Diagnosis, Classification Management of Chronic low back pain

From a mechanism based bio-psycho-social perspective

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Abbreviations :

PF – pelvic floor
TrA - transversus abdominis
IO - internal oblique
EO - external oblique
RA - rectus abdominis
LM - lumbar multifidus
ES - erector spinae
NZ - neutral zone
IAP - intra-abdominal pressure
Z Jt - zygapophyseal joint
SI Jt - sacro-iliac joint
CLBP - chronic low back pain
LMS- local muscle system
GMS - global muscle system
CNS – central nervous system
M/S – musculo-skeletal

* Low back pain the clinical problem

* Low back pain (LBP) affects over 80% of the population
* LBP frequently develops in adolescence
* many people recover within 4-6 weeks (50-80%)
* 80% chance of a recurrence within the next 12 month
* some go on to develop recurrent and chronic low back pain
* 45 – 60 % with pain and minor disability
* 8-15% with severe disability
* predisposition to recurrence of back pain has been linked to a specific muscle dysfunction (LM) in a sub-group (Hides, 2000).

Back pain and pregnancy

* 50% of women will develop LBP and pelvic pain during pregnancy
* most will recover spontaneously
* some go on to develop disabling CLBP and pelvic pain

Risk factors for developing a CLBP disorder

*** physical and psycho-social (Paris task force (2000))**

- total bed rest for greater than 3 days
- prior history of back pain
- poor muscle fitness (reduced back muscle endurance)
- manual work

- fear avoidance behaviour - people who fear to move due to the perception they will damage themselves
- presence of prior or co-existing psychological disorder (anxiety and depression)
- seeking financial compensation

CLBP

- 45% of people who develop acute LBP will develop CLBP - this is where the pain persists after 3 months
- recurrent episodic low back pain that becomes chronic
- 8 – 10% will develop disabling chronic low back pain

Diagnosis of LBP

Identify red flag pathology – cancer, inflammatory disease, infection

15% specific pathology

- unstable spondylolisthesis
- spinal and foraminal stenosis with nerve pain
- IVD herniation with radicular pain
- lumbar degenerative disc disease with modic changes
- based on radiology
- Note - 40% abnormality exists in those without LBP with MRI
- Even in the presence of pathology classification is necessary to determine whether the patient has an adaptive or mal-adaptive response to the disorder.

85% non-specific low back pain - (no radiological basis to pain condition)
- the challenge is to identify sub-groups (sub-classification)
- diagnosis needs to be based on different criteria than radiology

Proposed classifications of CLBP – contributions / limitations

* patho-anatomical - important in small group - limited to 15% (Nachemson 1999)
* pain source – important and can identify the majority of pain disorders but doesn't identify mechanism driving pain
* pain area - many structures can refer to the same site – differentiates nerve root from somatic referred pain, peripheral pain (localised) from central nervous system pain (generalised / regional)
* exercise intolerance – may be secondary to disorder.
* articular - joint mobility does not correlate to pain. Increased spinal ROM does correlate to positive outcomes in CLBP. Manual therapy treatment in isolation is limited in CLBP disorders.
* Mckenzie – validity in acute / subacute LBP - limited in CLBP.
* muscle dysfunction – present in CLBP - non-homogenous response to pain (highly variable presentation). The cause / effect dilemma.
* motor control – altered motor control is present in CLBP - the cause / effect dilemma.
* movement impairments (Sahrmann) - the cause / effect dilemma
* bio-mechanical model - biomechanical factors contribute to provocation of LBP, considered in isolation is limited and uni-dimensional

* Neurophysiological factors
– pain can be peripherally generated with ongoing peripheral nociceptive input.
  - ongoing peripheral input can lead to spinal cord and central sensitisation
  - pain can also be generated centrally from mid brain and / or forebrain drive
  - the fore brain can generate and greatly influence pain modulation
  - (secondary to cognitive and emotional factors) (Zusman 2002; O'Sullivan 2005)

* Psycho-social model - negative cognitive and emotional factors can drive and amplify pain through the CNS via the forebrain (Zusman 2002; O'Sullivan 2005)

  -Negative forebrain factors – anxiety, fear, negative beliefs, passive coping, over-active coping, hyper-vigilance, lack of awareness of pain mechanisms, -ve emotions, external locus of control
  - Positive forebrain factors - positive coping, distraction, realistic beliefs, awareness of pain mechanisms, +ve beliefs and +ve emotions

All factors that can contribute to a pain disorder must be considered (O'Sullivan, Man Ther. 2005) and the contribution and dominance of the different factors determined.

These may include: pathoanatomical, physical, signs and symptoms, social, environmental, psychological, neuro-physiological, genetic factors.

A bio-psycho-social model for LBP must be considered. On the basis of this a diagnosis and classification is made with consideration for the stage of the disorder.

All pain disorders have a component of organic and non-organic signs

- the aim of the examination is to determine the balance between the two and determine whether one is dominant

Diagnostics must consider :

1. diagnosis (specific vs non-specific)
2. classification (based on the underlying mechanism driving the disorder)
3. stage of disorder (acute, subacute, chronic)

This represents a bio-pyscho-social model. Classification is mechanism based and directs management of the disorder.

(Elvey and O'Sullivan 2004; O'Sullivan 2005)

* Motor control

Motor control describes the way a task is performed - movements and posture – it is not a muscle contraction!
Altered motor control describes the manner by which the movement or posture has changed.

It is described by kinematics and synergies of muscle control not muscles.
No muscles work in isolation.
The brain thinks of performing a task – not contracting a muscle!
Understanding how the different muscles act in synergy to control spinal kinematics helps us understand motor control.

Motor control may be adaptive or maladaptive.

Adaptive motor control
To perform a task maintaining a balance between... **maximal efficiency while protecting** the M/S system and the bodies physiological processes

Mal - adaptive motor control
To perform a task in a manner that results in **compromise** of the M/S system and / or the bodies physiological processes

To distinguish adaptive from mal-adaptive motor patterns in lumbo-pelvic pain disorders…. we need to understand the mechanisms that drive motor control.

* Basic sacro-iliac Jt biomechanics

(Pool-Goudzwaard, Vleeming et al. 1998)
Articular surfaces:
* auricular shaped joint
* development of ridges and grooves as adults (friction joint)
* sacral articular surface – fibro-cartilage
* ilium articular surface - hyaline cartilage

Stabilising ligaments:
* interosseous – primary stabiliser (extremely strong)
* posterior SI
* long dorsal SI – tensioned under counter-nutation
* sacro-tuberous – tensioned under nutation / ilium posterior nutation
* sacro-spinous
* ilio-lumbar – stabilises L5 on ilium

Movements:
* SIJ is a very stiff joint with minimal movement (1-2mm)
* designed for stability and load transference rather mobility
* sacrum – nutation is associated with extension L5/S1
  - counter-nutation is associated with flexion L5/S1
* ilium movement on sacrum – anterior rotation / posterior rotation

Supine – sacrum lies in counter-nutation
Standing - sacrum nutates due to the influence of gravity and muscle forces
Lumbar spine flexion – sacral nutation (self locking)
Lumbar spine extension – sacral nutation (self locking)
Hip flexion – posterior ilium rotation
Hip extension – anterior ilium rotation

Stability mechanisms: (Vleeming / Schneiders)
(a) Form closure - " the stable position of the joint with closely fitting joint surfaces."
* Stable position is nutation of the sacrum relative to the ilium. Nutation winds up most SI Jt ligaments and enhances joint compression.
* Loose packed position is counter-nutation of the sacrum relative to ilium.

(b) Force closure - is necessary when form closure is absent or insufficient. The SI jt is stabilised by compression (on a friction joint) imposed by the action of muscles and fascia.

* Basic lumbar spine biomechanics

(a) Flexion - anterior translation + anterior rotation arrested by Zjts
(b) Extension - posterior translation + posterior rotation arrested by Zjts
(c) Rotation - coupled movement with sidebending / flexion or extension

- the Zjt's glide not gap
- gapping represents rotational loosening of the joint 2nd to pathology (Mcfadden and Taylor, 1989, Spine)

(d) Side bending - coupled motion as with rotation

(e) Compression - The lumbar spine is well designed to sustain compressive load

- vulnerable to buckling, shear stress (translation and rotation) and hypermobility.

NB. extension is the close packed position for the lumbar spine
Sustained compression may be detrimental

(f) Arch Model - stability of an arch is provided by generating compressive forces along its length in such a way that extrinsic forces are not required to maintain equilibrium (Aspden, 1992).

If the thrust line lies outside the arch, then stability is compromised

Rested upright standing – lumbar lordosis with load share between Zjts and IVD

Increasing compressive load – lordosis reduces with load being borne through the vertebral bodies and disc (neutral zone) rather than through the ZJts

*** Neutral Zone (NZ)**

..... a region of high flexibility or laxity around the neutral position of a spinal segment (Panjabi, 1992; Panjabi, 1992)

- safe zone to load
- dependent on muscles for control (maximal co-contraction)
- dependent on proprioceptive feedback from muscle spindles for control
- loss of control of NZ with loss of motor control
- NZ increased with inter-vertebral disc injury and degeneration

*** Elastic Zone**

- towards the end of range increased load is placed on the passive structures
- greater risk of tissue injury in elastic zone
- relaxation of motor system as load shifts to the passive structures

*** Active neutral zones**

.... occur in-vivo with the influence of inter-segmental muscle forces across a motion segment (Panjabi 92)

– simulated segmental muscle forces across a damaged motion segment restored NZ of the segment to within normal limits (Panjabi, Abumi, Duranceau, & Oxland, 1989)
– NZ reduced by 83% with simulated segmental muscle forces
– LM has greatest influence (2/3 stiffness) (Wilke et al, 1995)
–increase of 1-2% maximum voluntary contraction of LM sufficient to restore stability (Cholewicke & McGill, 1996)
– no precise in vivo measurement currently available

*** Spinal stability** is dependent on the interplay between 3 systems (Panjabi 92)
- **passive** - comprising vertebrae, IVD, ZJt's, capsule, ligt.s,
- **active** - influence of muscle forces acting on the segment
- **neural** - comprises the central and peripheral nervous system that controls the active system in providing dynamic stability (Panjabi, 1992; Panjabi, 1992)
- psychological – emotions and psychological factors influence motor control (Vleeming & Lee, 2000)

*** When is the lumbar spine vulnerable to instability / segmental buckling?**
- neutral positions (NZ)
- low loads when the muscle forces are low
- reduced passive system stiffness (10%)
- poor NM control (timing)
(Cholewicke & McGill, 1996; Gardener-Morse, Stokes, & Laible, 1995)

*** Lumbar spine risk of tissue failure**
- high compressive load
- high levels of muscle forces
- sustained loading
- end range spinal loading - elastic zone (Cholewicke & McGill, 1996)

• Neuro-muscular system

*** Muscles involved in creating force closure of pelvis**
* lumbar multifidus - nutates sacrum
* TrA and lower anterior IO - compress joint via nut-cracker effect
* pelvic floor – internally compress SIJ

* gluteus maximus / piriformis - run perpendicular to the joint to compress it
* RA, EO, IO, ES, abdominal wall and quadratus lumborum – increase compression of the SIJ

*** Local muscle system (LMS) -** (Bergmark, 1989)
– all muscles that attach directly to lumbar spine vertebrae
– direct influence on active neutral zone (segmental stability)
- small er torque potential
- some of these muscles arise from and control the pelvis on the hips
- LM, Psoas, Lumbar ES, quandratus lumborum, TrA, IO
- motor control of these muscles greatly influenced by spino-pelvic posture, and range of spinal motion (elastic vs neutral zone)
- slump sitting and thoracic upright sitting inhibits the LMS
- lumbo-pelvic sitting activates the LMS (O'Sullivan, Grahamslaw et al. 2002)

*** Muscles that control IAP**
- pelvic floor, abdominal wall (TrA, IO, EO), diaphragm

- control influenced by spinal loading, stability demand, continence demands, respiration (Thompson, O'Sullivan et al. 2003)

*** Global muscle system (GMS) -** (Bergmark, 1989)
- consists of muscles that transfer load directly between the thoracic cage and the pelvis with no attachment to the lumbar spine
– indirect influence on NZ & segmental control
– large torque producers
- capable of exerting high levels of compressive load to the spine
– easily targeted by general exercise and strength training

*** Muscles that control the hip**
– the control of the hips is critical to lumbo-pelvic posture and activation of the LMS

Low load conditions:
LMS - tonic activation is associated with low levels of IAP and relaxed respiration
- tonic activity observed in pelvic floor,transverse abdominal wall (lower IO and TrA) and LM / PS
GMS - involved in movement initiation, postural alignment and gross stability in association with low levels of IAP and relaxed respiration

- levels of activation are influenced by posture and degree of loading of passive spinal structures

High load conditions:
- LMS work in co-contraction with GMS, in association with high levels of IAP, to act as stabilisers to 'splint' thorax to pelvis and restrict movement
- diaphragm acts as a stabiliser via control of IAP

Sudden loading:
- Feed-forward and feedback motor responses from motor system to stabilise the lumbo-pelvic region.

*** Thoraco-lumbar fascia**
- 3 layers
- middle and posterior wrap around ES muscles
- gives attachment to middle fibres of TrA, IO (posterior fibres), latissimus dorsi, gluteus maximus, lower trapezius, hamstrings
- capable of being tensioned longitudinally, laterally and obliquely

*** Transversus abdominus (TrA)**
- has respiratory and stabilisation function
- no direct joint moment
- high prop. type 1 fibres
- prime influence over IAP acting with the diaphragm and pelvic floor
- separate neural control / independence from other abdominal muscles
- provides lateral and rotary stability via the thoraco-lumbar fascia
- acts in co-contraction with LM, PF and lower IO

- Dysfunction is present in the CLBP population (Hodges & Richardson, 1996; Hodges, Richardson, & Jull, 1996)

*** Internal Oblique**
- function in part resembles TrA
- largest of the abdominal wall muscles
- lower fibres are transverse and continuous with TrA / common nerve supply
- stabilises SI Jt
- lateral fibres run vertically and act as flexors and stabilisers of the thorax
- only the posterior fibres attach to lumbar spine via thoraco-lumbar fascia
-important rotary stability muscle and driver of IAP
- dysfunction in CLBP (Hodges & Richardson, 1996; O'Sullivan, Twomey, & Allison, 1997b)

*** Diaphragm**
- respiratory and stabilisation function
- inspiratory muscle and controls IAP
- acting in isolation swells belly
- acting in co-contraction with TrA and increased IAP it displaces the lower rib cage laterally
- pattern of contraction changes depending on task stability demand
- acts as stabiliser to generate high levels of IAP under high load conditions and splinting of the spine
- dysfunction in CLBP – (O'Sullivan 2002)

*** Pelvic floor muscles** (levator ani and coccygeous)
- forms pelvic diaphragm, provides force closure for SI joint (A Poole 2005)
- controls sacral nutation (stabilises sacrum in co-contraction with LM)
- synergistic with LM, TrA, IO and Diaphragm with increases in IAP and lumbo-pelvic stability
- up-ward lifting motion of the pelvic floor muscles is associated with co-activation of TrA and lower IO and relaxed respiration – low load stabilising strategy
- bracing or splinting strategies – down ward motion of the pelvic floor associated with activation of the pelvic floor muscles and co-activation of the abdominal wall and diaphragm (O'Sullivan 2002)(Avery & O'Sullivan 2001)
- influenced by spinal posture (Thompson & O'Sullivan 2006)

*** Lumbar Multifidus**
– has a segmental innervation
- deep segmental fibres and more superficial multi-segmental fibres
– control of segmental lordosis and nutates sacrum
– provides up to 2/3 control over NZ
- minimal length change with ROM
- works in co-contraction with TrA to control NZ (spinal reflex)
- dysfunction in LBP and CLBP (Hides & Richardson, 1995; Hides, Richardson, & Jull, 1996b)
- may become hyper or hypo active!! Dankaerts et al, Spine (2006)

*** Psoas**
- synergistic action with LM to control lordosis and anterior pelvic rotation on hips
- action on lumbar spine depends on centre of rotation of the motion segment
- segmental stabilisation (anterior)
- controls eccentric control of backward bending
- critical muscle in sitting
- as with LM **may become hyper or hypo active!!**

*** Lumbar iliocostalis and longissimus**
- provides a stabilising function
- controls anterior shear forces in loaded lumbar flexion
- appears not as vulnerable to dysfunction as LM to pain

*** Thoracic component**
- attaches to Lumbar spine via Lumbar aponeurosis
- 70-80% extensor moment to upper Lumbar spine / 50% to low lumbar spine
- creates compressive loading
- tonic activation in upright postures

*** Quadratus Lumborum**
- lateral stabiliser of the lumbo-pelvic region
- may become hyper or hypo active!!
- closely linked to the pelvic / hip region control especially in single leg stand

*** Theories of dynamic stabilisation of lumbar spine:**
- patterns of co-contraction between the LMS and GMS
- compressive pre-loading increases joint stiffness / stability
- arch of spine provides stability
- IAP - increases with increasing stability demands

*** How do the LMS and GMS function under different loading conditions?**
- static postures
- dynamic movement at low load
- assymetrical movements
- rapid or sudden movements
- assymetrical loading
- heavy loading
- gives insight into appropriate rehabilitation (O'Sullivan et al 1998)

*** Relationship between the neural control of the respiratory and stability systems**

* supine – no abdominal tone – belly breathing (diaphragm un-opposed)

* low load – tonic activation of PF, TrA, lower IO, LM, ES, relaxed breathing upper belly, low levels of IAP
* moderate load - tonic activation of PF, TrA, IO, EO, LM, ES, relaxed breathing upper belly and lateral thorax, moderate levels of IAP

* high load – co-contraction of LMS and GMS, high levels of IAP, diaphragm fixed, apical breathing (splinting)

***Factors influencing motor control**

- Posture (gravitational influence)
- End range spinal motion – flexion relaxation
- Respiration
- Stability demand
- Continence demand
- Psycho-social factors (anxiety / depression)
- Pain

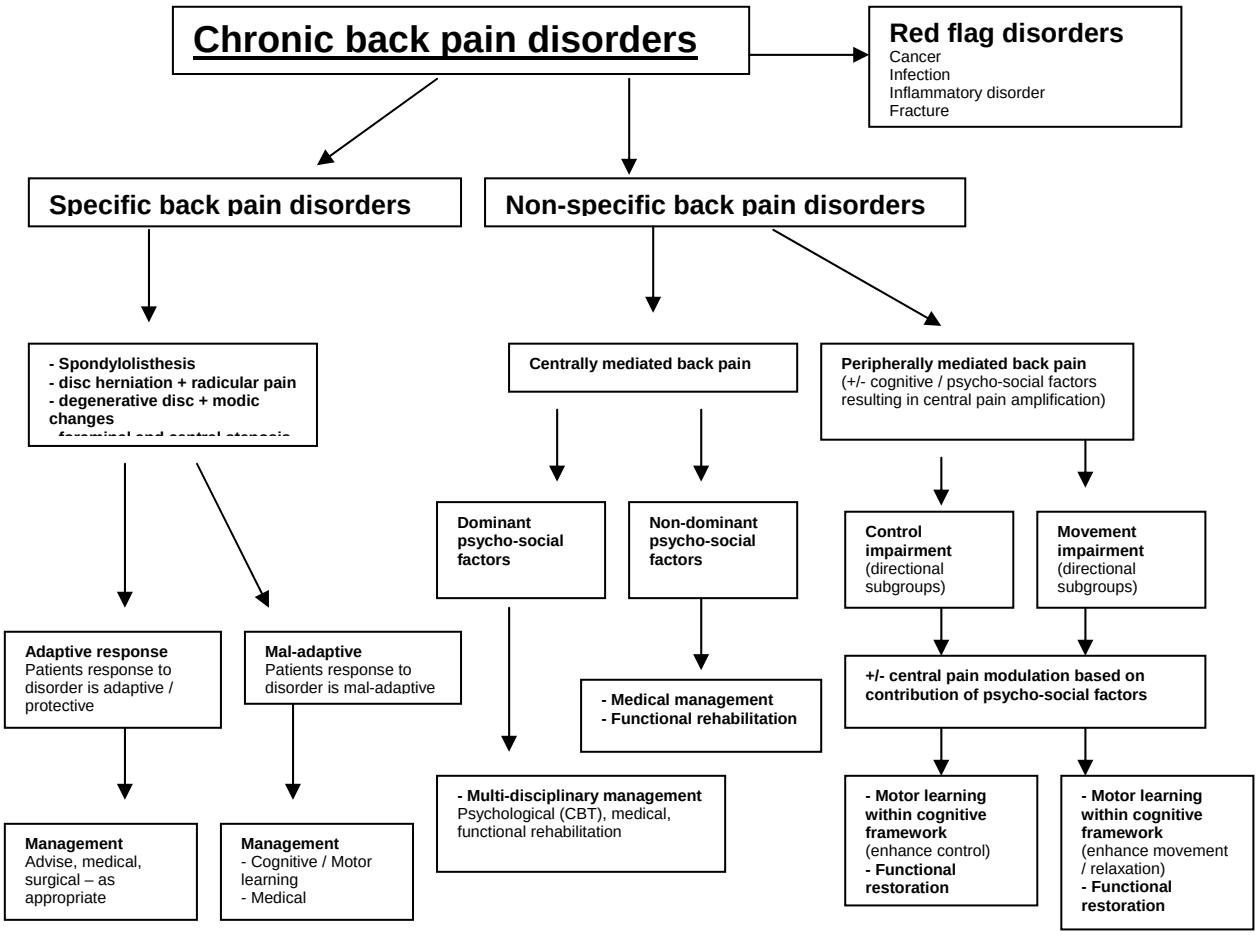
*** Pain influences motor control - nature of the disruption influenced by:**

- Mechanism and nature of injury
- Structures involved - neural, bone, connective tissue
- Neuro-physiological factors
- Hormonal factors
- Activity / postural / lifestyle factors
- Genetic factors
- Neuro-developmental factors

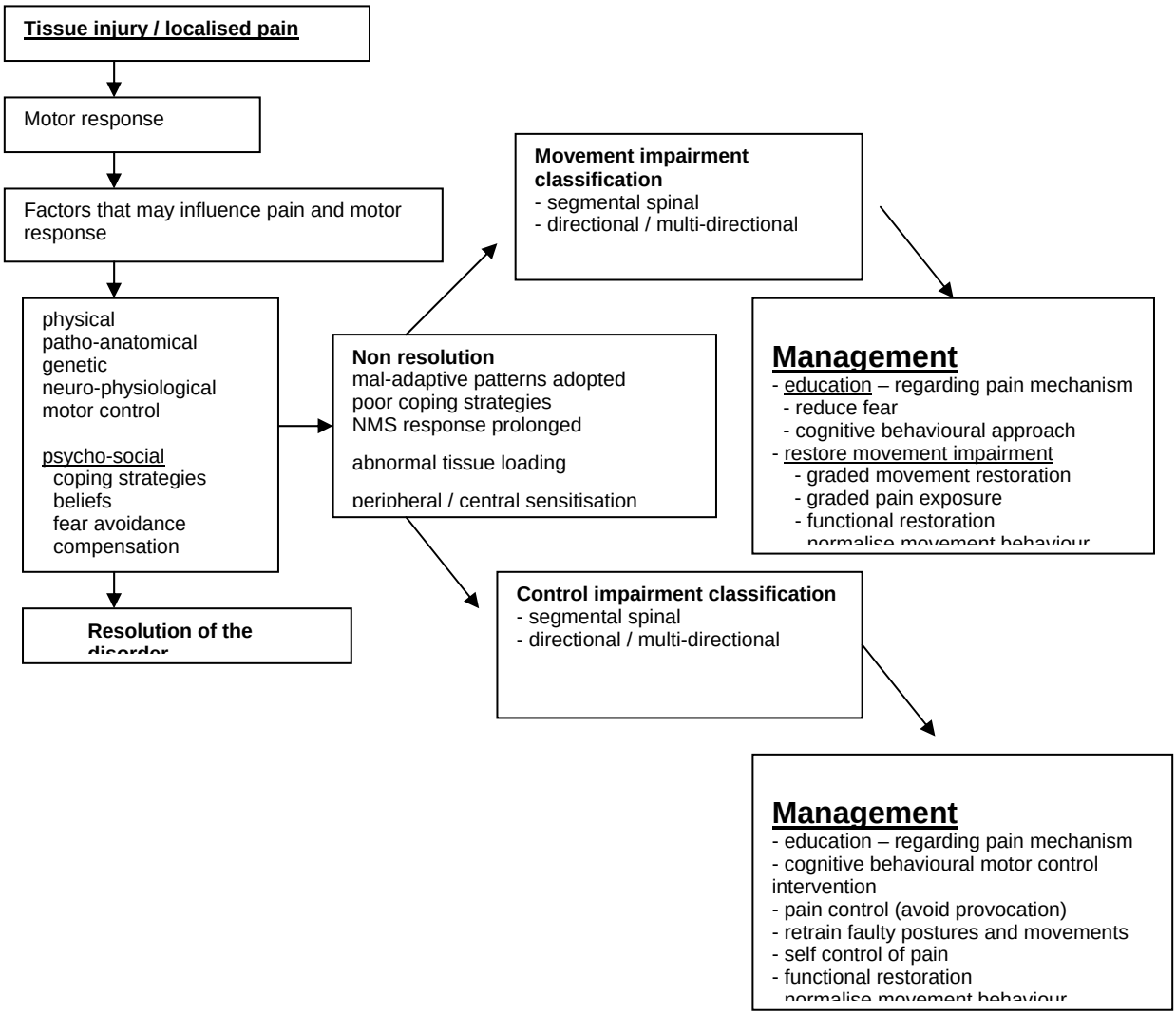
- Psycho-social factors
- Beliefs / advise
- Fear / anxiety
- Compensation

*** CLBP results in changes to the motor program – highly variable**

- strength (variable)
- loss of endurance (selective)
- altered patterns of recruitment between synergists
- altered timing of co-contraction
- segmental changes
- altered neural control


Chronic back pain disorders
Red flag disorders
Cancer
Infection
Inflammatory disorder
Fracture
Specific back pain disorders
Non-specific back pain disorders
- Spondylolisthesis
- disc herniation + radicular pain
- degenerative disc + modic changes
Centrally mediated back pain
Peripherally mediated back pain
(+/- cognitive / psycho-social factors resulting in central pain amplification)
Dominant psycho-social factors
Non-dominant psycho-social factors
Control impairment
(directional subgroups)
Movement impairment
(directional subgroups)
Adaptive response
Patients response to disorder is adaptive / protective
Mal-adaptive
Patients response to disorder is mal-adaptive
- Medical management
- Functional rehabilitation
+/- central pain modulation based on contribution of psycho-social factors
- Multi-disciplinary management
Psychological (CBT), medical, functional rehabilitation
- Motor learning within cognitive framework
(enhance control)
- Functional restoration
- Motor learning within cognitive framework
(enhance movement / relaxation)
- Functional restoration
Management
Advise, medical, surgical – as appropriate
Management
- Cognitive / Motor learning
- Medical

Mal-adaptive CLBP disorders - where 'movement' and 'control' impairments …. Dominate and represent underlying mechanism for pain



Movement impairment pain disorders

– peripheral NS pain associated with a painful loss of normal physiological movement of a spinal segment in one or more directions.

Classification based on:

- Sub-acute or chronic LBP history secondary to traumatic injury or repetitive strain
- History supports that normal movement wasn't restored following acute pain episode either due to early return to normal activity, fear avoidance behaviour, belief that pain is damaging, advise not to provoke pain
- Intermittent localised LBP
- Pain induced went posturing and moving into movement impairment (low inflammatory component)
- Loss of active movement of pain sensitive spinal segment when moving in direction of pain
- Loss of passive movement pain sensitive spinal segment when moving in direction of pain
- Impairment may be due to a loss of compliance of passive spinal structures and / or muscle guarding of antagonist muscles to the direction of pain provocation.
- Fear and muscle guarding associated with provocation of pain
- Relieved with gentle activity, heat, stretching
- Pain with provocation testing in direction of impairment
- Repeated movements active and passive (if gentle, non-threatening and not associated with muscle guarding) reduces pain
- Associated with muscle guarding in direction of pain

- flexion, extension, side bending, rotation, multi-directional

- **Note:** if a patients disorder is associated with advanced degenerative disc disease – then impaired movement may be a normal consequence of morphological change to the spinal segment. Mobilising this segment beyond the new physiological limits is likely to exacerbate the disorder. These disorders may in fact be control impairment disorders – that have 'normal' movement impairment when the morphology is considered. These disorders are commonly mistaken for movement impairment disorders. Mobilisation toward painful range exacerbates the disorder – establishing segmental spinal control within small margin of physiological movement is required.

Flexion	Pain disorder resulting from a movement impairment of the lumbar segment into flexion (associated with a painful loss of segmental flexion).
Lateral Shift (flexion > extension)	Pain disorder resulting from a movement impairment of the lumbar segment in the frontal plane (side bending / rotation). This pattern is also associated with a movement impairment into either flexion or extension.
Extension	Pain disorder resulting from a movement impairment of the lumbar segment into extension (decreased segmental lordosis).
Multi-directional	Pain disorder resulting from a multi-directional movement impairment of the lumbar spine (combinations of above).

Control impairment disorders

- peripheral NS pain disorder resulting from a loss of functional control of a spinal segment in one or more directions.

Classification based on:

- Sub-acute or chronic LBP history secondary to traumatic injury or repetitive strain injury
- Intermittent dominant LBP with minimal peripheral referral
- Clear mechanical basis of disorder: specific postural and functional movements that aggravate and ease symptoms
- Pain induced with posturing and moving in direction of control impairment
- No movement impairment in direction of pain (symptomatic segment)
  - Active movements
  - Passive movements
- Impairments in the control of the motion segment(s) in the provocative movement direction(s)
- Pain reduced with movement or loading away from direction of control impairment (unless multi-directional pattern)
- Relief of symptoms with enhancing motor activation of the symptomatic spinal segment in the provocation direction
- Pain relieved with enhancing motor control of symptomatic segment in direction of provocation
- Repeated movements active and passive in direction of pain provocation increases pain

Flexion	Pain disorder resulting from a loss of motor control of the lumbar segment into flexion (associated loss of segmental lordosis).
Lateral Shift (flexion > extension)	Pain disorder resulting from a loss of motor control of the lumbar segment in the frontal plane (lateral shift pattern). This pattern is also associated with a loss of control into either flexion or extension.
Active Extension	Pain disorder resulting from the lumbar segment being 'actively' held into extension (increased segmental lordosis).
Passive Extension	Pain disorder resulting from a loss of motor control of the lumbar segment into extension. This is associated with a tendency to passively over extend (hinging) at the symptomatic segment of the lumbar spine.
Multi-directional	Pain disorder resulting from a multi-directional loss of control of a lumbar spinal segment (combinations of above).

Three aspects to the motor control examination

(i) Functional movement tests:

These involve a series of functional movement tests analysing the aggravating postures and functional movements reported by the patient
Aims:
- analysis of motor control strategy adopted in primary functional impairments as reported by the patient
- analyse the motor control strategy that the patient has adopted to perform aggravating tasks, and its relationship to the pain disorder.
- to identify whether there is a directional basis to the pain disorder.
- observe avoidance and provocative pain behaviours
- observe for habitual movement habits

Functional movement test (observe pain response)	Flexion	Lateral shift (eg. flexion)	Extension (passive)	Extension (active)	Multi-directional
Standing posture	Flattened lumbar lordosis at 'symptomatic' segment	Flattened lumbar lordosis at 'symptomatic' segment Lateral shift	Thorax posterior to pelvis Increased segmental lordosis at 'symptomatic' segment	Thorax anterior to pelvis Increased segmental lordosis at 'symptomatic' segment	Variable
Stabilising strategy	Thoracic ES Upper abdominal	Asymmetrical Thoracic ES, quadratus lumborum, Upper abdominal wall ipsilateral to shift	Upper abdominal wall (RA, EO, upper IO)	Lumbar ES, Psoas +/- LM	Co-contraction / guarding of global trunk muscles
Spinal segment loading	Anterior	Anterior / lateral	Posterior	Posterior	Variable / alternating
Forward bending in standing	Increased flexion at 'symptomatic' segment Extension thoraco-lumbar spine Increased posterior pelvic rotation (+/- arc of pain)	Increased flexion and lateral deviation of 'symptomatic' segment Deviation accentuated in mid range of movement	-	Delayed or loss of reverse lordosis (delayed or absence of flexion relaxation) Hyper-extension of 'symptomatic' segment Excessive anterior pelvic rotation	Increased flexion at 'symptomatic' segment
Return to neutral from forward bending	Extension thoraco-lumbar spine 'symptomatic' segment remains flexed (+/- arc of pain)	Extension thoraco-lumbar spine 'symptomatic' segment remains flexed and deviated (+/- arc of pain)	Tendency to hinge at 'symptomatic' segment and sway pelvis anteriorly on assuming upright position	Tendency to hyperextend 'symptomatic' segment early on return to upright position (+/- arc of pain)	Variable / alternating
Lumbar:hip ratio	3:1	3:1	-	1:3	3:1
COR	Anterior	Anterior / lateral	-	Posterior	Anterior
Extension in standing	Increased extension above 'symptomatic' segment Reduced extension at 'symptomatic' segment	Increased extension above 'symptomatic' segment with lateral deviation Reduced extension at 'symptomatic' segment	Increased extension at 'symptomatic' segment Reduced extension above 'unstable' segment Excessive pelvic sway	Increased extension at 'symptomatic' segment Anterior pelvic rotation	Increased extension at 'symptomatic' segment
Lumbar:hip ratio	1:3	1:3	3:1	3:0	3:1
COR	Anterior	Anterior / lateral	Posterior	Posterior	Posterior
Single leg stand (gait)	-	Lateral shift of thorax +/- trendelenberg	Anterior pelvic sway +/- trendelenberg without sway Internal hip rotation	Posterior pelvic sway Internal hip rotation	Variable / alternating
Squat	Increased flexion at 'symptomatic' segment Posterior pelvic rotation	As with flexion pattern + Lateral deviation	-	Increased extension of 'symptomatic' segment Anterior pelvic rotation	Variable / alternating
Lumbar:hip ratio	3:1				
Sitting	Flexed lower lumbar spine Posterior pelvic rotation Extended thoraco-lumbar spine	As with flexion + deviation	Slumped posture	Lordotic lumbar posture	Variable / alternating
Sit-Stand	Increased flexion at 'symptomatic' segment Extension thoraco-lumbar spine Increased posterior pelvic rotation (+/- arc of pain)	Increased flexion and lateral deviation of 'symptomatic' segment (+/- arc of pain)	Extension 'symptomatic' segment and excessive anterior pelvic sway on assuming erect position	'symptomatic' segment maintained in hyper-lordosis throughout the movement (+/- arc of pain)	Either flexed or extended
Lumbar:hip ratio	3:1	3:1	-	1:3	Variable / alternating

(ii) Specific movement tests

The application of these tests is based on the findings of the functional movement tests.
These movement tests seek to identify specific movement faults of the lumbo-pelvic region and to determine the relationship between the observed movement fault and the pain disorder (whether the altered motor control is adaptive or maladaptive). For example if pain is reproduced in a specific posture or during a movement during the functional movement test battery, then correction of the posture of movement pattern allows assessment of the relationship between the symptoms and the pain disorder. If the correction of the pattern results in a reduction of the pain, then this supports that the movement disorder is a cause of the pain disorder. If on the other hand the symptoms are exacerbated this may indicate that the motor control deficit is being 'driven' by some other process.

Specific movement tests (establish pain response and motor control flexibility)	Flexion	Lateral shift (eg. flexion)	Extension (passive)	Extension (active)	Multi-directional
Standing posture correction (test for reduction inloading pain)	Anterior rotation of pelvis Increase lower lumbar lordosis Correct sway	As with flexion + correct deviation	Correct sway posture Extend upper lumbar spine Observe low abdominal reflex	Reduce lordosis / posterior pelvic rotation / relax thorax Correct sway	As indicated
Forward bending correction (for movement pain)	Anterior rotation of pelvis Increase lower lumbar lordosis Flex thoraco-lumbar spine	As with flexion + Correct deviation	–	Enhance posterior pelvic rotation and lumbar flexion Enhance return to neutral with gluteal activation	As with flexion
Backward bending correction (for movement pain)	–	Correct deviation	Reduce sway Enhance extension of upper lumbar spine with control of sway and posterior pelvic rotation to minimise hinging	Enhance posterior pelvic rotation via hips	As with 'passive' extension
Single leg stand correction (for loading pain)	Enhance anterior rotation of pelvis Increase lower lumbar lordosis	Correct deviation with focus on keeping head central with weight transference via hip	Correct postural sway aligning thorax over pelvis	Reduce lordosis / posterior pelvic rotation / relax thorax	As indicated
Squat correction (for loading +/- movement pain)	Enhance anterior rotation of pelvis Maintain lower lumbar lordosis	As with flexion + Correct deviation with focus on keeping head central with weight transference via hip	–	Reduce lordosis / posterior pelvic rotation / relax thorax	As indicated
Sitting correction (for loading pain)	Anterior rotation of pelvis Increase lower lumbar lordosis Relax thorax	As with flexion + correct devaiation	–	Reduce lordosis / posterior pelvic rotation / relax thorax	As indicated
Erect and slump sitting (movement test)	Erect sitting associated with thoraco-lumbar extension. 'symptomatic' segment remains in flexion	As with flexion + deviation	Hyper extension 'symptomatic' segment	Erect sit associated with hyper-lordosis Inability to slump sit	Hyper extension lower lumbar spine
Neutral zone re-positioning test place into neutral lordosis – (a) fully slump and ask to return to neutral position	Tendency to reposition into flexion at 'symptomatic' segment	Tendency to reposition into flexion and deviation	Tendency to reposition into extension	Tendency to reposition into extension	Variable
(b) maintain corrected position and bend forward through the hips	Tendency to flex at 'symptomatic' region	Tendency to flex and laterally deviate at 'symptomatic' region	Tendency to extend at 'symptomatic' region	Tendency to hyperextend lumbar spine	Variable
Sit-stand Place spine in neural lordosis – assess ability to hold spinal position during task (for loading and movement pain)	Tendency to flex at 'symptomatic' region	Tendency to flex and laterally deviate at 'symptomatic' region	Tendency to extend at 'symptomatic' region	Tendency to hyperextend lumbar spine at 'symptomatic' segment	Variable
Sit- stand – single leg (movement test)	-	Excessive lateral shift of thorax over the pelvis when loading the affected side	-	-	-
Anterior / posterior pelvic rotation (supine) (movement test)	Inability to anterior rotate pelvis and extend low lumbar spine independent of thorax	As with flexion + asymmetrical pelvic rotation	Inability to extend thoraco-lumbar spine independent of pelvis	Inability to posterior rotate pelvis and flexion lumbar spine independent of hip flexion	
Lumbo-pelvic lateral rotation independent from hip and thorax (movement test)	–	Inability to rotate lumbo-pelvic region independent of thorax and hip - on side of shift	–	–	As with lateral shift
Prone hip extension (movement test)	–	–	Excessive segmental extension Absence of gluteal activation	Excessive lumbar lordosis and trunk rotation Minimal hip extension	Excessive segmental extension
Four point kneeling Anterior / posterior pelvic rotation (movement test)	Inability to anterior rotate pelvis and extend lumbar spine independent of thorax	As with flexion with associated lateral deviation	Inability to extend thoraco-lumbar spine independent of pelvis and 'symptomatic' segment	Inability to posterior rotate pelvis and flexion lumbar spine	Variable
Lateral leg lower (movement test)	-	Inability to maintain lumbo-pelvic position on side of shift Asymmetrical rotation	Tendency to hyper-extend and rotate lower lumbar spine and flex thoraco-lumbar spine	Tendency to hyper-extend and rotate lumbar spine	Excessive rotation and extension of lumbar-pelvic region

(iii) specific muscle testing

Specific muscle testing forms the third part of the neuro-muscular examination.

It should be note these are cognitive and non-functional and therefore lack diagnostic specificity. This seeks to specifically examine the patients ability to consciously isolate the activation of the local muscle system without dominant activation of the global muscle system under low load conditions. More specifically it tests the ability of the patient to co-contract the transversus abdominis, transverse fibres of internal oblique and pelvic floor with segmental multifidus in a neutral lordotic posture while controlling respiration. This aspect of the examination seeks to identify the capacityto initiate a low load stabilising strategy. This form of examination has been described in detail previously [Richardson, 1995 #825; Richardson, 1999 #984]. Fig 4

Specific muscle tests (test local muscle system)	Flexion	Lateral shift (eg. Flexion	Extension (passive)	Extension (active)	Multi-directional
Pelvic floor and transverse abdominal wall (supine, prone, side ly, four point kneel, sitting)	Global abdominal wall contraction with tendency to flex lower lumbar spine and posteriorly rotate pelvis (loss of LM co-contraction)	As with flexion + lateral deviation Assymmetrical weakness	Tendency to flex thorax and upper lumbar spine Dominant upper abdominal wall activation Associated breath holding or apical breathing	Tendency to hyperextend lower lumbar spine Anterior pelvic rotation Global bracing of the abdominal wall Breath holding or apical breathing	Variable
Lumbar multifidus with co-contraction with transverse abdominal wall muscles in neutral lordosis (prone, side ly, four point kneel, sitting)	Inability to activate LM Tendency to flexion lower lumbar spine and posteriorly rotate pelvis	Asymmetrical activation of LM Deficit on opposite side to shift	Inability to activate LM above unstable segment	Inability to co-contract LM with TrA in neutral spine position Tendency to hyper-extend lower lumbar spine with dominant ES +/- LM activity	Inability to co-contract in neutral lordosis
Gluteus maximus (prone)	Bilateral weakness	Unilateral weakness	Bilateral weakness	Inner range weakness	Bilateral weakness
Iliopsoas (hip flexion sitting)	Inner range weakness Tendency to posterior rotate pelvis and flex lower lumbar spine	Unilateral inner range weakness Excessive lateral deviation and rotation on side of shift	Inability to maintain upper lumbar lordosis	Over-active psoas Tendency to hyper-extend lumbar spine and anterior rotate pelvis	Variable
Hip flexor length test (Thomas position)	Long 'short hip flexors'	Long 'short hip flexors	Long 'short hip flexors'	Short hip flexors	Long 'short hip flexors'

Factors suggestive of a loss of force closure of SIJ

- habitual passive postures (slump sit, sway standing, hang off one leg)

- +ve ASLR – PF / low abdominal wall / LM
- excessive lateral pelvic and lower trunk rotation with limb loading
- inability to 'lift' pelvic floor and initiate a low abdominal wall contraction with controlled respiration (non-weight bearing and weight bearing)
- abdominal bracing strategies
- poor gluteal, LM function
- piriformis often hyper-activation (in compensation for loss of SIJ force closure)
- poor loading strategies in weight bearing
- compromised urinary continence

Factors suggestive of excessive force closure of SIJ

- upright rigid postures
-ve ASLR
- increased muscle tone – abdominal wall, QL, LM, gluteal muscles
- inability to 'relax' pelvic floor and abdominal wall
- excessive cognitive contraction of the pelvic floor and abdominal wall
- aggravation with pelvic compression
- relief with stretching, massage, relaxation, passive postures
- compromised urinary continence
- anxiety disorder

Patient interview

Understanding the mechanisms that drive pain

Screen for red flags
Questionnaires: Pain drawing / oswestry disability scale / fear avoidance scale / orebro (coping / stress / anxiety)
Area and nature of pain (central vs peripheral pain generation)
History of disorder (especially early - where did it go wrong?)
Treatment history - success and failure
Functional Impairments (level of disability / directional basis to pain)
Provocative factors (both physical and psycho-social)
Easing factors (both physical and psycho-social)
Nature of impairment - social, work, family, emotional
Coping strategies (active vs passive)
- pacing / distraction / exercise / rest / medication / passive Tx

Patient understanding / beliefs as to cause of pain disorder
Patients beliefs fears re disorder / the future
Beliefs as to whether they are likely to get better
Anxiety / depression
Reflect inconsistencies back to patient in a gentle manner
Correct false beliefs or information
Observe posture, presentation, pain behaviour, movement behaviour, respiration etc

* Management of control impairment disorders

* Evidence to support motor control intervention:

(O'Sullivan, Twomey, & Allison, 1997a and 1997b, O'Sullivan, 2000, O'Sullivan 2001)

* Treatment aim is to change the motor program

Aim is to change movement behaviour that acts as an underlying mechanism for the CLBP disorder
(O'Sullivan, Twomey, & Allison, 1997a)
(Lindgren, Sihvonen, Leino, & Pitkanen, 1993)
(Cresswell, Blake, & Thorstensson, 1994)
This approach does not replace general exercise and rehab programs - it precedes and in some cases is integrated with them.

Motor learning is **not exercise training**.
It combines **cognitive strategies** with **retraining movement behaviour.**
Changing motor control requires changing cognitive behaviour (beliefs, awareness, coping strategies) and movement behaviour (habitual postures and movement patterns).

*** Motor learning principles** (Shumway-Cook and Woollacott 1995)
1. cognitive stage - demands a high level of cognition and awareness
2. associative stage - focus on refining a particular movement pattern
3. autonomous stage - low degree of attention required to perform task correctly so it becomes automatic

Cognitive strategies
- providing insight into the mechanism of the disorder
- teaching active control over pain (active coping)
- reducing fear and anxiety via pain control and enhanced function
- changing negative beliefs
- reduce pathological focus on pain / enhance function focus on pain
- reduce hyper-vigilance
- functional activation

Motor learning
- movement control
- focus on quality
- inhibit unwanted motor activity
- less motor units recruited
- maximal focus / awareness
- focus on learning
- change environment
- precedes conditioning
- daily training (6-12 weeks)
- change movement behaviours
- training effect remains once intervention has ceased

Conditioning programs
- strength
- endurance
- cardio-vascular fitness
- work hardening
- maximal motor units recruited
- less focus on learning
- may be contra-indicated if motor control deficits are present and dominant
- train 3-4 times per week / 6 weeks +
- training effect is lost once intervention has ceased

Motor learning intervention
- classification based
- identify faulty motor control maintaining pain disorder
- retrain faulty movement patterns
- train LMS co-contraction functionally with controlled respiration
- neutral zone control
- functional integration
- focus on quality and control of movement
- pain control
- integrated with general exercise (cardio-vascular exercise)

Identify movement faults and break down into components

* Stages 1. Cognitive stage
– critical stage
– education re pain mechanism
– brain exercise (change the hard wiring)
– identify key faulty movements and postures linked to the pain disorder
– retrain components of these movements and postures
– no set holds or numbers initially- specificity is critical
– to point of fatigue or substitution
– no increase in pain
– feedback and awareness is critical
– 1-4 weeks if very chronic!

*** Early key strategies**
* train lumbo-pelvic movement from hips / independent of thorax
* initiate diaphragm breathing
* functional activation of LMS in weight bearing
NB. watch for substitution

Problems during stage 1?
* Must first train pelvis and Lx in neutral lordotic position independent to thorax

1. Problems breathing (apical breathing)
- relax and align thoracic postures

- slow nose breathing / focus on expiratory phase of respiration
- train upper belly / lateral costal breathing
- begin training supine (inverted) and move to weight bearing

2.Inability to isolate anterior pelvic rotation and lower lumbar extension from thorax (flexion pattern)
- supine crook lying - isolate anterior pelvic rotation from hips (relax thoracic ES)
- train with breathing control
- train with thoracic spine flexion (supine / sitting supported / kneeling)
- progress to →sitting →sitting to standing→squat→flexion →lifting

2. Inability to isolate posterior pelvic rotation and lumbo-sacral flexion from thorax and hip flexors (active extension pattern)
- supine crook lying - isolate posterior pelvic rotation from hips (relax upper belly and hip flexors)
- progress to supine →sitting →standing→step standing→gait etc

3. Lx ES / superficial LM / Psoas hyperactivity (active extension pattern)
- train posterior pelvic control with relaxed thorax and hip flexors (from lower limb)
- inhibit hyperactive muscles - ST massage, stretch, muscle energy techniques
- train pelvic floor / TA in flexed postures initially (4 point kneeling, sitting)
- encourage passive postures

4. Inability to isolate lateral pelvic rotation from thorax and hip adductors (lateral shift pattern)
- supine uni-lateral crook lying - isolate lateral pelvic rotation from hip adductors and thorax , also performed in side lying
- single leg stand – correct load transfer (spino-pelvic / hip control)

5. Dominance of thoracic stabilisation strategy
- train diaphragm breathing
- avoid cognitive muscle training
- soft tissue release of upper abdominal wall
- focus on spinal position rather than muscle activation
- train neutral thoracic postures (relax thorax and shoulder girdle)
- train independent lumbo-pelvic control

* Stage 2. Associative stage

– identify and retrain faulty provocative postures and movement patterns
– train endurance of static postures
– avoid provocative postures and movements
– break down movement components and integrate into functional tasks
– integrate movement and muscle control into functional holding postures and movements
– change habitual postures and movement patterns

– frequent activation
– control pain
– individual specific
– encorporate low level aerobic exercise

Stages of motor learning for different clinical presentations

1. flexion

* train anterior pelvic tilt from hips / independent of thoracic spine extension
    - supine crook lying / sitting / kneeling
* train control of neutral lordosis (with relaxed thorax) with forward trunk loading
    - sitting with forward bend through the hips
    - sit to stand
    - squat
    - gait
* train control of neutral lordosis (co-contraction) with independent thoracic spine flexion / rotation and independent hip control
    - sitting
    - standing
    - squat
* train through range segmental control with co-contraction
    - sitting lumbar flexion
    - squat with lumbar flexion
    - standing flexion
    - functional activities / lifting / add load etc
* mirrors / palpation

2. (a) extension (passive)

* train neural thoraco-lumbar postures
    - sitting, kneeling, standing
    (postural correction critical - correct sway / shift COG anterior)
    - focus on neutral lordosis segmentally with upper lumbar lordosis
    - inhibit dominant upper abdominal wall activity with postural control
* train control of neutral lordosis with forward trunk loading
    - sitting with forward bend through the hips
    - sit to stand
    - squat
* train control of neutral lordosis standing, single leg stand, gait
    - focus on anterior shift of thorax relative to pelvis
* train control of backward bending via the hips and upper Lx with control of lordosis – kneeling, standing
* train functional activities / add load etc

(b) extension (active)

* train posterior pelvic tilt independent of hip flexors and thoracic spine flexion
    - supine crook lying, supine, sitting, standing
* train upright postures with reduced anterior pelvic tilt and lumbar lordosis
* train more passive postures (focus on pelvis)

* train segmental control into forward loading and bending (avoid segmental hyper extension - focus on lower abdominal and gluteal control)
    - sitting lumbar flexion
    - sit to stand (lumbar flexion)
    - squat (avoid segmental extension)
    - forward bending in standing
* train standing posture (shift centre of load posterior, 'lower' buttocks, posterior pelvic tilt isolated to the hips and lower lumbar spine)
* train backward bending
    - train via posterior pelvic rotation (enhance hip extension)
* train functional activities / add load etc

3. lateral (recurrent lateral shift)

* train independent lateral pelvic rotation from hip adductors and thorax
* train balanced thoraco-lumbar alignment
    - sitting with trunk flexion / lateral weight shift
    - sit to stand
    - stand
    - squat
    - particular emphasis is placed on symmetrical movement and even weight bearing through limbs
* train correct spino-pelvic alignment with weight transference
    - single leg stand
    - weight transference with squat
    - stepping, gait

4. multi directional

- train lumbo-pelvic movement (mid range) independent to thorax depending on pattern (as above)
- train neutral lordosis and relaxed thorax
- sitting
- sit to stand
- stand – single leg stand - gait
- squat
- functional tasks

- only once pain control is achieved with all the functional activities through range control is taught

5. impaired force closure

- facilitate funtional control of pelvic floor elevation, transverse abdominal wall, LM , gluteal muscle
- inhibit dominant substituting muscles
- facilitate active postures – target provocative postures and activities
- functional integration

5. excessive force closure

- relaxation of pelvic floor, transverse abdominal wall, LM , gluteal muscle
- diaphragm breathing - relax spinal postures

- avoid cognitive contractions
- target provocative postures and retrain without breath holding and reflex activation
- cardiovascular exercise

- **3 Automatic**

- **Increase the speed, load and complexity of training as required**

– dependent on individual demands
– postural control / general activity levels
– functional training ++
– coordination, speed work, proprioceptive work, limb loading, ball work, gym work, work hardening, functional training, hydrotherapy, cardio-vascular exercise
as indicated and required

*** Facilitation strategies**
– cogntive
– education and awareness
– feedback ++ (visual)
– imagery
– pain feedback
– palpation, mirrors, video, taping

*** Inhibition strategies**
– education and awareness
– feedback ++ (mirror)
– postural and breathing control, avoid cognitive muscle training, palpation, pain control, myofascial inhibitory techniques, low load training
– correct alignment, taping

*** Treatment success dependent upon:**
- some patients may never reach stage 3 for a number of reasons
– accuracy of classification
– cognitive factors (dominant psycho-social factors)
– specificity of exercise training (skill of therapist)
– level of central sensitisation
– multi-directional LSI (more difficult to train)
– compressive loading painful in all positions (poorer prognosis)
– proprioceptive awareness
– compliance
– compensation issues
– developmental factors

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