Disclosures

• Small honorarium
• No other financial interest
Spine Care Issues

- Common clinical presentation
- There is no agreed upon treatment for many spine problems
- Expensive care, yet outcomes have not improved
- Following reasonable guidelines would help improve patient outcomes and probably satisfaction
My Objective:

- To add a little bit of knowledge to your already overflowing cup, so that you can be a little more efficient and effective in the office evaluation of your spine patients
My Objective:

- To highlight and discuss:
  - Anatomy
  - Clinical presentations and evaluations
  - Treatments available and their clinical efficacy
  - Best Recommendations
Disc Biology

CELL MECHANICS
- cell or nucleus deformation
- volumetric changes
- membrane stretch or deformation
- altered polarization
- electrokinetic effects

EXTRACELLULAR MATRIX MECHANICS
- matrix stresses and strain
- hydrostatic pressure
- interstitial fluid-flow
- osmotic pressure
- streaming potentials
- streaming currents

UNIAXIAL COMPRESSION

BIAXIAL LOAD

SHEAR

aggrecan

hyaluronan

collagen fibril

cation

anion
The 3 Joint Complex
Early degenerative changes:
- Degeneration of articular cartilage with synovial inflammation or capsular swelling may result in referred pain.

Late degenerative changes:
- Facet joint and capsule innervated by dorsal rami from two spinal levels.
- Joint capsule.
- Synovial membrane.
Pain: What Gives?

- Nociceptors
- Dorsal roots → dorsal columns of cord
- Afferent pathways: spinothalamic, spinoreticular, others
- Thalamus, hypothalamus, midbrain
TISSUE DAMAGE

Inflammation

Kinins

Kinin B1 receptor induction

COX-2

fibroblasts

TNF-1

IL-1

IL-6

IL-8

Cytokines

Kinin B1 receptor induction

PGs

NGF

Gene transcription

EXCITE

SENSITISE

5-HT

Histamine

Histamine

Blood vessels

Extravasation

Vasodilation

Substance P

CGRP

NGF

Blood vessels

Histamine

Histamine

Histamine

Histamine

Histamine

Histamine

Histamine

Histamine

Histamine

Histamine

Histamine

Histamine

Histamine

Histamine

Histamine

Histamine
Inflammation

The Exudative Component

• Vascular Changes: Dilatation and permeability

• Plasma Systems Mediators: Complement, Kinin, coagulation, fibrinolysis system

• Cellular component: leukocytes and cell derived mediators, e.g. histamines, leukotrienes, nitrous oxide, prostaglandins, TNF
Pain: What Gives?

Inflammation

Phospholipase A2
Arachidonic Acid
Leukotrienes
Thromboxane

Cytokines: TNF-α, others
induces nitrous oxide production
Pain: What Gives?

Neurotransmission

Substance P
Neurokinin A & B
Glutamate

c-Fos gene:
Produces protein
Fos, crucial to CNS response to pain
What do we know?

Low Back Pain

- Prevalence 22-48%
- 80% of population will be affected
- Total costs >$100B/year
- Costs per year: Majority by 10% of patients
- Many forms of treatment
Spine Care Challenges

- JAMA 2008: $86B in 2005, a 65% increase from 1997
- Yet no improvement in self reported outcomes for spine related problems
- Tremendous regional variations in rates of care, e.g. spine fusion rates by a factor of 20
- Reimbursement is independent of outcomes and cost of care typically not considered
Spine Pain

- Mechanical Axial
- Initial Presentation
- Radicular
- Both
History

Key Components

• Systemic Disease?
• Evidence of neurologic compromise?
• Social or psychological distress?
Red Flags

• History of cancer, IV drug use
• Age >50
• Unexplained weight loss, constitutional sx
• Pain > one month
• Nighttime pain
• Hx AAA, use of corticosteroids
• Unresponsive to treatment > 6 weeks
Mechanical Pain

Etiology

• Any anatomical structure: disc, facet, muscular, ligamentous, vertebral

• The pain generator = The Holy Grail
Axial Pain

Risk Factors

• Carragee, Spine, 2005. LBP disability predicted by baseline psychosocial testing, when using MRIs, discograms, PE, work history, psych testing

• Psychosocial variables strong predictor of disability

• Cultural differences

• Structural spine changes only weakly associated with adverse outcomes
Axial Pain
Risk Factors

• Smoking, obesity, older age, female, strenuous work

• Sedentary work, psychologically strenuous work, low education, Worker’s Comp

• Job dissatisfaction, somatization disorder, anxiety, depression

• Poor physical health
Psychosocial Factors
Poorer Outcomes

- Depression
- Passive coping strategies
- Job dissatisfaction
- High disability levels
- Disputed compensation claims
- Somatization
Compressive Radiculopathy

- Secondary to nerve root compression
- Disc herniation
- Spondylosis
- Rule out myelopathy
Disc Pathology
What’s Going On?

Annular disruption *can* give way to mechanical weakening, microvascular invasion, disc herniations.

Three components to a disc herniation:
Mechanical
Immune
Inflammatory
Herniated Disc
Mechanical Compression

Pressure

- Decreased conduction velocity
- Decreased blood flow to the DRG
- Demyelination

- Edema
- Inflammation
- Hypersensitivity
Herniated Disc
Immune

• Glycoshingolipid antibodies
• Glial cell and nerve damage markers: several proteins elevated in CSF of HNP patients
Non Compressive Radiculopathy

- Diabetes
- Infectious/granulomatous
- Infiltrating Neoplastic
Conservative Treatment

Mechanical/Radiculopathy

• Medications/Oral Analgesics
• Short course oral corticosteroids
• Physical therapy, chiropractic care, acupuncture
• Epidural Injections: radicular
• Cervical traction
• Collar (??), Cervical pillow
Surgical Indications

Radiculopathy

- Unremitting pain despite full non surgical management
- Progressive weakness
- Cervical myleopathy
Cervical Myelopathy

• Exam based
• Ataxia, posterior columns
• Lack of fine motor coordination
• Numbness/pinprick/vibration
• Weakness, not dermatomal
• Upper motor neuron findings: hyperreflexia, positive Babinski,
Cervical Radiculopathy

Natural History

- Brain, 1994, Natural History. Rochester, MN, 561 patients
- Physical exertion or trauma: 14.9%
- C7 > C6
- 4.9 yr f/u. 31% recurrence, 26% underwent surgery
- Final Follow up: 90% asymptomatic or mildly symptomatic
Low Back Pain
Lumbar Radiculopathy
Outcome

• Many recover, but variable
• At 1 yr, 72% completely recovered. 86% little or no disability.
• Annular tears: 38% asymptomatic individuals
Is it Shoulder or Neck?
Cervical Spine Exam
The Shoulder

- ROM: active/passive
- Impingement/RC signs: Neer, Hawkins, Empty beer can, Speed’s
- Cervical salute: generally a cervical, not a shoulder problem. Shoulder abduction sign
Lumbar Spine Exam
The Hip

- IR/ER seated and/or supine
- Foot on opposite knee
- Getting in and out of car
- Limp with ambulation
Non Organic Signs

Waddell’s Criteria

• Suggest behavioral component to pain, psychological distress
• Superficial tenderness
• Distracted straight leg raise (seated v supine)
• Non dermatomal sensory or strength exam
• Cogwheel give way
• Axial loading, truncal rotation
Imaging
Who Needs It?

• Red Flag patients
• Trauma
• Lumbar radiography in LBP doesn’t change the outcome
• Radiographic findings do not correlate with clinical outcomes
Imaging

What Do We Find?

• Asymptomatic pts have DDD, increased with age

• 60-70% pts age 40-80 had facet degenerative changes and no correlation to LBP

• HNPs by MRI in 22-36% asymptomatic adults, 2/3 of HNPs resolve within 12 months

• Disc dessication, protrusions, bulges, decreased disc height common in asymptomatic pts

• Annular tears in 35-40% asymptomatic pts
Imaging
Consequences

• Early MRI in LBP associated with increased cost of care and incidence of surgery

• Incidental finding are common, but often lead to further tests or interventions
Imaging
What To Do

• Plain films: AP and lat, not obliques. >4-6 weeks axial pain with no improvement

• MRI: neurologic urgent conditions, radiculopathies unresponsive to conservative care, worsening weakness, infection, tumor/metastasis, compression fracture

• MRI: children with consistent LBP >3 months, adults >6 months
Treatment Effectiveness
Medications
The Data

• NSAIDS better than placebo
• Acetaminophen about the same
• Muscle relaxants help for acute LBP
• Opioids lack of definitive data, efficacy and addiction and side effect issues
NSAIDs

- Cyclo-oxygenase: key enzyme in making prostaglandins
- COX-1 and 2 inhibitors
- COX-1 Stomach lining protection
- Increased risk of vascular issues
Epidural Injections

The Data

• Efficacious for acute radiculopathy
• Numerous trials have given mixed results
• Meta analysis of data from multiple studies shows favorable results
• Results short lived. Some studies show no reduction in surgery rates
• Questions about efficacy, total number to give, content, best route
CAM Therapies
Acupuncture, Massage, Manipulation

• Furlan, 2010, Cochrane review. 265 RCTs back, neck, thoracic pain

• Acupuncture mixed results

• Manipulation and massage better than placebo for immediate or short term pain reduction. Better than PT in reducing LBP and disability
For Back Pain

Consider Chiropractic First

A recent study on work-related back injuries of equal severity, conducted by a collaboration of prestigious institutions, including two schools of medicine and two schools of public health, along with the Washington State Department of Labor and Industries, concluded:

42.7% of workers who first consulted a surgeon underwent surgery

1.5% of workers who first consulted a doctor of chiropractic underwent surgery
Surgical Effectiveness

• Cervical radiculopathy: better at 4 months, no difference at one year

• Lumbar radiculopathy: SPORT study
  • High quality evidence for discectomy
  • Good evidence for laminectomy in stenosis
New Therapies

• Medications: block nitrous oxide, TNF production
• Disc regeneration, gene expression
• Intra-discal therapies
• Non fusion technology
Clinical Evaluation

Systematic Approach

• History and Physical: Red Flags?
• Mechanical and /or Radicular
• Initiate treatment of choice, informed decision making
• Re evaluate patient, 3-4 weeks: Mech/Rad
• More Rx, maybe image with neuro or rad sx
Patient Education

• They need to be responsible and actively participate

• Tell them “The sky is NOT falling”. I de-educate my patients. Layman’s terms. They are normal. Ignore the radiology report.

• You may hurt, but you are generally not damaging anything
Patient Responsibilities

• Exercise/Get in shape
  • cardiovascular
  • core flexibility and strengthening
• Yoga, Pilates, physio ball
• Quit smoking
• I don’t bother talking weight loss: the deer in the headlights
Referral
When and How Urgent

- “Come on down”
- Serious neurological problems
  - Myelopathy
  - Cauda equina
- Progressive neuro deficit: weakness
- Red flag issues
Referral
When and How Urgent

• “Happy to see”
• Unresponsive to conservative care
• MRI findings that match symptoms
• Stable neuro deficits
• Imaging study issues/findings
Referral
When and How Urgent

• “Happy to confirm your clinical expertise”
• Frustrated, unhappy, mad patients
• Imaging study findings of no clinical consequence
• The specialist’s opinion
Summary

• Develop an approach that is consistent
• Base it on the best data you know
• Manage patient education and expectations
• Minor adjustments to fit your practice patterns
Conclusions

• As Practitioners, we will need to demonstrate our value, or become irrelevant

• Reimbursement is going to flow to value and not volume, and will generally be less

• Currently are criteria to evaluate the treatments available
Thank You
Diagnostic algorithm of low back pain

1. **Low back pain**
   - Presence of sciatica? (occasionally without back pain)
   - **No**
     - Simple back pain (60 percent)
       - Age under 50
       - No signs of sx or systemic disease
       - No hx of cancer
       - (Likelihood of musculoskeletal cause ≈ 0.99)
       - Conservative care for 4 to 6 weeks
         - Improved
           - STOP
         - Not improved
           - Plain film and ESR*
             - If either abnormal, consider CT or MRI
             - Have high clinical suspicion in patient with known cancer and new back pain; or patient with IDU, fever and back pain
             - Close follow-up is warranted
   - **Yes**
     - Complicated back pain without radiculopathy (37 percent)
       - Age over 50
       - Systemic signs, sx, or risk factors: fever, weight loss, hx of prior cancer, hematuria, adenopathy, injection drug use
       - (Probability of systemic disease is 1 to 10 percent, depending on the findings. Most patients still have musculoskeletal pain [95 percent].)
       - Conservative care for 4 to 6 weeks
         - Improved
           - STOP
         - Not improved
           - Plain film and ESR*
             - If either abnormal, consider CT or MRI
             - Have high clinical suspicion in patient with known cancer and new back pain; or patient with IDU, fever and back pain
             - Close follow-up is warranted

2. **Radiculopathy** (3 percent)
   - Signs and sx of radiculopathy, w/o bladder or bilateral findings
   - May also have systemic signs, sx, or risk factors noted in complicated back pain
   - Plain film
     - ESR* if risks for osteomyelitis
       - If normal, conservative care for at least 4 to 6 weeks unless neurologic deficit is progressive
         - Improved
           - STOP
         - Not improved
           - Urgent consultation and CT or MRI to evaluate for cord or cauda equina compression
             - Noncontrast CT or MRI, choice depends on local availability
             - If 12 week failure, meets criteria for subacute low back pain

---

* ESR: erythrocyte sedimentation rate.
  * Some clinicians obtain C-reactive protein test in addition to ESR as an inflammatory marker.
CAM Therapies
Acupuncture, Massage, Manipulation

• Massage effective
• Manipulation small benefit
• Acupuncture was unclear
CAM Therapies
Acupuncture, Massage, Manipulation

• Evidence poor to moderate, difficult to draw definitive conclusions, very few studies with long term outcomes, inconsistent methodologies, clinical diversity, complicating interpretation of trial results
Neck Pain
Axial

- Cervical strain
- Whiplash
- Degenerative discogenic pain
- Cervical facet syndrome
Neck Pain
Treatment

- Little evidence from controlled trials
- Psychosocial factors significant in whiplash duration and severity
Neck Pain

Treatment

• Postural Modification
• Pharmacology
• Home exercises, traction, PT
• Manipulation, massage, low level laser
• Collar generally discouraged
• Trigger point injections, TENS, facet injections/medial branch blocks, botulism
Laboratory Testing

• CBC, ESR, CRP
• RF, ANA, HLA B27,
• Serum/immunoelectrophoresis
Low Back Pain Risk Factors

• Jarvik, Spine, 2005. 148 VA pts, no LBP for 4 months. 3 yr f/u, baseline and f/u MRIs.

• 3 yr incidence of pain: 68%. Depression best predictor of LBP.

• No association with MRI findings of: central stenosis, annular tears, disc deg., facet deg.
Degenerative Disc Disease

Risk Factors

• Genetics
• Age
• Smoking
• Vascular disease
• Heavy lifting, torsional stresses, motor vehicle driving/vibration
Cervical Radiculopathy
Treatment Effectiveness
Non Invasive

- Uncertainty regarding effectiveness
- “Not demonstrated benefit”
- “No better than sham/placebo”, “inconclusive”
- Low quality trails
- Methodologic flaws
Cervical Epidurals

Effectiveness

• Data are weak and inconsistent

• Several small prospective and retrospective observational studies show relief in 40-60% of pts


• 3 week f/u: no difference
Cervical Radiculopathy

Surgical Effectiveness

• Benefit has not been clearly established

• Persson, Eur Spine J, 1997. Prospective controlled study, surgery v. PT/collar

• Pain, sensory, weakness improved within 4 months of surgery

• No difference at one year
Cervical Radiculopathy

Surgical Indications

• Presence of radicular pain after 6-12 weeks of conservative treatment

• **Progressive** weakness

• Myelopathy

• Evidence of nerve compression by imaging *that accounts for* clinical symptoms
**NSAIDs (Non-Steroidal Antiinflammatory Drugs, COX-1 & COX-2)**

**Opiates (mu agonists)**

**Anticonvulsants (phenytoin), antidepressant (amitriptyline), antiarrhythmics (mexilitine)**

**Sumatriptan, Zomig etc for migraine**

**Gabapentin (off label)**

**Tramadol (mu opioid plus ‘your guess as good as mine’)**
Most widely used of all therapeutic agents

— Over 50 NSAIDs on the market

Three main effects

— anti-inflammatory

— antipyretic

— analgesic

Primary mechanism of action is inhibition of arachidonic cyclo-oxygenase (COX) and therefore reduction of prostaglandin levels

— most NSAIDs block both COX-1 and -2 e.g. naproxen, indomethacin, ibuprofen, aspirin etc

Recent selective COX-2 inhibitors — Mobic, Vioxx, Celebrex, Bextra
**Lack of efficacy**

- in chronic pain 40% efficacy in Visual Analogue Scores typical
- Nothing works well in neuropathic pain

**Dose limiting adverse effects**

- not only unpleasant but life-threatening as well

**NSAIDs**

- gastric haemorrhage, renal/kidney toxicity

**Opiates**
Cervical Disc Herniation
Physiological Pain

- ‘nociceptive pain’ activation of C and A\textsuperscript{TM} fibres
- related to actual or potential tissue injury
- initiates ‘protective’ reflexes or behaviour
  - withdrawal from stimulus or ‘guarding’ of affected area

Non-physiological or pathological pain

- pain which continues beyond the point where it serves a physiological purpose

Neuropathic pain

- pain associated with damage to the peripheral or central nervous system
glutamate, aspartate, (homocysteate)

vast body of literature supporting major role in transmission in spinal cord

primary afferent transmitters

UEAAs act on 4 main receptor types

3 ligand-gated ionotropic receptors

- kainate receptor

- AMPA receptor

- NMDA receptor

1 G-protein coupled receptor
Excitatory neuropeptides localised in nociceptive afferents

- Substance P, Neurokinin A,
- receptors NK1 and NK2
- ? transmitters or neuromodulators
- both central and peripheral role (Substance P)

- when released centrally - excitatory, contributes to central sensitisation ‘wind-up’
- when released peripherally - pro-inflammatory ‘neurogenic inflammation’

U Calcitonin Gene-Related Peptide (CGRP)

- localised in greater % of nociceptive afferents than Sub P
Gamma Amino Butyric Acid (GABA) and Glycine

- released from interneurons in spinal cord and supra spinal
- inhibitory by reducing transmitter release
- glycine also has role as modulator of NMDA receptor

5-HydroxyTryptamine (5-HT)

- transmitter in inhibitory neurones from supra-spinal nucleus raphe medialis

Noradrenaline

- inhibitory transmitter from supra-spinal locus ceruleus

Inhibitory/excitatory

Adenosine
Peripheral injury or inflammation initiates cascades of pro-inflammatory mediators released from many tissues.

These agents act on nociceptive nerve terminals - sensitisation:
- decrease in threshold to stimulation
- increase in responsiveness to stimulation

Sensory nerve terminals not only ‘passive’ but contribute actively to the inflammatory process:
- neurogenic inflammation
  - release of neuropeptides, Sub P, CGRP
  - vasodilation of blood vessels
  - activate immunocompetent cells
Two isoforms of COX

- Both produce prostaglandins (PGE$_2$, PGF$_2\alpha$, PGI)

COX-1 is constitutive, expressed in most tissues

- physiological and homeostatic role, cell signalling

COX-2 is inducible following inflammation, trauma etc

- found in immunocompetent cells (e.g. leukocytes)

- pathophysiologica role, initiates, maintains inflammation

Prostaglandins (particularly PGE$_2$) do not directly
Opioid receptors

- 3 subtypes: μ, δ, κ
- About 60% homology between subtypes
- G protein-coupled receptors
- The ‘Grandfather’ of all analgesics - Morphine - acts here
- Many synthetic opiates available
Transient Pain

Brief noxious stimulus

milli
secs

C-/ A\textsuperscript{TM} fibres

transmitter release

brief activation in sc transmission to brain

secs

Peripheral sensitisation

Increase in synaptic efficacy

Central sensitisation

Induction of early genes, c-fos

Upregulation of neuropeptides

Recruitment of A\textsuperscript{\textregistered} fibres

min

Hyperalgesia

Sustained pain

hours

Short term inflammation

Perioplex sensitisation

Inflammation

Central sensitisation

Induction of early genes, c-fos

Upregulation of neuropeptides

Recruitment of A\textsuperscript{\textregistered} fibres

milli
secs

Hyperalgesia

Allodynia

Chronic pain

Pathological

years

Pathological inflammation

Neuropathy

Phenotypic changes

Sprouting of terminals

Inappropriate innervation

Expression of new receptors

Cell loss

days

weeks

months
**EAA receptors**: AMPA, NMDA, mGluR

**Brief Depolarisation**: Excitation

- Glut
- Na$^+$
- Mg$^{2+}$

**Sustained Depolarisation**: Excitation

- Glut
- Na$^+$
- Ca$^{2+}$

PKC, NO
Illustration of an axial view of the cervical intervertebral foramen and adjacent structures at the level of C6 with a needle inserted parallel to the axis of the foramen along its posterior wall.

Adapted from Rathmell, JP, April C, Bogduk, N. Cervical transforaminal injection of steroids. Anesthesiology 2004; 100:1595.
Neck pain

Axial

Radicular

Both
"Red flags" for a potentially serious underlying cause for low back pain

<table>
<thead>
<tr>
<th>Reason</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trauma, cumulative trauma</td>
</tr>
<tr>
<td>Unexplained weight loss</td>
</tr>
<tr>
<td>Age &gt;50 years, especially women, and males with osteoporosis or compression fracture</td>
</tr>
<tr>
<td>Unexplained fever, history of urinary or other infections</td>
</tr>
<tr>
<td>Immunosuppression, or diabetes mellitus</td>
</tr>
<tr>
<td>History of cancer</td>
</tr>
<tr>
<td>Intravenous (IV) drug use</td>
</tr>
<tr>
<td>Prolonged use of corticosteroids, osteoporosis</td>
</tr>
<tr>
<td>Age &gt;70</td>
</tr>
<tr>
<td>Focal neurologic deficit(s) with progressive or disabling symptoms, cauda equina syndrome</td>
</tr>
<tr>
<td>Duration longer than six weeks</td>
</tr>
<tr>
<td>Prior surgery</td>
</tr>
<tr>
<td>History of abdominal aortic aneurysm</td>
</tr>
</tbody>
</table>

Imaging
What To Do

• CT: best for bone windows, not best for HNP
• Bone scan: acute spondylolysis, facet degeneration
Physical Exam

- Inspection
- Palpation
- Range of motion
- Straight leg raise, Spurling’s
Physical Exam

Neurological Exam

• Sensory: know the dermatomes

• Motor: heel and toe walk, single toe raises, seated quads

• L4-5 and L5-S1 discs > 90% lumbar disc herniations

• Deltoids, biceps, triceps, wrist flexors/extensors, intrinsics
Is it Shoulder or Neck?
Low Back Pain

- 2.5% medical office visits, 15% population
- 44 million office visits in 2004
- Katz, JBJS 2006, total annual costs exceed $100B. 2/3 costs are indirect, lost wages/productivity
- 5% pts account for 75% costs
Physiological and Non-Physiological Pain

PHASE 1

PHASE 2

Inflammation

PHASE 3

Nerve or CNS damage

CNS

BRIEF

PERSISTING

ABNORMAL
Conservative Treatment

Radiculopathy

• Medications/Oral Analgesics
• Short course oral corticosteroids
• Physical therapy, chiropractic care, acupuncture
• Traction
• Collar (??), Cervical pillow
Guidelines

• ACP: 2007, Chou et al
Top view of a vertebra and a disk

- Nerve leaving the spinal canal
- Disk
- Foramen
- Ligament
- Lamina
- Nerves in the spinal sac
- Spinal canal
Cross Sectional view of a Lumbar Vertebrae

Vertebrae

Dural Sac

Facet Joint

Lamina
Spine Pain
Low Back and Cervical

• Ubiquitous
• 84% of adults have LBP at some point
• 26% within past 3 months, 14% day of survey
• Prevalence 22-48% in several surveys
Opportunity or Threat?

• $2.6 Trillion U.S. health care expenditures in 2010

• Annual direct and indirect bone and joint expenses in 2008 estimated at $849B.

• 7.7% U.S. GDP

• The “Boomers”