THE CERVICAL SPINE Injury, Diagnosis, & Treatment

Sharon Bassi, MD
The Spine Center
New England Baptist Hospital
Harvard Medical School, Dept of PM&R
Neck Injuries from Trauma
Outline

• Neck pain
  – Epidemiology
  – Causes
  – Cervical degeneration; natural history

• Acute Neck Pain
  – Natural history
  – Physical exam
  – Treatment

• Cervical Radiculopathy
  – Epidemiology
  – Causes
  – Symptoms
  – Physical Exam
  – Natural History
  – Treatment

• Chronic Neck Pain
  – Low threshold pain/sensitization
  – Clinical manifestations
  – Treatment
Anatomy and Pathophysiology

NECK PAIN

Pain perceived in the neck

CERVICAL RADICULAR PAIN

Pain perceived in the upper limb

The causes, mechanisms, investigations, and treatment are different
Neck Pain
Epidemiology of Cervical Pain

• 23% of population have neck pain annually
• 70% of people report neck pain at some point in life
• 10% have chronic neck pain
• 3 - 4% of the adult population presents to allopathic health care providers with neck pain every year
Epidemiology of Occupational Neck Pain

- Highest frequency in hospital and office workers
  - 24% incidence in computer workers
- Lowest in industrial/service and forestry sectors

Epidemiology of Occupational Neck Pain

- Course of Neck Pain in workers
  - Persistent or recurrent in majority
    - Slightly higher in women
  - >60% will report neck pain 1 year later
    - This is consistent with findings about the course of neck pain in the general population and WAD

Epidemiology of Occupational Neck Pain

Prognostic Factors in predicting recovery

- **Poor Prognosis**
  - Little perceived influence over own work situation
  - Prior neck pain and/or prior sick leave
  - Blue collar workers

- **Favorable Prognosis**
  - Workers who engaged in general exercise and sporting activities
  - White collar workers

*Carroll, et al. SPINE. Course and Prognostic Factors for Neck Pain in Workers. 2008*
Epidemiology of Occupational Neck Pain

Prognostic Factors in predicting recovery

- **No or Little association**
  - Age
  - Specific workplace or physical job demands unassociated

- **GAPS in Literature**
  - Impact of coping style, anger, frustration in predicting outcome not looked at
  - Few lifestyle factors (smoking, BMI) considered
  - Role of work related vibration (jackhammers) or work related driving (truck/bus drivers)

Occupational Neck Pain

- Often similar to non-work related neck pain in onset and clinical course
Occupational Neck Pain

• Tension-neck –syndrome in computer workers
  – Limited evidence to suggest causal relationship for overall computer use and mouse use
  – Insufficient evidence to support causal relationship with keyboard use

• Insufficient evidence to suggest that workplace interventions alter the clinical complaints
  – Aas, RW. Cochrane Database Syst Rev 2011
Conclusions

• Neck pain is common!
• Not much difference between frequency of occupational and non-occupational neck pain
• The course of neck pain in work similar to gen population
• Encourage sedentary workers to get active & exercise
• Infrequently associated with work absence
• Most people with neck pain do no seek medical care
What produces symptoms?

• *Idiosyncratic* (and generally benign) interaction between cervical structures (degeneration ?) and the central nervous system
What produces symptoms?

A new Conceptual model of Neck Pain; Bone and Joint Decade
Spine 2008

A biopsychosocial model of health and disease
creating a framework for the study of neck
pain that underscores that many
environmental and personal factors combine
to cause neck pain and to influence its
course....
What produces symptoms?


5 major components

1. Factors affecting the onset and course
   - Any attribute of the person or their environment
     - Genetics
     - Health Behaviors
     - Demographic and socioeconomic
     - Cultural
     - Prior health/prior pain/comorbidities
     - Collision/workplace factors
     - Psychological/social
     - Compensation/laws/societal
What produces symptoms?


5 major components

2. The Care Complex
   - The majority of people with neck pain do not seek health care for neck pain
     - Access to care, personal factors influence decisions
     - Options: no care, self care, professional health care
     - Preferences for certain type of management strategy
     - Patient-provider nexus
What produces symptoms?


5 major components

3. The Participation Complex

- The patient needs to decide whether to modify his/her participation or involvement in life situations
  - Employment
    - Type of job, degree of control over job tasks, incentives to maintain participation, available accommodations, personal factors
  - Leisure
  - Household responsibilities
What produces symptoms?


5 major components

4. The Claim Complex
   - Filing a claim to access financial benefit in the case of insurance
     • Healthcare claims
     • Disability claims
     • Workers compensation claims
     • Personal Injury claims

Likely that the person with neck pain will have to spend time negotiating the insurance system, usually while feeling unwell and in pain
5 major components

5. The Impacts and Outcomes of Neck Pain

- Impact of Neck Pain in 5 Domains
  - Impairments - impact on body structure and function; signs/symptoms
  - Activities - accomplish tasks
  - Participation - involvement in life situations
  - Wellbeing – subjective impact
  - Resource use
What produces symptoms?


5 major components

5. The Impacts and Outcomes of Neck Pain

- **Short term outcomes**: changes in these domains over short term (ie. Post cervical facet injection)

- **Long term outcome**: global personal appraisal of impact of neck pain over months to years → resolution, readjustment, or long term disability (unsatisfactory deficits in activity/participation)
What produces symptoms?

Factors: Demographic and Socioeconomic; Prior Health/Prior Pain/Comorbidities; Collision/Workplace; Psychological and Social; Compensation/Laws/Societal; Genetics; Health Behaviours; Cultural.

EPISODE OF INTERFERING NECK PAIN

Options:
- No Care
- Self-Care
- Health Care

Impacts:
- Impairments
- Activities
- Participation
- Well-being
- Resource Use

Resolution
- Chronic Pain and Disability

Readjustment or Redefinition
- Long-term Outcomes

Still Troublesome
- Short-term Outcomes

As Usual
- Modify Stop

No Claim
- Care Claim
- Disability Claim

No neck pain
- Interfering neck pain
- Non-interfering neck pain

Environment

Guzman et al. A new Conceptual model of Neck Pain; 2000-2010
Task force on Neck Pain; SPINE 2008
What produces symptoms?

Factors: Demographic and Socioeconomic; Prior Health/Prior Pain/Comorbidities; Collision/Workplace; Psychological and Social; Compensation/Laws/Societal; Genetics; Health Behaviours; Cultural.

EPISODE OF INTERFERING NECK PAIN

Options:
- No Care
- Self-Care
- Health Care

Impacts:
- Impairments
- Activities
- Participation
- Well-being
- Resource Use

Options:
- As Usual
- Modify
- Stop

Resolution
- Chronic Pain and Disability

Long-term Outcomes
- Readjustment or Redefinition

Short-term Outcomes
- Still Troublesome

No neck pain
- Interfering neck pain
- Non-interfering neck pain

Guzman et al. A new Conceptual model of Neck Pain; 2000-2010
Task force on Neck Pain; SPINE 2008
Sources of Neck pain

• How to determine the site from which nociception can be generated?
  – must be innervated

  – Physiologic evidence required
    1. normal volunteers: apply noxious stimulation to structures with injections of hypertonic saline
    2. Patients: anesthetize potential pain generator to see if doing so relieve the pain
Sources of Neck pain

- Pain from **facet joints** tends to follow recognizable patterns
- Similar pattern of pain is produced by mechanical stimulation of **cervical disks**
- Therefore, structures innervated by the same cervical segmental nerves have the same distribution of pain
Sources of Neck pain

• Atlanto-occipital and lateral atlantoaxial joints
  – Additional sources of neck pain and headaches
  – Suboccipital pain typically

• Other innervated structures potential sources of neck pain
  – Formal evidence/studies lacking
    • Posterior neck muscles, ligaments
    • Cervical dura mater
    • Median atlantoaxial joint and its ligaments
    • Vertebral artery
Sources of Neck Pain

• Other purported causes of neck pain are extensive
• For most of these conditions, an objective test is not available or has not been applied
• No real evidence therefore
• Most Likely / Valid sources
  – Serious but rare
    • Neoplasm, infection (<0.4% incidence)
    • Vascular
      – carotid artery dissection (6% with only neck pain)
      – Vertebral artery dissection (50-90% the first presenting complaint is neck pain, but usually also with headache)
Sources of Neck Pain

• Most Likely / Valid sources
  – Valid but rare
    • Inflammatory arthropathies
      – Rare causes of neck pain alone
      – RA, ankylosing spondylitis, crystal arthropathies/gout
    • Polymyalgia Rheumatica
      – Not isolated neck pain; a systemic disorder
    • Longus colli tendonitis
      – Aka retropharyngeal tendinitis; inflammation and edema of upper portions of longus colli
    • Fractures
      – Not all fractures cause neck pain (<0.4% incidence)
Sources of Neck Pain

- Detectable but of Questionable validity
  - DISH (diffuse idiopathic skeletal hyperostosis)
    - Often asymptomatic, or with dysphagia/stiffness
  - Ossification of PLL
  - Pagets Disease
  - Spondylosis/degenerative disease
  - Osteoarthritis
  - Synovial cyst

Most commonly applied diagnosis; neither is valid
Sources of Neck Pain

• Neurologic
  – Cause upper limb, not neck, symptoms
    • Thoracic outlet syndrome
    • Spinal cord tumors
    • Nerve injuries
    • Myelopathy
    • Radiculopathy
Sources of Neck Pain

• Spurious or Vague
  – These labels often applied to most patients with neck pain
    • Soft tissue injury “something has been injured but no fracture”
    • postural disorder
    • Whiplash; describes possible cause of pain but not the source
    • myofascial pain; pathophysiology not fully understood; debated
    • Cervical strain “something went wrong with the neck to produce pain”
    • fibromyalgia
    • Psychogenic “I don’t know what’s wrong”
Sources of Neck Pain

- Detectable but of Questionable validity
  - DISH (diffuse idiopathic skeletal hyperostosis)
    - Often asymptomatic, or with dysphagia/stiffness
  - Ossification of PLL
  - Pagets Disease
  - Spondylosis/degenerative disease
  - Osteoarthritis
  - Synovial cyst

Most commonly applied diagnosis; neither is valid
The cervical spine changes over time.

Age related changes
These changes are weakly...if at all, associated with pain

Age related changes
Prevalence of “degenerative” spine imaging findings in ASYMPTOMATIC populations

<table>
<thead>
<tr>
<th>IMAGE FINDING</th>
<th>20</th>
<th>30</th>
<th>40</th>
<th>50</th>
<th>60</th>
<th>70</th>
<th>80</th>
</tr>
</thead>
<tbody>
<tr>
<td>Disc Degeneration</td>
<td>37%</td>
<td>52%</td>
<td>68%</td>
<td>80%</td>
<td>88%</td>
<td>93%</td>
<td>96%</td>
</tr>
<tr>
<td>Disc Signal Loss</td>
<td>17%</td>
<td>33%</td>
<td>54%</td>
<td>73%</td>
<td>86%</td>
<td>94%</td>
<td>97%</td>
</tr>
<tr>
<td>Disc Height Loss</td>
<td>24%</td>
<td>34%</td>
<td>45%</td>
<td>56%</td>
<td>67%</td>
<td>76%</td>
<td>84%</td>
</tr>
<tr>
<td>Disc Bulge</td>
<td>30%</td>
<td>40%</td>
<td>50%</td>
<td>60%</td>
<td>69%</td>
<td>77%</td>
<td>84%</td>
</tr>
<tr>
<td>Disc Protrusion</td>
<td>29%</td>
<td>31%</td>
<td>33%</td>
<td>36%</td>
<td>38%</td>
<td>40%</td>
<td>43%</td>
</tr>
<tr>
<td>Annular Fissure</td>
<td>19%</td>
<td>20%</td>
<td>22%</td>
<td>23%</td>
<td>25%</td>
<td>27%</td>
<td>29%</td>
</tr>
<tr>
<td>Facet Degeneration</td>
<td>4%</td>
<td>9%</td>
<td>18%</td>
<td>32%</td>
<td>50%</td>
<td>69%</td>
<td>83%</td>
</tr>
<tr>
<td>Spondylolisthesis</td>
<td>3%</td>
<td>5%</td>
<td>8%</td>
<td>14%</td>
<td>23%</td>
<td>35%</td>
<td>50%</td>
</tr>
</tbody>
</table>

*Ishimoto Y, et al
Osteoarthritis and Cartilage 2013*
Etiology of Spine Degeneration

Initiation of process

- Genetics - 73% of degeneration of cervical spine is predicted by heritability
  - (Sambrook, Arthritis Rheum. 1999)

- (Epigenomics - changes in expression of DNA that are induced by factors other than DNA sequences)
  - gene interactions, prior gene activation, environmental factors
Etiology of Spine Degeneration

• Altered Cell Function
  – Altered cell phenotype
  – Cell senescence
  – Apoptosis

• Reduced synthesis of correct matrix and increased catabolism
Etiology of Spine Degeneration

• Cell mediated changes in disc matrix
  – Loss of proteoglycans
  – Abnormalities of the collagen fibrils

Lemaitre CL. Arthritis Res Ther, 2007
Etiology of Spine Degeneration

- Structural changes to the disc
  - Nucleus pulposus
    - Desiccation
    - Fissuring
    - Fibrosis
  - Annulus fibrosis
    - Weakening of lamellae
    - Tears that propagate through annulus (radial tears)
    - Delamination (concentric tears)
  - End plate separation

Natural History of Cervical Degeneration

• Begins during the second decade of life!
Natural History of Cervical Degeneration

• Twenties
  – disc degeneration/
    Fissuring
Etiology of Spine Degeneration

- Thirties and Forties
  - Changes in disc morphology
    - Loss of disc height
    - Distortion of the annulus
    - Failure of the annulus to contain the nucleus pulposus → HNP
    - Becomes more common in 30s

- Most noted at C4-5, C5-6 and C6-7
Etiology of Spine Degeneration

- Cell mediated changes in facets
  - Cartilage degeneration
  - Joint capsule degeneration
  - Bony hypertrophy
  - Reduced function

- Ligament degeneration

- Activation of reactive changes in adjacent bone
Natural History of Cervical Degeneration

Fifties, Sixties and beyond

“Cervical Spondylosis”
- Vertebral osteophytes
- Bridging osteophytes
- Facet Degeneration
- Foraminal stenosis
- Central Canal Stenosis
Spine Degeneration

- CAUSED BY LOSS OF CELL FUNCTION
- Occurring in everyone as part of aging
- Individual factors affect the onset and progression

- *Doesn’t greatly affect the spine*
  - Negligible impact on motion
    - ROM $\downarrow$ 4°/decade (Chen 1999)
  - No impact on muscle function
  - Rarely interferes with neurological function
  - Occasionally stimulates the pain system
Causes of Spinal Degeneration

• No evidence that office work accelerated cervical spine degeneration
Are pain symptoms important?

Probably not!

• 30% of the population never experience neck pain
  – These people seem to develop degeneration normally
  – Monitoring neurons seem resistant to chemical and mechanical stimulation generated during spine degenerative, and by physical exposures
  – These people are not harming themselves by functioning normally in the presence of degeneration and exposures

  – These people are not missing anything!
Determinants of pain experience

1. Genetic differences in pain sensitivity
2. Speed of development of degenerative changes
   – Abrupt changes may be more likely to produce symptoms
3. Ability of the pain systems to adapt to the development of spine degeneration
   – Changes in gene expression
4. Intellectual and emotional response to pain.
   – May influence gene expression

Onset of Neck Pain/Radiculopathy

- Pain neurons are stimulated by (often sudden) degeneration of spine structures, such as progression of annular tears or acute disc herniation, inflammation of facet joint, end plate change, soft tissue change, etc

- Onset
  - 85% spontaneous
  - 15% with exertion, trauma, etc
    - (Radhakrishnan, 1994)
Acute Neck Pain

• Posterior or lateral neck
• Often radiate to upper back or shoulder(s)
• Can produce occipital headaches
• Pain influenced by neck positions and movements
Examination – Cervical Motions
Note range and association with pain
Common shoulder disorders can mimic neck pain

- Rotator cuff tendinopathy/tears
- “Impingement” Syndromes
- “Bursitis”
- Bicipital tendonitis
- Acromio-clavicular joint problems
- Adhesive capsulitis
- Glenohumeral arthritis
- Glenohumeral instability
- Glenoid labrum tear
Cervical Vs Shoulder Disorders

**Cervical**

- Pain frequently referred to scapula (trapezius) area
- Pain increases with neck movements and positions

**Shoulder**

- Pain usually in lateral shoulder radiating to lateral upper arm
- Pain increases with shoulder movements and use (elevation)

A secondary myofascial pain component can occur with either
Acute Cervical Pain – Natural History

- Fairly Constant - Severe
  - Intermittent - Positional
    - Highly variable - Positional
      - Resolved
Acute Neck Pain

- Symptoms resolve after several weeks to months
- Recurrence rate is high (30%)
- 10% result in chronic symptoms
- Most cases recover despite treatment
  - Advice to resume normal activities, provide home exercises
Acute Cervical Pain

• **Diagnostic test** - None

• **Treatment**
  - Education
  - Reassurance
  - Advise to return to activities as tolerated
  - Pain management - NSAIDS, Analgesics (Tylenol)
  - Physical Therapy - only if symptoms persist or pain behaviors are severe
Natural History - Symptom Resolution

• Favorable change in pain stimulus threshold
  – Healing (?)
    • Reduction of inflammation associated with inciting process
    • Symptoms resolve – degeneration persists
  – Adaptation of pain neurons
    • Less responsive to stimulus over time
    • Recalibrate to ignore the degenerative changes
Acute Neck Pain $\rightarrow$ 10% Chronic

- Chronic neck pain
  - for these patients, knowledge of sources and possible causes of pain is pertinent
  - Cervical zygapophysial joint pain is common, only valid data for this dx
    - Most common source of pain post whiplash injury
  - Chronic neck pain without history of trauma
    - More challenging to diagnose
The causes, mechanisms, investigations, and treatment are different.
Cervical Radiculopathies

Radiculopathy: neurologic condition with objective signs of neurologic Function loss; sensory, motor, impaired reflexes; block of conduction along axon; loss of nerve function

Versus....

Radicular pain: involves compression/irritation of DRG or possible inflammation of nerve roots; pain is perceived deeply
Cervical Radicular Pain

- Perceived deeply
- Unlike sensory loss, pain is not dermatomal → more myotomal distribution
- C5: pain in arm
- C6-C8: arm, forearm, hand
- Pain involves cutaneous, and deeper muscle, and joint afferents
  - Segmental innervation of deep tissues is not the same as skin
  - Ie. Shoulder girdle muscles innervated by C6/C7
# Cervical Radiculopathies

<table>
<thead>
<tr>
<th>Structure</th>
<th>Condition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intervertebral disc</td>
<td>Protrusion, Herniation, Osteophytes</td>
</tr>
<tr>
<td>Zygapophysial joint</td>
<td>Osteophytes, Ganglion, Tumor, Rheumatoid arthritis, Gout, Ankylosing spondylitis, Fracture</td>
</tr>
<tr>
<td>Vertebral body</td>
<td>Tumor, Paget’s disease, Fracture, Osteomyelitis, Hydatid, Hyperparathyroidism</td>
</tr>
<tr>
<td>Meninges</td>
<td>Cysts, Meningioma, Dermoid cyst, Epidermoid cyst, Epidural abscess, Epidural hematoma</td>
</tr>
<tr>
<td>Blood vessels</td>
<td>Angioma, Arteritis</td>
</tr>
<tr>
<td>Nerve sheath</td>
<td>Neurofibroma, Schwannoma</td>
</tr>
<tr>
<td>Nerve</td>
<td>Neuroblastoma, Ganglioneuroma</td>
</tr>
</tbody>
</table>
Cervical Radiculopathy – common causes

- Cervical disc herniation
  - younger

- Neural Foraminal Stenosis
  - older

- Incidence – 80 / 100,000
  - Increases 3 fold during early 50s
  - Higher incidence in men

Cervical Radiculopathies - Symptoms

• Onset frequently abrupt and spontaneous
  – Only 15% with history of preceding physical exertion or trauma (Radhakrishnan, 1994)

• Shoulder – arm – hand pain - often severe; shooting, stabbing, electric
Neurological Symptoms

• Ill defined numbness and tingling
  – radial aspect of forearm/thumb (C-6)
  – index & middle finger tips (C-7)
• Very rarely involves little finger (C-8)
• Ill defined weakness
  – Hand feels weak for lifting items, opening jars
  – Most people are unaware of weakness even when present!
Cervical Radiculopathies

• Majority involve C-6 and C-7 roots (85%)
  – Lesion at C5-6
  – Lesion at C6-7

• Rarely affect C-8 roots (less than 5 %)
  – C7-T1 lesion

• Occasionally affect C-5 Root
  – C4-5 lesion

\[ C7 > C6 > C8 > C5 \]
## Cervical Radiculopathy

<table>
<thead>
<tr>
<th>Nerve Root</th>
<th>Pain Distribution</th>
<th>Motor</th>
<th>Sensory</th>
<th>Reflex</th>
</tr>
</thead>
<tbody>
<tr>
<td>C4</td>
<td>Lower neck, trapezius</td>
<td>None</td>
<td>Cape distribution (lower neck and shoulder girdle)</td>
<td>None</td>
</tr>
<tr>
<td>C5</td>
<td>Neck, medial scapula, shoulder, lateral arm</td>
<td>Shoulder abduction, elbow flexion</td>
<td>Lateral upper arm</td>
<td>Biceps</td>
</tr>
<tr>
<td>C6</td>
<td>Neck, lateral forearm, first and second digit</td>
<td>Elbow flexion, wrist extension</td>
<td>Lateral forearm, first and second digit</td>
<td>Brachioradialis</td>
</tr>
<tr>
<td>C7</td>
<td>Neck, medial scapula, dorsal forearm, third digit</td>
<td>Elbow extension, wrist flexion</td>
<td>Dorsal forearm, third digit</td>
<td>Triceps</td>
</tr>
<tr>
<td>C8</td>
<td>Neck, medial forearm, fifth digit</td>
<td>Finger flexors, finger abduction and adduction</td>
<td>Medial forearm, fourth and fifth digit</td>
<td>None</td>
</tr>
</tbody>
</table>
Cervical Radiculopathy

A

C5

Medial to the shoulder blade

B

C6

C

C7

D

C8
# Neurological Examination for Cervical Radiculopathies

<table>
<thead>
<tr>
<th>Level</th>
<th>Sensory</th>
<th>Motor</th>
<th>Reflexes</th>
</tr>
</thead>
<tbody>
<tr>
<td>C-5</td>
<td>Lateral Elbow</td>
<td>Elbow flexion</td>
<td>Biceps</td>
</tr>
<tr>
<td>C-6</td>
<td>Radial aspect of Thumb to Wrist</td>
<td>Forearm pronation</td>
<td>Brachio-radialis</td>
</tr>
<tr>
<td>C-7</td>
<td>Tips of Index finger</td>
<td>Elbow extension</td>
<td>Triceps</td>
</tr>
<tr>
<td>C-8</td>
<td>Little finger</td>
<td>Intrinsic hand muscles</td>
<td>Finger flexors</td>
</tr>
</tbody>
</table>
C5 – Elbow Flexion (biceps)

“Pull up.”
C6 – Wrist extension

“Hold your wrist back.”
C6 (better) – Forearm pronation

“Flip your hand over so that your palm is facing downward.”

Rainville, Spine  2007
C7 – Elbow Extension (triceps)

“Push your hand downward.”

“Don’t let me punch you in the nose.”
C8 - Hand and fingers

“Hold your fingers apart.”
(Ulnar nerve)

“Point your thumb towards the floor.”
(Medial nerve)

“Hold your finger straight.”
(Radial nerve)
Sensory – Pin prick in distal extremes of dermatomes

C5

C6

C7

C8
C-5
Biceps
C-6
Brachioradialis
C-7
Triceps
Nerve tension signs

- Spurling’s Test – extension, side flexion and rotation of the neck to the symptomatic side (can add axial compression)

93% specific, 30% sensitive
Natural History of Cervical Radiculopathies

• Cervical Disc Herniation
  – Symptoms severe for several weeks
  – For 85% - symptoms improve over 2-3 months
    • (Saal ,1996; Busch, 1997; Mochida, 1998; Swezey, 1999)
  – Resolution of symptoms
    • Reduction of inflammatory reaction
    • Re-absorption of herniation (Maigne, 1994; Mochida, 1998)

• Neurological adaptation
Treatment for Cervical Radiculopathies

• Education
  – Explanation of pathology
  – Explanation of recovery process
  – Reassurance –
    • Excellent prognosis
    • Progression of neurological symptoms is rare

• Advice
  – Stay as active as possible
  – No advantage for inactivity
Treatment of Radiculopathies

Time
Adaptation Requires **Time**

- **Keep busy** - *spread the misery around*
- **Keep moving** - *prevent deconditioning*
- **Keep Distracted** — *time passes more quickly*
- **Keep working** - *make some money while you’re miserable*
Treatment of Radiculopathies

Medications

• Analgesics
  – NSAIDS
    • Celecoxib/meloxicam (less GI SE’s)
    • Naproxen (less cardiac SE’s)

• Oral Steroids

• Muscle Relaxants

• Antidepressants

• Anticonvulsants

*Most conservative treatments are based on case reports and anecdotal evidence rather than robust RCT’s*
Treatment of Radiculopathies
Physical Therapy

• Usually not needed
  – Not effective for reducing pain or improve strength deficits

• Can be useful to encourage activation
  – Exercises
    • Minimize secondary deconditioning
    • Treat kinesiophobia
Favorable outcomes have been reported; short term and in some cases long term relief.

Some risk of serious complications (<1% risk).

Interlaminar approach.

Treatment of Radiculopathies

- Surgical Decompression
  - Persistent severe symptoms
  - Progressive motor loss
  - Less than 10% of cases
- Anterior cervical discectomy & fusion (ACDF)
- Posterior decompression

Unclear whether long term outcomes are improved compared with non-operative tx

Treatment of Cervical Radiculopathies

• Alternative treatments
  – Chiropractic
    • Manipulations?
  – Modalities
    • electrotherapy/TENS
    • Cryotherapy
    • Thermotherapy
  – Manual therapy
  – Traction

Supportive literature – none

• Acupuncture

Supportive literature – none

Chronic Neck Pain

• Nothing unique about degeneration
• Common problem with poorly understood pathophysiology
• 50% show moderate disability at long term follow up
• Neurological Phenomenon
  – *Low threshold pain* - pain that is generated by stimuli that are not harmful, nor of adequate intensity to stimulate the pain neurons when they are functioning normally
Chronic Neck Pain

• Neurological Phenomenon
  – Low threshold pain
  – Central Sensitization
    • an amplification of neural signaling within the central nervous system that elicits pain hypersensitivity
    • increased responsiveness of nociceptive neurons in the central nervous system to their normal or subthreshold afferent input
    • an augmentation of responsiveness of central neurons to input from unimodal and polymodal receptors
Low pain threshold is not low pain tolerance!!!
Low Threshold Pain

**Components**

An alteration in the manner in which the nervous system encodes and processes sensory information, rather than ongoing nociception from tissue injury.
PAIN PROCESSING

Cognitive appraisal
emotional reaction
attention
behavioral response

Thalamocortical relays
Pain neuromatrix: ACC, Insula, PFC, amygdala, Hypothalamus, sensory cortex

Autonomic reactivity
Spinothalamic transmission

Noxious stimuli
Dorsal horn

PAIN NEUROMATRIX [e.g., ACC, insula, PFC, amygdala, hypothalamus, sensory cortex]
Chronic Pain; Low Threshold

- Chronic pain is associated with structural nervous system/brain changes.

- A wide range of areas in the brain, called the PAIN MATRIX:
  - Amygdala, hypothalamus, periaqueductal gray, basal ganglia, cortex, insula, anterior cingulate cortex.

- So that the threshold for generating pain falls and its duration, amplitude, and spatial distribution increase.
Primary problem appears to be changes in the spinal cord

- **Sensitization of Wide Dynamic Range Neurons**
  - Reduced pain threshold
  - Amplification of pain response
  - Spread of pain sensitivity to non-injured areas

Ji, Trends Neurosci, 2003
Enhanced Central Pain Processing

- Generalized reduction in pain threshold
- CLBP similar to Fibromyalgia

Giesecke et al, Arthritis and Rheum, 2004
Low Threshold Pain Influences, and is Influenced by the Brain

- Pain gets our attention and causes us to worry
- Chronic pain is discouraging
- Cognitive / emotional factors lower the threshold to painful stimuli
- anterior cingulate, insular, and secondary somatosensory cortices showed reduced activation when pain was perceived to be controllable

Salomons et al, *J of Neuroscience, 2004*
Amygdala

- Amygdala integrates pain information with our emotional state

- Amygdala either inhibits or facilitates pain depending on our thoughts and emotion

Neugebauer et al, Neuroscientist 2004
Tendency to develop low threshold pain may be genetically based

- Humans pain susceptibility genes
  - Mogil
  - Macgregor
  - Woolf
Chronic Neck Pain

Lack of evidence for central sensitization in idiopathic, neck pain; Systematic review


- Majority of patients with chronic traumatic neck pain (i.e. whiplash) are characterized by CS

- chronic idiopathic neck pain. The available evidence suggests that CS is not a major feature, however individual cases might have CS pain

- Limitations: only 6 article met criteria ; all case control
Chronic Pain – Key Points -

• Pain is a biopsychosocial experience that goes well beyond mere nociception

• Complex process by which somatosensory information is transformed into the physiological, cognitive, affective, and behavioral response labeled as pain

• magnitude of tissue damage may be out of proportion to the reported pain experience
Chronic Pain – Key Points -

A psychophysiologica phenomenon

Pain, whether linked with injured tissue, inflammation, or functional impairment, is mediated by processing in the nervous system. In this sense, all pain is physical.

Yet, regardless of its source, pain may result in hypervigilance, threat appraisals, emotional reactions, and avoidant behavior. So in this sense, all pain is psychological.

Value of Low Threshold Pain

None!
Manifestations of Low Threshold Pain

• **Hyperalgesia** - heightened response to painful stimulus

• **Mechanical allodynia** – pain produced by non painful stimulus

• **Kinesiodynia** – pain produced by harmless movements, positions and physical stresses
Treatment

• For all treatment, effectiveness is based on the ability to improve the interaction of the pain system with peripheral tissues

• Stimulus threshold must be raised so that pain is not easily induced during benign activities
Treat the Symptoms

• Pain Medications

• Anti-inflammatory medications

• Muscle Relaxants
• 660 % increase in use for spinal pain over 10 years (Martin, Spine, 1996)

• Evidence of effectiveness is minimal if any (Martell, Ann Intern Med 2007)

• **Opioid for > 7 days increases risk for chronic back pain and disability** (Webster, Spine 2007; Franklin, Spine 2008)

• Opioid usage may block pain improvement from other treatments and lead to opioid induced hyperalgesia
Anti-inflammatory Medications

- Over 50 Trials
- Slight advantage over placebo
- No advantage over acetaminophen
- Various types are equal in effectiveness
  - Van Tulder, Spine 2000
Muscle Relaxants

• Usually reserved for acute pain

• No Advantage over anti-inflammatory medication

• No additive benefits over anti-inflammatory medications alone
  – Cherkin, Spine 1996
Advice about Activity

• Get back to normal activities/work

• No evidence that activity avoidance is therapeutic, or necessitated by chronic neck pain, or spinal degeneration

• No evidence of increased risk for additional injuries

  — Hagen, Spine 2002
Change Cognition through Education

• Cellular etiology of spine degenerative
  – Lack of impact of work/activities on progression of degeneration

• Lower pain threshold is the problem
  – Demonstrate
Change Cognition through Education


CENTRAL and PERIPHERAL SENSITIZATION
Movement and activity induced pain does not indicate harm!

• Pain indicates the stimulus threshold has been reached, not that harm is occurring

• Because pain inducing activities are harmless, we can *choose* to continue activities in the presence of pain without doing harm
Physical Therapy

Exercise has been the main winner in research for chronic neck pain

- Jensen I, Bergstrom et al., Pain, 2005
- Ylinen et al., JAMA, 2003 and J. Strength Cond Res 2006 (Finland)
Shoulder Girdle Exercises
Summary

• Most cervical pain is caused by benign, age-related degeneration (loss of cell function)

• Most episodes of cervical pain are brief, and can be managed with education, OTC medications, and reassurance and exercise
Summary

• Cervical radiculopathy has a very favorable prognosis, and can be managed with education, time and judiciously prescribed medications. Epidural steroid injections for refractory cases is reasonable.

• Chronic neck pain is best managed with reassurance, education, advice to stay active and exercise
References

8. Woolf. What is this thing called pain?. The Journal of Clinical Investigation. 2010
Thank you!