

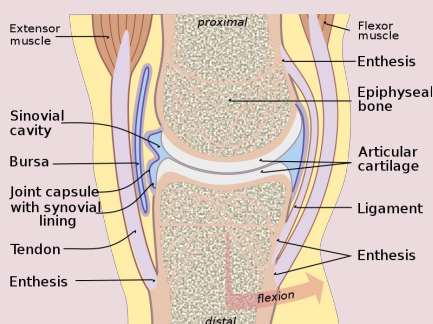
### Introduction

#### What is pathologic synovial disease?

- Inflammatory
  - Infectious
  - Degenerative
  - Traumatic
  - Haemorrhagic
  - Neoplastic
- => Leading to irreversible joint destruction

**What are arthritides?** Inflammation of joints due to infectious, metabolic, or constitutional causes

### Synovial joint



### Pathophysiology

- Physiology/ function** in abnormal states → specifically the **functional changes** that *accompany* a particular syndrome or disease
- It's about disordered function, i.e. there's function but it's abnormal
- When you wish to find/define the pathophysiology of a condition it's helpful to ask: **What's not functioning well?**

### Mechanisms

- Determined by the pathophysiology, i.e. disordered function
- They're the **defects in systems, organs, cellular & molecules** that constitute the **triggers** of specific diseases
- They **originate & explain** the clinical signs & symptoms
- When you wish to find/define the mechanism of disease, it's helpful to ask: **How is the specific pathophysiology leading to occurrence of these specific signs & symptoms?**

### Gout

#### Signs & symptoms:

- **Acute form:** painful, warm & swollen joint
- **Chronic tophaceous form:** topi in tendons, bursae & cartilages

#### Pathophysiology:

- **Hyperuricaemia & a ↓ in urinary excretion of uric acid** → both lead to *deposition & crystallisation* of uric acid in joints → followed by an inflammatory response with **release of enzymes** in joint space

#### Mechanism:

- **Deposition & crystallisation** occurs in previously traumatised or "-cooler" joints. *Neutrophil disruption* leads to enzyme release & inflammatory cascade

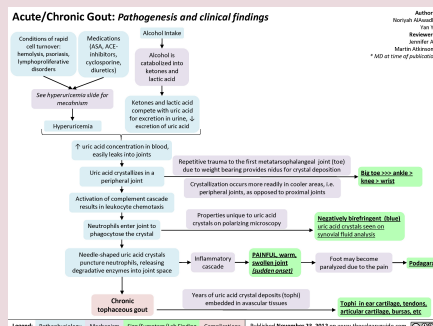
#### Does the pathophysiology explain/relate to the specific mechanisms of disease, how?

- Yes
- Hyperuricaemia leads to deposition of uric acid
- Previous trauma & location favours crystallisation
- Crystal deposition triggers immune response
- Damage to neutrophils produces the release of enzymes which irritate joints & cause an inflammation, i.e. arthritis

#### Do the mechanism explain the clinical signs & symptoms, how?

- Yes
- Inflammation causes painful, warm, swollen joints in the acute form
- In chronic tophaceous form: long term deposition of uric acid crystals in avascular tissues cause topi in tendons, bursae & cartilages

### Gout





### Ankylosing Spondylitis (cont)

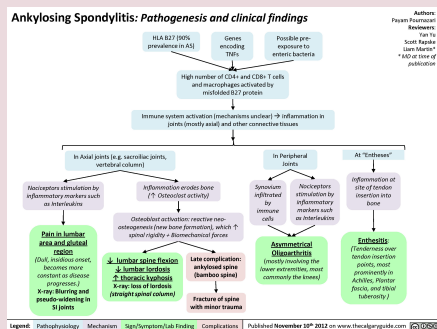
Does the pathophysiology explain/relate to the specific mechanisms of disease, how?

- Yes
- Axial joint arthritis (sacroiliac & vertebral column) causes **release of inflammatory substances** that stimulate **nociceptors**
- **Inflammation** leads to **osteoclast activation & erosion of bone**, which in turn causes **osteoblast activation with new bone formation**
- Leads to an **↑ in spinal rigidity**
- In **peripheral joints**, the arthritis causes **release of inflammatory substances** that stimulate **nociceptors**
- There's **infiltration** of the synovium by **inflammatory cells & inflammation of the entheses** (places of tendon insertions in bone)

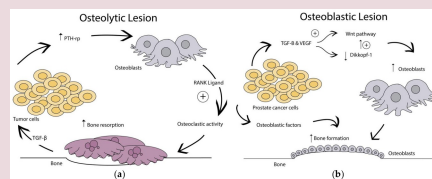
Do the mechanism explain the clinical signs & symptoms, how?

- Yes
- **Stimulation of nociceptors** causes **pain in axial & peripheral joints**
- **↑ in spinal rigidity** causes a **↓ in Lx spine flexion**, **↓ in Lx lordosis & ↑ in Tx kyphosis**
- Late **complications** include **spinal ankylosis & fractures**
- In peripheral joints, **stimulation of nociceptors** causes pain of lower limb joints & knees
- **Enthesitis** causes pain in achilles tendon, plantar fascia & tibial tuberosity\*

### Ankylosing Spondylitis



### Bone Remodelling Cycle



### Osteoarthritis

Signs & symptoms:

- Joint pain with loading & motion
- Palpable bone hypertrophy
- ↓ in ROM
- Crepitus (popping/crackling sound)
- Joint effusion

Pathophysiology & mechanism:

- Joint cartilage **destruction with inflammation**

Does the pathophysiology explain/relate to the specific mechanisms of disease, how?

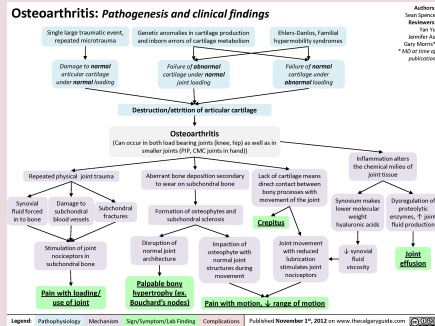
- Yes
- Cartilage **inflammation** in weight bearing joints (**knee, hip**) & smaller joints stimulates **nociceptors**
- Joint cartilage loss causes **wear** of exposed subchondral bone, which induces **defective new bone formation** leading to the appearance of **osteophytes & subchondral bone sclerosis** leading to **changes** in joint architecture
- During movement osteophytes & subchondral sclerosis are firmly pressed against normal joint structures
- Cartilage loss brings joint bones into direct contact between themselves with **reduction in joint movement** & stimulation of **nociceptors**
- **Joint inflammation** leads to **chemical changes** within the joint causing a **↓ in synovial fluid viscosity** & an **↑ in joint fluid production**

Do the mechanism explain the clinical signs & symptoms, how?

- Yes
- Stimulation of joint **nociceptors** causes **joint pain** whether upon **loading** or during **motion**
- Change in **joint architecture** consists of the appearance of **palpable bone hypertrophy**, i.e. **Bouchard's nodes**
- **Osteophytic & subchondral sclerosis** impaction against normal joint structures causes **pain & ↓ joint ROM**
- Joint bones into direct contact causes **friction & crepitus**, **pain & further ↓ in ROM**
- **↓ in synovial fluid viscosity** & an **↑ in joint production** produce joint **effusions**



### Osteoarthritis



### Polymyalgia Rheumatica

#### Signs & symptoms:

- Morning stiffness
- Aching of pectoral & pelvic girdle muscle structures
- Malaise
- Weight loss

#### Pathophysiology & mechanism:

- Aging combined with systemic (auto) immunologic/inflammatory process targeting structures in the walls of arteries with activation of macrophages

#### Does the pathophysiology explain/relate to the specific mechanisms of disease, how?

- More challenging to connect pathophysiology & mechanism
- The **inflammation of arteries** may lead to their **obstruction** potentially causing **hypoxia, ischemia & necrosis** of affected tissues
- This include muscles in the **shoulder girdle & pelvic girdle**

#### Do the mechanism explain the clinical signs & symptoms, how?

- **Yes** but challenging
- It's plausible that the **arteritis** & consequent **hypoxia & ischemia** of affected muscles together with the **systemic inflammation** **lead to** stiffness, aching, malaise & weight loss\*

### Polymyalgia Rheumatica

