FACULTY OF
PAIN MEDICINE
of the Royal College of Anaesthetists

Question examples:

1 MCQs

The first section of the FFPMRCA Examination will be 90 Multiple Choice Questions:

- 40 Multiple True False questions
- 25 Single Best Answers
- 25 Extended Matching Questions

Please find examples of these three question types below.

1.1 MTFs

These are the kinds of MCQ everyone is familiar with: a stem followed by five options each of which may be true or false. The example below is clinical but MCQs will be written to examine the entirety of the Pain Medicine and generic sections of the curriculum.

TOPIC: TRIGEMINAL NEURALGIA

Trigeminal neuralgia

A. Requires radiological imaging to establish the diagnosis (FALSE)

B. Has a peak onset in the fifth and sixth decade of life (TRUE)

C. Occurs in 20% of patients with multiple sclerosis (FALSE)

D. Is best treated with carbamazepine (TRUE)

E. Treated by microvascular decompression surgery is associated with a 5% mortality (FALSE)

1 Post-herpetic neuralgia is:

A a consequence of neurological damage caused by the Herpes Simplex virus (FALSE)

B more common in the elderly patient (TRUE)

C characterised by 'lightning' pain (TRUE)

D unresponsive to treatment with tricyclic antidepressants (FALSE)

E prevented by treating the acute phase with acyclovir (FALSE)

2 The Brief Pain Inventory (BPI) questionnaire:

A was originally designed to assess pain in cancer (TRUE)

B cannot be self-administered (FALSE)

C uses word scores to assess the quality of pain (FALSE)

D assesses pain over the last month (FALSE)

E assesses the interference from pain on work (TRUE)

3 Gabapentin:

A has a lower bioavailability than pregabalin (TRUE)

B acts primarily on GABA-A receptors (FALSE)

C causes seizures at high doses (FALSE)

D reaches its maximum plasma concentration 30 minutes after oral dosing (FALSE)

E is absorbed actively from the gut via a saturatable transport system (TRUE)

4 The coeliac plexus is :

A directly anterior to the crura of the diaphragm (TRUE)

B directly anterior to the inferior vena cava (FALSE)

C directly anterior to the aorta (TRUE)

D at the level of the L3 vertebra (FALSE)

E directly posterior to the pancreas (TRUE)

5 The descending pain inhibitory system includes:

A periaqueductal grey matter (TRUE)

B gamma efferent system (FALSE)

C locus coeruleus (TRUE)

D nucleus tractus solitaries (FALSE)

E nucleus raphe magnus (TRUE)

6 Glutamate:

A is an excitatory peptide neurotransmitter (FALSE)

B reuptake is modulated by glial cells (TRUE)

C binds to AMPA, kainate and NMDA receptors (TRUE)

D binds to G-protein coupled receptors resulting in sustained depolarisation (TRUE)

E is metabolised in the synaptic cleft (FALSE)

1.2 SBAs

These are now being used in the RCoA exams and are well described in the article by Dr Liam Brennan, Chair of the Final FRCA examination:
http://www.rcoa.ac.uk/docs/Bulletin57.pdf.

CATEGORY: CLINICAL PAIN

A 76 year old man with low back pain has been referred to your clinic. The pain has been well controlled for several months with immediate release morphine. He says the morphine is no longer working so well. The most likely reason for this is:

Options:

A A change of preparation

B Addiction

C Inappropriate use of a short duration of action opioid

D Tolerance

E Underlying malignancy

Answer: D

A 57 year old man has complained of a shooting pain over his right cheek for 6 months. This can happen several times a day. The pain is worse in cold weather and can wake him at night. He is pain free between attacks. There are no abnormal findings on examination.

The most appropriate investigation is:

A CT scan of the head

B MRI angiography of the head

C orthopantogram (OPG) X-ray

D skull X-ray to include the zygoma

E upper cervical spine X-ray

Answer: B

Reasoning and Comments: This is trigeminal neuralgia.

The highest dose of ionising radiation to the patient occurs during:

A bone density scan

B chest X-ray

C CT head

D CT spine

E isotope bone scan

Answer: D

A 28 year old man comes to your pain clinic. He has had severe headaches for the last six months. The headaches are precipitated by drinking lager with his friends. He had similar headaches a few years ago but these got better.

The most likely diagnosis is:

A cerebral metastasis

B chronic daily headache

C cluster headache

D migraine

E tension-type headache

Answer: C

1.3 EMQs

Extended Matching Questions are used by the Royal College of Obstetricians & Gynaecologists for their major examinations. Two Pain Medicine example are given below but please also visit the two online documents below to see some O&G examples.

http://www.rcog.org.uk/files/rcog-corp/uploaded-files/Ex-Diploma-Format.pdf
http://www.rcog.org.uk/files/rcog-corp/uploaded-files/Ex-Part-2-Format-March2011.pdf

Facial pain

Options for questions n-n

A. Dental pain

B. Migraine

C. Post herpetic neuralgia

D. Sinus pain

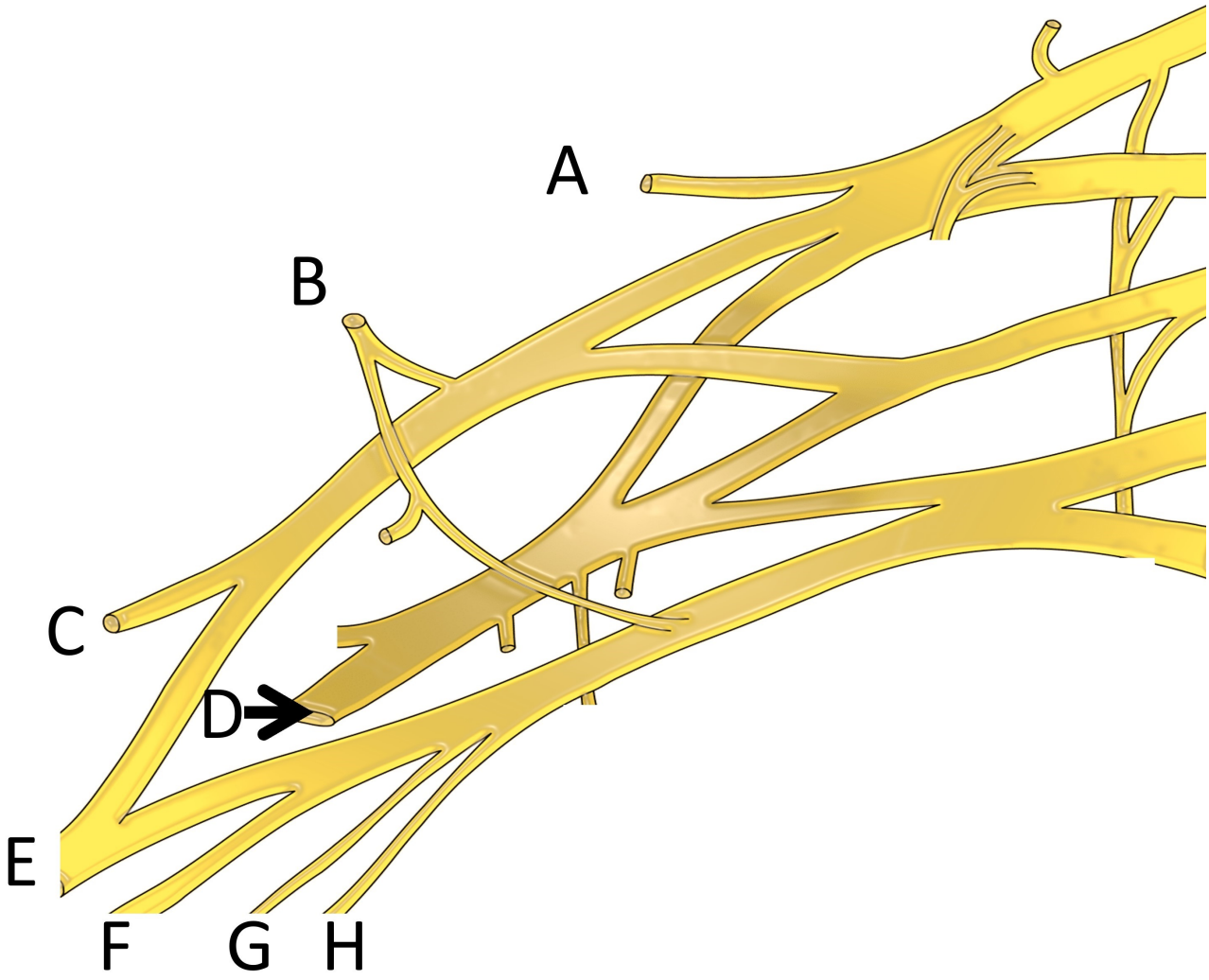
E. Temporo-mandibular joint pain

F. Trauma

G. Trigeminal neuralgia

H. Tumour related pain

Instructions: For each scenario described choose the single most likely diagnosis from the list above.



The radial nerve is labelled in the above diagram as D

The musculocutaneous nerve is labelled in the above diagram as C

The medial cutaneous nerve of the arm is labelled in the above diagram as H

The ulnar nerve is labelled in the above diagram as F

The suprascapular nerve is labelled in the above diagram as A

Question:
A 65 year old woman has been referred to the pain clinic by the maxillofacial l surgeons; no cause of pain inside or outside the mouth has been found. She has had unilateral pain over the right cheek for a year. The pain is described as a constant ache with shooting sensations. She has no history of a rash. On examination there are no sensory changes but there is widespread tenderness on both sides of her face.

Answer D Sinus pain

Question:
A 38 year old woman has been referred to the pain clinic with brief spasms of facial pain. She has recently had an episode of blurred vision and ataxia.

She is awaiting review by the neurologists. The most likely diagnosis is.

Answer G Trigeminal neuralgia (secondary to multiple sclerosis)

Headache

Options:

A cerebral secondary malignancy

B cluster headache

C chronic daily headache

D migraine

E occipital headache

F secondary headache

G tension type headache

H trigeminal neuralgia

For each of the scenarios below, choose the item that provides the most appropriate answer from the above options. Each option may be used once, more than once or not at all.

1 A 20 year old student complains of generalised pain in the back of her head and neck that feels like a 'tight band'. Her headaches normally come on during the day, and get worse as the day goes on. (Answer: G)

2 This headache is commonly associated with an aura. (Answer: D)

3 This headache is commonly unilateral. (Answer: D)

4 This headache causes attacks that occur over several weeks or months and then remit for several months or years. (Answer: B)

5 This headache is the most common primary headache . (Answer: G)

SOE QUESTION BANK.

The decision was made late in 2021 to publish the title in short form of the entire SOE bank of questions. This was done with the aim of assisting candidates to prepare better for the examination, by demonstrating the broad scope of questions which they will encounter in their exam, and will direct them in their learning, as well as reassure them of the clear clinical relevance of questions linked to the practice of a pain consultant.

It is important to note that the candidate should be prepared for the high likelihood/certainty that new questions, similar in relevance and scope to those here, and not present on this list, will form part of their exam, as new questions are added in a continuous process of development.

It is also important to note that the question list used by the Examinations department will change over time, and that the FPM makes no undertaking to refresh this list on a regular basis- the purpose of this list is to guide the trainee in their preparation by illustrating the breadth and depth of topics covered.

Furthermore, while every effort is made to produce a balanced paper without clear duplication, there may appear, within the same set of examination questions in a single (Clinical and Science SOE) paper, some overlap.

As explained elsewhere, the paper will consist of 1 long clinical case in 3 parts (21 mins in total), 3 short clinical cases (7 mins each), and 4 Science questions, each 7.5 mins long, 1 drawn from each section of pharmacology, Physiology, Anatomy, and Miscellaneous, all as below in alphabetical order within each section.

Clinical Long Qs

Acute pain and opioid addiction

NICE NG 59 Back Pain Guidelines

Paediatric knee pain

Palliative care (Acute pain aspects/Interventional aspects)

Pelvic pain

Rheumatoid arthritis

Shoulder pain

Spinal inflammatory disease

Whiplash Associated Disorder

Clinical short Qs

Abdominal pain in children with cerebral palsy

ACT and Mindfulness

Acute pain management of opioid tolerant patient

Acute pain management of Sickle Cell disease

Bio-psycho-social aspects of paediatric pain

Buprenorphine

Cancer pain interventions

Cancer related bone pain

Cannabinoids

Cannabis

Capacity and consent

Carpal tunnel syndrome

Chronic Low Back Pain

Chronic pain after surgery

Classification and interactions of antidepressant drugs

Coccydynia

Complications of SCS

Deep Brain Stimulation

Depression

Diabetic peripheral neuropathic pain

Disability

Epidural abscess

Epidural steroids

Fear-avoidance model of pain

Fibromyalgia

Flail segment

Generalised joint hypermobility

Headache

Intra-thecal Drug Delivery

Ketamine

Lidocaine infusion

Lower limb radicular pain

Lumbar sympathectomy

Medico-legal topics

Methadone

Multiple sclerosis

NAP3 Audit

Nerve root blocks / transforaminal epidural

Neuropathic pain and post herpetic neuralgia

Neurosurgical treatments for chronic pain

Non-obstetric pain in pregnancy

Opioid addiction

Opioid induced hyperalgesia

Opioid misuse predictor tools

Opioid prescribing - best practice in Cancer pain

Opioid side effects

Opioids - equianalgesic doses

Opioids aware

Oro-facial pain

Paediatric pain assessment

Pain assessment in children

Pain in the elderly

Pain management in adenotonsillectomy

Pain Management Programmes

Paracetamol

Physiotherapy

Post Dural puncture headache

Post-mastectomy pain

Post-vasectomy pain

Prescribing for children

Procedural pain management in children

Psychological approaches to chronic pain

Radiation safety

Radiofrequency in low back pain.

Risk measurement tools in opioid use

Serotonin syndrome and drug interactions

Sickle Cell Crisis

Sleep

Spinal cord injury

Spinal stenosis

Stellate ganglion block

Telehealth

The Fear-Avoidance Model of Pain

Transition

Tricyclic antidepressants - clinical use

Trigeminal Autonomic Cephalalgias

Ultrasound

Vulvodynia

Whiplash Associated Disorder

Science

Pharmacology:

Antidepressants

Botulin toxin

Buccal fentanyl

Cannabinoids

Cytochrome P450

Future novel drug targets

Gabapentinoids

Intravenous lidocaine in acute pain management

Lidocaine /EMLA

NMDA receptors

Nociceptors and capsaicin

NSAIDs, COXIBs

Opioid induced constipation and hyperalgesia

Pharmacology in the elderly

Serotonin Syndrome

Steroid preparations in pain

Tramadol

Transdermal delivery of drugs

Physiology:

Addiction

Cancer-induced bone pain (CIBP)

Central Sensitisation (CS)

Complex Regional Pain Syndrome (CRPS)

Developmental neurobiology

Fibromyalgia

Mechanisms of inflammation

Neuropathic pain

Obesity

Placebo

Pre-emptive and preventative analgesia

Principles of Neuromodulation waveform

Sex/gender and pain

Visceral vs. somatic pain

Vitamin D and pain

Anatomy:

Anatomy of sympathetic block

Anatomy of the abdominal and thoracic wall

Anatomy of the pudendal nerve

Anatomy of the sacroiliac joint

Brachial plexus

Cervical spine anatomy

Coeliac plexus block

Cranial nerves

Inguinal region

Intercostal nerve

Intervertebral discs and disc disruption

Lumbar facet joint

Occipital nerves

Pudendal neuralgia

Sacrum

Spinal cord blood supply

Trigeminal nerve

Miscellaneous:

Cognitive behavioural therapy

Genetic factors in pain

Imaging of the brain

Measurement of neuropathic pain

Measurement of validity and reliability

Mindfulness and pain management

Neuropathic pain – diagnosis /screening tools

NNTs & NNHs

Pain assessment in children and the elderly

Physics - neurostimulation

Physiology and measurement of QST

Principle of radiofrequency denervation

Psychiatric diagnoses and pain

Psychology factors in low back pain

Radiation safety

Sleep

Stellate ganglion

Tests for nerve function

Trial design for non-drug therapies

Ultrasound

Xray generation and safe use of X-ray equipment

FULL SOE EXAMPLES- CLINICAL

SOE Clinical Long Case

The long case will have a 10 minute preparation time during which the candidate will be given the opportunity to read a case history and view relevant investigation results provided. During this time the candidate may make notes on paper provided for this purpose. Thereafter, during the next 20 minutes, the clinical long case will examine a candidate's in-depth knowledge of the assessment and management of a complex chronic pain patient. Knowledge of clinical assessment tools and investigations relevant to clinical practice and available treatments will be required.

Example 1:

Scenario:

A 78 year old woman was admitted on medical take with severe headache, fever and nausea.

MRI scan of brain was normal and she was discharged with analgesics after 2 days. Five days later she was admitted for 2 weeks with painful vesicular rash and was treated with acyclovir. Her discharge medication was Tramadol modified release 200mg bd, amitriptyline 20mg OD, carbamazepine 100mg bd. Urgently referred to you at the pain clinic by her GP after 4 weeks with severe pain.

Supporting information:

GP had stopped Tramadol.

What is her diagnosis?

- Persistent pain after Acute herpes zoster with prodromal systemic symptoms (can also include neck rigidity, encephalitis, myelitis)
- Pathophysiology of HZ could be discussed & risk factors
- opinion about when pain becomes post-herpetic neuralgia

What is the annual incidence of HZ and PHN (pain >12 mo) in her age group?

- HZ: Age 40 2.5/100,000 age 75 7:100,000 Overall 1:4 of population
- PHN: Age 40 7.4% Age > 70 c. 50%

Where is she most likely to have developed the rash?

- Unilateral thoracic or ophthalmic dermatome (this lady

Was ophthalmic extending into cervical occipital dermatomes)

What type of pain would she complain of?

- Neuropathic constant aching and may have burning, itching and stabbing
- Often paroxysmal. May complain of numbness and/or hypersensitivity
- This lady had ache, numbness & stabs and no sensitivity apart from ear pinna.
- Quality of life impact high

What would you look for on examination and link with the pathophysiology

- Allodynia, hyperalgesia , hyperaesthesia, scarring and numbness
- Irritable nociceptor, deafferented allodynia and deafferented non-allodynic subtypes
- Eye signs

What is your management?

- Depends on pain history and examination findings – 'aching pain worsened when GP stopped Tramadol and no recent stabs'.
- Could restart Tramadol (helped) and follow NICE neuropathic guidelines, Stop small dose carbamazepine and start pregabalin to 150mg bd and try topical agent

You reviewed after 2 weeks and background ache has improved but stabbing pain has become much worse? What do you do?

- Carbamazepine 100mg BD restarted and with other medication had minimal pain
- Topical agents were unhelpful

Does the stabbing pain mean she has Trigeminal Neuralgia?

- No, TGN is a specific diagnosis made on a history of stabbing pain, triggering factors and periodicity

Is there any role for interventions in treating PHN?

- Probably no evidence of effectiveness

Can we reduce the incidence of post-herpetic neuralgia?

- Early antiviral therapy & early neuropathic pain drugs after start of HZ may help
- Adult vaccination with Varicella vaccine (at >60yrs) reduces incidence of HZ by 51 % and of PHN by 66% in placebo controlled trials.

Example 2:

Category Clinical Long Case

Case Description:

A thirty year old man is referred to the Pain Clinic with pain in his stump and painful phantom sensations 9 months after left above knee amputation following a motorcycle accident. Pain is interrupting his sleep. On examination he has allodynia over the base of the stump which is

preventing him being able to wear an artificial limb. He has phantom sensations with a feeling that his toes on the phantom and clenched downwards digging into the sole of his foot. His GP has started him on Gabapentin 400mg tds which has helped his sleep but not the stump pain or phantom sensations.

Supporting information:

1. Photo of a stump with area marked in pen delineating area of as allodynia

2. Picture of a Mirror Box or mirror held so remaining limb is reflected over amputated limb.

A) Can candidate describe difference between Phantom Sensations and Phantom Pain?

Phantom sensations experienced by almost all amputees usually within the first few days. Telescoping happens in around 1/3 of patients. Pain may be described as neuropathic pain associated with the phantom or the phantom may be described as being in a painful position.

B) What Central Nervous System Changes has been found in Amputees in research using Functional Magnetic Resonance Imaging?

Alterations in areas of representation e.g. mouth and chin invade the cortices representing the arms and digits that have been lost in functional magnetic resonance studies.

Degree of phantom pain relates to the degree of cortical reorganisation in a linear way.

C) How may a mirror be used in a therapeutic trial for this patient's phantom pain?

A parasagittal mirror has been used to modulate pain and has allowed the patient to "move" the position of the phantom by moving the unaffected limb while looking at the visual feedback in a mirror.

D) Describe any topical therapies that may be offered to help allodynic pain in the stump

NB- These are 'off license' indications with NO RCT evidence.

(1) Topical local anaesthetics – lidocaine 5% plasters, topical local creams (EMLA, AMETOP).

(2) Topical Capsaicin Creams (0.0025% or 0.0075%).

(3) Others – topical ketamine cream, ticyclic antidepressant creams etc.

E) Supplementary Question. In a patient with a traumatic amputation resulting from a road traffic accident what concurrent psychological pathology may be present?

(1) Depression.

(2) Anxiety.

(3) Post Traumatic Stress Disorder.

(ICD 10 Classification of above 3 conditions available to examiners if this question is to be used)

(1) Reference. Ramachandaran VS, Altschuler E L. Reflections on hand. Pain 149 (20101) 171172

(2) Nikolajsen L, Jensen T. Phantom Limb. In Textbook of Pain 5th Edition, 2006, Elsevier. 961969

(3) ICD 10 Classification of Mental and Behavioural Disorders. WHO, Geneva. Churchill Livingstone. ISBN 0-443-04909-2

Syllabus: PM_AS_01, PM_AS_02, PM_AS_04, PM_AK_01.

Example 3:

SOE long case: central post stroke pain (CPSP)

A) Explore knowledge of central post stroke pain (CPSP) and demonstrate understanding of the psychosocial issues of CNMP

Candidate information:

A 75-year old man complains of pain in left arm and leg which started 1 month after a CVA about 2 years ago. He has mild COPD and controlled hypertension. He also complains of nocturia and is under investigation by the urologists. He has longstanding frequent headaches. His GP prescribed Gabapentin 100mg BD and Morphine Sulphate Controlled Release 10mg BD.

He is also taking:

- Bendroflumethlazide
- Ramipril
- Salbutamol
- Aspirin
- Simvastatin
- Oxybutynin
- Lactulose

Findings on examination:

- Limited movement of the neck especially rotation.
- Mild residual weakness of the left upper limb > lower limb (dominant side)
- Mild sensory loss of the left arm (pinprick and temp)
- Allodynia over the left side of his body
- Reflexes- no abnormality
- His wife reports he has withdrawn from social interactions, his sleep is disturbed andhe has lost interest in his usual hobbies.

Supporting information:

Na 136, K 4.1, Urea 12.1, Creatinine 195. His GFR is 30.

What is the most likely diagnosis?

Central Post Stroke Pain (CPSP)

Are there possible alternative causes for his pain?

Unlikely since started post CVA and involves arm and leg –possible, if mainly arm pain then he could have OA neck or cervical disc prolapse.

Do the other ongoing problems contribute to the pain?

Discussion of pain and depression causing sleep problems and inability to manage the pain.

What do you know about CPSP?

Pain from a primary lesion or dysfunction of the central nervous system after stroke. Pain can be spontaneous or evoked and continuous or paroxysmal. Described as burning, pricking, shooting, squeezing and throbbing. Allodynia and hyperalgesia associated with it and probably essential parts of the syndrome.

Can occur with ischaemic and haemorrhagic lesions at any level. Anywhere in spinothalamic pathway and its cortical projection. (Thalamic pain described after thalamic stroke in 1906). Probable that half of those with CPSP have lesions involving thalamus.

Pinprick, temperature and touch are impaired in 2/3 of CPSP cases. Distribution of pain in terms of frequency are arm, leg, trunk and face. Most common is hemi-body. Problems are usually contralateral to the side of the cerebral event.

Incidence 8%- 35% of all CVA. (Variation in inclusion criteria). 25% patients after CVA have somatosensory deficits. 85% of strokes are due to infarct so more CPSP seen after infarct.

When does it normally present?

Most in first month (60%) but can occur up to three years.

What is the likely pathophysiology?

Not well understood but central disinhibition, imbalance of stimuli and central sensitisation suggested. 'Wind up' and denervation hypersensitivity. Discriminative sensory deficit in CPSP results in disinhibition which results in spontaneous pain and allodynia. Central sensitization. Importance of sodium channels in central pain.

B) Pharmacological treatment

Would you expect to see any benefit from the dose of gabapentin prescribed?

Discussion of starting low and needing to increase, also discussion of dose modification in renal failure.

Other Rx?

Discussion pregabalin e.g. tolerance and cost issues.

Role of TCA?

Amitriptyline effective but may be contraindicated for this case. (50-70% will benefit)

Role of sodium channel blockade?

Lamotrigine moderately effective in CPSP. Regarded as second line treatment after TCA. Little evidence for carbamezepine or phenytoin.

Role of NMDA antagonists?

Use of oral ketamine, possible as short term measure.

Role of opioids?

Morphine ineffective. Limited evidence for tramadol.

Is there a role for sympathetic blockade?

TENS-useful in patients who have not lost touch and vibration in painful area.

Is there a role for intrathecal drugs?

Limited evidence of value. Baclofen may be useful for spasticity when quality of life is affected

C) Need for multidisciplinary assessment treatment of the psychosocial issues How many patients have fatigue and mood changes?

- 50% disturbed sleep and fatigue
- 87% mood changes

What questionnaires are you aware of that might help with the diagnosis of depression?

- HAD (Hospital Anxiety and Depression score) o 14-item scale – 7 depression, 7 anxiety. o Each item scored 0-3. Maximum score 21 for each. Likert scale. Validated. Cut off is 8/21.
- BDI (Beck Depression Inventory) o 21-question self-report scale. Severity and depth of depression symptoms over the previous week.
    - 0-3 for each question. Score over 17 indicates mild depression, over 40 severe depression. Not a diagnostic tool. Developed in mental health care setting. Used to evaluate therapy.
- Centre of Epidemiology study of Depression Index (CESDI)
- PSQ 9
- Zung
- Hamilton

Need for psychological intervention?

- Essential part of care as depression and other psychological sequelae are common.
- Psychosocial assessment to evaluate the perception of pain and disability and potential barriers to treatment. Influence of mood on pain. Anger/depression/distress.
- Cognitive factors – beliefs and attributions, coping strategies.
- Adjustment – familial, social.
- Possible options for treatment e.g. CBT, cognitive restructuring, relaxation techniques, mindfulness
- Imagery

What role do you see for acupuncture in neuropathic pain states?

Limited but sometimes useful.

Non- pharmacological treatment?

Motor cortex stimulation, deep brain stimulation and transcranial magnetic stimulation have been tried. Need for careful selection / in drug resistant only.

1.4 SOE CLINICAL PAIN MEDICINE: SHORT CLINICAL QUESTION EXAMPLES

The final 21 minutes of the Clinical SOE will be given to the three short clinical questions in equal proportions. The topics of these questions could be any aspect of clinical pain medicine.

Example 1

Scenario:

50 year old woman referred GP with neck pain, altered sensation left hand and wasting thenar eminence.

Supporting information:

Artefact: Nerve conduction studies showing a median nerve sensory and motor deficit.

Question:

How would you examine this patient and what is the differential diagnosis?

Examiners looking to find candidate can describe an appropriate neck and upper limb musculoskeletal and neurological examination. Patient has Carpal Tunnel Syndrome.

Ask them to discuss Differential Diagnosis (from common to uncommon)

- **Cervical radiculopathy** (especially C6/7)— look for local neck pain on movement and neurological signs outside the territory of the distal median nerve
- **Generalised peripheral neuropathies** — these should be recognised from the wider distribution of symptoms and reflex changes
- **Tendonitis** — specific tests may help in diagnosis, such as Finkelstein's test for De Quervain's tenosynovitis
- **Osteoarthritis of the metacarpophalangeal joint of the thumb** — this can produce a spurious appearance of thenar wasting but not true weakness or sensory deficit
- **Raynaud's phenomenon**—this should be recognisable from a history of symptoms related to cold exposure
- **Vibration white finger**—suspect this if the patient uses vibrating hand tools at work
- **Motor neurone disease**— this can present with wasting in one hand but does not produce sensory symptoms.
- **Syringomyelia**—features such as prominent loss of temperature sensation in the hands should give a clue -Multiple sclerosis.

Also looking for description of:

Phalen's Sign – Flex the wrist for a few minutes and wait to see if symptoms provoked.

(Sensitivity ranges10% to 73%; Specificity from 55% to 86%).

Tinel's sign – lightly tap over the flexor retinaculum to provoke symptoms. (Sensitivity range 8% to 100%; Specificity from 55% to 87%)

Supplementary question: Describe the contribution of the sensory and motor supply to the hand of the median nerve

Sensory – skin of the palmar side of the thumb, index, middle and half of the ring finger.

Motor - First and second lumbricals ; muscles of the thenar eminence by recurrent thenar branch. (LOAF – Lumbricals, Opponens Pollicis, Abductor Policis Brevis, Flexor Pollicis Bevis.

Final supplementary question: What is the role of Steroid Injection?

- Carpal tunnel syndrome has been shown to respond to both systemic steroids and to local steroids given at (or near) the wrist by injection.
- Local steroid injection has no discernible systemic effects and a very low incidence of local

  Complications. - Median nerve damage from intraneural injection has been reported in eight cases, the risk may be estimated at<0.1% in competent hands.
- The initial response rate to a single steroid injection is about 70%, but relapse is common. - No adequate long term studies exist to allow precise quantification of the relapse rate beyond the first few months.
- Pessimistic estimates suggest that 92% may have relapsed by two years. At the other extreme is a series in which half of injected patients remain in remission at seven years.
- No evidence is available to guide policy on treatment after relapse following a successful first injection, though it is common practice to inject a second or sometimes third time.

Example 2

Short Clinical Question

Category Oro-facial pain

Opening question: What is the differential diagnosis?

Supporting information:

A 46 year old woman with constant diffuse burning mouth pain for 18 months referred from

Maxilo Facial Surgery. No pathology found on investigation. Symptoms had improved on 75mg Desulepin but had to discontinue because of daytime sedation. She reports feeling low in mood pervasively for 6 weeks, struggling to work and having suicidal thoughts

Guidance to examiners:

Differential Diagnosis (Diagnosis likely to be Burning Mouth Syndrome)

"Suggested Classification Chronic Orofacial Pain"

- Neuropathic Pain – Primary Neuropathies e.g. Trigeminal Neuralgia; Glossopharyngeal Neuralgia and Secondary Neuropathies e.g. PHN, Diabetes, Multiple Sclerosis, HIV, Postsurgical and lingual inferior alveolar nerve injuries.
- Idiopathic - Burning Mouth Syndrome ; Idiopathic (atypical) facial pain ; Temperomandibular Joint Pain.
- Neurovascular – Tension headache ; Migraine ; Cluster Headache ; Giant Cell (temporal) arteritis; SUNCT(short lasting unilateral neuralgiform conjunctival irritiation and tearing)
- Acquired Intra Oral Pain : 'Surgical Sieve'

(NB- Odontogenic Pain = pain from teeth or supporting structures i.e. mucosa ; gingival; periodontal membranes).

- Infection toothache caused by inflammation of the dental pulp or apical abscess; Gingivitis ; Alveolar Ostitis (food in a socket can cause recurrent infection and inflammation) and referred pain from Maxillary Sinusitis.
- Trauma (tooth fracture). Malignancy.

Supplementary questions:

1. **Discuss the common side effects of Tricyclic Antidepressants.**

- Mainly relate to antimuscarinic properties of TCA's – Dry mouth ; blurred vision ; constipation ; tachycardias ; cognitive impairment ; sexual dysfunction.
- Caution – Epilepsy (lower seizure threshold) ; Cardiac Toxicity in Over dosage (risk of ventricular arrhythmias) ; Prostatic Hypertrophy ; Glaucoma ; History of Bipolar Affective Disorder or Psychosis (caution can precipitate mania).

2. **How can you further assess her report of low mood and suicidal ideas?**

- Need to explore by undertaking a more in depth Mental State Assessment and possibly using Mood Rating Scales.
- Important the examiners use the Faculty Psychology Document as the basis for the standard of assessment and ascertain that the candidate can safely make a basic risk assessment in order to make an appropriate urgent referral to Mental Health or to triage to an outpatient Psychiatric or Psychological Assessment but the key is to SEEK TELEPHONE ADVICE FROM ON CALL MENTAL HEALTH TEAM IF UNSURE. May be a time link between discontinuing the Desulepin and onset of Depressive Symptoms even though the therapeutic level for depression for TCA's usually seen as higher than 75mg.

(1) Not all examiners may agree with 'The suggested classification of chronic orofacial pain' referenced and it has not been adopted by IASP but it is included to provide a framework of reference for differential diagnosis.

(2) A better candidate will have a structured approach to differential diagnosis.

(3) Glossopharyngeal Neuralgia : Characterised by pain attacks similar to Trigeminal Neuralgia but located unilaterally in the distribution of the Glossopharngeal Nerve. Pain felt usually in the posterior pharynx, soft palate, base of the tongue, ear, mastoid or side of the head.

Example 3

Short Clinical Question

Opening question: When consenting a patient for lumbar sympathectomy, what possible side-effects to you warn the patient about?

Supporting information:

75 male with widespread arterial disease unsuitable for further revascularisation referred for a Lumbar Sympathectomy.

Guidance to examiners:

Side Effects

1. Intravascular Injection (reduced if fluoroscopy used).
2. Genitofemoral neuralgia (reported 4-15%).
3. Psoas Muscle Necrosis.
4. Renal / Ureter damage.
5. Impotence failure of ejaculation.

Supplementary questions:

1. What is the effect of Phenol on nerves?

Phenol causes neurolysis by denaturing the proteins of axons and perineural blood vessels

2. What other chemicals are used to achieve neurolysis and how do they work?

- Alcohol - Alcohol extracts cholesterol, cerebrosides and phospholipids from nerve tissue and causes precipitation of lipoproteins and mucoproteins – i.e. changes typical of Wallerian Degeneration.
- **Glycerol** – Glycerol results in myelin sheath swelling, axonolysis and severe inflammatory response. Wallerian degeneration.
- Ammonium Compounds – Acute Degenerative neuropathy of all fibre types.

Example 4

Short Clinical Question

What are the mechanisms of action of epidural steroids?

- Steroids primarily exert their inhibitory function via the lipoxygenase pathway to reduce theformation of leukotrienes.
- Cells exposed to glucocorticoids synthesize and release a phospholipase A2inhibitoryglycoprotein, lipomodulin. The inhibitory action of lipomodulin reduces the formation ofarachidonic acid.
- Glucocorticoids stabilise leucocyte lysosomal membranes and prevent the release of destructive acid hydrolases from leukocytes
- Inhibition of macrophage accumulation in inflamed areas
- Reduce fibroblast proliferation, collagen deposition, and scar tissue formation.
- Suppress the production of inflammatory lymphokines and monokines including IL-1 and TNF

What are the systemic and local side effects of steroids?

- Endocrine- adrenal suppression, hyperglycaemia, hypokalaemia, amenorrhoea, menstrual disturbances, growth retardation
- Cardiovascular- hypertension, fluid retention, CHF, DVT
- Musculoskeletal- osteopenia, osteoporosis, avascular necrosis of bone, pathological fracture, muscle wasting and atrophy, muscle and joint pain
- Psychological- mood swings, insomnia, psychosis, anxiety, euphoria, depression
- Gastrointestinal- dyspepsia, GI bleed, diarrhoea, constipation
- Ocular- retinal haemorrhage, increased intraocular pressure, exopthalmos, glaucoma
- Dermatological- facial flushing, impaired wound healing, hirsutism, petechiae, ecchymosis, dermatitis, hyperpigmentation, cutaneous atrophy, hypopigmentation
- CNS - headache, vertigo, insomnia, restlessness, increased motor activity, ischaemic
- PNS - neuropathy

Example 5

Short Clinical Question

Introduction:

Sickle cell disease is the name given to a group of lifelong inherited conditions of haemoglobin formation. Red blood cells in people with sickle disease behave differently under a variety of conditions including dehydration, low oxygen levels and elevated temperature. Patients with sickle cell anaemia may suffer from

- Anaemia and sequelae
- Pain – acute, chronic and acute-on chronic
- Ischaemic organ damage / infections and comorbid conditions.

Acutely painful sickle episodes (painful crises) are characterised by the effects of sickle cell vasoocclusion - micro +/- macro vascular which cause ischaemia, tissue damage and pain. Most crises last 5-7 days however the severity, frequency and duration can vary.

Scenario read to the candidate:

"A 18 year old female of Afro-Caribbean descent has attended the Accident and Emergency Department with hip pain from a sickle cell crisis. The advice of the pain team is sought by A+E staff to guide analgesia".

What information do you want to find when taking a clinical history from this patient?

- Pain history - locations of pain, current and previous analgesics / history of pain interventions and how helpful these were.
- Assessment of possible causes of the sickle cell crisis – hypoxia, infection, dehydration, bleeding, alcohol and drug misuse and pregnancy. NB Often there is no predisposing cause.
- Assessment of associated conditions secondary to end organ damage – May be history of anaemia, stroke, renal impairment, pulmonary hypertension. Possible history of splenectomy (susceptibility to encapsulated bacteria – may be on long term prophylactic antibiotics)

What would you look for on physical examination of this patient?

- Airway / Breathing - shortness of breath (Secondary to Infection or 'Acute Chest Syndrome' characterised by SOB, fever and chest pain - may be hard to differentiate from chest infection).
- Circulation - Hypovolaemia may be manifested by tachycardia, hypotension, venous collapse (Note: splenic sequestration crisis - acute painful enlargement of the spleen, an emergency that may lead to circulatory collapse).
- Disability - Look for clinical signs of condition and associated comorbidities. Hip pain may be from avascular necrosis of the femoral head from vaso-occlusion or from osteomyelitis of the femur.

What Observations and Investigations would help you assess this patient?

- Vital signs and non-invasive SpO2 Blood Tests:
  - (I) blood gases
  - (II) (II) FBC, in particular to quantify the extent of the anaemia and white cell count. Reticulocyte count, this reflects red cell production. Infection (especially by parvovirus) may reduce red cell production and precipitate a crisis (aplastic crisis).
  - (III) (III) HbS % o (IV) U+Es
- IMAGING - X-Rays hip and femur / MRI hip and femur

Discuss acute pain management strategies for this patient?

(a) Analgesia

- Optimisation of analgesics stepwise from combined simple painkillers, through to strong opioids however renal impairment may contraindicate NSAIDS. Oral route preferred if possible however inpatient admission and intravenous opioids often needed (e.g. PCA). May be opioid tolerant already and PCA may need to be modified. Medication for neuropathic pain may be considered if indicated including ketamine acutely. Avoid Pethidine.

- Possible role for regional anaesthetic technique (various options including psoas compartment block), Trial of TENS.

(b) Condition – Specific

- Correction of any dehydration and hypoxia and dehydration.
- Aggressive treatment of infection if present.
- Interdisciplinary Care – Patients with acute sickle crises should by managed by acute medicine haematology specialists to treat underlying medical conditions and for further management such as blood transfusion to correct anaemia and its sequelae plus reduce the HbS %, also agents such as hydoxyurea (promotes fetal Hb production in place of HbS) may be indicated. Orthopaedic involvement for the treatment of AVN / osteomyelitis often from Salmonella and Staph. Aureus.

Full SOE Examples- Science

1.5 SOE SCIENCE

The Science Structure Oral Examination (SOE) of the FFPMRCA examination will comprise four sections, anatomy, physiology, pharmacology and a section covering psychology, epidemiology and clinical measurement. The total SOE will last 30 minutes, each section being 7.5 minutes and marks will be allocated proportionately to each section. There will be 2 examiners for the Science SOE. The importance of the scientific basis of Pain Medicine for the FFPMRCA examination must be emphasised.

Example 1

Category Physiology

Opening question: What is the role of glial cells in the nervous system? What role do they play in pain modulation?

Supplementary question(s): How do glia interact with opioids? What potential targets for pain treatment do glial cells present?

Scientific principle to be explored: Structure and function of glia in pain.

Clinical application: Potential treatments targeted at suppressing glial activation. Role in opioid tolerance.

Supporting information: Pain 2008 Updated Review IASP Press Ch27 pp249-268

***Background:**

- Astrocytes and microglia.
- Do not have axons or communicate from spinal cord to brain.
- Play no role in pain transmission until activated.
- Once activated release neuroexcitatory and neurotoxic substances.
    - These substances may then activate other glial cells in a +Ve feedback manner to create a state of neuroexcitation.
    - The primary afferent then releases more neurotransmitter (SP, glutamate) and the transmission neurons become more excitable – therefore increasing the "gain on pain".

- Involved with "sickness response" (sickness induced hyperalgesia) and pain facilitation.

- ***Microglia** 5-10% of all glial cells.
  - Usually scavenger role.
  - When activated produce pro-inflammatory mediators o Proliferate in nerve injury (by mitosis, conversion of monocytes into glia)

- ***Astrocytes** 40-50% of all glial cells (outnumber neurons).
  - Encapsulate synapses
  - Play a supportive role (trophic, axon guidance, synapse formation)
  - Modulate neural transmission when activated
  - Involves NMDA & AMPA

***Substances released:**

Proinflammatory cytokines (IL-1,IL-6, TNF)
NO
Oxygen radicals
EAAs
PGs
ATP.

Glial interaction with morphine: microglia express opioid receptors and produce proinflammatory IL1 and IL-6 in response to morphine. Interleukins released from opioid-activated glia may induce neuronal excitation and result in tolerance.

How does viral illness produce sickness induced hyperalgesia?

- Vagal responses to nucleus tractus solitarius to VM medulla to spinal cord. In this process, medullospinal neurotransmitters are released (such as substance P, CCK or glutamate) which activate glia.

Treatments: Glial modulators have been shown to suppress most pathological pain states in animals.

- old molecules (ie: pentoxifylline, minocycline) decrease microglial activation & inhibit proinflammatory cytokines
- new molecules include; Etanercept (TNF glial modulator), IL-1 antagonists, CB2 agonists, naloxone - AV411 (ibudilast) is an astrocyte inhibitor

Example 2

Category Psychology

Opening question: When assessing a patient presenting with low back pain of 6 weeks duration, what factors would suggest that they may be at a high risk of developing chronic pain?

Supplementary question(s): What examination findings might suggest a high degree of distress in a patient?

Scientific principle to be explored: Risk factors for chronicity in low back pain (yellow flags)

Clinical application: Decision making regarding appropriate treatment modalities in CLBP

Supporting information:

- Linton SJ A review of psychological risk factors in back and neck pain. Spine 2000; 25:11481156
- Kendall NAS, Linton SJ, Main CJ. Guide to assessing psychosocial yellow flags in acute low back pain: Risk factors for long term disability and work loss. Wellington, NZ: ACC and NHC 1997
- Waddell G, McCulloch J, Kummel E, Venner R Nonorganic physical signs in low-back pain.
- Spine 1980; 5 (2): 117–125
- Vlaeyen JW and Linton SJ Fear-avoidance and its consequences in musculoskeletal pain: a state of the art. Pain 2000; 85: 329

*** Candidate must emphasize a biopsychosocial approach, including mention of risk factors in the following domains:**

1) Cognitive: Fear-avoidance beliefs, catastrophizing, low self efficacy
2) Emotional: Stress, distress and anxiety and depression
3) Behavioural: Passive coping strategies, high levels of pain behaviour, dysfunction
4) Work: social support at work, poor job satisfaction (x3.5 OR for non-return to work)

Psychological factors explain 69% of the variance in the development of back pain problems at one year in a review of 37 prospective studies (Linton 2000).

*** Awareness of "flags" system (Kendall, Linton & Main 1997):**

1) Yellow flags - Aspects of normal psychological processing
    a) Pain seen as a threat
    b) Depressed mood
    c) Low self efficacy
    d) Activity patterns that repeatedly aggravate pain
    e) Lack of acceptance of pain persisting
    f) High drug use / reliance on aids (smoking x5 OR for non-return to work)
    g) Overly solicitous doctor or family
    h) Poor relationship with employer (work related injury x3.8 OR for non-return to work)
2) Orange flags – Frank psychiatric disorder inc. addiction
3) Blue flags – Work perception of work
4) Black flags – Work related organisational factors (conditions of employment)

Examination findings Waddell's signs (1980):

Any individual sign marks its category as positive. When three or more categories were positive, the finding was considered clinically significant. Important for the candidate to be aware these are not tests of malingering.

1) Tenderness tests: superficial and diffuse tenderness and/or non-anatomic tenderness

2) Simulation tests: these are based on movements which produce pain, without actually causing that movement, such as axial loading and pain on simulated rotation
3) Distraction tests: positive tests are rechecked when the patient's attention is distracted, such as a straight leg raise test
4) Regional disturbances: regional weakness or sensory changes which deviate from accepted neuroanatomy
5) Overreaction: subjective signs regarding the patient's demeanour and reaction to testing

Opening question: Tell me about the different domains in which pain can be assessed or measured.

Supplementary question(s): Give an example of a test or measure in each domain and indicate its usefulness (reliability and validity)

Scientific principle to be explored: Valid and reliable measurement of symptom domains relevant to pain

Clinical application: Pain Measurements in clinical assessment

Supporting information:

- Textbook Of Pain, 4th ed. Wall and Melzack.
- Pain Management- an interdisciplinary approach. Main and Spanswick.
- The measurement and valuation of health status using EQ-5D: a European Perspective. Richard Brooks et al

Pain is a personal subjective experience that comprises sensory-discrimitive, motivational-affective and cognitive-evaluative components. Measures/ tests in these domains add to the quality of assessment gained in clinical interview, but do not supplant it. Can highlight barriers to intended treatments, and measure progress toward stated goals.

1) **Pain intensity rating scales**
2) **Pain multidimensional scales**
3) **Mood**
4) **Function/ Disability**
5) **Quality of Life**
6) **Cognitive appraisal**

1. Verbal and Numerical rating scales

Reliable, valid, sensitive to change, easy to administer unidimensional measure of intensity. VRS (eg 4 point Likert: no pain, mild, mod, severe) Pro- instant rating Con-less reliable than VAS, comprehension/language issues. NRS 0 □10/100 (no pain worst possible pain) Pro- instant rating, Con NRS 11pt scale less sensitive. VAS 10 cm line, (no pain□worst possible pain) Pro- "Gold standard" Con non-instant rating (needs measurement in mm), explanation on use (especially old, mentally frail) influences outcome.

2. Magill Pain Questionaire (MPQ), Brief Pain Inventory (BPI)

MPQ. Scoring in 20 subclasses of grouped words (sensory, affective, evaluative, and miscellaneous items) summed to give total score, with 6 point present pain intensity scale (word descriptor). Pro-widely used, reliable, valid, sensitive to change across wide range of clinical applications, discriminating. Con-complex scoring (not immediate, error prone), requires fair standard of English comprehension.

BPI. 17 items, including 0-10 NRS measuring sensory, functional/pain interference elements. Also body diagram, % relief. Developed for cancer, validated for non-cancer pain. Pro- Sensitive to change, correlation with SF-36.

3. **Mood and affect**
   HADS (Hospital anxiety and depression scale) 14 items (7 each of A&D), each scored 0-3, total /21 for A & D. Purpose- A & D detection in medical IP/OP. Pro- Quick to fill-in, structure valid across numerous settings. Con- depression items anhedonia based lacking cognitive content. Higher sensitivity than specificity. BDI (Beck Depression Inventory) 21 items, scored 0-3, total /63. Purpose- depression severity in general and psychiatric populations. Pro- includes cognitive content (eg blame, failure). Con-applicability to pain patients, ? higher sensitivity than specificity.

4. **PDI (Pain Disability Index)**
   PDI 7 items, 0-10 scale , total /70. Application to general self assessment of function. Pro- quick to administer and score, multidimensional, correlates with other disability self reports Con- test retest reliability modest, ? sensitivity to change.
   Roland Morris, Oswestry and others- specific functional self assessment measures.

5. **EuroQOL EQ-5D**
Measures Health related QOL. Simple descriptive profile with 5 domains (mobility, self-care, activity,pain,mood) scored 1-3 □ 5 digit score. Also QOL VAS (0-100) "Your health state today"(worst imaginable□best imaginable). Pro- wide applicability, easy to administer, sensitive to change. Con- ? less discriminitive at low QOLs.

6. **PSEQ (Pain Self Efficacy Scale) PSEQ**
   10 items, score 0-6, on perceived confidence (performing tasks of daily living despite pain), total /60.

7. TSK (Tampa Scale for Kinesiophobia) 17 items, score 0-3, on degree of agreement (testing beliefs on pain, fear and injury) , total /51.

Guidance for examiners: The candidate should demonstrate a specific knowledge of a number of scales commonly used to measure different elements of the pain experience. The list is not exhaustive- conversely it is accepted that the candidate may not know all the tests above in detail.

Clinical Science Viva

Category Pharmacology

Opening question: What are the adverse effects of long-term opioid use on the endocrine & immune systems?

Supplementary question(s): What are the signs & symptoms of hypogonadism? How do opioids affect the HPA axis?

Scientific principle to be explored: mechanisms and treatments of common and/or important opioid sideeffects

Clinical application: management of common and/or important opioid side-effects

Supporting information:

1. Katz, N. The impact of opioids on the endocrine system. Clin J Pain 2009 25(2) 170-175
2. Vuong, C. The effect of opioids and opioid analogs on animal and human endocrine systems. Endocrine review 2010 31(1) 98-132

1) Endocrine effects:

>40% of patients on long term opioids may suffer from hypogonadism. Opioids bind to receptors in hypothalamus, pituitary and testis leading to stimulatory or inhibitory effects on hormone release. Opioid use primarily leads to hypogonadism but may also affect the secretion of other pituitary hormones.

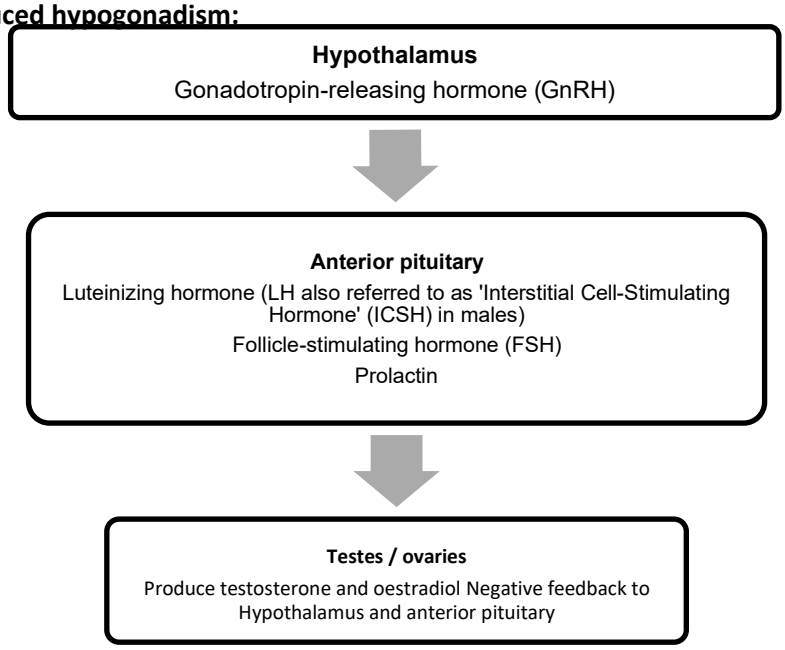
Long-acting opioids >100mg morphine equivalent/day for >1 month will impair endocrine function.

Hypogonadism:

- Decreased libido
- Decreased muscle mass
- Anaemia
- Anxiety
- Depression
- Menstrual abnormalities (secondary amennorhea)
- Osteoporosis
- Fractures
- Infertility

HPA axis:

Findings in opioid induced hypogonadism:



Findings in opioid induced hypogonadism:
Decreased: hypothalamic GnRH, pituitary LH (+FSH), adrenal DHEA & testosterone, testicular testosterone (men), oestradiol & progesterone (women), oxytocin, cortisol

Increased: prolactin, TSH

What to test:
Testosterone, LH, FSH, Prolactin if male
Oestradiol, LH, FSH, Prolactin if female

Differential effects:
Tramadol & buprenorphine have less effect than morphine in animal studies.
Buprenorphine-treated patients had normal hormonal levels
Effects reversed if opioids stopped

2) Immunosupression:
Animal evidence: accelerated onset of simian immunodeficiency virus in animals on opioids.
Human evidence: malignancy and rate of spread of tumours higher in opioid users.

Opioids with a high affinity for the opioid receptor and hydroxyl group at C3 and C6 are most immunosuppressive – morphine. Methadone, fentanyl, remifentanil and pethidine all have an effect but less marked. Less affinity results in less effect – buprenorphine.
Modification to C3 results in less effect – eg: codeine, dihydrocodeine
Modification to C6 abolishes the effect – eg: hydromorphone, oxycodone, tramadol

Naloxone & naltrexone enhance immune responses

Mechanism either indirect via HPA axis or SNS activation or directly via opioid receptors on immune cells.

Opioid peptides modulate chemotaxis, cytokine production & phagocytic activity.

Endogenous opioids inhibit IL2, Exogenous opioids inhibit IL10 and IL12 production by macrophages – increases susceptibility to bacterial and viral infections

Macrophage production by bone marrow & T-lymphocyte proliferation reduced by morphine

Tumour spread:

NK Cells play a vital role in immunosurveillance. Pain reduces NK cell activity, as does the surgical stress response with activation of SNS & HPA axis.

1.6 NEUROPATHIC PAIN – DIAGNOSIS AND SCREENING TOOLS

Scientific principles: Pain assessment in a subsection of the chronic pain population

Clinical application of Scientific Principles: Neuropathic pain assessment

Question: What is the definition of neuropathic pain?

IASP NeuP SIG 2007 : ''pain arising as a direct consequence of a lesion or disease affecting the somatosensory nervous system".

How does this differ from the previous 1994 definition?

Disease instead of dysfunction to exclude vague terms, 2) somatosensory instead of nervous system to exclude pure motor nerve pathology, which is not NeuP

Can you categorise NeuP into Possible, Probable & Definite?

- Possible - history of pain that fits characteristics and neuro-anatomical distribution
- Probable - requires the above plus either the demonstration of a neural deficit or gain (sensory, motor, autonomic) or diagnostic tests which document a specific nerve disease process or nerve lesion.
- Definite - requires all 3 of the above features.

What Special investigations might be done to confirm NeuP?

- Function - QST (Quantitative Sensory Testing)
- Pathology - blood (B12/folate, diabetic, alcoholic induced etc), CT, MRI, NCS/EMG, skin/punch biopsy.

What assessment tools are you aware of for NeuP?

Non specialised "generic" tools to measure multidimensional pain presentation. However SF Magill Pain Questionnaire relatively insensitive as discriminative tool for neuropathic pain.

Specific neuropathic pain assessment tools do not supplant clinical evaluation, can suggest /support the diagnosis. Most useful for non-specialists. All based on neuropathic descriptors and two of them (LANSS and DN4) also on simple examination findings. Validity issues due to "questionable" gold standard (demonstration of lesion as per neuropathic pain definition).

LANSS- Leeds Assessment of Neuropathic Symptoms and Signs. British. Physician administered. 7 weighted items, dichotomous: 5 sensory items, 2 clinical examination findings. Available as (self report) S- LANSS. Summed total 24, threshold 12 (>12 "neuropathic mechanisms are likely to be contributory"). Validated for NeuP in Cancer (multiple studies). Sensitivity 80%, specificity >90% (Similar to DN4).

DN4 – Dolor Neuropathique en 4 questions. French. Uses 7 interview questions and 3 physical tests

PainDetect- German. Self report. 9 items- 7 sensory, 2 measuring spatial/ temporal characteristics. Includes a drawing. Sensitivity 85%, specificity 80%. Validated for NeuP in back pain

NPQ - Neuropathic Pain Questionnaire. 10 sensory items (history and examination findings), 2 affect related.

66% sensitivity, 74% specificity. "Short form" of 3 sensory items similarly discriminative.

- StEP (Standardized Evaluation of Pain) combines six interview questions and ten physical tests for distinguishing neuropathic from nociceptive pain in low back pain. 90% sensitivity & specificity

NPS- Neuropathic Pain Scale- first specific neuropathic pain questionnaire. 11 point scales for pain intensity, quality, temporal characteristics etc. Validated for response to treatment, NOT for diagnosis.

Filler - Epidemiology. How common is NeuP?

Neuropathic pain commonly reported to be around 2-4% of general population
Prevalence of POPNO "pain of predominantly neuropathic origin" 8% in UK – based on S-LANSS.
Prevalence data- possibly inaccurate related to diagnostic difficulty
27% pain clinic attendees have neuropathic pain- higher proportion in pain clinics compared with population prevalence- may reflect higher pain intensity/refractoriness to treatment/ associated disability.
Highest proportion most likely post surgery/accident related trauma.
Diabetic (25% DM develop NeuP), MS, post stroke pain (8% develop NeuP), PHN, TN Approx 25% peripheral neuropathies no cause found.

1.7 INTERVERTEBRAL DISCS AND DISC DISRUPTION

Scientific principles: Structural lesions and pathology

Clinical application of Scientific Principles: Diagnosis & treatment options of disc disease. Candidate must have good understanding of the anatomy and pathology of intervertebral discs.

Question: Describe the anatomy of the intervertebral discs (IDs):

Structure?

- Thick outer ring of fibrous cartilage (annulus fibrosus), inner gelatinous nucleus pulposus, sandwiched by cartilage endplates
- Nucleus contains collagen fibres, organized randomly, and elastin fibres, arranged radially.

- Annulus is a series of 15-25 concentric lamellae, the collagen fibres lie parallel within each lamella.

Blood supply?

- Healthy disc has few blood vessels supplying it, most of the vascular supply terminates at the adjacent longitudinal ligaments.

Nerve supply?

- Ggrey rami communicantes, from the lumbar sympathetic trunks, join the ventral rami of the lumbar spinal nerves to form a mixed nerve, the sinuvertebral nerve, which then supplies the posterior and posterolateral annulus fibrosus, and the posterior longitudinal ligament.

Describe intervertebral disc disruption (IDD) and its causes:

- IDD and disc herniations are common causes of low back and/or lower extremity pain.
- In chronically damaged IDs, leak nuclear material from annular tears - promotes inflammatory process.
- Mediators include matrix metalloproteinases (MMP), phospholipase A2 (PLA2), cyclooxygenase (COX), prostaglandins, nitric oxide (NO), cytokines, and interleukins.
- Infiltration of macrophages and other inflammatory cells may promote neovascularization in outer regions of the annulus.
- Central sensitization.
- Prolapsed lumbar discs account for less than 5% of all low-back problems, but are the most common cause of nerve root pain. Most lumbar disc prolapses resolve naturally

How can Disc degeneration be classified?

- Grade 0: Normal non-leaking nucleus.
- Grade 1/2/3: Annular tearing confined to inner 1/3,2/3 or 3/3 of the annulus fibrosus.
- In grade 3 during discography, contrast material leaks out of the back of the disc into the epidural space.
- The presence of a disc bulge and/or disc herniation is also included in this category.

What are the Risk factors/causes?

- Genetic inheritance accounts for 70%
- Ageing changes: proteoglycans bind water, when they fragment lead to dehydration and decompression of nucleus. Mechanical stress concentrated on annulus. Collagen crosslinks of annulus form, stiffer, less able to absorb energy with loading, less able to remodel i.e. more vulnerable to injury.
- Repetitive loading, microscopic damage
- Impaired nutrition, already avascular ie at risk; assoc. with smoking
- Supplementary if earlier covered: Interventional treatments possible based on above pathologies but vital to demonstrate appreciation of limited and controversial evidence for:
    - Provocative discography – for diagnosis o Discectomy – removal of damaged portion of disc o IDET/ nucleoplasty/ bipolar annuloplasty etc o Intervertebral fusion

1.8 ANTIDEPRESSANTS

Scientific principles: Putative mechanisms of action of antidepressants

Clinical application of Scientific Principles: Rationale for antidepressant prescriptions for pain. Knowledge of side effects and serotoninergic syndrome.

Question: What classes of antidepressants do you know, and give some examples?

- Tricyclic antidepressants (TCA): e.g. amitriptyline, nortriptyline, imipramine
- Serotonin-Noradrenergic Re-uptake inhibitors: SNRI: e.g. duloxetine, venlafaxine
- Selective-Serotonin Reuptake Inhibitors (SSRI): e.g. Fluoxetine, paroxetine, citalopram
- Monoamine Oxidase Inhibitors: e.g. Phenelzine, Tranylcypromine
- NaSSAs : (also sometimes called Tetracyclics) Noradrenergic and specific serotonergic antidepressants (NaSSAs) are a class of antidepressants. They act by antagonising various adrenergic and serotonin receptors, typically a1-adrenergic and a2-adrenergic, and 5-HT2A, 5-HT2C, and 5-HT3, respectively. For example: Mirtazapine

Why may antidepressants act as analgesics for people in pain?

- Mode of action:thought to act via several mechanisms which include increased supraspinal availability of noradrenaline (thought to enhance descending inhibitory bulbospinal control), activation of endogenous mu opioid and delta-opioid receptors, sodium channel blockade, and NMDA receptor inhibition, plus activation of potassium channels, and calcium uptake inhibition. Tricyclics may also have a peripheral action via P2X receptors.
- Tricyclics can also cause sedation and may help if patient not sleeping at night.

Are tricyclics more effective than other antidepressants for pain?

- NNT for tricyclic antidepressants for neuropathic pain is about 3.1.
- SNRIs (Venlafaxine, Duloxetine) are used for neuropathic pain but have an NNT=5 approx. SSRIs (Fluoxetine) do not appear to be good analgesics NNT=7
- Tricyclics have more diverse modes of action-hence more effective, but also more side effects Duloxetine effective in DPN, fibromyalgia, Generalised Anxiety Disorder, stress incontinence.

What are the adverse effects of tricyclics?

- Postural hypotension, somnolence, weight gain, constipation. Anticholinergic effects, drug mouth, caution in glaucoma, hesitancy, caution in prostatic hypertrophy. Increased risk of arrhythmias. Caution in epilepsy Contraindicated after MI for one year.
- NNH for TCAs: 15.9 (11-26) similar to SNRIs: 13.1 (9.6-21). NNH for SSRI > 25 -better tolerated, less effective

Can Tricylics be used in children? What are the reasons for using Tricyclics in children?

- Yes. Starting dose amitriptyline: 200-500 micrograms/kg, then increased as necessary. Used for nocturnal eneuresis and neuropathic pain. No evidence for efficacy in depression. Caution in heart disease, esp. arrhythmias.

Tell me about serotoninergic syndrome? What drug combinations can cause this?

- Serotonin syndrome (SS) is caused by excess serotonin (5-hydroxytryptamine; 5-HT) availability in the CNS at the 5-HT1A-receptor. Suspect it in the setting of the recent addition of a serotonergic agent.
- Constellation of symptoms. Hunter criteria are simpler and more sensitive than older Sternbach's Clonus, agitation, sweating, tremor, hyperreflexia +. If have hypertonicity or T > 38°C require hospital admission

Most common drug combinations causing the serotonin syndrome are:

- Monoamine oxidase inhibitors (MAOIs) and serotonin selective reuptake inhibitors (SSRIs), MAOIs and tricyclic antidepressants
- MAOIs and tryptophan MAOIs and pethidine.
- More recently, tramadol plus tricyclics, SNRI or SSRI antidepressants. Many patients are on this combination.

SS is generally associated with a favourable prognosis. The management of SS encompasses five basic principles. What are they?

- Provide necessary supportive care
- Discontinue all serotonergic agents
- Anticipate potential complications
- Consider administering anti-serotonergic agents (cyproheptadine), and
- Reassess the need for psychopharmacologic therapy before reinstituting drug therapy.
- Approximately 42% of patients in published case reports of SS required admission to an intensive care unit and 24% of patients required intubation and ventilatory support. Most patients show some improvement within the first 24 hours after symptom onset with supportive care alone.

1.9 PRE-EMPTIVE AND PREVENTATIVE ANALGESIA

Scientific principles: Pre-emptive vs preventive analgesia

Clinical application of Scientific Principles: Peri-operative/ acute pain management

Question: What is meant by pre-emptive analgesia, How does it differ from preventive analgesia?

- Pre-emptive analgesia: Analgesia given prior to an injury or adequate noxious stimulus. It prevents the nociceptive barrage that leads to central sensitisation, thereby preventing or reducing ongoing pain.
- Preventive analgesia: An analgesic is said to have a preventive effect if its administration leads to a reduction in pain or analgesic consumption that extends beyond its expected duration of action (usually arbitrarily set at 5.5half lives).
- The principal difference is that in pre-emptive analgesia the focus is on the timing of an intervention i.e. before or after an incision, whereas for preventive analgesia the focus is on the effect of the intervention on the expected duration of analgesia, regardless of when the administration takes place in relation to an injury.

Can you briefly describe the underlying physiology and theory/ theories?

- Mechanisms.
    - C-fibre mediated nociceptive barrage from the periphery at site of injury induces secondary changes leading to central sensitisation. Abolition of this initiating event should prevent secondary changes and thereby reduce ongoing pain.
    - Theory supported by in vitro and in vivo laboratory investigations.
    - However, nociceptive barrage continues throughout surgery, relative importance of initiating or subsequent inputs in producing sensitisation not clear.

- Preventive analgesia is a much broader concept acknowledging that multiple factors may be involved in the sensitisation process. Preventive effects may be clinically more relevant, a number of drug combinations have been shown to have preventive effect.
- If an analgesic is capable of reducing sensitisation then its effects would be expected to extend beyond its normal duration of action, this may or may not be related to timing of administration.

Has pre-emptive analgesia proven to be clinically useful, discuss the reasons for this? What about preventive analgesia?

- Clinical usefulness of pre-emptive analgesia disappointing. Research evidence weak and contradictory. Reasons: too simplistic theory, differences in clinical trial design, difficulties in completely blocking nociceptive inputs, use of many different outcomes.
- Preventive probably more useful; broader concept and better defined outcomes i.e. reduction in pain scores or analgesic consumption at a time point beyond expected duration of target drug(at least 5.5 half-lives).

Discuss NMDA receptor antagonists and preventive analgesia.

- Ketamine (0.1 to 0.5 mg/kg/hr infusion) reduces hyperalgesia/allodynia and opioid consumption after surgery. Effect lasts longer than expected duration. Useful for chronic/cancer pain patients or uncontrolled acute pain.

Describe how you would design clinical trials to investigate these two effects.

- Pre-emptive; active treatment vs placebo, before or after incision (after group might be intra or postop), discuss use of negative (placebo vs placebo) and positive (active vs active) controls.
- Preventive; might include above design(s) and co administration of 2 active treatments etc. Importance of clear outcome definition.