Treatment Modalities in Rheumatology

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Biopsychosocial vs Biomedical Management
Management

• Multidisciplinary

• Education!!!!!!!!!!!!!
Physiotherapy

• Temperature:
  – Cold:
    » Reduced enzyme activity, decreased metabolic rate → analgesia, muscle relaxation, vasoconstriction
  – Heat
    » Superficial or deep (ultrasound)
    » Better perfusion, relaxation, analgesia
Physiotherapy

• Electrical: TENS, Interference

• Physical:
  – Massage: myofascial pain, stress relief, pain gating
  – Mobilization-manipulation
  – Ischaemic pressure
  – Dry needling
Physiotherapy

• Rest vs exercise: Acute inflammation:
  – Rest (splinting)
  – ROM 1-2 X per day (active/passive)
  – Isometric exercise to maintain muscle bulk

• Exercise therapy: Subacute disease
  – Range of motion: maintain & improve
  – Muscle strength esp quadriceps (isometric, isotonic, resisted)
  – Aerobic capacity: CHD risk, central analgesia eg fibromyalgia

• Hydrotherapy
Occupational Therapy

- Aids and appliances: dressing, kitchen etc
- Splints:
  - night splints eg hands, knees
  - functional splints eg wrist extensions
- Energy conservation
- Joint protection
- Hand joint exercises
Orthotics

- Insoles: medial arch supports, metatarsal pads, medial/lateral wedges
- Heel inserts for plantar fasciitis
- Knee/ankle braces: instability
- Neck braces: Soft/Philadelphia
- Corsets
- Wrist splints
- Mobility aids: sticks, crutches, frames, W/C
Psychological support

- Depression very common
  - Loss of role
  - Loss of job
  - Financial problems (↓ income, ↑ medical expenses)
  - Poor sleep
  - Sexual problems
- Relaxation techniques
- Psychological support
- Relationship counselling
Psychological/social Intervention

- Relaxation techniques:
  - Breathing
  - Progressive relaxation
- Distraction techniques:
  - Mindfulness
  - Guided imagery
  - Meditation
- Coping skills-stressors, pain
  - Cognitive behavioural techniques
  - Psycho analysis
  - Hypnotherapy etc
- Relationship therapy
Social Work

- Psychological support
- Family/relationship problems
- Employment problems: liaise with employer
- Disability grant / early pension / medical boarding
Dieticians

- Avoid obesity - stress on hips, knees, ankles
- Fibre - prevent constipation due to medication
- Inflammatory diseases
  - Anti-oxidants: eg Vit C and E
  - Omega 3 fatty acids: eg fatty fish, fish oils
  - Essential fatty acids: evening primrose oil
  - Calcium - osteoporosis prophylaxis
  - Low cholesterol - prevent IHD
  - Fasting: RA improves!
- Gout: low purine diet
Medical Treatment
Analgesia
Types of Pain

- **Nociceptive pain:**
  Tissue damage - trauma, inflammation

  - Somatic (musculoskeletal):
    - local pain
    - referred pain
    - NB Myofascial pain syndromes

  - Visceral structures: referred pain
EXAMPLES OF TRIGGER POINTS AND THEIR REFERRED PAIN PATTERNS IN MYOFASCIAL PAIN SYNDROME

- Pain radiating from:
  - subscapularis muscle trigger points
  - upper trapezius
  - lower trapezius
  - trigger point

[Diagram showing pain patterns in the shoulder and back areas]
Types of Pain

• **Neuropathic pain**: Damage to peripheral nerves or CNS
  - Stabbing, burning or shooting
  - Often poor response to opioids
  - Usually some sensory defect (usually thermal)
  - Often autonomic instability
  - Allodynia to touch, cold and movement
Analgesics

- Paracetamol
- Cox inhibitors: Aspirin, NSAID’s, Coxibs
- Opioids
- Atypical analgesics
  - Antidepressants
  - Anticonvulsants
  - Antiarrhythmics
Pain Treatments under Investigation

- Cannabinoids
- Ion channel blockers
- Adrenergic agonists
- Excitatory amino acid antagonists (NMDA)
- Neurokinin antagonists
- Neurotrophin antagonists (e.g., nerve growth factor)
Pharmacologic Therapy

- **Timing**
  - Give ‘by the clock’: more stable blood concentrations minimizes risk of breakthrough pain
  - Give according to T $\frac{1}{2}$: frequent enough to maintain stable concentration
  - ‘When needed’ : administration of each dose contingent on reappearance of pain
Pharmacologic Therapy

• **Dose**
  – Too small: not effective
  – Excessively large: toxicity or side-effects overwhelm beneficial pain relief

• **Analgesic Type**
  – Decrease generation of pain impulses at sites of inflammation: anti-inflammatories
  – Suppress nervous system transmission of pain impulses: opioids, atypical analgesics
Hyperalgesia (inflammation)

Analgesia

Anti-inflammatories

Opioids

DOSE-RESPONSE CURVE FOR PAIN
Arachidonic acid

Cyclo-oxygenase (COX)

PG E2  PG F2α  TX A2  PG D2  PG I 2
Gastroprotection  Platelet aggregation  Vasoconstriction  Vasodilatation
Arachidonic acid

**Cox 1**
Constitutively expressed:
*Stomach, intestines, platelets, kidney*

- **PG E2**: Gastroprotection
- **PG F2α**: Platelet aggregation
- **TX A2**: Vasoconstriction
- **PG D2**: Vasodilatation
- **PG I 2**: Vasodilatation
Arachidonic acid

**Cox 1**
Constitutively expressed:
Stomach, intestines, platelets, kidney

**Cox 2**
Constitutively expressed:
Brain, kidney

**Induced:** sites of inflammation

**PG E2**
Gastroprotection

**PG F2α**
Platelet aggregation
Vasoconstriction

**TX A2**

**PG D2**

**PG I 2**
Vasodilatation
Arachidonic acid

Cox 1
Constitutively expressed:
Stomach, intestines, platelets, kidney

Cox 2
Constitutively expressed:
brain, kidney
Induced: sites of inflammation

PG E2
Gastroprotection

PG F2α

TX A2
Platelet aggregation

PG D2
Vasoconstriction

PG I 2
Vasodilatation

Cox 1 & 2 inhibition:
• ↓ PG E2 in stomach → parietal cell damage
• ↓ TX A2 → bleeding
• ↓ PG I 2 in kidney → ↓ kidney function and ↑ BP
• ↓ inflammation
**Arachidonic acid**

**Cox 1**
Constitutively expressed:
- Stomach, intestines, platelets, kidney

**Cox 2**
Constitutively expressed:
- Brain, kidney
  **Induced:** sites of inflammation

---

**PG E2**
Gastroprotection

**PG F2α**

**TX A2**
Platelet aggregation
Vasoconstriction

**PG D2**

**PG I 2**
Vasodilatation

---

**Cox 1 & 2 inhibition:**
- ↓ PG E2 in stomach → parietal cell damage
- ↓ TX A2 → bleeding
- ↓ PG I 2 in kidney → ↓ kidney function and ↑ BP
- ↓ inflammation

**Cox 2 selective inhibition:**
- ↓ PG I 2 in kidney → ↓ kidney function and ↑ BP
- ↓ inflammation
1. Paracetamol

- Action: Inhibits cyclo-oxygenase centrally
  - Analgesic
  - Antipyretic
- Rarely produces gastric irritation
- No inhibition of platelet function
- Can be given in combination with NSAID’s, coxibs or aspirin
- Inexpensive
- Up to 4 g per day if normal LFT- liver toxicity in high dose
- Regular dosing to achieve steady state levels
2. Anti-inflammatory Drugs

- Inhibit cyclo-oxygenase (COX)
  - Centrally
    - Analgesic
    - Antipyretic
  - Peripherally
    - Anti-inflammatory, especially at higher dosages
    - Secondary analgesic effects
• **Aspirin:**
  - POTENT anti-inflammatory!!
  - Significant side effects esp gastric
  - Permanent inhibition for life of platelet

• **Conventional NSAID’s**
  - Gastric, renal/cardiovascular side effects
  - Reversible platelet inhibition
  - “Safer”: misoprostol, PPI (not H2 antagonists, sucralfate)

• **Cox 2 selective drugs**
  - Less gastric side effects
  - Renal/cardiovascular effects similar to conventional NSAID’s
  - No effect on platelet function
Cox inhibitors

• Musculoskeletal pain
• Headaches
• Dysmenorrhoa
• Pre-emptive analgesia
• Post operative pain
3. Opioids

- Most potent pain-relieving drugs
- Most reliable drugs for rapidly relieving pain
- Inhibit pain impulse transmission within CNS
- Opioid receptors and effects
  - Mu and Kappa: analgesia, sedation, respiratory depression and euphoria
  - Delta and Sigma: dysphoria, hallucinations
Opioids: Indications

• **Severe acute pain**
  – Large and frequent doses

• **Chronic pain**
  – Malignancy: use of opioids well accepted
  – Chronic non-malignant pain: careful - nociception not sole source of suffering
  – Use only if
    • Opioid sensitive pain
    • All other treatments have failed
    • Pain has major effect on quality of life of patient
    • Usually: medical confirmation of diagnosis
    • Full explanation and agreement of patient
Relatively opioid-insensitive pain

- Neuropathic pain
- Bone pain
- Superficial ulcer pain
- Bladder and rectal tenesmus
- Perineal pain
- Pancreatic pain
- Isolated sharp pains (constant pain more reliably relieved)
Opioids: Contra-indications

- Raised ICP: CO2 retention leads to further raise in pressure
- Pulmonary disease eg COPD
- Renal impairment: accumulation (codeine, dihydrocodeine, Pethidine, morphine)
- Gross liver function disturbance
- Hypothyroidism
- Other sedative drugs
- (Previous substance abuse)
Pharmacology of Opioids

- Opioids classified according to:
  - Potency: kind of pain relief (mild/moderate/severe)
  - Receptor effect at mu and kappa receptors
Opioid Potency

• **Strong:**
  – Morphine, Diamorphine (Heroin), Methadone, Fentanyl

• **Intermediate:**
  – Meperidine (Pethidine), Pentazocine, Tramadol, Tilidine, Dipipanone, Buprenorphine

• **Low:**
  – (Dextro)propoxyphene (0,66x), Codeine (1x), Dihydrocodeine (2x).
  
  – *Unacceptable side effects at high doses required to provide equivalent analgesia to high potency opioids: eg 200 mg codeine required to achieve the same effects as 30 mg morphine po*
4. Antidepressants

- Analgesic effect of tricyclics
  - Mechanism of analgesia is unknown
  - More rapid onset than antidepressant effects
  - Lower dose than antidepressant effects
  - Patients with chronic pain who are not depressed also obtain pain relief with antidepressants
  - Potentiate opioid analgesia: useful adjuncts for treatment of severe persistent pain eg malignant tumors
Antidepressants

- Tricyclic antidepressants
  - Particular valueable in management of neuropathic pain eg diabetic neuropathy, postherpetic neuralgia
  - Significant side effects
  - Depression: adequate dosage for 2-4 weeks - lag effect
  - Pain: lower doses
  - Dose: 10 to 25 mg nocte, increase weekly to maximum 150 mg
Antidepressants

- Serotonin-selective reuptake inhibitors: Fluoxetine (Prozac)
  - Fewer and less serious side effects
  - Not been shown to provide pain relief

- Venlafaxine (Efexor)
  - Nontricyclic antidepressant
  - Blocks serotonin and norepinephrine reuptake
  - Appears to be useful in patients who cannot tolerate tricyclics
5. Anticonvulsants

• Useful for
  – “stabbing pain”
  – “shooting pain”
  – “lancinating pain”
  – “electric shock like pain”

• Less likely to have an effect on constant burning pain
Anticonvulsants

• Carbamazepine
  – Most commonly used
  – Start 100mg/day and increase gradually to 1600 mg per day
  – Toxic symptoms: nausea, vomiting, and unsteady gait
  – Take with food

• Gabapentin (Neurontin) 600-1200 mg/d
  – Increases brain gamma-aminobutyric acid levels
  – Broad range of neuropathic pains

• Clonazepam (Rivotril) 1 mg qid

• Phenytoin (Epanutin) 300 mg per day
6. Antiarrhythmic drugs

• Neuropathic pains

• Block spontaneous activity of damaged primary afferent nociceptors

• **Drugs and doses (max)**
  – Mexiletine (Mexitil) 150-300 mg tds
  – ((IV Lignocaine))
7. CNS Depressants:

- Sedative-hypnotics, tranquilizers, anxiolytics, muscle relaxants eg benzodiazepines, barbiturates

- If some ‘pain relief’: non-nociceptive factors at least partially responsible for suffering
Sedative hypnotics

- Mood elevation and dysinhibition briefly allay suffering (like alcohol)
- Physiologically beneficial sleep only for a few weeks, then significant disturbance of REM sleep
- Cognitive defects similar to those of organic brain disease: months to recover
- Cause / aggravate depression
- Effects on driving more subtle than alcohol - may persist > 24 h after single dose
- Tolerance and habituation
Specific Disease Treatments
Osteoarthritis
OA: Glucosamine Sulphate

**Effects**
- Pain relief
- Chondropotence (DMOAD) in knee OA
  - After 3-5 y: slight ↑ in joint space, ↓ in placebo groups
  - Increase proteoglycan synthesis: building block of glycosaminoglycans (cartilage ground substance)

**Shrimp and crab shells**
**1500 mg per day**
**Which one??**
- Sulphate (not chloride)
- No quality control regarding bioavailability of active ingredient
OA: Other DMOAD’s

- **Chondroitin Sulphate**
  - Studies not definitively conclusive
  - Bovine product - higher cost

- **Diacerhein**
  - Reduce OA symptoms
  - Decrease cartilage loss in hip compared to placebo
  - Not yet in SA

- **Tetracycline: ongoing trials**
  - Block synthesis of nitric oxide and metalloproteases involved in cartilage destruction

- **Antimalarials: ongoing trials**
Gout
Gout

- Colchicine
  - High dose in acute attack
  - Low dose as prophylaxis
- Urate lowering drugs: increase slowly until urate <0.3 mmol/l
  - Allopurinol
  - Probenecid
Inflammatory Arthritis

Rheumatoid arthritis
Spondylarthritis
Connective Tissue Diseases
Corticosteroids

- Weight gain
- Hypertension
- Cataract
- Diabetes mellitus
- Arteriosclerosis
- Osteoporosis (weight bearing exercise, Calcium + vit D, HRT, bisphophonates)
Corticosteroids

- Dramatic improvement in pain and stiffness

- Use (in lowest dose, shortest time possible):
  - Acutely ill
  - Ineffective response to optimal DMARD
  - Significant systemic disease
  - Serious social problems: breadwinner losing job
Corticosteroids

• Intra-articular
  – Pain (Flare)
  – Sepsis: aseptic technique
  – Cartilage softening?? 3-6 month intervals between injections

– Duration of effect varies ++
<table>
<thead>
<tr>
<th>Drug Strength</th>
<th>Rank Order</th>
<th>Potency</th>
</tr>
</thead>
<tbody>
<tr>
<td>Auranofin</td>
<td>1</td>
<td>Weak</td>
</tr>
<tr>
<td>Hydroxychloroquine</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Minocycline</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>Chloroquine</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>Sulphasalazine</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>Gold IM</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>D-penicillamine</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>Methotrexate</td>
<td>4/5</td>
<td>Strong</td>
</tr>
<tr>
<td>Leflunomide</td>
<td>4</td>
<td></td>
</tr>
</tbody>
</table>
DMARD’s

- No single therapeutic regimen consistently halts disease progression
- Most cartilage loss in first 18 months of disease (irreversible): Early and aggressive treatment leads to better long term outcome
- RA: begin early with a therapy that will downgrade disease to a less destructive form of RA without causing undue morbidity from the therapy
RA: Minocycline

• Trials contradictory but balance showed benefit
• Used in milder disease - no decrease in radiological progression
• 100 mg bd
• Effect after 3-6 months
Chloroquine

- Chloroquine base average dose 200mg/d
- Lower dose in older and smaller patients (eg 5 days per week)
- Irreversible maculopathy
- 6-12 monthly Ophthalmology reviews
**Gold preparations**

- Allergic reactions
- GIT: stomatitis, diarrhoea
- Bone marrow suppression/eosinophilia
- Renal: GN, nephrosis
- Lungs: pneumonitis
- Peripheral neuropathy

- Regular FBC and urine Dipstick
- Not available in SA anymore
D-Penicillamine

- Expensive
- Side effects:
  - Bone marrow suppression
  - Renal: nephrosis
  - Rash
  - Drug induced lupus
- Specialist use: vasculitis, lung disease, Felty
- Regular FBC and urine Dipstick
Sulphasalazine

- GIT: Nausea, liver function disturbance
- Dizziness, headaches
- Bone marrow suppression
- Impaired spermatogenesis

- Do regular FBC and LFT
- 500-2000 mg per day in divided doses
Methotrexate

• First line treatment
• Safe use
  – With folic acid every day
  – Once a week
  – 7.5-15 mg per week (higher with specialist use)
  – Regular full blood count and liver function test
**Methotrexate**

- Nausea
- Liver fibrosis
- Bone marrow suppression
- Pneumonitis
- Increases size of RA nodules
RA: Leflunomide (ARAVA)

- Pyrimidine synthesis inhibited
  - decreased T cell activation
  - decreased T cell proliferation
- Reduction
  - inflammation
  - radiologic erosions
- Dose:
  - Loading dose 100 mg per day for 3 days
  - 10-20 mg per day
RA: Leflunomide

- Side effects (serious in 20%)
  - GIT: diarrhoea
  - Skin rash
  - Alopecia
  - Raised liver enzymes
  - Bone marrow suppression
  - Severe teratogenicity
  - Cost!

- Pregnancy and lactation: contraindicated for 3 YEARS M+F
IL-1 and TNF-α Are Central Mediators in Rheumatoid Arthritis

Etanercept (Enbrel®)
TNF-α receptor fusion protein

**TNFα**

**TNFα receptor**

**p75 TNFα receptor (Recombinant)**

**Fc of IgG**

**IgG**
Infliximab (Revellex®)
Dimeric (murine + human) anti-TNF-alpha antibody

TNF-α receptor

Dimeric IgG against TNF α
Adalimumab (Humira ®)
Humanised anti-TNF-alpha antibody

TNF- \( \alpha \) receptor
TNF- \( \alpha \)
IgG against TNF \( \alpha \)
Anakinra
Recombinant human IL 1 receptor antagonist

IL-1 receptor

IL-1

IL1 receptor antagonist
Anakinra molecule
Efficacy of biological treatment

ACR20  ACR50

MTX  aTNF+MTX  l1ra+MTX
Medical management: Disease modification

- Immunosuppressants in severe RA, CTD
  - High doses IV steroids
  - Azathioprine (Imuran)
  - Cyclosporine (Neoral)
  - Cyclophosphamide (Endoxan)
  - Chlorambucil
  - Mycophenolate Mofetil
**Immunosuppressants**

- Severe disease especially if vasculitis or systemically unwell
- Could lower daily requirement of steroids
Surgical Interventions
Surgical Management

- Arthroscopy: knee, shoulder
- Arthrodesis: wrist, ankle, IP
- Excision arthroplasty: elbow, wrist
- Arthroplasty: hip, knee, MCP’s
- Synovectomy: knee
- Tenosynovectomy: wrist, trigger finger
- Osteotomy: OA
Common surgical interventions

- Cervical spine: stabilization
- Shoulder: decompression, replacement
- Elbow: excision of radius head, replacement
- Wrist: excision of ulna head, synovectomy, arthrodesis, carpal tunnel release
- MCP: synovectomy, Swanson prosthesis
- Fingers: soft tissue release, Swanson’s, arthrodesis
Common surgical interventions

- Hip: replacement
- Knee: synovectomy (open/arthroscopic), replacement
- Ankle: arthrodesis, replacement
- Hind/midfoot: arthrodesis
- Toes: excision of MT heads, soft tissue release, arthrodesis
Injection therapy
Corticosteroids
Indications for steroid injections

Any lesion with an inflammatory component
## Properties of Steroid drugs

<table>
<thead>
<tr>
<th></th>
<th>Glucocorticoid</th>
<th>Mineralocorticoid</th>
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<tbody>
<tr>
<td>Fludrocortisone</td>
<td>10</td>
<td>125</td>
</tr>
<tr>
<td>Cortisone</td>
<td>0.8</td>
<td>0.8</td>
</tr>
<tr>
<td>Hydrocortisone</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Prednisolone</td>
<td>4 Potent</td>
<td>0.8</td>
</tr>
<tr>
<td>Methylpred</td>
<td>5 Potent</td>
<td>0.5</td>
</tr>
<tr>
<td>Triamcinolone</td>
<td>5 Potent</td>
<td>0</td>
</tr>
<tr>
<td>Betamethasone</td>
<td>25 Very potent</td>
<td>0</td>
</tr>
<tr>
<td>Dexamethasone</td>
<td>25 Very potent</td>
<td>0</td>
</tr>
</tbody>
</table>
Steroids used in soft tissue injections

<table>
<thead>
<tr>
<th></th>
<th></th>
<th>Duration</th>
<th>Conc</th>
<th>1 ml eq to pred (mg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>TC</td>
<td>Ledercort</td>
<td>1-4 W</td>
<td>25 mg/ml</td>
<td>25 mg</td>
</tr>
<tr>
<td></td>
<td>Lederspan</td>
<td>3-4 W</td>
<td>20 mg/ml</td>
<td>20 mg</td>
</tr>
<tr>
<td>MP</td>
<td>Depo Medrol</td>
<td>½ D-4 W</td>
<td>40 mg/ml</td>
<td>40 mg</td>
</tr>
<tr>
<td></td>
<td>Medrol</td>
<td></td>
<td>80 mg/ml</td>
<td>80 mg</td>
</tr>
<tr>
<td>BM</td>
<td>Celestone Diprosone</td>
<td>3-4 D</td>
<td>4 mg/ml</td>
<td>21.3 mg</td>
</tr>
<tr>
<td></td>
<td>Celestone Soluspan</td>
<td>1-2 W</td>
<td>6 mg/ml</td>
<td>32 mg</td>
</tr>
<tr>
<td>DM</td>
<td>Decadron</td>
<td>3 D-3 W</td>
<td>4 mg/ml</td>
<td>21.3 mg</td>
</tr>
<tr>
<td></td>
<td>Oradexon</td>
<td>3 D-3 W</td>
<td>5 mg/ml</td>
<td>26.6 mg</td>
</tr>
</tbody>
</table>
Solubility of Steroid Preparations

- Very soluble: Hydrocortisone
- Moderately soluble: Betamethasone
- Poorly soluble:
  - Triamcinolone
  - Methylprednisone

- Soluble preparations → soft tissue injections (less likely to provoke tissue damage)
- Poorly soluble steroids → joint injections
Systemic side effects

- Skin flushing (>20 mg MP)
- Menstrual irregularity (>40 mg MP)
- Muscle wasting and myopathy
- Impaired glucose tolerance
- Osteoporosis
- Psychological upset
- Steroid arthropathy
- Adrenal suppression
- Immunosuppression
Local Side Effects

- Post injection flare:
- Infection: Increased pain & local signs, febrile and ill
- Subcutaneous atrophy: Too large dose/ volume, too frequent injection, subcutaneous injection
- Skin depigmentation: Too large dose/ volume, too frequent injection, subcutaneous injection
- Tendon rupture: Too large dose/ volume, too frequent injection, bolus injection
Local Anaesthetics
Indications for use of Local Anaesthetics

• Aid to diagnosis
• Diminish pain (mix with steroid)
  – Solubility: BM>MP
• Provide volume for injection of steroids (large joints, bursae)
<table>
<thead>
<tr>
<th>Local anaesthetic characteristics</th>
<th>Time to onset</th>
<th>Duration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lignocaine</td>
<td>1-2 min</td>
<td>1 h</td>
</tr>
<tr>
<td>Bupivacaine</td>
<td>&lt; 30 min</td>
<td>&lt; 8h</td>
</tr>
</tbody>
</table>
Local effects of local anaesthetics

- Loss of pain
- Loss of all sensation
- Loss of motor power

Increasing dose of local anaesthetic
### Safe doses of local anaesthetics

<table>
<thead>
<tr>
<th>Concentration</th>
<th>Max volume</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Lignocaine</strong></td>
<td></td>
</tr>
<tr>
<td>1%</td>
<td>20 ml</td>
</tr>
<tr>
<td>(200 mg)</td>
<td>10 ml</td>
</tr>
<tr>
<td>2%</td>
<td></td>
</tr>
<tr>
<td><strong>Bupivacaine</strong></td>
<td></td>
</tr>
<tr>
<td>0,5%</td>
<td>30 ml</td>
</tr>
<tr>
<td>(150 mg)</td>
<td></td>
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</tbody>
</table>
Side effects of local anaesthetics

• Allergic reactions (observe for 30 min)

• Toxic reactions (Overdose/IVI)
  – CNS: peri-oral paresthesiae, agitation, convulsions, coma, respiratory arrest
  – CVS: cardiovascular collapse
OA: Hyaluronan injections

- Made from rooster comb
- Supplement the reduced endogenous hyaluronan in joint
- Efficacy due to effects on synovial cytokines
- No definite long term efficacy in trials, but some patients get symptomatic relief lasting months
- Use in patients who require, but do not want, surgery and who failed to respond to corticosteroid injection
Summary

- Multidisciplinary approach
- Rational use of medical, surgical and allied health interventions