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

ANNULUS FIBROSUS

- 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]

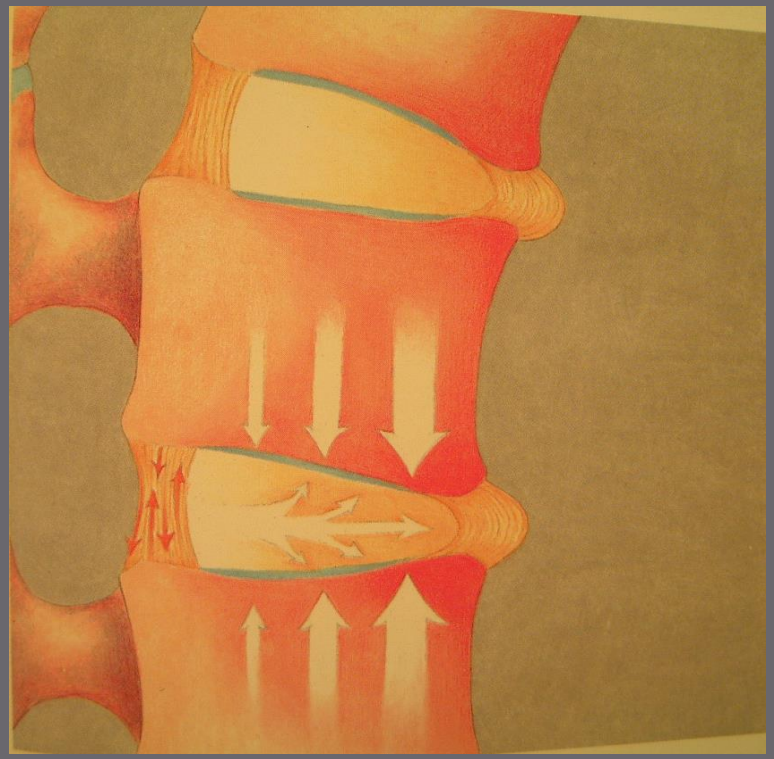
- THE INTEGRITY OF THE DISC RELIES ON THE PROPER BALANCE BETWEEN MATRIX SYNTHESIS AND DEGRADATION
- REF [1]

AGEING DISC

- STRUCTURAL ANNULAR CHANGES
- CONCENTRIC FISSURES
- LOSS OF CELLULAR DETAIL
- NUCLEUS BLENDS WITH ANNULUS
- NUCLEUS = TANGLED MASS OF FIBROUS TISSUE

DISC INJURY

- FLEXION FORCES CAN PRODUCE ANTERIOR DISC INJURY WITHOUT SCIATICA
- LEADS TO ACCELERATED DEGENERATIVE OR AGEING CYCLE



DISC CONSTITUENTS

- COLLAGEN = 70% ANNULUS DRY WT.[6-25% NUCLEUS]
- PROTEOGLYCANS ATTRACT WATER VIA OSMOSIS – GEL
- WATER = 80% NUCLEUS , 65% ANNULUS
- SOME ELASTIN

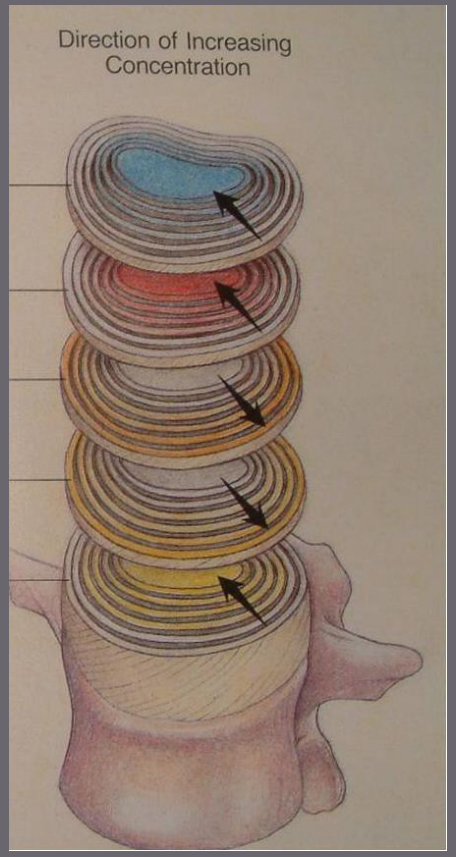
WATER

PROTEO GLYCANS

TOTAL COLLAGEN

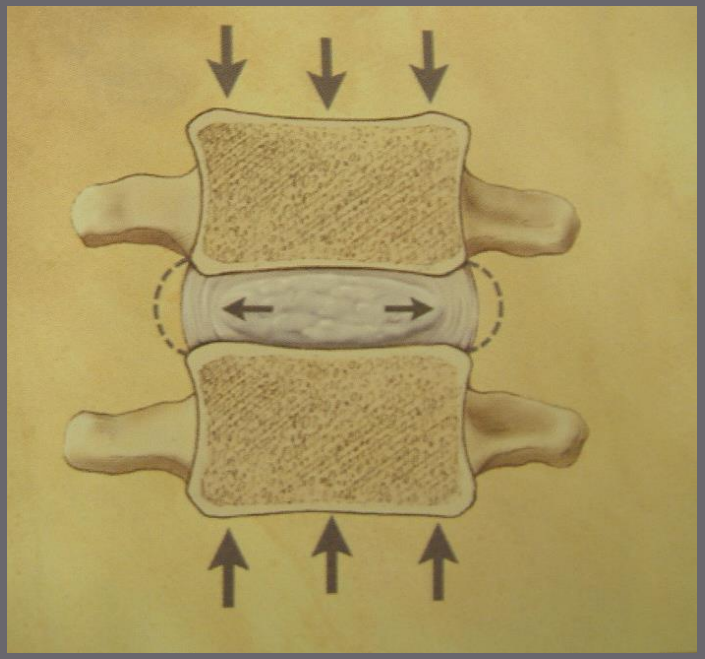
COLLAGEN 1

COLLAGEN 2



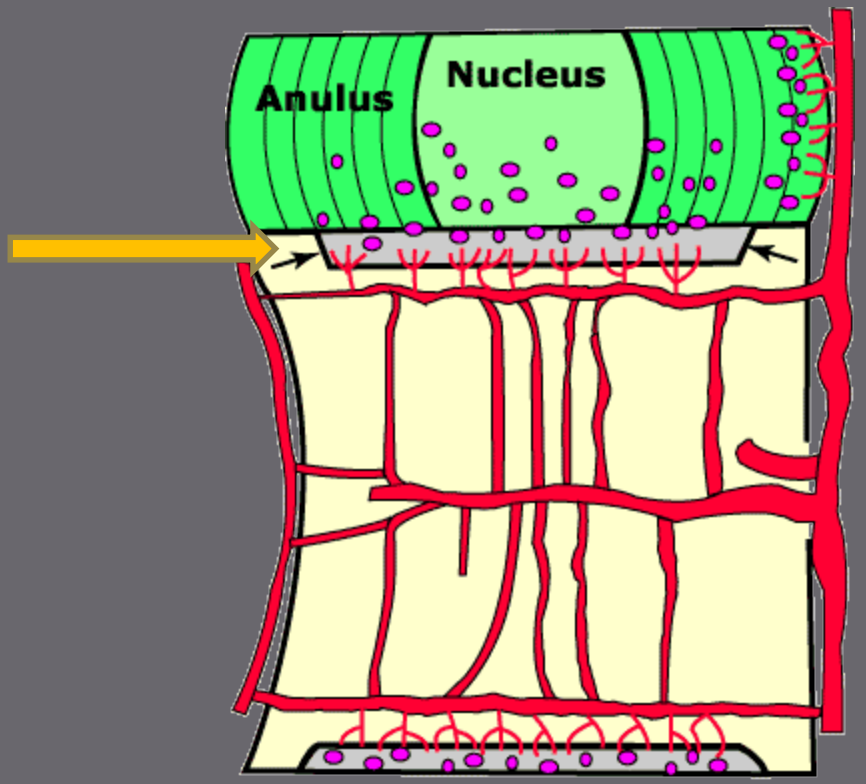
ROUGHLEY et al
Eur Spine J (2006)

- ABILITY OF THE DISC TO RESIST COMPRESSION DEPENDS UPON ITS HIGH PROTEOGLYCAN CONCENTRATION [MAJORITY IS AGGRECAN]
- OSMOTIC PRESSURE
- REF[2]



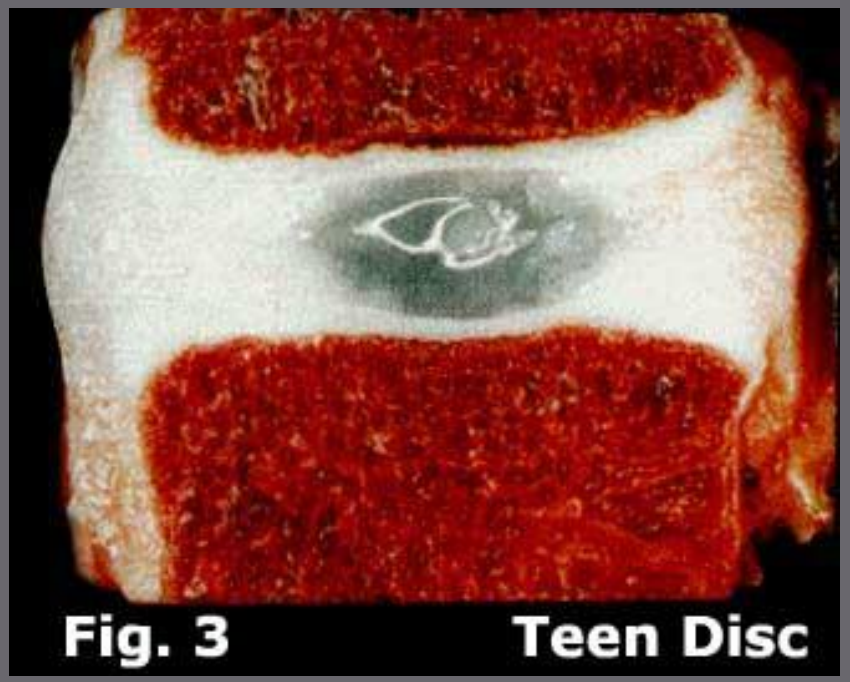
THE VERTEBRAL ENDPLATE

- THE VERTEBRAL ENDPLATES ARE CRITICAL FOR MAINTAINING DISC FUNCTION REF[3]
- MINOR DAMAGE TO A VERTEBRAL END PLATE LEADS TO PROGRESSIVE STRUCTURAL CHANGE IN THE ADJACENT DISC REF[4]



IMMUNOHISTOCHEMICAL STUDIES

- **NERLICK** et al SPINE 1997
- EVIDENCE SHOWS CHANGES OCCUR EARLY
- 8 – 13 YEARS
- AUTOPSY EVIDENCE

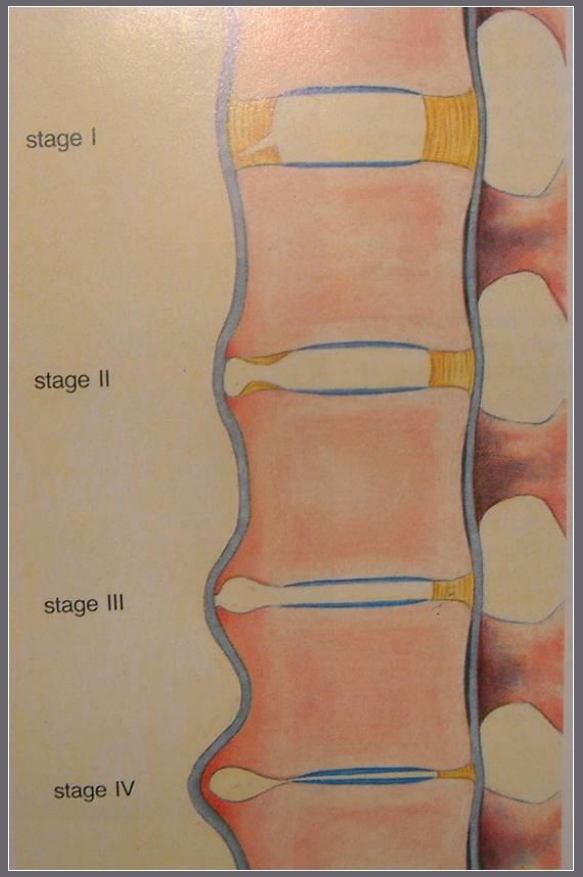


DISC STRUCTURE WITH AGEING

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

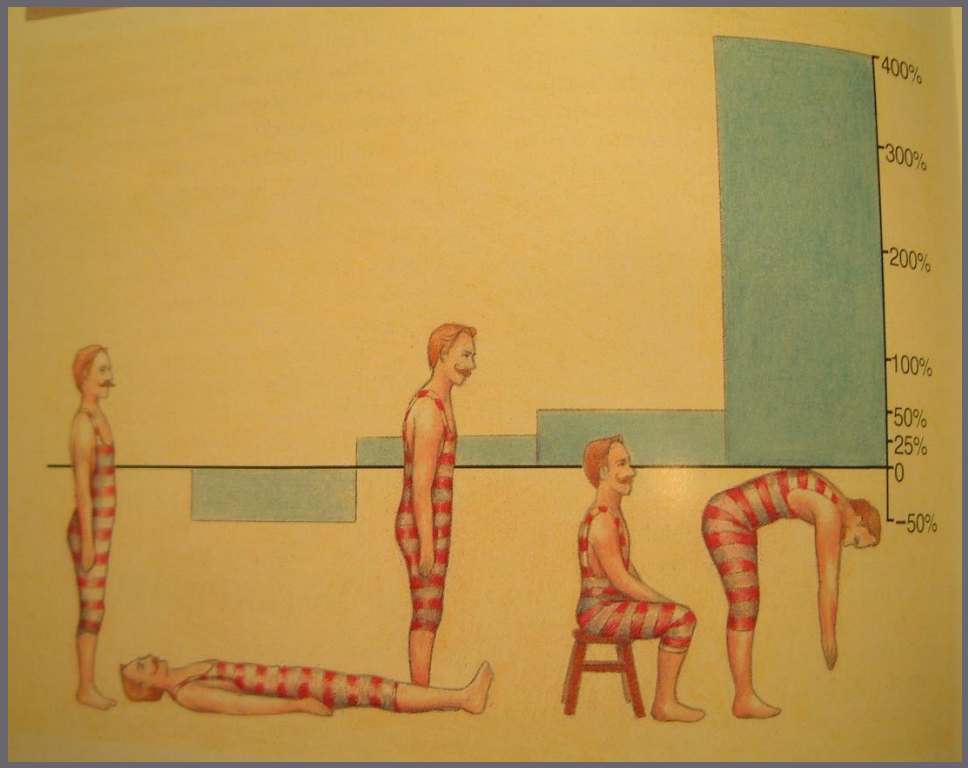
SPONDYLOSIS DEFORMANS

- DISC DESSICATES
- DISC STIFFENS
- MICROSCOPIC ANNULAR TEARS
- FIBROCARTILAGE METAPLASIA
- CLEFTS IN DISC TISSUE ON X-RAY = VACUUM PHENOMINA
- 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]

MECHANICAL CONDITIONS THAT ACCELERATE INTERVERTEBRAL DISC DEGENERATION

- 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

GENETICS OF DISC DEGENERATION

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, MULTIFACTORAL DISEASE DETERMINED BY INTERPLAY BETWEEN GENES AND ENVIRONMENT

REF 7

LUMBAR DISC DEGENERATION, EPIDEMIOLOGY AND GENETIC INFLUENCE

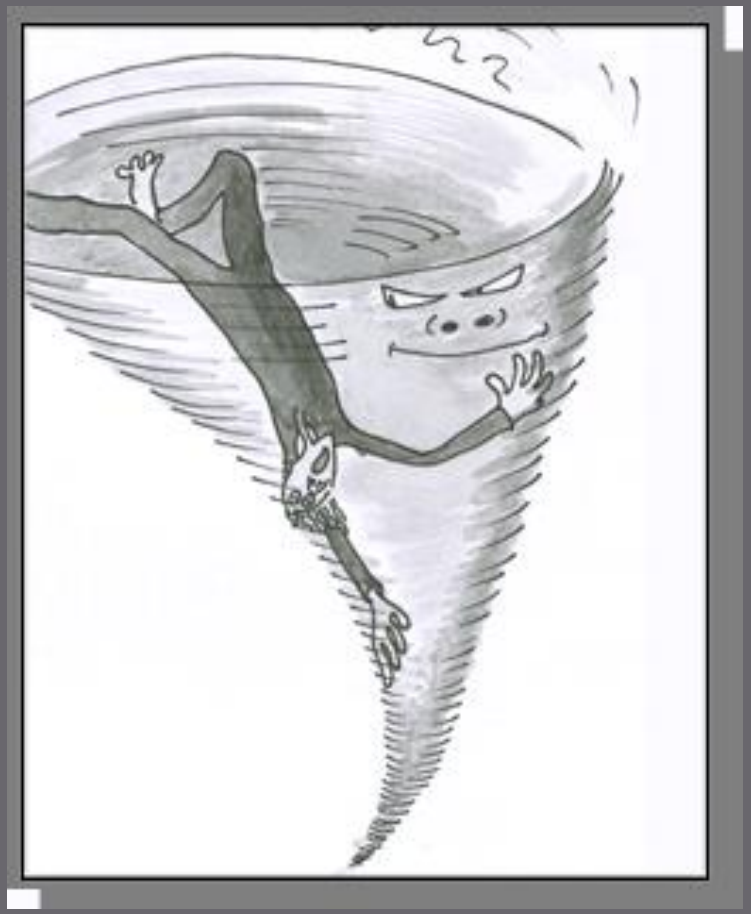
- BATTIE et al 2004 + 2009
- MONOZYGOTIC TWIN STUDIES = HEREDITY WITH A DOMINENT ROLE IN DISC DEGENERATION. ENVIRONMENT HAS A MODEST ROLE REF[8]
- STUDY FROM 1991 SUBSTANTIAL EFFECT OF HEREDITY ON LUMBAR DEGENERATION BUT LOADING HAS LITTLE EFFECT. LARGER EFFECT FROM MUSCLE STRENGTH AND BODY WEIGHT
- 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 IHERITANCE, 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



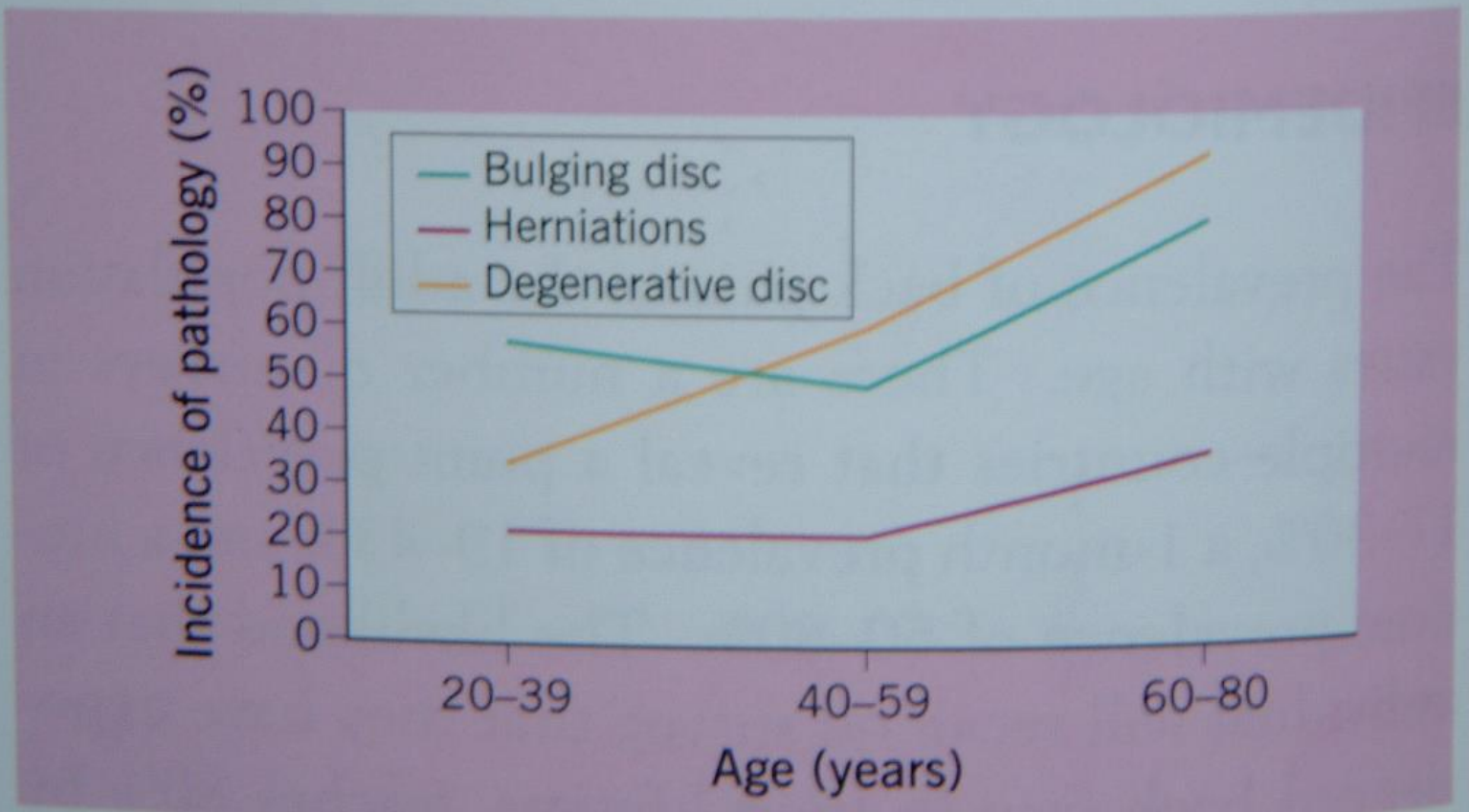
DEGENERATIVE DISC AND ENDPLATE CHANGES ON MRI

NATURAL HISTORY

- 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

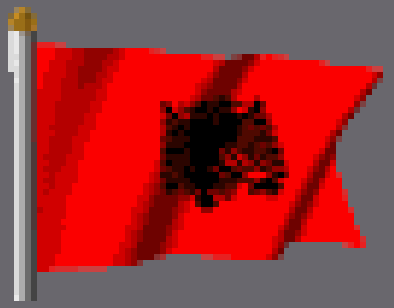


BODEN et al ABNORMAL MRI IN NORMAL SUBJECTS; JBJS 1990

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

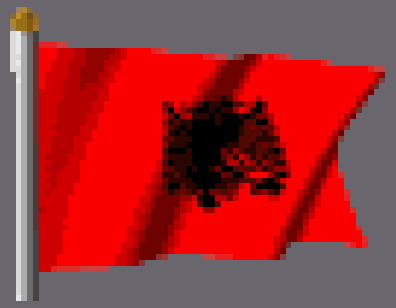
SERIOUS PATHOLOGY [RED FLAGS]

- POSSIBLE SERIOUS SPINAL PATHOLOGY
- 1. AGE <20 or >55
- 2. VIOLENT TRAUMA
- 3. CONSTANT PROGRESSIVE NON-MECHANICAL PAIN
- 4. PMH CARCINOMA
- 5. WEIGHT LOSS



[RED FLAGS] CONTINUED

- 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)

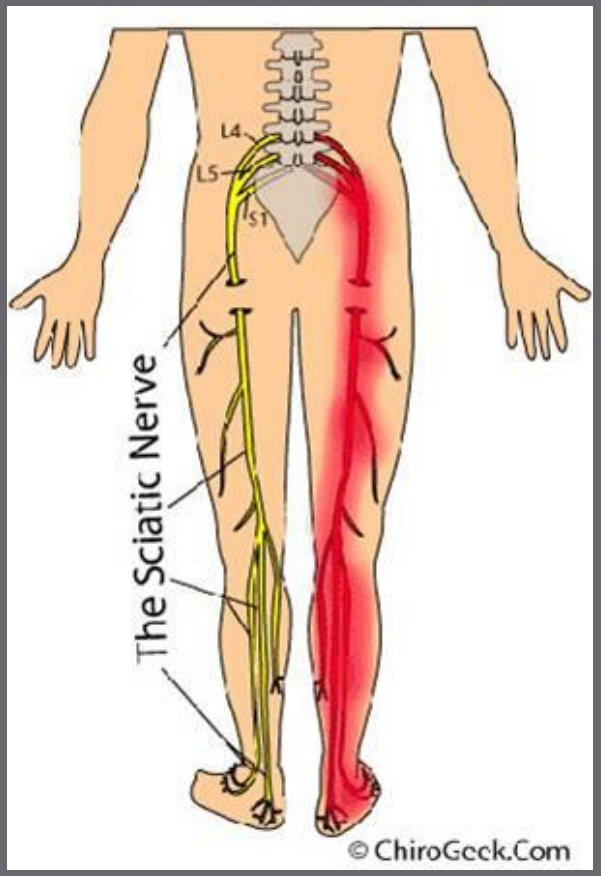
- If there are more than 3 of 5 present then there is high probability that patient has non-organic pain.

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

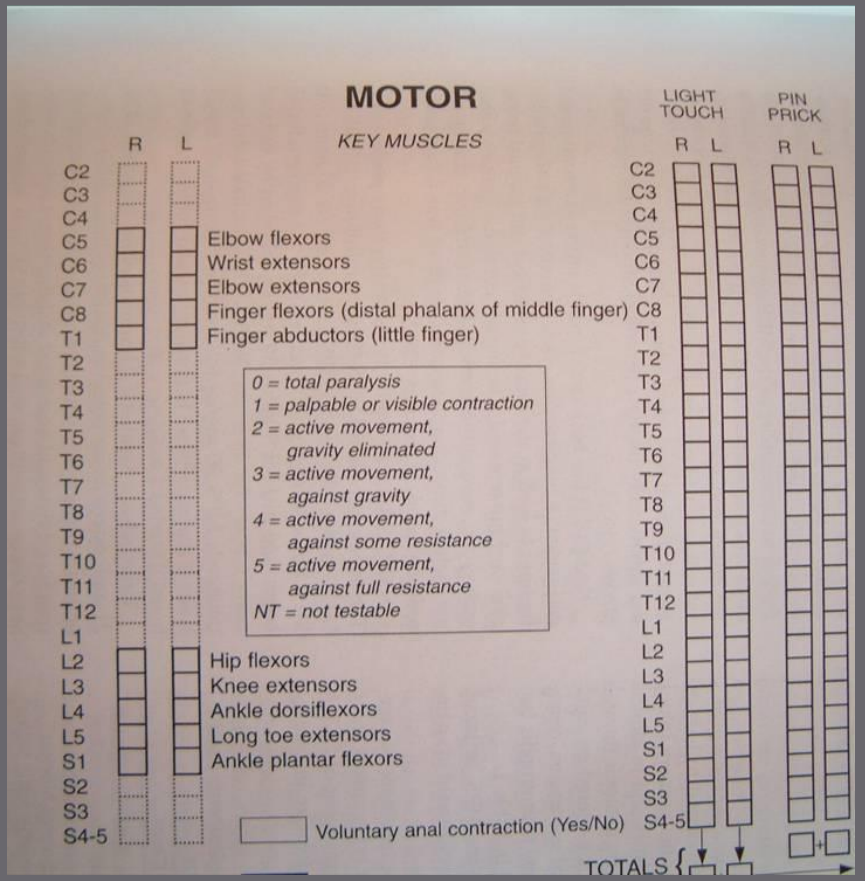
- OBSERVATION [POSTURE, PSYCHOLOGY]
- PALPATION [TENDERNES, SPASM]
- MOVEMENT
- GAIT [ANTALGIC = INVOLVED LEG FLEXED]
- SPECIAL TESTS [REFLEXES, SLR {GOOD PREDICTIVE VALUE IF > 30}, HIP JOINTS]
- CROSS OVER SIGN

EXAMINATION

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

MOTOR EXAMINATION

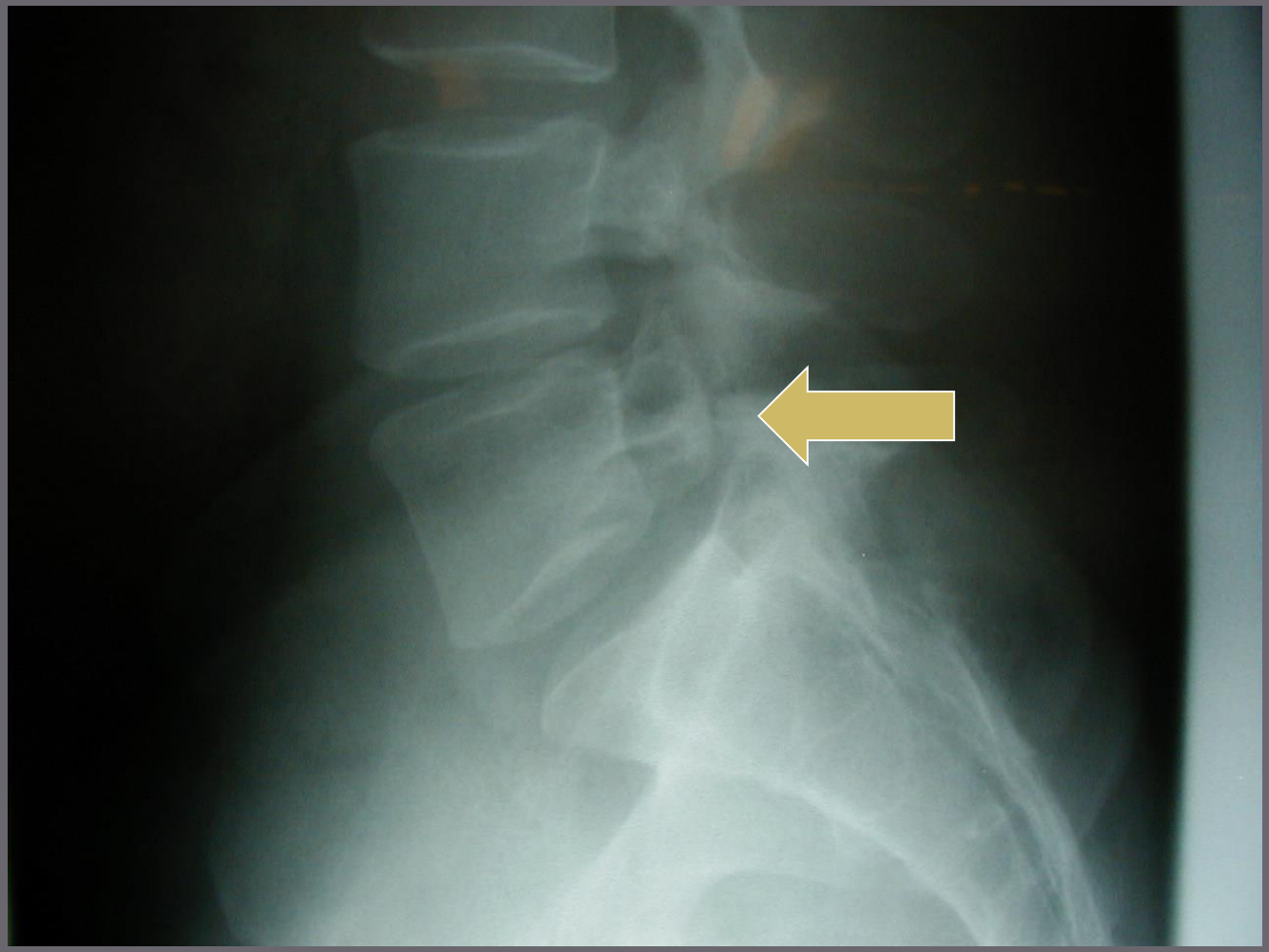
- S1 = CALF [GASTROCNEMIUS AND SOLEUS]
- L5 = HALLUX EXTENSION
- L4 = TIBIALIS ANTERIOR [DROPPED FOOT]
- L3 = QUADS



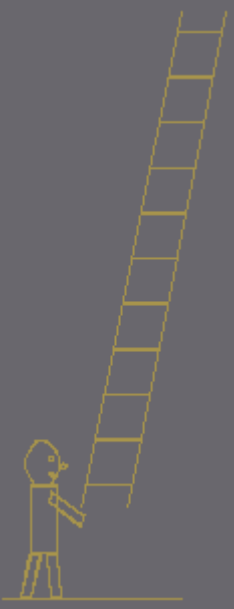
INVESTIGATIONS

- 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

MORPHINE

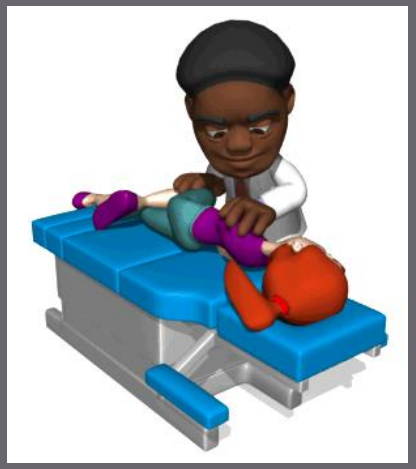
COMBINATION / WEAK OPIOID

PARACETAMOL+/- NSAID

JOINED UP APPROACHES

EXERCISE PROGRAMMES

NO INTELLECTUAL SNOBBERY IN GETTING BETTER!



MANIPULATIONS

PAIN RELIEF

SPINEWORKS

PSYCHOLOGICAL ASPECTS

COMBINED SPECIALITIES ADVISORY GROUP - 1994

- 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

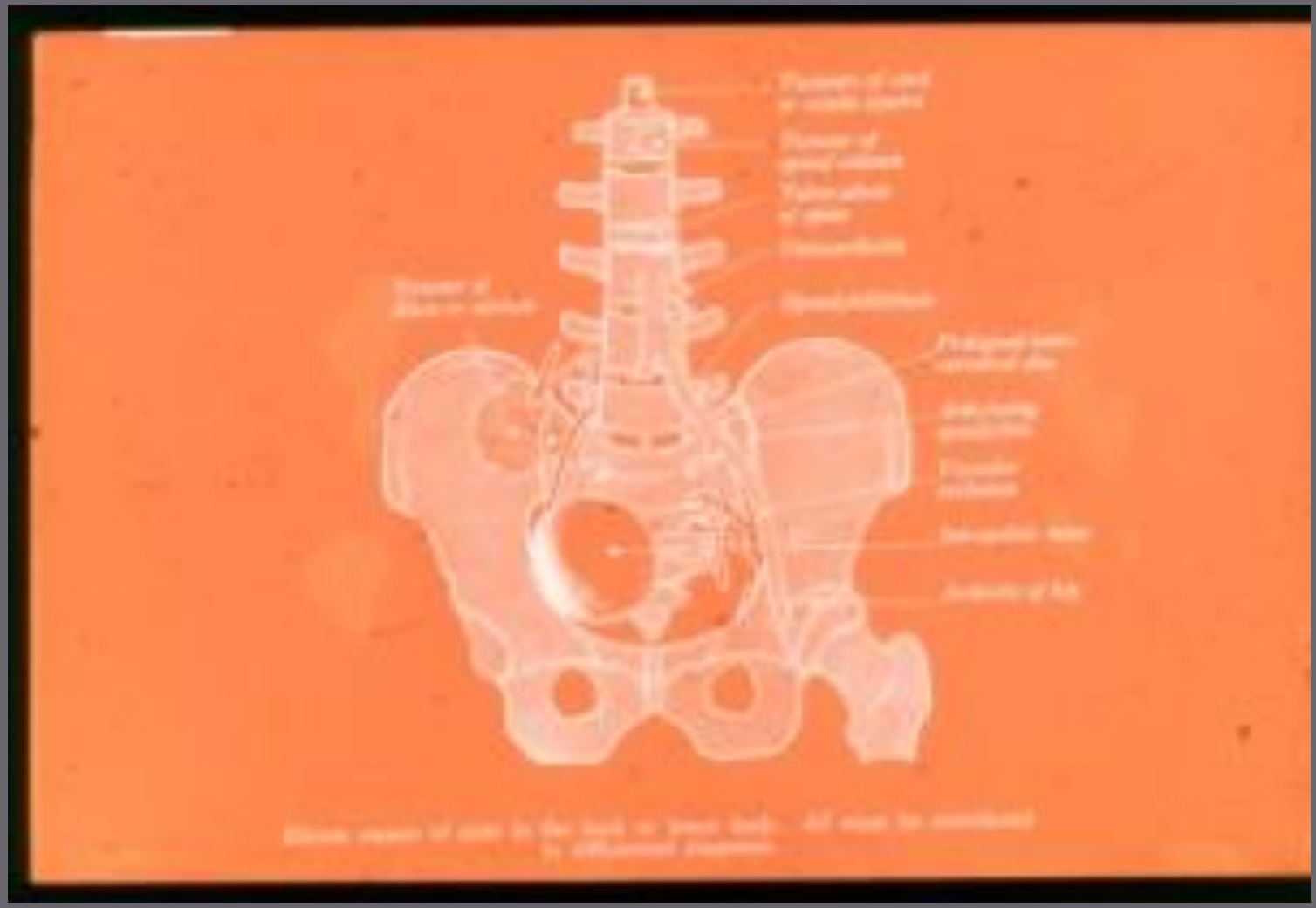
- EARLY TREATMENT IS DESIREABLE, MOST PRAGMATIC, EFFECTIVE AND COST EFFECTIVE.

  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

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

CAUDA EQUINA CONTINUED

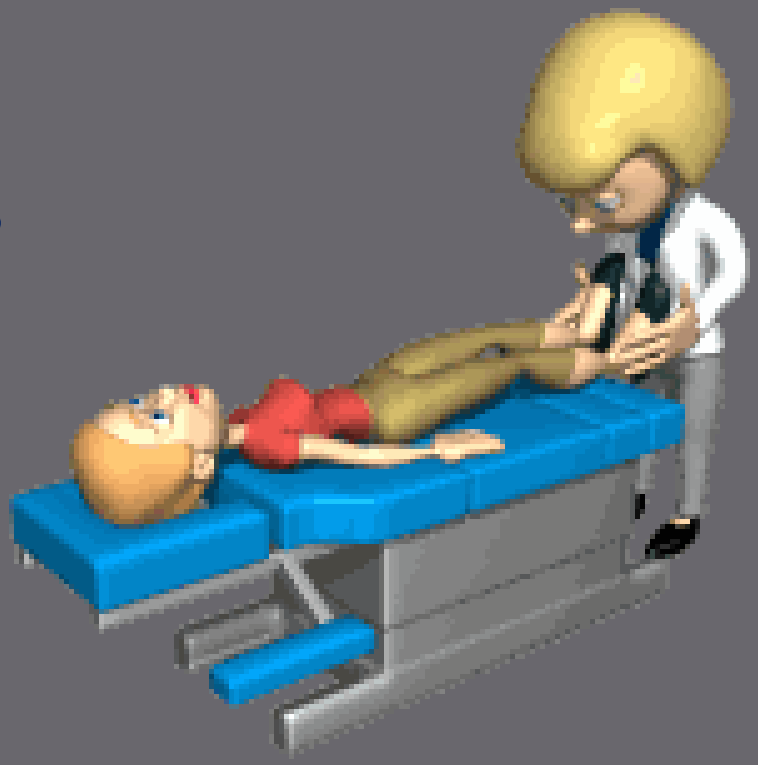
- DISTINGUISH FROM UPPER MOTOR NEURONE LESION
- INVESTIGATE AS EMERGENCY WITH MRI / CT
- IF CONFIRMED SURGERY ON NEXT AVAILABLE LIST

RECOMMENDATIONS FOR PRIMARY CARE

- ADVICE TO STAY ACTIVE
- ADEQUATE ANALGESIA
- REASSURANCE
- USE OF NSAIS
- EARLY REHAB

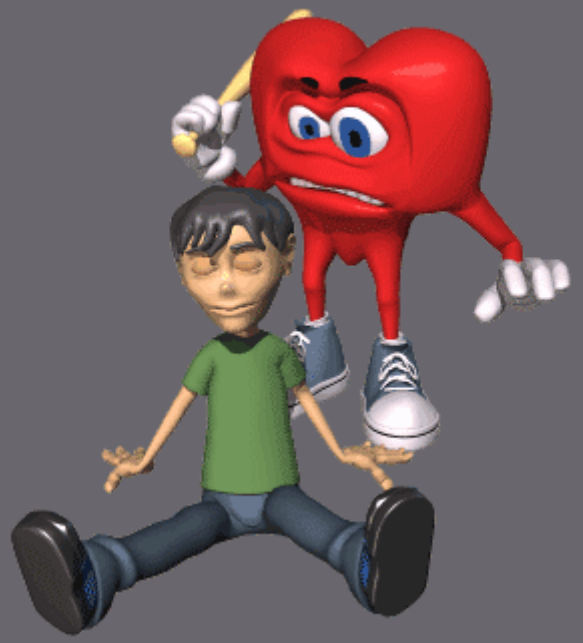
PLAN A - PHYSICAL THERAPY [JUST PHYSIO]

- 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

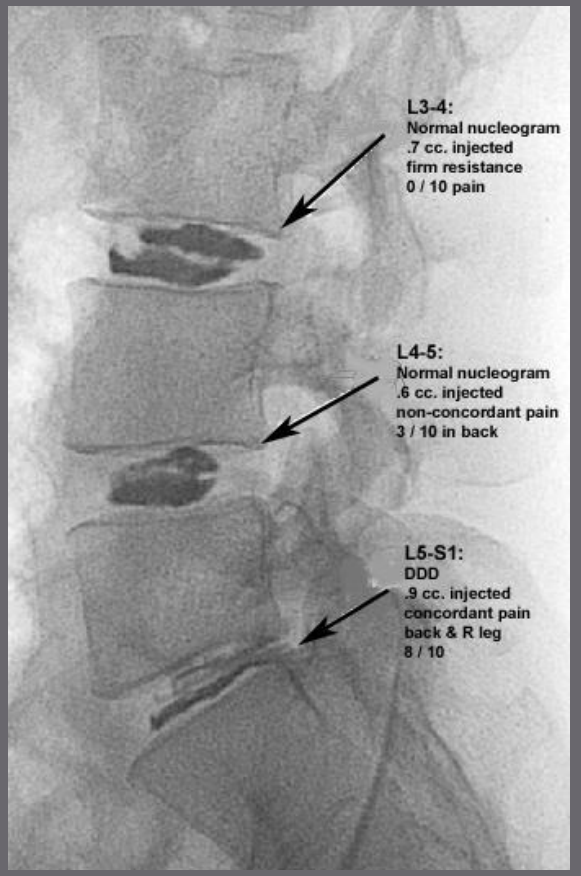
- CORE STABILITY VIA PILATES / FIT BALL
- AFTER AN ATTACK OF PAIN OFTEN TOO LATE
- PHYSIOS NEED TO SEE PATIENTS EVEN IF THEY ARE OVER THE LAST ATTACK

PLAN B - INJECTIONS

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

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



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

- [1] MOLECULAR THERAPY OF THE INTERVERTEBRAL DISC. YOON et al Eur Spine J (2006) 15 (Suppl 3) S379 – 388
- [2] THE STRUCTURE AND DEGRADATION OF AGGRECAN IN HUMAN INTERVERTEBRAL DISCS. ROUGHLEY et al. Eur Spine J (2006) 15 (Suppl 3) S326 - 332
- [3] THE VERTEBRAL ENDPLATE: DISC DEGENERATION AND REGENERATION. Eur Spine J (2006) 15 (Suppl 3) S333 – 337
- [4] MECHANICAL IMITATION OF INTERVERTEBRAL DISC DEGENERATION. ADAMS et al. Spine vol 25, 13,1625 – 1636
- [5] BIOLOGY OF INTERVERTEBRAL DISC AGEING AND DEGENERATION. ROUGHLEY. Spine (2004)vol 29, 23, 2691 – 2699
- [6] MECHANICAL CONDITIONS THAT ACCELERATE INTERVERTEBRAL DISC DEGENERATION: OVERLOAD vs IMMOBILISATION. STOKE et al. Spine (2004) 29, 23, 2724 – 2732
- [7] GENETICS OF DISC DEGENERATION. CHAN et al. Eur Spine J (2006) 15 (Suppl 3), S317 – S325
- [8] LUMBAR DISC DEGENERATION, EPIDEMIOLOGY AND GENETIC INFLUENCES. BATTIE et al. Spine vol29, 23, 2679 – 2690
- [9] THE TWIN STUDY: CONTRIBUTIONS TO A CHANGING VIEW OF DISC DEGENERATION. BATTIE et al. The Spine Journal. 9,(2009) 47 -59
- [10] WHAT IS INTERVERTEBRAL DISC DEGENERATION AND WHAT CAUSES IT. ADAMS et al. Spine (2006) vol 31, 18, 2151 - 2161

LETS HOPE THE PICTURE IS AS CLEAR!