UNIVERSA MEDICINA

September-December, 2010

Vol.29 - No.3

Biomechanical aspects of nonspecific back pain

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ABSTRACT

Low back pain (LBP) is a common problem in adult life, since despite its benign nature it is commonly associated with incapacity, productivity loss due to sick leave, and correspondingly high costs to the individual worker. Psychosocial and lifestyle factors and work-place exposures have been implicated in the onset of symptoms. Heavy physical work, static work postures, frequent bending and twisting, lifting and postural movements, repetitive work, and whole body vibrations are occupational factors associated with LBP. The usual classification of LBP is related to the duration of the complaints (acute, subacute, and chronic). However, these terms fail to take into account several clinically important aspects of the course of LBP, which is frequently recurrent and thus neither acute nor chronic. More realistically, LBP should be classified as specific and nonspecific. Approximately 90% of LBP cases have no identifiable cause and is designated nonspecific LBP. However, despite its high prevalence, the etiology and nature of nonspecific LBP are not yet well understood. Its pathophysiology remains complex and multifaceted. Multiple anatomic structures and elements of the lumbar spine (e.g. bones, ligaments, tendons, discs, and muscles) are all suspected of playing a role. Many of these components of the lumbar spine have sensory innervations that can generate nociceptive signals in response to tissuedamaging stimuli. Other causes could be neuropathic (e.g. sciatica). Some cases of LBP most likely involve mixed nociceptive and neuropathic etiologies.

Keywords: Nonspesific back pain, biomechanical, sciatica, nociceptive, neuropathic

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Univ Med 2010;29:177-87.

INTRODUCTION

Low back pain (LBP) is defined as a symptom complex consisting of pain and muscle tension or stiffness in the lumbar region localized below the costal margin and above the inferior gluteal folds, with or without pain radiating into the legs (sciatica) (Figure 1).⁽¹⁾ LBP is a frequently occurring disorder, with more than 85% of individuals having ever experienced LBP in her/his lifetime.^(1,2) About two thirds of adults suffer from low back pain at some time. Low back pain is second to upper respiratory problems as a symptom-related reason for visits to a physician.⁽³⁾ The highest prevalence of LBP occurs in the age range of 45-64 years among workers of both genders.⁽⁴⁾ Harrianto



Figure 1. Mannequin with shaded area indicating site of LBP symptoms.⁽¹⁾

According to the National Center for Health Statistics LBP poses important socioeconomic problems to the workers' community, the most frequent being disability of persons below 45 years of age.⁽⁵⁾ Adults with low back pain are often in worse physical and mental health than people who do not have low back pain: 28% of adults with low back pain report limited activity due to a chronic condition, as compared to 10% of adults who do not have low back pain. Also, adults reporting low back pain were three times as likely to be in fair or poor health and more than four times as likely to experience serious psychological distress as people without low back pain.⁽⁵⁾ Other studies state that in the United States LBP is responsible for the annual loss of 149 million working days, with 102 million working days being lost due to occupational LBP. It is estimated that the compensation costs for loss of working time amounts to \$3000 per insurance claim.⁽⁴⁾

Patients with an attack of LBP commonly recover spontaneously after one month at the latest,⁽⁶⁾ while several studies report that 90% of LBP cases recover within 6 weeks, 77% within 7 weeks, and the remaining cases become chronic LBP cases.⁽⁶⁾ However, the study conducted by Van den Hoogen et al. suggests that 70% of patients still suffered from back pain after 4 weeks, 48% after 8 weeks, 35% after 12 weeks, and 10% after 1 year. ⁽⁷⁾ A population-based, prospective cohort study conducted by Cassidy et al. showed that only 1.0% developed intense and 0.4% developed disabling LBP after 12 months follow-up. Resolution occurred in 26.8%, and 40.2% of episodes persisted. Of those that recovered, 28.7% had a recurrence within 6 months, and 82.4% of it was mild LBP.⁽⁸⁾

Only around 5% of acute LBP becomes chronic LBP and results in disability. Pengel et al. reported an improvement in disability of 58% in acute LBP, and found that 82% of patients returned to work within one month.⁽⁹⁾ Cases of LBP with recovery times of less than 6 weeks are designated acute, those with recovery times between 6 weeks and 3 months are called subacute, while chronic cases do not recover within 3 months and not infrequently lead to disability, such that the individual is unfit for work.⁽¹⁰⁾ However, these terms fail to take into account several clinically important aspects of the course of LBP, which is frequently recurrent and thus neither acute nor chronic. More realistically, LBP should be classified as specific and nonspecific.⁽⁷⁾

LBP may occur as a result of excessive physical stress on normal spinal structures, or of normal physical stress on abnormal spinal structures. LBP cases with underlying organic disease, either spinal or nonspinal, generally identifiable by radiological abnormalities of the spine, are classified as specific LBP.⁽²⁾ The study by Koes et al. in the Netherlands found that of all patients with LBP in primary care, 4% was identified with compression fractures, 3% with spondylolisthesis, 0.7% was caused by tumors or tumor metastases, 0.3% was due to ankylosing spondylitis, and 0.01% due to infection, while the remaining 90% had nonspecific LBP (NSLBP).⁽¹¹⁾

Epidemiology

The large variation in methods and diagnostic criteria for LBP, and the wide range of work types and physical activity performed, result in the wide range of figures obtained by epidemiological studies. The point prevalence of LBP in the general population has been reported by several studies as being in the range of 14-28,⁽¹²⁾ while the reported 12-month prevalence is 35%.⁽⁷⁾ The overall prevalence of LBP in workers, as reported by a number of studies, ranged between 15% and 30%,⁽¹²⁾ while the overall 12-month prevalence in first-time workers and first-time sufferers of LBP was 19%.⁽¹³⁾

The study conducted by Omokhodion and Sanya on administrative workers with LBP in Ibadan, Nigeria, found a 12-month prevalence of 38% and a point prevalence of 20%.⁽¹⁴⁾ The 12-month prevalence of LBP in Iranian industrial workers is 21%,⁽¹³⁾ in construction workers 30%⁽¹⁵⁾ in long-distance taxi drivers 51%,⁽¹⁶⁾ and in personal car drivers 53%.⁽¹⁷⁾ Several studies of the nursing profession yielded high prevalence rates, with 12-month prevalences of 30%-76%,⁽¹⁸⁾ while the point prevalences were 15.5-54.7%.^(15,17)

Risk factors

The risk factors of influence are age, gender, education, body mass index, and length of employment.^(3,8,10) Daily habits may also constitute risk factors for LBP, such as smoking, alcohol consumption, sports and daily activities of living.^(8,10) Other factors, such as repetitive movements, vibration, parity and psychosocial stress, may also play a role in the development of LBP.^(4,13) Although a number of studies on various specific industrial groups suggested that the role of the above risk factors was less significant, most studies indicated a strong association between LBP on the one hand and mechanical exposure and poor working posture on the other.^(11,19)

Approximately 80-90% of these disorders were caused by lumbar strain/sprain,⁽⁵⁾ that could be triggered by acute or cumulative trunk injuries.⁽²⁾ Several highly important risk factors for LBP have been reported, such as manual material handling, which is work requiring strenuous physical activity, e.g. lifting, lowering, pushing, pulling, throwing, supporting, moving of loads (40%), or involving postures with frequent bending or stooping at work (20%), and prolonged static sitting or standing (20%).^(13,20) The study by Tousignanti et al. reported that the types of work necessitating frequent exposure to manual material handling activities have a 2.05 times higher risk for LBP than work with frequent exposure to manual material handling activities.⁽²¹⁾ Harkness et al. reported that workers lifting loads of more than >24 lb with two hands, those lifting loads of more than >23lb at or above shoulder level, those pulling loads of \geq 55 lb, and those kneeling or squatting at work for ≥ 15 minutes had twice the risk for LBP than workers who never performed these activities.⁽¹⁾ Other investigators reported that work involving frequent flexing and/or rotation of the trunk had a 2.2x higher risk, while carrying loads of >25 kg had a 1.5x higher risk.⁽²²⁾ Although various physical activities in the workplace have been identified as significant risk factors for LBP prevalence, the quantitative relationship of both intensity and duration of exposure with LBP prevalence is still unclear. One of several studies in the Netherlands reported that there is a doseresponse relationship between lifting and carrying loads of more than 10 kg and LBP risk. Workers who performed lifting and carrying of loads for 7.5-15 minutes daily had a 2.13 times higher risk for LBP than those who performed lifting and carrying of loads for 0 - 7.5 minutes daily, while for workers

who performed lifting and carrying of loads for 15-30 minutes and for more than 30 minutes, the respective risks were only 1.38 and 1.33 times the risk for workers who performed these activities for 0 - 7.5minutes.⁽²³⁾ The study of Chen et al. on longdistance taxi drivers in Taiwan reported that static sitting (driving > 4 hours/day) had a 1.78 fold risk for LBP.⁽¹⁶⁾ However, the same group of investigators in their 1998 - 2006 review reported that although 8 studies found evidence for an association between static sitting at work and development of LBP, one study failed to find such an association, thus Chen et al. concluded that in the latter study static sitting was not the sole risk factor for LBP.

Anatomy of the locomotor apparatus of the trunk

The spinal column is S-shaped, being concave in the thoracic region and convex in the lumbar region. This shape confers elastic properties on the trunk for absorbing downward compression forces when jumping and lifting loads.

Intervertebral disc (IVD)s are situated between two adjacent vertebrae, starting at IVD C2-C3 (between cervical vertebrae C2 and C3) down to IVD L5-S1 (between L5 and S1), giving a total of 23 IVDs. An IVD consists of an outer annular rim, known as the annulus fibrosus, which is a plate of concentrically arranged fibrous connective tissue sheets, with the collagen fibers running obliquely in each sheet, but in different directions in each subsequent sheet, thus forming a strong fibrous ring. The central part of the disc is called nucleus pulposus, consisting of soft fibrocartilage-like tissue with the consistency of foam rubber, thus exerting strong pressures to hold apart the vertebrae immediately above and below. This pressure develops in reaction to the weight of the body and the load being lifted.

At the posterior part of each vertebra there is an opening in apposition with similar

openings of the vertebrae above and below, forming the spinal canal for protection of the spinal cord. The spinal canal also contains openings for the passage of spinal nerve roots to all parts of the body.

The risk factors for LBP are closely associated with the anatomical structure and function of the trunk and with the intensity of physical activity. The role of these risk factors can be explained by biomechanical mechanisms of the locomotor apparatus of the human body. Heavy physical work involving frequent truncal movements requires a larger amount of energy or physical strength, resulting in compressive stress loading on muscles, ligaments, nerves, blood vessels, bones and joints, particularly in the lumbar region. The compressive stress loading in turn gives rise to fatigue and constitutes repetitive microtraumas to these anatomical structures. Muscles, nerve roots, dura mater, posterior longitudinal ligaments, facet joints, joint capsules, periosteum, vertebrae, and the fibers of the outer layers of the annulus fibrosus have a somato-sensory innervation and are therefore sensitive to pain stimuli. Stimulation of the locally distributed pain fibers of the sensory nerves produces the sensation of pain in the lumbar region. Several anatomical structures at these sites, such as the fibers of the inner layers of the annulus fibrosus, nucleus pulposus and ligamentum flavum are resistant to pain stimuli.

Biomechanics of manual material handling and truncal motion

In a person standing upright, the load of the body mass is carried by the five lumbar vertebrae and is particularly concentrated at IVD L5-S1. Even without the person carrying a load, the compression forces exerted by the body mass results in a forward moment of forces, because the center of mass of the body is located slightly anterior to IVD L5-S1. Therefore, maintaining the upright position of the body requires contraction of the erector spinae muscles (sacrospinal, quadratus lumborum, longissimus dorsi, and multifidus muscles) and the flexors of the lower limb (gluteal and hamstring muscles), leading to traction and torsional forces on all spinous processes of the lumbar vertebrae, especially around IVD L5-S1 (Figure 2).⁽²⁴⁾

When an individual is lifting a heavy load, the above mechanism causes IVD L5-S1 to receive larger compression forces that ultimately result in tearing of the annulus fibrosus and allow prolapse of the nucleus pulposus, known as hernia nucleus pulposus (HNP), with pain radiating into the back due to compression of the spinal nerve roots in this



B=load, S=sacrospinal muscles, G=gluteal muscles, H=hamstring muscles.

Figure 2. Compression forces of body mass load and the load supported by all spinous processes of the lumbar vertebrae, especially around intervertebral disc L5-S1.⁽²⁴⁾ location. Although cases of HNP capable of inducing neurogenic pain are rare, mild cases are a relatively frequent occurrence. Slight stretching or tearing of the outer layer of the annulus fibrosus, partial prolapse of the nucleus pulposus compressing the posterior longitudinal ligaments, or degeneration of the intervertebral articular surfaces, may lead to irritation of fine unmyelinated sensory nerve fibers in these locations, thus causing lumbar pain, termed mechanical or discogenic pain, which occasionally may be as severe as neurogenic pain. Ong et al. in their Australian study ⁽²⁵⁾ reported that athletes participating in the Olympic Games who suffered from lumbago actually had degeneraon of the lumbar IVDs, 36% of this abnormal group having severe degeneration. The degeneration became more severe in the caudal direction, and was most frequently found in the L5-S1 segment. IVD narrowing was also found mainly at IVD L5-S1, while HNP was most common in the lower lumbar IVDs. In 58% of L5-S1 IVDs there was displacement of the disc, in most cases causing disc bulging.

Flexion, extension and rotation of the trunk of an individual at work is a cumulative motion of the whole trunk, but actually the greater part of the motion is performed by the lumbar vertebrae. The lumbar IVDs assume the role of synarthroses, with the nucleus pulposus functioning as rotational axis of the vertebrae when performing rotation, flexion, extension, lateral bending, and pulling and pushing movements, with 80-90% of these movements occurring at IVDs L4-L5 and L5-S1.

Rotation of the vertebrae sets up shear stresses that rotate the external portion of the IVD, most of the shear occurring at the outer layers of the annulus fibrosus. Flexion, extension, and lateral bending exert compression stresses and tension in the annulus fibrosus at the sites of apposition. Several force components parallel to the IVD, such as pushing and pulling of loads, produce shear stresses that give rise to slipping in the direction of the forces.

In load lifting the lumbar vertebrae act as levers, therefore contraction of the muscles of the back and buttock also sets up compressive and rotational stresses in the IVDs. The combination of lifting and truncal motion causes larger intradiscal stresses at L5-S1 in comparison with those caused solely by lifting movements. Axial loading of short duration is resisted by tensioning of the annular ligament fibers in the IVDs, but axial loading of longer duration leads to stretching of the annulus fibrosus and induces larger compression stresses in the surface layers of the vertebral body, leading to pain due to injury to these anatomical structures.

Ligaments act as fixation points for joints, thus limiting their movements. In lumbar flexion the greatest tension is found within the interspinous and supraspinous ligaments, followed by the intrascapular ligaments and the ligamentum flavum. In lumbar extension the greatest tension occurs in the anterior longitudinal ligament. Lateral bending produces the greatest tension in the ligaments contralateral to the direction of bending, while rotation results in the capsular ligaments undergoing the greatest tension.⁽¹³⁾ The strains occurring in these ligaments increase the pain induced by the compressive and rotational stresses in the IVDs.

Biomechanics of prolonged static standing and sitting

The IVD has no blood supply and obtains its nutrients by diffusion from the adjacent tissues, for which process motion is a necessary condition, while static body posture, such as prolonged sitting or standing, decreases the nutrient flow.

The lumbar vertebrae are positioned vertically on the sacrum, thus the magnitude of lumbar lordosis depends on the vertical position of the sacrum and is indicated by the magnitude of the lumbosacral angle. The vertical position of the sacrum depends on the rotation of the pelvis. In individuals standing upright, the thorax has a convex anterior curvature; when the lumbosacral angle is $>40^{\circ}$, there is lumbar hyperlordosis, while with increasing relaxation of the upright position the pelvis rotates posteriorly and the lumbosacral angle diminishes. In the sitting position the pelvis rotates posteriorly and the magnitude of the lumbosacral angle decreases to -5° , decreasing with increasing slumping. In the standing position the pelvic flexor muscles and the erector spinae muscles contract, while the pelvic extensors undergo relaxation (stretch) in order to stabilize the pelvis. The result is that the pelvis is tilted forward, thus increasing the lumbosacral angle. In this condition the center of equilibrium of the body tends to move forwards, and lumbar lordosis occurs, so as to maintain the upright position of the trunk and head.

Prolonged contraction of the erector spinae muscles (quadratus lumborum, longissimus dorsi, and multifidus muscles), and the pelvic flexors (gluteal and hamstring muscles), results in weakness or stiffness and occasionally cramps in the pelvic muscles and sustained stretching of the sacrospinal ligament, giving rise to pain.⁽⁷⁾

The superior articular processes of a vertebra form facet joints with the inferior articular processes of the vertebra immediately above. The facet joints make an angle of 45° with the horizontal plane, which causes them to resist intervertebral shear forces, while the IVDs resist compressive stresses. In a person lifting a load, the compression forces cause narrowing of the IVDs and are transmitted to the facet joints, such that these sustain a heavy load, resulting in shear stresses. In the standing position, lumbar hyperlordosis of the vertebrae leads to slackening of the supraspinous ligaments, such that the ligaments cannot prevent forward displacement of the lumbar vertebrae, causing the two adjacent vertebrae to slide against one another due to the greater compression forces on the superior endplates of the IVD. Narrowing of the facet joints leads to friction between their articular processes, facilitating the development of facet joint osteoarthritis, and giving rise to back pain.

In the sitting position the pelvic extensors (iliopsoas) and the abdominal wall muscles contract, while the pelvic flexors (hamstring and gluteal muscles) and the erector spinae undergo relaxation (stretching), such that the pelvis is tilted backwards, resulting in a decreased lumbosacral angle and decreased lumbar lordosis, which may even turn into a kyphosis in order to maintain the upright position of the trunk and head. In this position the potential elasticity of the trunk in absorbing downward forces is lost, the compression forces are directly transmitted downwards, such that the intradiscal stresses at L5-S1 are greater in sitting than in standing.

The large intradiscal stresses at L5-S1 in kyphosis compress the anterior portion of the IVD, while the posterior portion stretches, such that the annulus fibrosus (degenerated due to poor nutrition) prolapses posteriorly, compressing the posterior longitudinal ligaments and giving rise to pain.

Correlation of organic abnormalities with clinical manifestations of nonspecific back pain

On the basis of the organic abnormalities underlying NSLBP and for clinical purposes, several diagnostic entities are recognized, such as low back strain, piriformis syndrome, iliolumbar syndrome, discogenic pain, facet joint syndrome and sacroiliac syndrome.

Myofascial pain syndrome/Low back strain

Muscles that are exposed to prolonged physical stress, because of considerably long periods of contraction and relaxation in a static position, extremely rapid repetitive movements, or forced vigorous contractions, may undergo spasm and shortening, thus increasing their tone and tension, such that they may contract of their own accord without neural stimulation. These contractions may be prolonged and demonstrable on EMG. In these conditions the muscle fibers undergo injury and inflammation, with a compromized blood supply. The injured muscles may regenerate if the damage is slight and the physical stress is discontinued, but with severe damage or persistent physical stress, the muscles degenerate and are replaced by fibrotic tissue. These fibrotic sites may become trigger points (TrPs), palpable as tender nodules 3-6 mm in diameter. The pain arising from stimulation of these TrPs is called myofascial pain, and may be due to direct or indirect trauma, excessive or repetitive physical activity, or faulty posture.⁽¹⁾ The condition is called myofascial pain syndrome/chronic thoracolumbar syndrome (low back strain), which appears as sudden pain or as mild back pain that gradually worsens. The pain usually persists on one side of the back, and results in marked tenderness in the gluteal region and/or paralumbal regions, with a positive Patrick test.

Piriformis syndrome

The piriformis muscle runs from the facies pevina of the sacrum to the greater trochanter of the femur, dividing the greater sciatic foramen into the suprapiriformis and infrapiriformis foramina, through the latter of which the sciatic nerve passes from the pelvic cavity into the leg.

In blunt trauma to the sacroiliac or gluteal region, e.g. in a person falling on the buttocks, there is hemorrhage and hematoma in or around the piriformis muscle, followed by spasm and stiffness of this muscle, thus irritating the sciatic nerve and resulting in the piriformis syndrome.⁽²⁶⁾

According to several studies, the incidence varies between 0.33% and 6%.⁽²⁷⁾ This syndrome may also result from arthritis and periarthritis of the sacroiliac joint, from excessive use of the pelvic rotators (e.g. in individuals with severe physical activity, soccer players, or athletes), and from repetitive

injury to the sciatic nerve, such as occurs in workers with prolonged sitting postures.

The resulting pain, muscle spasm, or stiffness around the sacroiliac joint, greater trochanter, or iliopsoas muscle, is occasionally accompanied by pain radiating into the leg, leading to difficulty in walking. The pain may also appear on stooping or lifting. On physical examination a sausage-shaped mass and tenderness are found around the injured piriformis muscle, with positive LRS test and occasionally fibrosis of the gluteal muscle. The lesions are usually unilateral, but may be bilateral; paresthesia and numbness are rarely found. The syndrome is difficult to differentiate from ischialgia due to HNP.

Iliolumbar syndrome

The iliolumbar ligament is a strong ligament connecting the transverse process of L5 with the internal lip of the iliac crest. Injury or inflammation of this ligament results in acute LBP, subsequent to execution of an inapproriate movement or to blunt trauma. This syndrome is the most frequent cause of LBP, with a prevalence of up to 43%.⁽²⁸⁾ The iliolumbar syndrome is also frequently called iliolumbar ligament syndrome, iliac crest pain syndrome, and multifidus triangle syndrome. The pain is felt on the medial side of the iliac crest, with the patient generally being able to indicate the precise location of the pain. The pain may be induced or increased by lateral flexion of the trunk or by the stair-step test. The syndrome commonly occurs in individuals whose occupation requires prolonged standing or sitting. There is marked tenderness at the site of pain, with positive LRS and Patrick tests.

Discogenic pain

Repetitive compressive and rotational stresses on the IVDs, particularly at L5-S1, lead to in degeneration of the annulus fibrosus, with solitary or multiple tears. The tears may be marginal, tangential, or radial, but fortunately may undergo autorepair. In the nucleus pulposus the degenerative process is manifested by dehydration and fragmentation of the nucleus into sequestra, with its normally firm consistency (resembling that of lobster meat) turning into a soft mass mixed with gas bubbles. Tears of the annulus fibrosus, particularly radial tears, facilitate prolapse of the sequestra of the nucleus pulposus. The posterolateral angle is the thinnest and weakest portion of the annulus fibrosus, such that IVD bulges due to prolapse of the nucleus pulposus compresses the posterior longitudinal ligament, stimulating the fine unmyelinated sensory nerve fibers in these locations, and inducing back pain known as mechanical or discogenic pain. Kuslich et al. conducted a study on 193 candidates for HNP or spinal stenosis surgery. The investigators were able to elicit the pain by means of blunt surgical instruments or lowvoltage electric current in 30% of those stimulated at the paracentral area of the annulus fibrosus and in 15% of those stimulated at the central part.⁽²⁸⁾ Mechanical or discogenic pain may ultimately cause tears in the annulus fibrosus, manifesting as HNP, due to compression of the spinal nerve roots.

Sacroiliac syndrome

Compression forces from the spinal column are transmitted to the pelvic (innominate) bones by way of the sacroiliac joint. Although apparently locking the pelvic girdle for transmission of downward loads to the femoral head, actually the sacrum tends to be displaced forwards and out of the pelvic girdle, due to its trapezoid shape that is narrower at the rear. In the sacroiliac syndrome the forward shift of the sacrum results in stretching of the ligaments inserting into the sacrouberous ligaments, giving rise to sudden pain radiating from the sacroiliac joint to the back and the posterior thigh(Figure 3).⁽²⁹⁾

In contrast to discogenic pain, the pain is never centrally located, but commonly to one



Figure 3. The sacrum tends to be pushed forwards when lifting loads.⁽²⁹⁾ A =sacrum, b =ilium, c =pubis, d =position of intervertebral disc between S1–L5

side. There is pain upon standing on one leg, limited movement at the sacroiliac joint, and tenderness over the joint, with positive Patrick test.

Facet joint syndrome

Prolonged and continuously applied compression forces on the IVD result in IVD degeneration, described as narrowing of the IVD. The biomechanical axis of the IVD promotes the transmission of compression forces posteriorly to the facet joints. Therefore the facet joints receive a larger distributed load from compression forces, in comparison to other parts of the vertebra. Prolonged injury to the articular surface results in inflammation of the joint followed by formation of osteophytes to compensate for the increased biomechanical stress in order to stabilize the joint. Osteoarthritis of the facet joints manifests itself as LBP and rigidity of the joints, accompanied by secondary muscle spasm.⁽¹¹⁾

Similar to the case of facet joints, compressive loading on the sacroiliac joint also

allows the formation of osteophytes and the development of osteoarthritis in the joint. Parmar et al. found one case of an osteophytic bridge across the anterosuperior border of the left sacroiliac joint, ⁽³⁰⁾ while Kumar et al. found 4 cases of osteophytes at the inferior border of the sacroiliac joint, where all cases complained of back pain and recovered after surgery.⁽³¹⁾

Prevention

Health education and training for applying biomechanical principles of the body and adequate medical care of the back need to be instituted as primary and secondary preventive measures.

Analysis of daily physical activities needs to be carried out to study the relationships between the disease symptoms and influencing factors, such as occupation, hobbies, and sports, by evaluation of postures in standing, sitting, driving, load lifting, or performing other physical activities.

Work-related activities should be planned to minimize the amount of work to be

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performed requiring prolonged sitting or standing, monotonous motions, and repeated stooping and truncal rotation.

Particularly for the working community, in the future jobs should be redesigned, by minimizing the frequency and duration of work postures capable of inducing physical stress, eliminating monotonous jobs, regulating rest periods to regain use of the muscles, joints and ligaments. There is also a need for training to improve work techniques. Furthermore, there should be a stage by stage evaluation of performance.

Clearly worded instructions for preventing LBP symptoms should be mandatory reading material for all manual handling workers. Strength testing should be carried out particularly when hiring new workers for manual handling jobs. Every LBP event experienced by the workers should be reported, to allow early medical evaluation in order to reduce the occurrence of more severe and irreversible health problems. Adequate evaluation and counseling performed on workers with previous LBP events is urgently required.

CONCLUSIONS

In general NSLBP is due to heavy work in connection with manual material handling, such as lifting, lowering, pushing, and pulling of heavy loads, and is also associated with frequent or prolonged bending of the body, stooping, sitting, and standing, or other unnatural postures. Although the organic abnormalities of NSLBP are difficult to clarify, it is firmly believed that biomechanical stress of the lumbar vertebrae due to changes in the center of gravity of the body, followed by compensatory changes in posture, will give rise to pain. Tension and strains or injury to muscles, ligaments, vertebral and pelvic articular surfaces, spinal cord, and spinal nerve roots, are some of the causes of the symptoms. On the basis of the pathophysiology and the

clinical implications, NSLBP may be categorized into a number of diagnostic entities.

Analysis of daily physical activities and training should be carried out, to institute positive behavioral changes in work-related postures, presumably leading to reduced rise of recurrent LBP.

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