Question examples: (updated July 2012)

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
B. Has a peak onset in the fifth and sixth decade of life
C. Occurs in 20% of patients with multiple sclerosis
D. Is best treated with carbamazepine
E. Treated by microvascular decompression surgery is associated with a 5% mortality

Answer: A:F B:T C:F D:T E:F

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

1.3 EMQs

Extended Matching Questions are used by the Royal College of Obstetricians & Gynaecologists for their major examinations. One Pain Medicine example is 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	**I.**	
B.	Migraine	**J.**	
C.	Post herpetic neuralgia	**K.**	
D.	Sinus pain	**L.**	
E.	Temporo-mandibular joint pain	**M.**	
F.	Trauma	**N.**	
G.	Trigeminal neuralgia	**O.**	
H.	Tumour related pain	**P.**	

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

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)**

2 SOE

2.1 SOE Clinical Pain Medicine

The Clinical Structured Oral Examination (SOE) of the FFPMRCA examination will comprise a long case and three short clinical questions (SCQs). No patients or actors will be participating in the Clinical SOE of the FFPMRCA examination. There will be two examiners for the Clinical SOE.

2.1.1 SOE Clinical Pain Medicine: 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:

Clinical Viva	
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)

Guidance for examiners:
Long Case Phantom Limb Pain

(1) Reference. Ramachandaran VS, Altschuler E L. Reflections on hand. Pain 149 (20101) 171-172

(2) Nikolajsen L, Jensen T. Phantom Limb. In Textbook of Pain 5th Edition, 2006, Elsevier. 961-969

(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.

2.2 SOE Clinical Pain Medicine: Short Clinical Question

The final 20 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.

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—t**his 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 – **L**umbricals, **O**pponens Pollicis, **A**bductor Policis Brevis, **F**lexor 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.*

Scientific principles to be explored:

NA

Clinical applications of scientific principles:

(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	
Category	Lumbar Sympathectomy, procedures, neurolytic substances

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.*

Scientific principles to be explored:
A sound understanding of the mode of action of neurolytic agents

2.3 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
    - 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 IL-1 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; 25:1148-1156
- 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

Clinical Science Viva	
Category	
Question number	**1400**

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.
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. Nick Plunkett 8/11

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 side-effects

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.

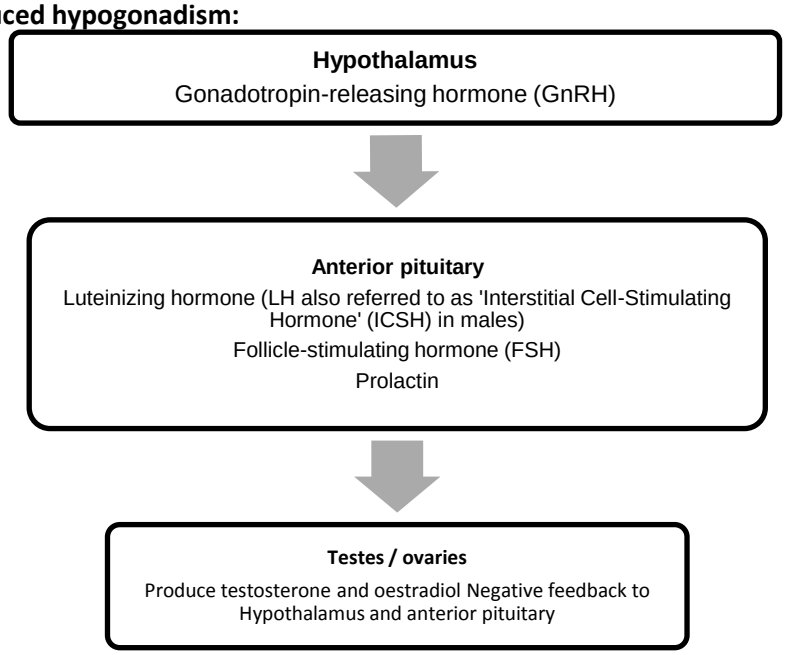
Long-acting opioids >100mg morphine equivalent/day for >1 month will impair endocrine function.

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 μ 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 μ 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.

Guidance for examiners: