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Excerpt from upcoming APTA publication…

MSD PATHOPHYSIOLOGY

Today's workplace MSD problems are typically the result of repetitive tasks… performed in static postures. These create a scope of structural and biochemical effects locally at the primary affected musculoskeletal structures, as well as systemically more widespread effects due to neurophysiological responses and systemic inflammatory biochemical effects. Several of these pathophysiological processes can interact to spread symptoms widely beyond the original localized pain complaint, such as is often seen in the clinic with injured workers complaining of spreading pain problems throughout one or even both upper extremities.

CELLULAR AND SYSTEMIC EFFECTS

The pathophysiology of MSD can be extensive. The ongoing repetitive or sustained tissue loading from muscle contraction, tendon tension, joint compression, and neurovascular stress can create chemical and structural responses within the affected tissues. These physical demands may reduce perfusion of the working tissues. Reduced perfusion encourages anaerobic metabolism and its chemical consequences, along with the structural damage to tissue fibers and cells being loading. The effects are a **loss of nutrient pathway**: ischemia, anaerobic metabolism, increasing damage and reducing healing.

Collagen microstructure is disrupted or altered. Cells are damaged. Matrix hydration is reduced. Several inflammatory events begin to unfold. The initial acute inflammation seen in tendinitis quickly evolves into non-inflammatory degenerative processes such as fibrosis, as seen in tendinosis. Nociception leads to a range of neuroplastic changes within the peripheral and central nervous system resulting in increased pain sensitivity. Our prevention interventions seek to reverse these pathophysiologic processes at the cellular level.

Load intensity (weight), load duration, and task repetition can stress tissues beyond their tolerance and ability to adapt to these work demands. Damage occurs from mechanical overload that breaks fibers, and from hypoxia-ischemia damage from sustained loads blocking tissue perfusion. Muscles undergoing long periods of sustained contraction during static posture may experience hypoxia, ischemic damage, change in fiber type, inflammation, and scarring. Static loading hypoxia may force anaerobic metabolism and its accompanying biochemical stresses. Damage to sarcolemmae and sacromeres allows leakage of intracellular components in the extracellular matrix. This stimulates proliferation of collagen around the myofibers and leads to fibrosis.

Tendons experience similar collagen dysplasia, fibroblast and macrophage proliferation, fibrosis, and release of proinflammatory proteins. Some of these chemical releases can stimulate a systemic inflammatory response that may contribute to more widespread symptoms, sometimes in all extremities. Cell damage releases cytokines that can lead to proliferation of macrophages which can produce further tissue damage… thus creating a vicious cycle of damage leading to inflammation leading to more damage, particularly as ongoing daily work demands continue to load these tissues as they struggle to repair and adapt. This is a cellular model of workplace MSD.

The effects of pain often create a neuroplastic changes that increase levels of nociceptor sensitivity throughout the peripheral nervous systems and, eventually, at the spinal cord and cortex. Affected tissues become sensitized. Pain sensitivity is upregulated. Sensitivity is increased at the primary injury site, as well as within and along the peripheral nerve fibers fibers and at the dorsal horn of spinal cord itself. Cord changes cause non-pain stimuli to stimulate pain tracts. This leads to hyperalgesia and allodynia. MSD symptoms become chronic and progressively more severe as a result of these neuroplastic changes in response to sustained tissue loading and pain. Primate brain research also suggests changes in the brain in the presence of repetitive tasks at the arm and hand. These changes include de-differentiation of cortex areas representing the hand and arm, distorting motor control.

Our interventions seek to reverse these various pathophysiological processes by reducing workload forces, reducing load duration, reducing task repetition rates, dispersing loads over a wider variety of tissues, and by enhancing tissue perfusion through posture control, work task rotation, and even stretch breaks.

MACRO-TRAUMA VS. MICRO-TRAUMA

Decades ago, work typically required heavy physical efforts performed in a variety of movement patterns using many large muscle groups and a variety of work postures. Injuries from that era were mostly overexertion strains and sprains caused by heavy efforts, as well as from macro-trauma injuries on jobs that were more hazardous than those seen today. Today's jobs are safer from macro-trauma accidents but are more automated, specialized, and narrowly focused. Many work tasks today are performed repetitively by smaller sets of small muscles working with minimal variety of movement patterns performed in static confined postures for prolonged periods.

Today's MSD risk jobs may be described as highly focused lightweight repetitive tasks performed in sustained postures. This creates prolonged periods of tissue compression, tension, and loading. These periods of sustained loading compromise perfusion and nutrient pathway, leading to anaerobic metabolism with accumulating chemical byproducts. This can lead to fatigue, pain, and inflammation. Fatigue and pain then degrades posture and movement efficiency to further stress working tissues. Damage accumulates slowly. This cumulative micro-trauma eventually evolves into today's clinical overuse MSD.

Micro-trauma accumulates gradually over long periods until it reaches symptomatic levels, late in the disease process after damage has become extensive. This is why early prevention efforts are so important, rather than waiting to react to an established clinical MSD in later, more difficult stages of the disease process. This is what we seek to accomplish with proactive primary prevention. Primary prevention can be extremely cost-effective.

Upper Extremity MSD Risks

The typical collection of upper extremity MSD includes rotator cuff and related shoulder dysfunctions, lateral and medial epicondylalgia at the elbow, tendinitis-tendinosis at the wrist or thumb, plus a variety of neurovascular entrapment disorders such as carpal tunnel syndrome, pronator syndrome, cubital tunnel syndrome, radial tunnel syndrome, and thoracic outlet compression. Many workers suffering MSD problems present with multiple dysfunctions by the time they reach the clinic.

Neurovascular "double-crush" phenomena are common, such as carpal tunnel syndrome accompanied by pronator syndrome and/or thoracic outlet compression. Neurovascular entrapments often accompany tendinitis-tendinosis problems such as radial tunnel compression accompanying lateral epicondylalgia, cubital tunnel compression accompanying medial epicondylalgia or, in the lower extremity, tarsal tunnel compression accompanying plantar fasciitis and/or piriformis-sciatic entrapments. MSD problems seldom exist in isolation.

Rotator cuff structures are challenged by reaching tasks that are vertically high, horizontally far, heavily loaded, repetitive, or sustained. Lateral epicondylalgia is at risk with heavy, repeated or prolonged lightweight loading at extensor carpi radialis working across the wrist, as with repetitive lifting tasks or with prolonged computer mouse use. Loading or tension across the pronator teres may risk the medial epicondylalgia or entrap nearby neural structures. This often occurs when loading the hand or wrist with the forearm postured in a fully supinated position, drawing taut the pronator teres (such as with a restaurant waitress carrying a tray loaded with dishes).

Carpal tunnel neurovascular compression is at risk from grip or pinch that is repeated, heavy, or sustained, especially if grip diameters are extreme (small or large) or to surfaces that are slippery or accompanied by vibration. Wrist flexion or deviation that is extreme, sustained, repetitive, or loaded during grip also risks neurovascular compression at the carpal tunnel. Thumb extensor tension or loading such as when working thumb switches or with pinch during ulnar deviation. This risks deQuervain's tenosynovitis or irritate nearby CMC-saddle joint.

Many of these upper extremity problems are worsened by a degree of thoracic outlet-inlet compression, which is commonly seen with forward head posture or inadequate proximal motor control during upper extremity tasks. This proximal neurovascular entrapment often contributes to distal upper extremity MSD symptoms. Proximal posture risk factors, especially forward head posture, are often overlooked as significant contributors to distal upper extremity symptoms. Seating ergonomics and the workers' sitting posture habits become critical in this context, with forward head posture virtually epidemic among workers who sit.

Tendinitis, tendinosis, and various sites of neurovascular entrapment can become widespread as multiple MSD problems emerge and become chronic. By the time an MSD problem is officially reported, one dysfunction has commonly led to another, often complicated by nociceptor upregulation and systemic inflammatory responses. Musculoskeletal tissue loading responses often stress peripheral nerves, risking entrapments and upregulated

nociception. Studies imply that neuropathodynamics problems are an underlying problem for many, if not most workers suffering MSD.

Lower Back MSD Risks

Common lower back MSD diagnoses include intervertebral disc degeneration or derangement, zygoapophyseal joint sprain or degeneration, ligament injury, muscle strain, sacro-iliac dysfunction, and hip dysfunctions. Lower back problems are the most common MSD overall, especially where workers endure bending, twisting, sitting, overhead work, and materials handling (lifting, pushing, pulling, or carrying). Flexion is usually the predominant work demand to the lower back, risking flexion-related dysfunctions and derangements such as intervertebral disc herniated nucleus pulposus. Work that requires spinal flexion that is repeated, sustained, extreme, or loaded would stress intervertebral discs and posterior ligament structures. Lumbar flexion loading would also include prolonged sitting.

Excessive extension or twisting would stress zygoapophyseal or sacroiliac joint structures. Standing, overhead tasks, or work ladder work (such as performed by factory maintenance workers) often create loading stresses that risk zygoapophyseal joint irritation symptoms. Asymmetrical lower extremity weightbearing, such as footswitch operation while standing, risks hip region and lumbo-pelvic problems such as piriformis or sacroiliac dysfunctions.

Other work stresses would include whole body vibration, inadequate seating design, and the spectrum of material handling variables such as lifting, pushing, pulling, or carrying. These various work stresses all contribute to accelerated aging and degenerative processes. Degenerative changes to one structure can increase degenerative stresses to other structures. Disc degeneration or herniation alter axes of motion and shift increased weightbearing forces to the zygoapophgyseal joints. Reduced disc space can also alter ligament tension and support and may contribute to spinal segment instability.

Lifting is the most obvious workplace risk factor for lower back MSD. While employers may focus on the weight lifted, they may overlook other factors. The many lower back materials handling risk variables go beyond the weight lifted to include frequency lifted, from how low, to how high, how far horizontally from worker's center of gravity, duration of day spent on lifting task, and how cumbersome is handling the load (coupling).

These are the external requirements of the job; the ergonomics demands. But we must also consider worker behaviors such as lifting technique (body mechanics) in the spectrum of material handling MSD risks. It is not merely "what" they lift; it is also "how" they lift. Individual worker flexibility, strength, stamina, skills, core stabilization and motor control also affect vulnerability to injury.

Training and Education: Back School and MSD School

Some risks are presented by ergonomics of the job, workstation, tools, or procedures. Other risks are presented by worker behaviors, posture habits, and individual flexibility deficits. Ergonomics design corrections must, therefore, be accompanied by effective worker education on ergonomics skills and personal behavior risk factor controls. Training and education facilitates actual implementation of all the proposed prevention interventions. Workplace training and education is the core of prevention. OSHA ergonomics program recommendations strongly encourage training and education as vital to any ergonomics program. This is not unlike the critical role of patient education in patient care and recovery of function.

This calls for the physical therapist to provide a structured education program once workplace evaluation has been completed. One example of this is the workplace "Back School." For decades, workplace back schools have been a traditional back injury prevention program provided to industry by physical therapists. This was initially pioneered by David Apts, PT, and Keith Blankenship, PT, of the American Back School in the late 1970's and later expanded upon by others. Studies of preventive back school effectiveness show varied outcomes based on what tactics were being taught (e.g., pelvic tilt method of lifting versus maintaining lordosis when lifting) and how it was taught (live presentations by experts versus film strip presentation by safety managers) and on instruction content (address a wide scope of risk factors versus isolated attention to lifting only).

The physical therapist can expand upon the "Back School" model to assemble a wider-scope "MSD School" program of workplace education encompassing risk factor education addressing both low back and upper extremity MSD. An MSD School teaches workers and managers the scope of their MSD risk factors, pathomechanics, and

prevention interventions. The physical therapist presents the findings of the workplace evaluation, explains MSD pathomechanics, describes the recommendations for ergonomics improvements, and other prevention tactics. This class teaches the workers posture fatigue control, body mechanics techniques, and self-care of the aging working body. The MSD School becomes the primary vehicle by which prevention and ergonomics interventions are actually inserted into the workplace knowledge base, culture, and processes. It becomes the vehicle by which the MSD prevention interventions are actually implemented.

WORKPLACE STRETCHING

Not all MSD risks come from ergonomics deficits. And not all ergonomics deficits can be corrected. How may workers be protected in those situations? One approach is to reduce exposure time through job task rotations and expanded work task variety. Another tactic may be brief but frequent pauses to stretch musculotendinous structures that are under ongoing loading, seeking to restore tissue perfusion. Workplace stretching to prevent fatigue, pain, and MSD is controversial. Published studies vary widely in their conclusions, study quality, and potential bias. "Exercise" and "stretching" are generic non-specific terms. We need to assess the effects of workplace stretching based on specific exercise selection (which muscle groups) and implementation (how, when, how often, how instructed, and how enforced) to identify what workplace stretching strategies may or may not be effective in reducing MSD.

An MSD School can introduce job-specific, brief but frequent workplace "micro-stretches" to enhance perfusion to working tissues through the workday. Micro-stretching typically calls for a concise and targeted set of ten-second stretches performed every two hours to at-risk working tissues. A typical plan may include axial extension and scaleni stretches at the neck, Codman's pendulum at the shoulder where there may be risks to the rotator cuff, forearm flexor and pronator group stretch, forearm dorsal extensor group stretch, standing back-bends, seated hamstring stretch, and calf stretch. Modifications may be made to this list to target other specific risks.

The objective of micro-stretching is to allow brief but frequent interruptions in static posture and repetitive tasks to allow the working tissues to relax and enjoy a period of enhanced perfusion. Such brief and gentle stretches likely do not actually lengthen tight tissues but, rather, seek an inhibitory receptor response to encourage relaxation of the targeted musculotendinous group, to restore perfusion. Such a brief micro-stretch program like this seldom requires more that two minutes, minimizing management concerns over production. Some companies have even seen improvements in productivity after implementing a micro-stretching program, ostensibly due to reduced worker fatigue as a result of frequent stretch breaks. Micro-stretching is an especially important consideration where jobs have a high risk of MSD but ergonomics improvements are not available.

Body Mechanics Training: Safer Lifting Behaviors

The industry terminology is "materials handling" when referring to lifting, pushing, pulling, and carrying of loads. One critical intervention for preventing back injury is body mechanics education for worker lifting behaviors. This has long been the topic of traditional Back School workplace education programs, the vehicle by which many physical therapists are initially brought into the workplace for on-site prevention efforts. Involving workers in evaluation, risk problem solving, and customized body mechanics training can be highly effective, particularly where ergonomics improvements in work design are limited.

Workplace Back School training typically teaches spinal anatomy, biomechanics, mechanisms of injury and degeneration, personal ergonomics skills, and self-protection tactics with a focus on body mechanics material handling techniques. Safer materials handling techniques can be both generic and customized to each work area. The longstanding debate between posterior pelvic tilt versus maintaining lumbar lordosis when lifting appears to lean in favor of utilizing stabilizing the natural lordosis posture for lumbar loading (per McKenzie, originally adapted from Cyriax).

Workers will offer challenges and suggestions for address specific materials handling risks in their own work tasks. These special challenges may involve cumbersome shapes, unstable loads, obstacles, and other complications. This is where the consulting physical therapist must call upon his-her best biomechanical problem solving, in collaboration with the skills and suggestions of the workers, to identify best practices. This becomes especially important with training of new employees.

TENDINITS AT THE CELLULAR-MOLECULAR LEVEL…

Hertling & Kessler (MGT OF COMMON MUSC-SKEL DISORDERS)… pp138-140 (paraphrased)

During continual loading stress, if nutrition is compromised or if damage outruns repair, tissues may atrophy, weaken and fail. Tissues with poor vascularity (such as tendons, ligaments, cartilage) are more susceptible to this degeneration, especially if nutrient pathway is compromised.

When loading cycles are intermittent enough to allow for adequate repair and adaptation (conditioning), no pathology will result. Improved ability to attenuate loads comes from increased collagen, if that collagen is mobile enough to allow deformation in response to loading. If the collagen is unable to deform, the resulting internal strain can lead to damage. Collagen must form with the correct "weave" or direction that absorbs the loading stress.

Mildly increased loading stimulates laying down of collagen precursors that polymerize into collagen fibers that align in response to the direction of loading stress. High loading stresses or reduced nutrient support can result in inadequate adaptation. New collagen that has not had enough time to mature breaks down, resulting in inflammation. The new fibers are not mature enough to attenuate loads.

Tendinitis can be attributed to continued abnormal stresses to a tendon, which preclude adequate modeling and create structure that is not sufficiently deformable to attenuate those stresses.

In tendinitis, rest alone will often not assure adequate maturation of collagen. Loading stress is also a stimulus to restore normal alignment of structural elements (collagen fibers), as well acting as a source of improved nutrient pathway (cyclical loading to improve perfusion). There must be a balance between rest to allow collagen fiber production to proceed, versus some ongoing loading stimulus to facilitate proper collagen fiber alignment.

SYSTEMIC & CNS EFFECTS of MSD (Barr, et al, Work-Related MSD of Hand & Wrist, JOSPT, Oct 04, 610-625)
Tissue injury (micro-trauma) leads to local inflammatory response, including edema, and production of pro-inflammatory cytokines and other inflammatory mediators by injured cells and by invading immune cells.

Systemic inflammatory processes… In rats, high repetition negligible force (HRNF) tasks increased immunohistochemical expression of hsp 72, an indicator of cellular distress. Macrophage infiltration was seen, not only in the working tissues, but also in the other non-work limbs (suggesting a systemic cytokine-mediated inflammatory response). This may be a vehicle for an increased susceptibility for MSD from previously innocuous tasks at other sites.

CNS effects… There is neural re-organization in response to repetitive movement demands. There was a degradation of motor behavior in these animals. Motor performance decline and maladaptive movement patterns were seen in 100% of test animals even weeks after inflammatory processes had declined. There can also be brain changes in the somato-sensory cortex. Primate studies of CNS response to repetitive forceful hand squeezing over 2-5 months showed de-differentiation of hand representation in the cortex area controlling hand function. In humans there is a loss of single touch localization and spatial discrimination, contributing to potential maladaptive movement patterns in WMSD's.

Similar sensorimotor changes are seen at the CNS on individuals with CTS. CNS reorganization, such as central cortical field expansion, following peripheral nerve compression may explain why there is often ulnar nerve findings accompanying CTS, as well as why there may be hyperalgesia and preserved motor function in the presence of impaired sensation. Plus, positive CTS findings in the non-dominant or less-used hand may indicate a systemic inflammatory response. MRI of brain cortex in patients with CTS show invasion of median nerve representation region with ulnar and radial nerve representation areas. Somatosensory cortex degradation is seen in rats, monkeys and humans exposed to repetitive tasks. There is de-differentiation of hand areas with shifted and degraded digit receptor fields.

The PATHOGENESIS of OVERUSE MSD

Mechanical-structural…
- Collagen fiber disruption-alteration-disorganization

Postural effects & consequences…
- Forward head, protracted shoulders, shoulder-elbow-forearm-wrist-thumb-hand

Nutrient pathway compromise…
- Proximally… TOC… lymphatic-venous-arterial
- Locally… sustained tissue tension-compression… exceeding perfusion pressure

Switch to anaerobic metabolism…
- Concentration of metabolites… chemical irritants

Inflammation…
- Chemical hostility
- Nociceptor facilitation

Fluid Dynamics…
- Effusion… lymphatic flow disruption… another nutrient pathway challenge

Pain…
- Nociceptor depolarization… via chemical… or mechanical distortion
- Sleep deficit effects, serotonin, anxiety, pain perception, ANS effects

CNS effects…
- Summation of nociceptor input to CNS… reduces nociceptor thresholds distally
- Motor cortex adaptation… de-differentiation-expansion… motor control distortion
- Anxiety responses… trigeminal spread

Radicular facilitation…
- Segmental referral of nociception-pain; facilitated segment
- Nerve root irritation

Systemic biochemical effects…
- Release of immunohistochemicals
- Widening susceptibility to inflammatory responses ssytemically

Spread of biomechanical dysfunctions… biomechanical lesion complexes…
- Forward head posture
- Shoulder girdle protraction
- Subacromial impingement
- Thoracic outlet-inlet compression
- Cranio-mandibular dysfunction
- LB facet-disc-iliolumbar ligament-sacroiliac-piriformis-hip joint-trochanteric bursa

THORACIC OUTLET COMPRESSION…

FHP.. reduced diaphragmatic breathing, increased accessory (scalene) breathing.
Narrow scalene triangle… shortened scalenes… elevates rib 1
Decreased type II fibers, increased type I fibers (posture)… (from 50-50 to 77-33%)
Connective tissue doubles …(from 14.5% to 36.6%)

Brachial plexus is C5 through T1… abnormal contribution of T2 reduces neural mobility
Slumped posture elongates cord, increasing length brachial plexus must travel to get through TO
Slump pulls down on cord at LB, taut at neck thru brach plexus = reach causes tension (C67T1)

Paradoxical breathing.. overworks scaleni… some nociception (facilitated)…
- somatic flex withdraw hypertonicity (background tension)… autonom vasoconstrict

Key weakness in TOC is ADDUCTOR POLLICIS & FCU (muscle test for D Dx)

Subclavian artery-vein become Bracial artery-vein.distal to rib 1.
Vein more vulnerable to compression. Vein squeezed more than artery… upsets fluid dynamics
- Especially at CT… “pooling of the swamp”

Nutrient pathway relies on pressure gradient… alter that = vein collapses = hypoxia & edema
- Later fibroblastic activity = fibrosis

Venous compromise far more common than arterial (cyanosis, edema, heaviness, fingers stiff)
May be cerv rib, fibrous band, elongated transv proves on C7.
Roos Test (AER) = loss of pulse; then open-close elevated arm stress test (EAST) clausdication sx
Many false positives but reasonably reliable… reproduction of sx is most reliable response
I combine Adson-hyperabduct-costoclavic positions to reproduce sx or drop pulse
Red flags… pancoast tumor

RX… diaphgr breathing; Jacobsen's Relaxation; stretch scaleni; neural mobs, mobil mid) & up thor
Workplace sit-stand, UE-WB, microstretching, ergo layout
KEY ELEMENT… UPPER EXTREMITY WEIGHTBEARING SUPPORT ON WORK SURFACE

POSTURE RISKS… CONSEQUENCES & EFFECTS…

FORWARD HEAD…
- subcranial hyper-ext,… occipital NV compress… shortening subcran muscles
- flat cervical lordosis… von Lusca loading…hypertrophy… spurring
- thoracic flexion… sustained… MF thickening-stiffening… supraspin lig stretch
- scaleni shortening… TOC
- craniomandibular shifts
- protracted shoulder girdle… impingements
- flattens LB lordosis

PROTRACTED SHOULDER GIRDLE…
- Retractor weakness... protractor tightening
- Subacromial compromise… impingement

SHOULDER REACH…
- Supraspinatus loading… Subacromial compromise

ELBOW FLEXION…
- Ulnar tension at cubital tunnel

FOREARM SUPINATION…
- Pronator-flexor tension… medial epicondyle… pronator syndrome (median)

WRIST FLEXION... DEVIATION… GRIP… PINCH…
- Carpal tunnel compromise

THUMB LATERAL PINCH WITH WRIST DEVIATION…
- DeQuervain's

STATIC POSTURE DISORDER vs REPET. MOTION INJURY…

Musculoskeletal system designed for locomotion, not sitting, not standing
- During locomotion, head is balanced upright over erect spine for level vision
- When locomotion ceases, head and shoulders roll, forward

Repetitive tasks are carried out in sustained postures (sitting – standing)
- "Repetitive motion injury" is a misleading term… usually more a static posture stress
- Even "good" posture is bad if it is sustained.
- Posture variety far more important that posture perfection
- Posture fatigue then distorts-deteriorates posture alignment

Sustained joint compression, disc compression, muscle contraction, tendon tension
- All these create tissue pressures exceeding tissue perfusion pressure
- Tissues switch to anaerobic metabolism… 19x more glucose … 19x more metabolic wastes
  - Forward head (see earlier notes)
  - Sustained Shoulder reach
  - Sustained Elbow flexion
  - Sustained Forearm supination
  - Sustained Wrist flexion or deviation
  - Sustained pinch or grip

MULTI-LEVEL NEUROVASCULAR COMPRESSIONS

Compression sx at one site increases susceptibility for compression sx at other sites along nerve
- Ischemia, plus blocks bi-directional flow of axonal fluids… swelling, biochem changes
- Alters blood-nerve barrier… more later on that
- "Double Crush" effects

Nerve root compression… Disc bulge… or joint of von Lusca… median-ulnar-radial
Scaleni… median – ulnar nerves… TOC… forward head posture
Pectoralis minor… median – ulnar nerves… TOC… protracted shoulder girdle
Pronator teres… median nerve… pronator syndrome, mimics CTS, except sensory loss over thenar … Ligament of Struthers at distal medial humerus, prox to pronator origin
Carpal tunnel.. more later
Radial extensor group… RTS… radial nerve, just distal to TE site… dorsum hand sensory loss
Elbow flexion… Cubital tunnel… ulnar nerve… noc sleep posture sx
Guyon's canal… ulnar nerve at wirst-hand

C.T.S. considerations…

TCL runs from pisiform and hamate medially to scaphoid tuberosity and trapezium laterally.
Pressures as low as 20-30 mm Hg will interrupt nerve blood flow… loss of nutrient pathway.. reduced drainage due to altered fluid dynamics.
Blood-nerve barrier is made up of the inner cells of perineurium and endothelial cells of the endoneurial microvessels.
Loss of blood-nerve barrier allows large proteins, macromolecules and macrophages to seep into nerve causing intra-neural edema.
Creates internal pressure on endoneurial capillaries… reduces blood flow.
Macrophage infiltration stimulates growth of collagen in epineurium and release of connective tissue growth factor… leading to fibrosis. Fibrosis occurs around and within nerve.

Grade 1… Neuropraxia… conduction block, but axon not injured… intermittent tingling
Positive compression tests, negative Tinel. Some loss of light touch and vibration
Grade 2… Axonotmesis… Axon injured and attempting regeneration… numbness
fibrillation on EMG… Tinel positive, reduced 2-point discrimination
Grade 3... Neurotmesis.. Loss of axons, scar in endoneurium... constant numbness..
thenar atrophy.. poor Px
EMG-NCV…There is no gold standard clearly diagnosing the presence of CTS… false + and –'s exist
Distribution of regions of sx (median) and day-noc sx patterns are reasonably diagnostic of dysfunction, if not disease. See **KAMATH & STOTHARD CTS DIAGNOSTIC SCALE**… much more sensitive & specific

N-V compression may come from…
- Proximal compression at c-spine or scaleni or pect minor or pronator teres
- Wrist flexion
- Wrist deviation
- Sustained, repeated, loaded tendon tension (grip-pinch)
- Fluid accumulation (lymphedema, hormonal effects)
- Altered collagen metabolism and health (nicotine, hormonal effects, thyroid imbalances)
- Direct external mechanical compression
- Bony changes (DJD… square wrist)
- Obesity
- Vibration

A "unified explanation" of LBP… "a cascade of patho-mechanics" ….

LBI-LBD is a Nutrient Pathway Disorder (discs, articular cartilage, ligaments)
Dysfunction leads to Degeneration leads to Derangement
- Spinal axis… Spinal segment..
- Facet-apophyseal joints... IV discs..
- Ligaments.. muscle control
- Lumbo-sacral jct… sacro-iliac… hip joint
- Intrinsic factors… extrinsic factors

Extrinsic factors
- Long, supf muscles of torso (ES, lats, traps, TL fascia)
- Hamstrings
- Piriformis
- Iliopsoas
- Rectus Fem
- ITB
- Calf and foot biomechanics

Dysfunction
- Mobility – flexibility – strength – stability

Degeneration
- Time – gravity – nutrient pathway
- DJD, DDD, collagen changes

Derangement
- Mechanical collapse
  - Disc
  - Sacro-iliac

A "unified explanation" of LBP… "a cascade of patho-mechanics" …. Specific structures…

Apophyseal Joints
- 15% WB when upright
- Joint surface compression loading
- Joint capsule tensile loading (bending)

Articular cartilage
- Water.. proteoglycans.. collagen matrix
- Nutrient pathway
- Cyclical loading-unloading
- Degeneration… loss of proteoglycan… dehydration... quality & quantity of collagen

Hypomobility
- Loss of cyclical load-unload
- Sitting – standing - flexed posture – static posture
- Loss of nutrient pathway… degeneration…
- Reduced elasticity at annulus, facet joint capsules, ligs… high risk for sprains
- Discs shrink.. increased facet loads… hypertrophy lamina… spinal stenosis

Disc… degeneration – dysfunction – derangement
- Disc avascular but well-hydrated… hydraulic… load transmitter
- Posterior annulus is actually a posterior ligament
- Degeneration blurs annulus versus nucleus
- Loss of hydraulic load resistance at nucleus
- Loss of tensile load tolerance at annulus… greater bulging
- Axis of flexion moves posteriorly from disc to facet… stresses both
- Flexion loading risks HNP… flexed loading to failure
- Nucleus loads transmitted to annulus overwhelms annulus tangential strain tolerance
- Also, if nucleus material contact blood supply (end-plate failure) = autoimmune response (nasty!)

The Sacro-iliac story !
- DDD or HNP…
- reduces disc volume …
- L4-L5-S1 vertebrae drop closer,,,
- Iliolumbar ligament slackens
- Loss of support to sacroiliac (a suspension joint)
- Sacrum tilts anteriorly around S3… SI derangement !
- Then…
- SI nociception
- Piriformis splinting or trigger or tension
- Sciatic entrapment… sciatica (independent of any HNP)
- Also piriformis tendon compresses trochanteric bursa… troch bursitis
- Troch Bursitis usually a piriformis-SI issue!

- Piriformis also reduces hip rotation ROM… accelerates hip DJD
- Hip DJD very responsive to mobil hip, stretch hip rotators, correct SI Rx

Motor Control
Strength
Stabilization… dynamic
Motor control… coordination… closed chain

Erector spinae
Multifidus (actually a sensory organ with lots of spindles)
Ilopsoas
Transversus abdominus
Glut max
Hamstrings… length and strength
Quadriceps

EVIDENCE… REFERENES… BIBLIOGRAPHY

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IMPORTANT JOURNAL SPECIAL ISSUE… JOSPT OCT 2004:

MacDermid J, Doherty T: Clinical & Electrodiagnostic Testing of carpal Tunnel Syndrome. JOSPT 2004 Oct.
Review of various simple non-invasive diagnostic testing for CTS, with correlations to electrodiagnostic testing. Includes valid testing per history, sensory screening, provocation tests, NVC-EMG.

Michlovitz S: Conservative Interventions for CTS. JOSPT, 2004 Oct.
Review of current PT approaches to clinical management of CTS.

Lee M, LaStayo P: Pronator Syndrome and Other Nerve Compressions That Mimic CTS. JOSPT, 2004 Oct.
Differential assessment of median nerve compressions of the upper extremity proximal to the wrist, mimicking CTS, along with treatment approaches for these.

Barr A, et al: Work-Related Musculoskeletal Disorders of the Hand and Wrist," JOSPT, 2004 Oct.
Extensive review of research on MSD pathophysiology, diagnosis, epidemiology. Of particular note is description of pathophysiology, especially describing histochemical changes that can trigger systemic inflammatory responses. Cytokines are released from local inflammatory sites may trigger systemic inflammatory responses, thus causing tendinitis symptoms to spreading to other sites. Of equal interest is the motor behavior degradation that can occur with repetitive motion tasks, causing loss of motor control and a resulting loss of coordination and movement efficiency that may increase work damage. This may be the result of changes at the brain where repetitive movement causes degradation of motor cortex, distorting motor control. This is reorganization of CNS control of movement.

Novak C, "Upper Extremity Work-Related Musculoskeletal Disorders: a Treatment Perspective," JOSPT, OCT 2004
This is an excellent discussion of various important pathomechanics of MSD. Of particular interest is the prevalence of multi-level neurovascular compressions (double-crush) {{a critical issue in prevention tactics where we address proximal posture risks to reduce distal repetitive motion problems}}. Static postures, particularly at the neck are also described {{another critical issue in prevention tactics where we address proximal posture risks to reduce distal

repetitive motion problems}}. Posture risks at various articulations are described. Treatment approaches are also well-described in this paper.

MORE IMPORTANT REFERENCES WITH ABSTRACTS & COMMENTS….

COSTS & EPIDEMIOLOGY

Lou, Pietbrobon, et al: Estimates and patterns of direct health care expenditures among individuals with back pain in the Untied States. Spine 2004; 1: 79-86.
It is estimated that over the course of an average lifetime, 80 percent of Americans will suffer at least one episode of back pain.

Maetzel A, Li L. The economic burden of low back pain: a review of studies published between 1996 and 2001. Best Pract Res Cli Rheumatol 2002; 16:23-30.
Low back pain results in approximately 149 million lost work days annually, with 2/3 of these caused by occupational injury and an annual lost productivity loss of $28 billion.

Pai S, Sundaram L. Low back pain: an economic assessment in the United States. Orthop Clin North Am 2004; 35: 1-5. …Estimated annual cost for low back pain is $20-50 billion.

Bernacki E. Factors influencing the costs of workers' compensation. Clin Occup Environ Med 2004; 4: v-vi, 249-57.
California's Worker Compensation data fro 1993 to 2000 showed that indemnity costs increased eightfold when there is legal involvement.

EXERCISES & REST BREAKS

De Vera Barredo R, Mahon K: The effects of exercise and rest breaks on musculoskeletal discomfort during computer tasks: an evidence based perspective. J Phys Ther Sci, 2007, vol 19, no 2: 151-163.
A review of research evidence on effects of exercise and rest breaks on musculoskeletal discomfort during computer work found that evidence supports use of rest breaks and exercise breaks to reduce discomfort. The evidence, in aggregate, suggests no additional benefits of exercises over rest breaks. This review identified widespread problems with research design, internal validity, statistical analyses, dropout rates and poor subject compliance. This serves to illustrate the potential value of rest breaks and exercise breaks but identifies problems with the research in allowing us to reach valid conclusions. This article is also an excellent demonstration of how to assess the value of research according to levels of evidence and internal validity.

Fenety A, Walker JM: Sort-term effects of workstation exercises on musculoskeletal discomfort and postural changes in seated video display unit workers. Phys Ther, 2002, 82: 578-589.
Authors examined short term effects of an exercise program on eleven subjects, involving an exercise break every 30 minutes. Exercises done by video display unit operators resulted in short-term decrease in both musculoskeletal discomfort and postural immobility. This is a non-randomized trial where participants acted as their own controls, resulting in evidence level IV with moderate internal validity.

Saltzman A: Computer user perception of the effectiveness of exercise mini-breaks. In: Proceedings of the Silicon Valley Ergonomics Conference and Exposition. Silicon Valley, CA, 1998, 147-151.
Authors examined effects of an ergonomics exercise software program of frequent short stretch breaks. Participants reported stretch breaks were effective in reducing workplace discomfort. 23 percent of participants reported increased productivity. 34 percent of participants dropped out of the study. This study is level V evidence with weak internal validity.

Thompson D: Effects of exercise breaks on musculoskeletal strain among data entry operators: a case study. In: Promoting Health and Productivity in the Computerized Office: Models of Successful Ergonomics Intervention. Taylor and Francis, 1990, 118-127.
Employees were asked to perform five-minute exercise sessions during their two regular break periods. Operators reported reduced discomfort and generally improved physical condition. There were no Worker Compensation claims for one year into the program. Productivity had increased by 25 percent during the first four months of the program. Level of evidence is V, with weak internal validity.

Linton S, van Tulder M: Preventive interventions for back and neck pain problems. Spine, 2001 vol 26, no 7: 778-787.
A review of 27 controlled trials demonstrated back schools to be ineffective for prevention, with exercises being the only effective preventive intervention.

Karas B, Conrad K: Back injury prevention in the workplace: an integrative review. AAOHN J, 1996; 44(4): 189-96.

Review of 15 experimental and quasi-experimental studies showed some positive evidence for back belts, back schools, stretching programs, and educational classes. Back schools and stretching programs were studied more frequently and showed the greater proportion of positive results.

Moore T: A workplace stretching program. Physiologic and perception measurements before and after participation. AAOHN J, 1998, 46(12): 563-8.
Participants who completed a structured stretching program had zero occurrences of MSD during the two-month period. There was statistically significant improvements in flexibility and the Fox Self Perception Profile, suggesting that flexibility exercises may reduce workplace MSD.

Silverstein B, Armstrong T, et al: Can in-plant exercise control musculoskeletal symptoms? J Occup Med, 1988, 30 (12): 923-927.
After one year of an on-the-job exercise program to control musculoskeletal symptoms in the neck and upper limb, there were no statistically significant differences in localized postural discomfort scores or in the proportion of those whose discomfort decreased based on exercise participation. Although no clear reduction in discomfort was achieved by the exercise program alone, at least 67 percent of respondents who participated in the exercise program reported that the program made them feel better.

Galinsky T, et al: Supplementary breaks and stretching exercises for data entry operators: a follow-up field study. Am J Indust Med. 2008, 30(7); 519-527
Workers in the supplementary breaks group scored significantly lower in discomfort and maintained output despite extra break periods. The group that was to add exercises to their break period reported only 25 percent exercise compliance during break periods, preventing valid assessment of stretching effects.

Balci R, Aghazadeh F: The effect of work-rest schedules and types of task on the discomfort and performance of VDT users. Ergonomics, 2003, 46: 455-465.
and
Balci R, Aghazadeh F: Effects of exercise breaks on performance, muscular load, and perceived discomfort in data entry and cognitive tasks. Computers & Industrial Engineering, 2004, 46(3):399-411.
Comparing various work time versus rest time periods revealed that very brief very frequent micro-breaks resulted in less discomfort and higher accuracy and productivity than did longer work periods with longer breaks. This study was level II evidence with strong internal validity.

Hess J, Hecker S: Workplace stretching programs: the rest of the story. Appl Occup Envir Hyg, 2003; 18(5): 331-8.
Several studies are examined and analyzed in detail, describing positive and negative findings among a variety of studies and exercise approaches. One study review included flexibility program for firefighters. Showing no significant reduction in incidence of LBI, but injury costs significantly reduced ($85,372 for stretch group versus $235,131 for control group) from Hilyer 1990.

Hilyer J, et al: A flexibility intervention to reduce the incidence and severity of joint injuries among municipal firefighters. J Occup Med 1990, 32(7): 631-637.
A flexibility program for firefighters showed no significant reduction in incidence of musculoskeletal injury, but total injury costs significantly reduced ($85,372 for stretch group versus $235,131 for control group)

SIT-STAND

Hasegawa T, et al: Effects of a sit-stand on a light repetitive task. Int J Indust Ergo, 2001, 28 (3-4): 219-224.
Subjects performed light repetitive tasks in sitting and standing. More frequent switches between postures resulted in better work performance and less fatigue that with less frequent switch.

BACK SCHOOLS

Schenk R, Doran R, Stachura J: Learning effects of a back education program. Spine 1996; Oct 1; 21(19): 2183-2189.
The American Back School was compared to a video education group and a control group. Learning effect was assessed by examining gains in post-test results. No differences in post-test comparisons were seen in control and video groups. Significant differences were seen in the back school group at the .001 level, indicating that back school is an effective intervention for influencing lifting posture and conveying information on spinal mechanics and lifting technique. Additionally, video training may not be an effective prevention intervention.

Heymans M, van Tulder M, et al: Back schools for nonspecific low back pain: a systematic review within the framework of the Cochran Collaboration Back Review Group. Spine. 2005; 30(19): 2153-63.
There is moderate evidence suggesting back schools in an occupational setting reduce pain and improve function and return-to-work status.

Karas B, Conrad K: Back injury prevention in the workplace: an integrative review. AAOHN J, 1996; 44(4): 189-96.
Review of 15 experimental and quasi-experimental studies showed some positive evidence for back belts, back schools, stretching programs, and educational classes. Back schools and stretching programs were studied more frequently and showed the greater proportion of positive results.

Gatty C, et al: The effectiveness of back pain and injury prevention programs in the workplace. Work 2003; 20(3): 257-66.
Review of nine studies showed that positive outcomes were associated with studies reporting high compliance that used job-specific and individualized/small group education and training approaches.

van Poppel M, et al: An update of a systematic review of controlled clinical trials on the primary prevention of back pain in the workplace. Occup Med (Lond), 2004; 54(5): 345-52.
There is no evidence for effectiveness of back supports or education in primary prevention of LBP. There is limited evidence of moderate efficacy or exercise.

LIFTING WITH LORDOSIS

Hart D, Stobbe T, Jaraiedi M: Effects of lumbar posture on lifting. Spine 12:138-145, 1987.
Describes the advantages of lifting with lumbar spine positioned in a lordotic loading position versus in a kyphotic position (posterior pelvic tilt). They noted the advantage of lordotic lifting creating higher protective muscle contraction, as evidenced by EMG, providing a protective muscle action to counter flexion moments on the disc. Also, flexion moments were higher in the kyphotic lifting position and lower in lordotic lifting position.

Delitto R, Rose S, Apts D: Phys Ther 1987; 67(9):1329-34.
This study examined the effects of two different alignments of the lumbar spine on electromyographic activity of the erector spinae (ES) and oblique abdominal (OA) muscles during squat lifting. Nineteen healthy subjects (8 men, 11 women) participated in this study. Each subject performed squat lifts both with the lumbar spine aligned in "back-bowed-in" (BBI), or normal, lordosis and with the lumbar spine aligned in "back-bowed-out" (BBO), or relatively less, lordosis. Based on total duration, the lift was divided into two equal phases. EMG activity of each muscle was quantified for each half of the lift and normalized to the total EMG produced by the muscle during a maximal voluntary isometric contraction. A three-way analysis of variance for repeated measures was used to analyze the effects of position of the lumbar spine, timing, and load on the amount of EMG activity during lifting. For all loads, ES muscle activity was greater during the first half of the BBI lift, whereas OA muscle activity was greater during the first half of the lift, regardless of the lifting style (p less than .01). The greater ES and OA muscle activity occurring during the crucial initial period in the BBI lift may provide the best protection for the lumbar spine.

Bazrgari B, Shirazi-Adi A, Arjimand N: Analaysis of squat and stoop lifting : muscle forces and internal spinal loads. Eur Spine J 2007; May; 16)5):687-99.
Findings advocate for squat lifting over stoop lifting as the technique of choice in reducing net moments, muscle forces and internal spinal loads (moment, compression, shear forces).

Hickey D, et al: Relation between the structure of the annulus fibrosus and function and failure of the intervertebral disc. Spine 1985; 5(2):106-114.
Annulus fibers have the same mechanical properties as tendon fibers, per tolerance to stretch and strain. Failure of the annulus is most likely to fail during forward bending loads and during torsion loads, whereas compression is more likely to cause end-plate failure.

POSTURE

Gerr F, et al: A randomized controlled trial of postural interventions for prevention of musculoskeletal symptoms among computer workers. Occup Environ Med. 2005 Jul; 62(7): 478-87.
This study assigned two intervention groups each a work posture to maintain during computer work, plus a control group with no posture intervention. Results showed no significant differences in the incidence of musculoskeletal symptoms among the three groups, suggesting that the two postural interventions are ineffective at reducing symptoms. {{Lauren adds: I would suggest that this may support the hypothesis that it is not the position that leads to posture-related pain but, rather, the time spent in a sustained position that leads to symptoms. Position may matter less than time spent in the position.}}

Melhorn J: A prospective study for upper extremity cumulative trauma disorders of workers in aircraft manufacturing. J Occup Environ Med, 1996; 38(12): 1264-71

8000 employees underwent prospective study in a four-way experimental design to assess several interventions showed that only posture training had a beneficial risk reduction. Combination of posture training and exercise had beneficial effect for two of the groups.

UE SUPPORT

Nag P, et al: Influence of arm and wrist support on forearm and back muscle activity in computer keyboard operation. Appl Ergo. 2009; 40(2): 286-291,
Study examined muscle activity while using a wrist rest, forearm support and floating (unsupported) upper extremity work posture during keyboard work. Wrist rest showed mixed reductions in muscle activity, while forearm support showed widespread reduction in muscle activity compared to forearms unsupported.

Conlon C, Krause N, Rempel D: A randomized controlled trial evaluating an alternative mouse and forearm support on upper body discomfort and musculoskeletal disorders among engineers. Occup Environ Med. 2008 May; 65(5): 311-8.
In engineers using computers more than 20 hours per week, a forearm support board may reduce right upper extremity computer use.

Remple D, et al: A randomized controlled trial evaluating the effects of two workstation interventions on upper body pain and incident musculoskeletal disorders among computer operators. Occup Environ Med. 2006 May; 63(5): 300-6.
Comparing ergonomics training only with training plus computer trackball with training plus forearm support revealed that training plus forearm support was effective in preventing upper body MSD among call center employees.

ERGONOMICS INTERVENTIONS

Haukka E, et al: A randomized controlled trial on whether a participatory ergonomics intervention could prevent musculoskeletal disorders. Occup Environ Med. 2008 Dec; 65(12): 849-56.
A cluster randomized trial of 504 kitchen workers in 119 workplaces over 11-14 month period with 402 ergonomics changes implemented revealed no systematic differences in any outcome variable between intervention group and control group. There was no reduction in perceived physical workload or MSD among these workers.

Karsh B, Moro F, Smith M: The efficacy of workplace ergonomic interventions to control musculoskeletal disorders: a critical analysis of peer-reviewed literature. Theor Iss Ergo Sci, 2001, vol 2, no 1: 23-96.
An analysis of 101 peer-reviewed studies examining the efficacy of a range of workplace ergonomic interventions concluded that the most effective interventions were multiple-component interventions. Of the studies considered 84 percent found some positive results, although most had mixed outcomes. Only 32 percent of the studies used experimental or quasi-experimental designs.

PATHOLOGENESIS

Byng J: Overuse syndromes of the upper limb and the upper limb tension test: a comparison between patients, asymptomatic keyboard workers and asymptomatic non-keyboard workers. Man Ther 1997, 2: 157-164
The ULTT was positive in 100 percent of the patient group, supporting the hypothesis that the pathology of occupational upper limb overuse is neurogenic in origin. Furthermore, the asymptomatic keyboard users (intended to be a control subgroup) also had a significantly higher positive ULTT compared to asymptomatic non-keyboard users (the other control subgroup).

Porterfield J, DeRosa C: Mechanical Low Back Pain: Perspectives in Functional Anatomy. 1998; W.B. Saunders.
Describes functional anatomy, biomechanics, pathomechanics, degenerative changes at the lower spine at the zygoapophyseal joints, intervertebral discs and sacroiliac joint.

Riley G, et al: Tendon degeneration and chronic shoulder pain: changes in collagen composition on the human rotator cuff tendons in rotator cuff tendinitis. Ann Rheum Dis. 1994; 53(6): 359-66
Degenerated tendons show changes consistent with repeated minor injury and fiber damage consistent with reduced vascular perfusion, tissue hypoxia and influence of cytokines, weakening the tendon.

Lundborg G, Dahlin L: Anatomy, function, and pathophysiology of peripheral nerves and nerve compression. Hand Clin 1996; 12(2):185-93.
The clinical stages of nerve compression lesions can be related to changes in intraneural microcirculation and nerve fiber structure, alterations in vascular permeability and subsequent formation of edema. The double crush and reverse double crush syndromes are related to disturbances in axonal transport induced by compression, followed by morphological and functional changes in nerve cell bodies.

Edgelow P: Ch.6; Neurovascular consequences of cumulative trauma disorders affecting the thoracic outlet: a patient-centered approach. In Donatelli R (ed): Physical Therapy of the Shoulder; 1997; Churchill-Livingstone.
Comprehensive description of pathomechanics and pathophysiology of thoracic outlet compression.

ON-SITE PT

Sadi J, et al: A 13-year study of musculoskeletal disorders treated in an autoplant , on-site physiotherapy clinic. J Occup Rehabil. 2007; 17(4): 610-22.
On-site physiotherapy services can provide early cost-effective management of MSD in the automotive sector.

Rothstein, et al: The hypothesis-oriented algorithm for clinicians II (HOAC II): a guide for patient management. Phys Ther 2003, 83 (5): 455-47.
Describes the clinical decision-making process defined by HOAC-II as a structured process of patient evaluation, problem-identification, goal-setting, intervention selection, and outcomes assessment.

BACK BELTS

Kraus J, et al: Reduction of acute low back injuries by use of back supports. Intl J Occ Envir Hlth, 1996; 2(3).
The study was sponsored by UCLA School of Public Health and the Southern California Injury Prevention Research Center. The study of 36,000 employees at 31 Home Depot stores in California encompassing 101,000,000 work hours revealed a decrease of 34% in low back injuries following a mandatory policy on use of back supports among employees. Favorable effects were seen in both genders, young and old, new or experienced workers, low lifting intensity and high lifting intensity jobs. The study provides evidence that proper use of back supports as part of a comprehensive back injury prevention program may be effective in reducing back injuries.

Kraus J, et al: Back supports and low back injuries: a second visit with the Home Depot cohort study data. Intl J Occ Envir Hlth, 1999; 5: 9-13.
This paper revisits data for the study suggesting favorable low back injury prevention effects of wear back supports in the workplace, in response to letters to the Journal editor questioning the study's findings. The issues were examined and answered, supporting the validity of the findings.

Warren L, et al: Effects of soft lumbar support belt on abdominal oblique muscle activity in nonimpaired adults during squat lifting. JOSPT 2001; 31.
Wearing a soft lumbar support during squat lifting significantly decreased activity of abdominal obliques. This study shows back belts reduce muscle activity and this should be consistent with decreased loads on the spine, likely due to increased intra-abdominal pressure. This is consistent with other studies that, taken together, demonstrate the both abdominal and back muscles produce less force during lifting with a back support. Decreased coactivation of the muscles around the spine suggests that spinal compressive forces are decreased. Loads on the spine during lifting tasks are positively correlated with increased muscle activity. Intra-abdominal pressure is significantly increased with wearing a back belt during lifting. Increased intra-abdominal pressure is believed to result in decompression of the spine during loading and decreased load on spinal muscles. There is also an increased recruitment of quadriceps muscle action during lifting with back belts, suggesting they encourage improved lifting technique. This paper mentions numerous studies in its text and its bibliography that add support to the hypothesis of the authors.

Van Duijvvenbode, et al: Lumbar supports for prevention and treatment of low back pain. Cochrane Database Syst Rev. 2008; (2):CD001823.
There is moderate evidence that back supports are not more effective than no intervention or training. There is conflicting evidence of their effectiveness when combined with other interventions.

Van Tulder M, et al: Lumbar supports for prevention and treatment of low back pain. Cochrane Database Syst Rev. 2007; (2):CD001823.
There is moderate evidence that back supports are not more effective than no intervention or training. There is conflicting evidence of their effectiveness when combined with other interventions. Authors also point out that quality of the research reviewed was poor and that one essential issue in the research was inadequate subject compliance

Ammendolia C, et al: Back belt use for prevention of occupational low back pain: a systematic review. J Manip Phys Ther. 2005;28(2): 128-34.
As of 2003, conflicting evidence and the lack of high quality trials imply there is no conclusive evidence to support the use of back belts to reduce lost time from occupational LBP.

Butler, David: THE SENSITIVE NERVOUS SYSTEM; Noigroup Publ, Adelaide, Australia, 2000.
The is an excellent text on the neurophysiology of pain, nerve mobility and adverse neural tension, neurovascular

entrapment, AIGS, double crush, central excitation, upregulation of pain, and other issues that closely relate to extremity pain syndromes, particularly those involved with work disorders. Excellent description of underlying issues important to dealing with injured workers. Addresses mobilization of the peripheral nervous system, which may be a part of the stretching tactics we may consider for the workplace.

IMPORTANT TEXT:

Bullock M, ed.: Ergonomics: The Physiotherapist in the Workplace, Churchill-Livingstone, 1991.

pp 214-215 and 228-229. This text describes the role of the physical therapist as highly qualified industry resource for ergonomics. The physical therapist is described as providing a valuable mix of musculoskeletal medical training with human biomechanics and physics of movement and posture as the basic science foundation of this profession. Most large industry settings in Europe employ on-site physical therapists as their ergonomics resource (in contrast with US industry which relies primarily on engineers.) In Scandinavian countries ergonomics is chiefly the domain of physical therapists.

pp 52-53: Posture workload is described as a primary risk for CTD. Static muscle contraction reduces circulatory irrigation to muscle, decreasing oxygen and creating metabolic wastes accumulation in the tissues, leading to pain and tissue hardening. Muscle posture work should not tolerate more than 5-6% of MVC in work conditions exceeding one hour. Rapid repetitive motion and high accuracy demands create increased background tensions in working muscles and tendons, as do noise and cold, creating similar stresses as seen in excessive posture load demands and high MVC. Similar stresses are seen when repetitive loading exceeds 50% of MVC and peak loading exceeds 75% of MVC. Similar stresses are seen in extreme positions and sudden jerky motions.

pp 108-115: Posture load is described as a severe risk, particularly in the presence of short rest breaks. Static posture loads with short rests is a strong risk. Posture load is a risk especially when posture is awkward or needed to manipulate tools distally. Speed of repetition increases static posture loads (Waersted 1986). Forward head posture is seen as a pain source in many static posture jobs according to one study, suggesting pain is a result of mechanical deformation of passive tissues (Harms-Ringdahl 1986). Static loading is identified as stress causing fatigue and reduction in blood flow at a time when wastes are increasing and oxygen demands are increasing (IBID). VDT work is discussed in this context, with up to 95% demonstrating symptoms (Bjorksten 1984). Management of this risk calls for improving posture habits, early symptom reporting, redeployment of work, and task alternation. Fatigue is the precursor to musculoskeletal pain problems. Repetitive light tasks produced significant changes in EMG and serum creatine kinase (SCK) in local muscles. These changes were not seen in heavy aerobic tasks (Hagberg and Jonsson 1982, 1984). The quality and frequency of rest affects the ability of muscles to recover (Rhomert).

pp 134-143: Forward head posture produces stress through muscles posture load and passive tissue loads at ROM extremes, where EMG may be quiet. Posture loads at neck and shoulder are affected by motions, postures and loads in the arms and hands. Precision, speed, psychological stresses all increased muscle posture loads. Neutral upright head posture uses approximately 2% of MVC; slightly flexed uses 10%; much flexed posture uses 17% of MVC. Trapezius fatigue is reached where MVC is at 2-5% for more than an hour. This load is greatly increased with arm flexion or abduction. Flexion was defined as lower cervical flexion with some compensatory extension at upper cervical, a protracted head posture over the shoulders (Harms-Ringdahl and Schuldt 1988). Work with arms unsupported greatly accelerates fatigue. The more they are elevated, the more fatigue changes are seen spreading from the upper traps to the lower traps, thoracic erector spinae, rhomboids and glenohumeral muscles. Symptoms are related to time spent in forward postures, shoulder elevation, and total duration of arm activity. Endurance also depended upon the worker's physical condition. Continuous arm activity entails no return to zero activity, so there is no relaxation of neck and shoulder muscles. Rather, movement is superimposed on static contractions. This is in light of the obstruction in perfusion of these working tissues (Schuldt, Jonsson, Christensen). The use of micro-pause loading breaks for only a few seconds are important to preserve comfort and work performance through the work day. When the hands and arms are supported during work activity, there is less pain in the neck and shoulder region(Hunting, 1981). Elbow support, properly designed, reduced activity in the traps, rhomboids, and erector spinae in both erect and flexed head positions among assembly workers (Schuldt, 1987).

Barr A, Barbe, M: Pathophysiological Tissues Changes Associated with Repetitive Movement: A Review of the Evidence. Phys Ther. 2002 February: 82(2): 173-187.

This article presents several key considerations defining MSD pathophysiological mechanisms taken from extensive literature review, particularly among animal studies describing neuromusculoskeletal responses to repetitive or sustained loading demands. Key points include:

CELLULAR CHANGES:

Muscle tissue biopsies of humans with hand overuse symptoms showed histological and muscle fiber structure changes consistent with denervation or ischemic loss of type II fibers with hypertrophy of type I fibers. Upper trapezius samples showed changes consistent with hypoxia and reduced blood flow. Cell membrane damage releases intracellular factors that stimulate infiltration of lymphocytes and macrophages. These processes stimulate regreneration, or scarring if that damage is ongoing. In tendons this can result in fibroblast proliferation leading to fibrosis and collagen dysplasia within the extracellular matrix. Ongoing mechanical or metabolic stress such as hypoxia, ischemia or inflammation leads to release of heat shock proteins (HSP) by cells such as neurons, glia, fibroblasts and muscle cells. This is a healing protective response whereby these HSP restore denatured proteins.

HSP are stimulated by ischemia or tears in cell membranes releasing cytokines, mediators of inflammation, cell proliferation and regeneration. These mediate proliferation of macrophages and fibroblasts. The phagocytic action of the macrophages can further increase damage and release more cytokines, thus creating a vicious cycle of chronic inflammation.

CNS CORD CHANGES:

Chronic pain can lead to neuroplastic changes in CNS and PNS. Sustained nociceptive afferent bombardment can increase release of excitatory neurotransmitters glutamate and substance P in the dorsal horn. These can activate and potentiate synapse activity both presynaptically and postsynaptically. This can also alter genetic expression in neurons to upregulate receptor sites. The end result is hyeralgesia (increased sensitivity to nociception) and allodonia (non-painful stimuli felt as pain). Clinicians often mistake this process as "symptom magnification" or psychological complications. Nerve constriction peripherally due to repetitive or sustained mechanical compression can also cause neuroplastic changes in the dorsal root ganglion that can increase nociception transmission.

CNS CORTEX CHANGES:

Repetitive tasks can induce changes in cerebral cortex, particularly de-differentiation of cerebral cortex representation of the hand. This is induced by constrained and repeated motions at the upper limb. Loss of specific hand field representation of the cortex causes loss of coordination and changes in movement behaviors toward less efficient motor control. This loss of movement efficiency increase fatigue and pain risks during repetitive tasks. This is maladaptive movement behavior. It may be that this motor control degradation precedes the onset of pain and may even precipitate it.

SYSTEMIC INFLAMMATORY REACTION:

Animal studies reveal increased cellular chemical changes: HSP-72, COX2, and macrophage infiltration at levels 1000 times above baseline. PLUS, these biochemical changes were seen also in the non-moving control limbs, suggesting a systemic inflammatory response to the high repetition low load tasks in the experiment. This suggests that repetitive task work can lead to not only local inflammatory reactions at the exposure site, but also leads to a wider systemic inflammatory response as well as neurological reorganization (neuroplasticity) centrally at the spinal cord, increasing nociception, and at the cerebral cortex, causing motor control degradation.

USEFUL WEB SITES:

http://www.emcins.com/lc/niosh.htm
Automated online NIOSH Lifting Equation calculator

http://www.osha.gov/pls/oshaweb/owadisp.show_document?p_table=federal_register&p_id=16305
This is the entire OSHA Ergonomic Final Rule; (29 CFR 1910.900, November 14, 2000).
Federal Register #: 64:65768-66078

http://personal.health.usf.edu/tbernard/HollowHills/WISHA_WMSD_Checklist.pdf
Washington State MSD hazards checklist format

http://personal.health.usf.edu/tbernard/HollowHills/WISHA_Lifting.pdf
Washington State lifting hazards calculator

http://www.ergo.human.cornell.edu/
Cornell University web site of numerous ergonomics research, guides, analysis tools