a meta-analysis of randomised controlled trials

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ABSTRACT

Objective The efficacy of low-level laser treatment (LLLT) for chronic back pain remains controversial due to insufficient trial data. We aimed to conduct an updated review to determine if LLLT (including laser acupuncture) has specific benefits in chronic non-specific low back pain (CNLBP).

Methods Electronic databases were searched for randomised trials using sham controls and blinded assessment examining the intervention of LLLT in adults with CNLBP. Primary outcomes were pain and global assessment of improvement with up to short-term follow-up. Secondary outcomes were disability, range of back movement, and adverse effects. A random effects meta-analysis was conducted. Subgroup analyses were based on laser dose, duration of baseline pain, and whether or not laser therapy used an acupuncture approach.

Results 15 studies were selected involving 1039 participants. At immediate and short-term follow-up there was significant pain reduction of up to WMD (weighted mean difference) −1.40 cm (95% CI −1.91 to −0.88 cm) in favour of laser treatment, occurring in trials using at least 3 Joules (J) per point, with baseline pain <30 months and in non-acupuncture LLLT trials. Global assessment showed a risk ratio of 2.16 (95% CI 1.61 to 2.90) in favour of laser treatment in the same groups only at immediate follow-up.

Conclusions We demonstrated moderate quality of evidence (GRADE) to support a clinically important benefit in LLLT for CNLBP in the short term, which was only seen following higher laser dose interventions and in participants with a shorter duration of back pain. Rigorously blinded trials using appropriate laser dosage would provide greater certainty around this conclusion.

INTRODUCTION

Chronic non-specific low back pain (CNLBP) not attributable to a recognisable, known specific pathology is common, with an estimated prevalence in developed countries of approximately 23%.[1] CNLBP is a major cause of medical expenses, absenteeism, and disability. There are concerns regarding the benefits and potential harms of medication such as paracetamol, non-steroidal anti-inflammatory drugs (NSAIDs), and opioids[2 3] for the treatment of chronic back pain, and non-drug treatments including exercise and multidisciplinary and behavioural treatment have been demonstrated to be of benefit.[4]

Low-level laser therapy (LLLT) is a light source treatment that may act via non-thermal or photochemical reactions in cells. It includes laser acupuncture (LA), which involves focused irradiation at specific points, most commonly traditional acupuncture points, with a low intensity laser.[5] LLLT for pain relief in medicine remains controversial with claims that apparent efficacy is due to the placebo effect.

Multiple mechanisms for LLLT analgesia may exist. There is experimental evidence suggesting that laser irradiation induces peripheral neural blockade, suppresses central synaptic activity, modulates neurotransmitters, reduces muscle spasm and interstitial oedema, and exerts anti-inflammatory effects.[6] The World Association of Laser Therapy (WALT) has published guidelines for LLLT dosage described in Joules (J) per point for arthritis and tendinopathy.[7]

A number of meta-analyses since 2003 have reported pain relief from LLLT in painful musculoskeletal conditions.[8–10] In 2008, a Cochrane systematic review of laser therapy focusing on non-specific low back pain (LBP)[11] included seven trials, considered both acute and chronic pain, did not restrict controls to sham laser, and excluded LA trials. At that point, there

Evidence based management in rehabilitation practice

Low-level laser therapy for chronic non-specific low back pain: a metaanalysis of randomised controlled trials

Summary of main results

15 studies were selected involving 1039 participants. Meta-analysis showed a clinically important pain reduction in laser versus sham lasting up to 12 weeks post-completion of treatment (WMD (weighted mean difference) −1.40 cm, 95% CI −1.91 to −0.88 cm).

Pain reduction occurred in subgroups with nonacupuncture laser interventions, laser dosage ≥3 J per point, and in participants with a shorter duration of baseline pain (30 months).

Global assessment showed a risk ratio of 2.16 (95% CI 1.61 to 2.90) in favour of laser treatment in the same groups only at immediate follow-up.

(Ref: Glazov G, et al. Acupunct Med 2016;0:1–14. doi:10.1136/acupmed-2015-011036)

Evidence based management in rehabilitation practice

Research Paper

PAIN®

Regular physical activity prevents chronic pain by altering resident muscle macrophage phenotype and increasing interleukin-10 in mice

Audrey Leung[a], Nicholas S. Gregory[a,b], Lee-Ann H. Allen[a,c], Kathleen A. Sluka[a,b,d,*]

Abstract

Regular physical activity in healthy individuals prevents development of chronic musculoskeletal pain; however, the mechanisms underlying this exercise-induced analgesia are not well understood. Interleukin-10 (IL-10), an antiinflammatory cytokine that can reduce nociceptor sensitization, increases during regular physical activity. Since macrophages play a major role in cytokine production and are present in muscle tissue, we propose that physical activity alters macrophage phenotype to increase IL-10 and prevent chronic pain. Physical activity was induced by allowing C57BL/6J mice free access to running wheels for 8 weeks and compared to sedentary mice with no running wheels. Using immunohistochemical staining of the gastrocnemius muscle to label regulatory (M2, secretes antiinflammatory cytokines) and classical (M1, secretes proinflammatory cytokines) macrophages, the

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Abstract

Regular physical activity in healthy individuals prevents development of chronic musculoskeletal pain; however, the mechanisms underlying this exercise-induced analgesia are not well understood. Interleukin-10 (IL-10), an antiinflammatory cytokine that can reduce nociceptor sensitization, increases during regular physical activity. Since macrophages play a major role in cytokine production and are present in muscle tissue, we propose that physical activity alters macrophage phenotype to increase IL-10 and prevent chronic pain. Physical activity was induced by allowing C57BL/6J mice free access to running wheels for 8 weeks and compared to sedentary mice with no running wheels. Using immunohistochemical staining of the gastrocnemius muscle to label regulatory (M2, secretes antiinflammatory cytokines) and classical (M1, secretes proinflammatory cytokines) macrophages, the percentage of M2-macrophages increased significantly in physically active mice (68.5% ± 4.6% of total) compared with sedentary mice (45.8% ± 7.1% of total). Repeated acid injections into the muscle enhanced mechanical sensitivity of the muscle and paw in sedentary animals, which does not occur in physically active mice; no sex differences occur in either sedentary or physically active mice. Blockade of IL-10 systemically or locally prevented the analgesia in physically active mice, ie, mice developed hyperalgesia. Conversely, sedentary mice pretreated systemically or locally with IL-10 had reduced hyperalgesia after repeated acid injections. Thus, these results suggest that regular physical activity increases the percentage of regulatory macrophages in muscle and that IL-10 is an essential mediator in the analgesia produced by regular physical activity.

Keywords: Pain, Exercise, Analgesia, Physical activity, IL-10, Cytokine, Antiinflammatory, Macrophage, Immune

Spinal, prespinal, and supraspinal gain control mechanisms and their clinical implications.

Mechanism	Neurons involved	Signaling	Duration	Spatial extent	Clinical manifestation	Implications for treatment
Pronociception						
Peripheral sensitization	Peripheral nociceptors	Inflammatory mediators	Hours to a few days	Strictly confined to site of injury (even part of peripheral RF)	Heat hyperalgesia at injury site, ongoing pain	NSAIDs, …
Wind-up	Spinal WDR neurons	Glutamate, substance P	Seconds	Specific to C-fiber input	Slow temporal summation	?
Central sensitization	Spinal nociceptive neurons and glia	Glutamate, substance P, …	Hours to a few days	Extending beyond injury site (within central RF)	Pinprick hyperalgesia surrounding injury site	Many signaling pathways under study
LTP	Spinal nociceptive neurons and glia, nociceptive afferents	Glutamate, substance P, …, ephrin B	Hours to a few days	Homosynaptic: input specific, heterosynaptic spread to other inputs	Pinprick hyperalgesia at and surrounding site of origin of nociceptive input	Many signaling pathways under study
Descending facilitation	On-cells in RVM, spinal nociceptive neurons, and glia	Serotonin, 5HT3 receptor, …	Hours to a few days	Widespread?	Mechanical hyperalgesia after nerve injury	5HT3 antagonists?
Cortical reorganisation	Primary somatosensory cortex	Glutamate, GABA, …	Minutes to years	Involves neighboring somatotopic representations	Referred sensations, phantom limb pain?	Retraining programs
Antinociception						
Peripheral adaptation and fatigue	Peripheral nociceptors	Intracellular calcium?	Seconds to minutes	Confined to stimulus site	Fatigue of response to mild painful stimuli	Deficient in migraine, cardiac syndrome X and others
Gate control	Spinal nociceptive neurons and tactile afferents	GABA?	Only during conditioning stimulation	Confined to stimulus site	Touch inhibits pain	TENS (high frequency, low intensity)
Long-term depression	Spinal nociceptive neurons, nociceptive afferents	Glutamate, …	Several hours	Confined to stimulus site	Human surrogate models	TENS (low frequency, high intensity)
DNIC	Off-cells in RVM	Norepinephrine, serotonin, …	A few minutes beyond conditioning stimulation	Widespread affects entire body	Pain inhibits pain	Enhanced by SNRI and tricyclic antidepressants, deficient in chronic widespread pain
Cortical pain inhibition through brainstem	Cortex, brainstem, spinal cord	Same as DNIC	?	Predicted to be widespread	Placebo	Cognitive control?
Intracortical pain inhibition	Cortex, thalamus	Glutamate, GABA?	?	Predicted to be according to somatotopic representation	Some psychiatric disorders	Cognitive control?

5HT, serotonin; DNIC, diffuse noxious inhibitory controls; GABA, gamma amino butyric acid; LTP, long-term potentiation; NSAIDs, nonsteroidal antiinflammatory drugs; RF, receptive field; RVM, rostral ventral medulla; TENS, transcutaneous electrical nerve stimulation; WDR, wide dynamic range.

Conclusion

- LBP is one of the most common symptoms and conditions motivating individuals to seek medical consultation.
- The effects of back pain on society are significant, both epidemiologically and economically, and this is likely to only further increase owing to a combination of shifting attitudes and expectations, medical management techniques, and social provision.
- Hence, LBP must always be addressed as a complex disease in which it is mandatory that an accurate diagnosis of pain generators is determined before starting any treatment.
- All the guidelines currently avalaible stress the importance of a multimodal and multidisciplinary approach in order to determine a strategy to solve the problem and not simply alleviate symptomatic pain.
- Finally, a careful follow up is important to adapt our therapeuthic strategies to dynamic clinical manifestations of CLBP.


•THANK YOU