THE AGING DISORDERS OF THE LUMBAR SPINE

HOW DISCS AGE AND THE INFLUENCE OF GENES AND THE ENVIRONMENT
IS IT THE DISASTER PATIENTS FEAR?
LOW BACK = MODERN EPIDEMIC

- 106 MILLION SICK DAYS PER YEAR
- £3.8 BILLION LOST PRODUCTION
- £1.4 BILLION IN BENEFITS
- 14 MILLION GP VISITS
- 1.6 MILLION HOSPITAL VISITS
INTERVERTEBRAL DISC

- SHOCK ABSORBER
- ALLOWS MOVEMENT
- CAR TYRE
FIBROUS TISSUE LAYERS
ATTACHED TO BONEY END PLATES VIA SHARPEY’S FIBRES
ANTERIOR ANNULUS TWICE AS THICK AS POSTERIOR
NUCLEUS PULPOSUS

- ECCENTRIC – CLOSER POSTERIORLY
- LARGE NO. OF CELLS
- BIOCHEMICAL FEATURES OF BOTH FIBROCARTILAGE AND HYALINE CARTILAGE
YOUNG DISC

- BULGING MUCOID NUCLEUS
- DENSE COLLAGENOUS ANNULUS
- WELL DEFINED CARTILAGINOUS END PLATES
MOLECULAR THERAPY OF THE INTERVERTEBRAL DISC [Eur Spine J 2006]

- THE INTEGRITY OF THE DISC RELIES ON THE PROPER BALANCE BETWEEN MATRIX SYNTHESIS AND DEGRADATION

- REF [1]
AGEING DISC

- STRUCTURAL ANNULAR CHANGES
- CONCENTRIC FISSURES
- LOSS OF CELLULAR DETAIL
- NUCLEUS BLENDS WITH ANNULUS
- NUCLEUS = TANGLED MASS OF FIBROUS TISSUE
FLEXION FORCES CAN PRODUCE ANTERIOR DISC INJURY WITHOUT SCIATICA

LEADS TO ACCELERATED DEGENERATIVE OR AGEING CYCLE
COLLAGEN = 70% ANNULUS DRY WT.[6-25% NUCLEUS]

PROTEOGLYCANS ATTRACT WATER VIA OSMOSIS – GEL

WATER = 80% NUCLEUS , 65% ANNULUS

SOME ELASTIN
ABILITY OF THE DISC TO RESIST COMPRESSION DEPENDS UPON ITS HIGH PROTEOGLYCAN CONCENTRATION [MAJORITY IS AGGRECAN]

- OSMOTIC PRESSURE
- REF[2]
THE VERTEBRAL ENDPLATE

- THE VERTEBRAL ENDPLATES ARE CRITICAL FOR MAINTAINING DISC FUNCTION REF[3]
- MINOR DAMAGE TO A VERTEBRAL ENDPLATE LEADS TO PROGRESSIVE STRUCTURAL CHANGE IN THE ADJACENT DISC REF[4]
NERLICK et al SPINE 1997

EVIDENCE SHOWS CHANGES OCCUR EARLY

8 – 13 YEARS

AUTOPSY EVIDENCE
DISC STRUCTURE WITH AGEING

- PROTEOGLYCAN POPULATION DECREASES
- REDUCED PG = REDUCED OSMOTIC PRESSURE
- COLLAGEN NETWORK + ELASTIN DISORGANISED
- REDUCED HYDROSTATIC PRESSURE = MORE STRESS IN ANNULUS / END PLATES
SPONDYLOYSIS DEFORMANS

- DISC DESSICATES
- DISC STIFFENS
- MICROSCOPIC ANNULAR TEARS
- FIBROCARTILAGE METAPLASIA
- CLEFTS IN DISC TISSUE ON X-RAY = VACUUM PHENOMENA
- END PLATE DISRUPTS [MICRO #]
- ENDOCHONDRAL OSSIFICATION
LOADS ON THE LUMBAR DISCS

- TAKING STANDING AS 0%
- 5 DEGREE TILT INCREASES PRESSURE BY 25%
- SITTING BY 40% [SEDENTARY OCCUPATIONS]
- FORWARD BEND WITH ROTATION UP TO 400%

INTRA DISCAL PRESSURE
THE RATE OF DISC AGING

- ACCELERATED AGING OCCURS:
  - 1. **INHERITED** [TWINS STUDY]
  - 2. LABOURING
  - 3. PREVIOUS INJURY
  - 4. SMOKING
  - 5. MEDICAL CONDITIONS [RA, DIABETES etc]
  - 6. ?WEIGHT
BIOLOGY OF DISC AGEING

- SPINE 2004
- IT IS NOW APPRECIATED THAT THE METABOLISM OF DISC CELLS IS INFLUENCED BY THEIR MECHANICAL ENVIRONMENT, PARTICULARLY COMPRESSIVE LOADING WHICH CAN INFLUENCE BOTH MATRIX TURNOVER AND CELL VIABILITY. IT DEPENDS UPON THE TYPE AND EXTENT OF LOADING FOR EACH INDIVIDUAL REF[5]
Spine 2004: It is concluded that probably any abnormal loading conditions, including overload and immobility, can produce tissue trauma and/or adaptive changes that may result in disc degeneration.

There appears to be a ‘safe window’ in which discs remain healthy.

REF 6
DISC STRUCTURE WITH AGEING

- PROTEOGLYCAN POPULATION DECREASES
- REDUCED PG = REDUCED OSMOTIC PRESSURE
- COLLAGEN NETWORK + ELASTIN DISORGANISED
- REDUCED HYDROSTATIC PRESSURE = MORE STRESS IN ANNULUS / END PLATES
Eur Spine J (2006)

**DDD = ACCUMULATION OF ENVIRONMENTAL FACTORS PRIMARILY MECHANICAL INSULTS IMPOSED UPON THE ‘NORMAL’ AGEING CHANGES.**

THESE INCLUDE OCCUPATION, SPORTING ACTIVITIES, SPINAL INJURY, SMOKING + ATHEROSCLEROSIS

**TWINS STUDY = GENETIC FACTORS [ RISK OF DEVELOPING DDD UP TO 6X GENERAL POPULATION**

**IT IS LIKELY THAT DDD IS A COMPLEX, MULTIFACTORIAL DISEASE DETERMINED BY INTERPLAY BETWEEN GENES AND ENVIRONMENT**

REF 7
BATTIE et al 2004 + 2009

MONOZYGOTIC TWIN STUDIES = HEREDITY WITH A DOMINANT ROLE IN DISC DEGENERATION. ENVIRONMENT HAS A MODEST ROLE \[\text{REF[8]}\]

STUDY FROM 1991 SUBSTANTIAL EFFECT OF HEREDITY ON LUMBAR DEGENERATION BUT LOADING HAS LITTLE EFFECT. LARGER EFFECT FROM MUSCLE STRENGTH AND BODY WEIGHT \[\text{REF[9]}\]
WHAT IS INTERVERTEBRAL DISC DEGENERATION AND WHAT CAUSES IT?

- Spine 2006
- THE PROCESS OF DISC DEGENERATION SHOULD BE DEFINED AS AN ABERRANT, CELL MEDIATED RESPONSE TO PROGRESSIVE STRUCTURAL FAILURE.
- DEGENERATIVE DISC DISEASE INFERS SYMPTOMS.
- UNDERLYING CAUSE IS TISSUE WEAKENING FROM GENETIC INHERITANCE, AGEING, NUTRITIONAL COMPROMISE AND LOADING HISTORY.
- THE PRECIPITATING CAUSE IS STRUCTURAL DISRUPTION FROM INJURY OR FATIGUE FAILURE
- REF 10
DOWNWARD SPIRAL OF PAIN AND DISABILITY

- Discs degenerate
- More load on facets
- Potential nerve entrapement
- Less function
- Weaker stabilisers
- Prone to more attacks
- Fear avoidance
- Frustration and depression
DEGENERATIVE DISC AND ENDPLATE CHANGES ON MRI
DISC AGING OCCURS IN MID 20’S

AKIN TO A CAR TYRE DEFLATING [STIFFER AND SHORTER]

80% OF POPULATION WILL HAVE SOME LBP

VAST MAJORITY IS SELF LIMITING

DEGENERATIVE DISC DISEASE SHOULD REFER TO SYMPTOMATIC DEGENERATION

NATURAL HISTORY!
Figure 1.3 The incidence of pathology in the normal population

- Bulging disc
- Herniations
- Degenerative disc

Incidence of pathology (%)

Age (years)

20-39 40-59 60-80

BODEN et al ABNORMAL MRI IN NORMAL SUBJECTS; JBJS 1990

SPINEWORKS
IT IS LIKELY DDD = COMPLEX, MULTIFACTORAL DETERMINED BY INTERPLAY BETWEEN GENES + ENVIRONMENT [CHAN et al]
POSSIBLE SERIOUS SPINAL PATHOLOGY

1. AGE <20 or >55
2. VIOLENT TRAUMA
3. CONSTANT PROGRESSIVE NON-MECHANICAL PAIN
4. PMH CARCINOMA
5. WEIGHT LOSS
6. DRUG ABUSE / HIV
7. WIDESPREAD NEUROLOGY
8. CAUDA EQUINA

SUSPICIOUS CLINICAL FEATURES INVESTIGATED VIA RADIOLOGY AND BLOOD TESTS
REFERAL
YELLOW FLAGS

- Yellow flags are psychosocial factors shown to be indicative of long term chronicity and disability:
  - A negative attitude that back pain is harmful or potentially severely disabling
  - Fear avoidance behaviour and reduced activity levels
  - An expectation that passive, rather than active, treatment will be beneficial
  - A tendency to depression, low morale, and social withdrawal
  - Social or financial problems

PROVINCE OF A CLINICAL PSYCHOLOGIST
BACK PAIN HISTORY TAKING

- MOST IMPORTANT TO LISTEN TO THE PATIENT
- PAIN QUALITY
- NIGHT OR RESTING PAIN
- EXACERBATING OR RELIEVING FACTORS
- FACET SYMPTOMS
- INSTABILITY PAIN
WADDELL’S SIGNS

- PURPOSE

Waddell's signs were developed to identify psychogenic, or nonorganic, manifestations of pain in patients that may have heightened emotional effects on their conditions. In order for these signs to be significantly correlated with disability, three of the five signs should be present, *Waddell et al. in 1980.*[^4] They have been also associated with detecting malingering in patients with complaints of lower back pain.
WADDELL’S SIGNS

TECHNIQUE

1. Superficial and Widespread tenderness or Non-anatomic tenderness. (Skin discomfort on light palpation or tenderness crossing over non-anatomical boundaries)

2. Stimulation tests: Axial loading and Pain on simulated rotation. (eliciting pain when pressing down on the top of the patient’s head or rotating the shoulders and pelvis together should not be painful)

3. Distracted straight leg raise. (if a patient complains of pain on straight leg raise, but not if the examiner extends the knee with the patient seated at another time during the initial evaluation)

4. Non-anatomic sensory changes: Regional sensory changes and Regional weakness. (sensory loss in an entire extremity or side of the body or weakness that is non consistent and jerky, ie "cog-wheeling")

5. Overreaction. (Exaggerated painful response to a stimulus, that is not reproduced when the same stimulus is given later)

If there are more than 3 of 5 present then there is high probability that patient has non-organic pain.
WADDELL’S SIGNS

- **EVIDENCE**

  There has been questions and research that has questioned the reliability of Waddell’s signs when trying to associate positive Waddell’s signs with non-organic signs and physiological signs. (Fishbain, Cole, Cutler, Lewis, Rosomoff, & Rosomoff, A structured evidence-based review on the meaning of nonorganic physical signs: Waddell signs. Pain Medicine. 4(2):141-81, 2003 Jun.)

  Although Waddell's signs can detect a non-organic component to pain, they do not exclude an organic cause. A high Waddell score (>3) is indicative only of symptom magnification or possible illness behaviour. Often the test has been misused (Main, Chris J. PhD; Waddell, Gordon DSc. MD. Spine. 23(21):2367-2371, November 1, 1998.). It does not signify malingering.

  **N.B. ORIGINALLY DESCRIBED FOR DETERMINING THOSE PATIENTS WHO WOULD DO POORLY WITH SURGERY**
NERVE SYMPTOMS

- ONSET
- NERVE DISTRIBUTION
- ASSOCIATED WEAKNESS AND NUMBNESS
- WALKING TOLERANCE
- EXACERBATING AND RELIEVING FACTORS
- BLADDER PROBLEMS
EXAMINATION

- OBSERVATION
  [POSTURE, PSYCHOLOGY]

- PALPATION
  [TENDERNESS, SPASM]

- MOVEMENT

- GAIT [ANTALGIC = INVOLVED LEG FLEXED]

- SPECIAL TESTS
  [REFLEXES, SLR {GOOD PREDICTIVE VALUE IF > 30}, HIP JOINTS]

- CROSS OVER SIGN
EXAMINATION

- DIFFICULT IN ACUTE PAIN
- COMBINED WITH HISTORY
- UPPER MOTOR NEURONE LESIONS
- VARIABLE
- NOTE ONLY 35% OF PIVD = SCIATICA
MOTOR EXAMINATION

- **S1 = CALF**
  - [GASTROCNEMIUS AND SOLEUS]
- **L5 = HALLUX EXTENSION**
- **L4 = TIBIALIS ANTERIOR**
  - [DROPPED FOOT]
- **L3 = QUADS**
INVESTIGATIONS

- BLOOD TESTS
- PLAIN X-RAY
- MRI
- CT
- MYELOGRAPHY
- BONE SCAN
- EMG
- ALL NEED INTERPRETATION WITH AND TO THE PATIENT
PLAIN X-RAYS
STEP LADDER APPROACH

4. SURGERY
3. INJECTIONS / NON OPERATIVE PAIN CONTROL
2. PHYSICAL THERAPY
1. ADEQUATE PAIN

_________________ +/-

+/- PSYCHOLOGICAL SUPPORT [THE F IN PAIN]
THE WHO ACUTE PAIN LADDER

NOT TOO MUCH CONTROVERSY HERE

PARACETAMOL+/-NSAID

COMBINATION / WEAK OPIOID

MORPHINE
JOINED UP APPROACHES

NO INTELLECTUAL SNOBBERY IN GETTING BETTER!

EXERCISE PROGRAMMES

MANIPULATIONS

PAIN RELIEF

SPINEWORKS

PSYCHOLOGICAL ASPECTS
THERE IS NO EVIDENCE FOR THE EFFECTIVENESS OF HOSPITAL BED REST WITH OR WITHOUT TRACTION FOR BACK PAIN

KEEP ACTIVE
C.S.A.G. RECOMMENDATIONS

- Early treatment is desirable, most pragmatic, effective and cost effective.
- Only 50% of patients return to work after 6/12 off.
- Consider return to work programs [Mon, Wed, Fri am etc]
- Note considered chronic if over 3/12
ONE STOP CLINICS

- PHYSIO PRACTITIONER [HISTORY, PAIN SCORES, EXAM]
- MRI SCAN
- CONSULTANT OPINION [EXPLAIN FINDINGS, ORGANISE TREATMENT]
- REASSURANCE
- RETURN TO WORK

"It was inevitable."
OTHER CAUSES OF SYMPTOMS
CAUDA EQUINA SYNDROME

- Usually caused by massive central P.I.V.D.
- Extrinsic pressure on thecal sac
- Rare causes = abscess, tumours, epidural haematoma, trauma
- Variable symptoms including reduction in perineal sensation, loss of bladder and/or bowel control, neurological deficit in one or both legs
DISTINGUISH FROM UPPER MOTOR NEURONE LESION

INVESTIGATE AS EMERGENCY WITH MRI / CT

IF CONFIRMED SURGERY ON NEXT AVAILABLE LIST
RECOMMENDATIONS FOR PRIMARY CARE

- ADVICE TO STAY ACTIVE
- ADEQUATE ANALGESIA
- REASSURANCE
- USE OF NSAIS
- EARLY REHAB
PLAN A - PHYSICAL THERAPY
[JUST PHYSIO]

- CHIROPRACTERS
- OSTEOPATHS
- PHYSIOTHERAPISTS
- SPORTS INJURY THERAPISTS
- MASSAGE THERAPISTS
- INDIVIDUAL MORE IMPORTANT THAN THE LABEL
PLAN A - ENCOURAGE PHYSICAL ACTIVITY

- PAIN DOES NOT = DAMAGE
- TABLETS CAN NOT MASK INJURY
- USE IT OR LOOSE IT / LONG TIME DEAD
- MUSCLES, JOINTS AND DISCS NEED MOVEMENT TO BE HEALTHY
RETURN TO WORK PROGRAMMES

- IF OUT OF WORK FOR 6 MONTHS 50% RETURN
- IMPORTANT NOT TO GET OUT OF HABIT
- LOOK AT INCREASING WEEK i.e. MONDAY WEDNESDAY FRIDAY am.s etc
PLAN A - PREVENTION BETTER THAN CURE

- CORE STABILITY VIA PILATES / FIT BALL
- AFTER AN ATTACK OF PAIN OFTEN TOO LATE
- PHYSIOS NEED TO SEE PATIENTS EVEN IF THEY ARE OVER THE LAST ATTACK
PLAN B - INJECTIONS

- WINDOW OF OPPORTUNITY FOR EXERCISE
- NOT CURATIVE
- CAN BE REPEATED
- CAN REVEAL PAIN SOURCE
- ALLOW NATURAL HEALING WHILST FUNCTIONAL

SEIZE THE OPPORTUNITY

SPINEWORKS
Surgery for Low Back Pain

- LBP from DDD poorly understood
- 4 likely factors of causation
- Investigate via:
  - MRI, CT, Discography, X-ray, Bone Scan,
  - Spinal probing.
- Need to find pain generator

LBP FROM DDD POORLY UNDERSTOOD

4 LIKELY FACTORS OF CAUSATION

INVESTIGATE VIA:

MRI, CT, DISCOGRAPHY, X-RAY, BONE SCAN,

SPINAL PROBING.

NEED TO FIND PAIN GENERATOR
MY OWN PRACTICE

- PATIENT SAMPLE = 1196 [SEPT 04 – JAN 06]
- MRI – 92%
- X-RAY – 15%
- INJECTIONS – 11%
- OPERATIONS – 6.78%

-------------------------------

2008 / 2009 HCI = 19%, SURGERY 5%
REFERENCES


LET'S HOPE THE PICTURE IS AS CLEAR!

SPINEWORKS