FACULTY OF PAIN MEDICINE
AUSTRALIAN AND NEW ZEALAND COLLEGE OF ANAESTHETISTS
ABN 82 055 042 852

EXAMINATION HELD ON 14th then 29th & 30th NOVEMBER 2014

at local centres (14 Nov 2014) then at Royal Adelaide Hospital, South Australia.

THIS REPORT IS PREPARED TO PROVIDE CANDIDATES AND SUPERVISORS OF TRAINING WITH INFORMATION ABOUT THIS EXAMINATION AND TO ASSIST WITH PREPARATION FOR FUTURE EXAMINATIONS. THE EDUCATIONAL RESOURCE CONCERNING EACH WRITTEN QUESTION IN APPENDIX A IS NOT A MODEL ANSWER FOR THE QUESTION. THE INFORMATION CONTAINED IN EACH WILL BE MORE INFORMATION THAN COULD BE COVERED IN THE FIFTEEN MINUTES. THE INFORMATION PROVIDED IS CONSIDERED CURRENT AND EVIDENCE-BASED, BUT MAY BE SUBJECT TO CHANGE IN THE FUTURE.

CANDIDATES SHOULD DISCUSS THE REPORT WITH THEIR TUTORS SO THAT THEY MAY PREPARE APPROPRIATELY FOR FUTURE EXAMINATIONS.

The Examination is an integral part of the Pain Medicine Training Program, leading to the award of Fellowship of the Faculty of Pain Medicine.

The Objectives of Training guide the range of content which may be assessed.

The Examination consists of written and oral sections and covers the theory and practice of Pain Medicine.

It is acknowledged that standardisation of the assessment process across the candidature is imperfect, vulnerable to an array of potential confounders. The Faculty of Pain Medicine attempts to optimise the accuracy and fairness of the examination process by ensuring that all stages are assessed by a pair of examiners; generally, at least one of the pair is an experienced member of the Court of Examiners. Where possible, members of the Court of Examiners are paired such that an examining duo will come from different primary specialties and different geographical locations. Efforts are made to reduce the likelihood of a candidate being examined by an examiner to whom he/she is known.

The very large candidature presenting for this examination presented a logistical challenge to the organisers. Of course, every effort was made to see that the 2014 Examinations were conducted to the standard well-established over the 15 years of Faculty of Pain Medicine examinations.

The 2014 FPM Examinations were observed by Dr Patrick Farrell, Chairman of Examinations, ANZCA.

1. EXAMINATION OVERALL PASS RATE 76.2%

This year, 42 candidates presented for the examination and 32 were successful.

2. WRITTEN SECTION PASS RATE 29/42 69.0%

See Appendix A for the educational resources regarding each question.

General information:
Always, candidates need to:

1. *Answer the question.*
2. Plan the answer in a logical fashion and demonstrate an organised approach.
3. Give succinct answers and avoid repetition.
4. Use headings and dot points if asked to list or discuss briefly.
5. Give definitions of specialist terms (e.g. neuropathic pain, placebo response or breakthrough analgesia). Examiners are unable to assume understanding or meaning of a particular term without clear definition. Similarly for abbreviations.
6. Start answer with "I would do…" if asked to "outline your approach to…"

Candidates were required to answer ten out of fifteen questions. The first five questions were compulsory and five questions were selected from the ten non-compulsory questions.

General comments from the Examiners:

General comments: Candidates should endeavour to introduce their responses with accurate definitions of the pivotal terminology in the question. Consideration of the broad sociopsychobiomedical aspects of the question must be provided. As always legibility of responses continues to challenge the markers. Needless to say, there is no substitute for a sound knowledge of the subject matter.

Many answers lacked the sophistication expected of specialist pain medicine physicians. Once again, there was concern about the lack of knowledge in some key and basic areas given the candidates are supposed to be specialists in the area and know more than the referrer. **It remains apparent that the trainees are not digesting the content of recent journals.**

Candidates are reminded to read the questions carefully, and to attempt to write legibly.

Many candidates obviously used a generic template to answer questions, and these were both not specific to task, and poorly adapted to it. Some questions had more than one section. All sections needed to be answered in order to pass these questions.

Abbreviations always, and specific medical terms generally, require definition when used for the first time.

Question 1 – Compulsory PASS RATE 39/42 93%

List the clinical signs and/or medical conditions for referral to a Spinal Surgeon for consideration of spinal surgery in a patient with (either acute or chronic) low back pain and leg pain.

Generally the responses provided by the candidature were disappointing. Remarkable was the focus on lumbar disease with very little reference being made to other areas of the spine.

Question 2 – Compulsory **PASS RATE 11/42 26%**

In a patient with renal or hepatic impairment, the changes may affect the choice and/or dose of medications prescribed. Summarise the key changes and indicate how they might influence your choice and/or the dose of medications used for pain management in these patients. Include both opioid and non-opioid drugs in your answer.

The responses of almost a half of the candidates were unacceptably poor, and overall it was clear that there was an inadequate grasp of the basic pharmacology pertinent to this question.

Question 3 – Compulsory **PASS RATE 32/42 76%**

Mr Smith has attended your clinic for six years due to severe CRPS (Type 1) of the left foot. Despite every possible pain clinic intervention including a multidisciplinary pain management program in which t he took on board the principals well, he remains distressed and unable to weight bear. The affected foot is an obstacle for him. He now seeks your advice about amputation.

Most candidates did not favour amputation. The responses to this question were generally adequate, although few mentioned postoperative adversities eg wound healing, important communication issues or rehabilitation matters. Many provided a somewhat generic response, and some did not explore the amputation issue

Question 4 – Compulsory **PASS RATE 40/42 95%**

Describe the risk factors for persistent post-surgical pain (PSP), and detail the steps you would follow to reduce the risk of this outcome

Whereas most candidates attained a pass mark in the question, responses were essentially superficial.

Question 5 – Compulsory **PASS RATE 32/42 76%**

Discuss the diagnosis and management of Major Depressive Disorder in a patient with persistent pain.

The responses to this question, a common pain medicine challenge, were surprisingly poor, with the allocated marks ranging from 6 down to 2; (the question was not marked by a psychiatrist.) The responses in the diagnosis area were unsophisticated, and the treatment comments were more focussed on depression in general, and failed to adequately address the issues related to pain comorbidity.

Question 6 **PASS RATE 9/11 82%**

Provide notes on the management of pain associated with Irritable Bowel Syndrome (IBS).

The responses to this question were generally brief, generic and superficial. Only two candidates mentioned formal diagnostic criteria such as the Rome Criteria.

Question 7 **PASS RATE 4/7 57%**

Describe the differences in nociception in the immature and mature nervous system.

This question apparently presented a challenge to the candidates. Whilst the loose wording of the question allowed much latitude for the candidates, many struggled.

Question 8 **PASS RATE 10/12 83%**

Discuss the role of cognitive-behavioural therapy (CBT) in the pain clinic. Include reference to specific patterns of cognition.

It was a little surprising that only 12 candidates attempted this question, likely indicating an inadequate level of confidence in discussing this area of pain management in any depth. None of the responses would be considered to have been good. All candidates chose to use catastrophisation as their example, but few were able to define and discuss this properly.

Question 9 **PASS RATE 3/7 43%**

In a typical <u>adolescent</u> chronic pain clinic, 80% of patients are female.

1. **What factors have been proposed in the literature to explain this?**
2. **What implications for pain experience and function in adult life does having a chronic pain syndrome in adolescence have?**

Few candidates discussed psychosocial factors, some mentioned laboratory data. Some candidates related the question to the incidence of pain in adult females. The menarche was not mentioned in any response. There was a paucity of information regarding adolescent pain presentations.

Question 10 **PASS RATE 23/25 92%**

Discuss the assessment, differential diagnosis and management of unilateral facial pain.

There were a number of borderline marks awarded to these responses. There was little comment correlating symptoms with disease process, nor was there adequate discussion of differential diagnoses. The management area was superficially addressed.

Question 11 **PASS RATE 16/23 70%**

Outline the metabolism of codeine and tramadol. Discuss how genetic differences may alter both the effect and incidence of adverse effects associated with each of these drugs.

Many candidates who attempted this question knew little of the metabolism of tramadol. Issue of variable rate of metabolism was rarely mentioned.

Question 12 **PASS RATE 4/27 15%**

Discuss pain in the patient with a spinal cord injury

Responses were lacking inn structure / organisation.

Question 13 **PASS RATE 39/40 98%**

Bone pain is common in patients with breast cancer. Discuss the management of this.

This question was popular with the candidature and was generally well done.

Question 14 **PASS RATE 15/42 63%**

Discuss the differential diagnosis and management of Medication Overuse Headache (MOH)

Few candidates provided a satisfactory definition og the syndrome. The genesis of MOH was similarly poorly described as was the management. Although only a third of those attempting the question were unsuccessful. Another third produced borderline performances.

Question 15 **PASS RATE 20/34 59%**

Describe the assessment and management of painful persistent peripheral neuropathy

Here again the examiners noted an inadequate application to the provision of a definition for the condition. Again the apparently high success rate in this question is misleading. There was considerable concern raised by the markers that the widespread reluctance of the candidates to incorporate psychosocial factors into the assessment and treatment should attract a heavy penalty

3. LONG CASES PASS RATE 28/42 66.7%

General comments:

The Long Case is an important part of the Examination because it aims to mirror a first consultation undertaken by a Specialist Pain Medicine Physician. This section remains a key part of the assessment process.

Candidates are advised to access the Trainee Support Kit and previous Examination Reports for the general instructions regarding the conduct of this section of the examination process.

The success rate in this section was particularly disappointing this year, although there were some outstanding candidates. Generally, the candidates were careful and respectful of their patients.

Examiners' comments:

Yet again, the patients were nice people who were cooperative and helpful without exception. Candidates should use this to their advantage, and follow up on any clues given.

Steering the interview and timing of history and examination parts remains problematic.

History:

Key points that were omitted in the history included patient demographics (income, living alone, type of house from functional aspect etc.), and background history such as substance use and psychiatric history.

Candidates are advised to refer to the outline of "How to take a Pain History" available in *Acute Pain Management: Scientific Evidence.* 3rd Edition, 2010 Macintyre et al., (Eds), ANZCA available at http://www.fpm.anzca.edu.au/resources/books-and-publications as a starting point for learning the elements of a pain history then practice under exam conditions as time management is essential.

Mental State Examination:

Whilst some level of Mental State Examination is generally indicated, an exhaustive MSE is rarely warranted in the FPM Fellowship Examination.

It is apparent that the "Mental State Examination (MSE)" is still incorrectly taken to mean different things to some candidates. The MSE is completely different to Folstein's *Mini Mental State Examination* (MMSE) http://www.ncbi.nlm.nih.gov/pubmed/1202204 ; the latter may be administered as a 30 point screen in the assessment of the different facets of cognitive function, and might therefore be included as a component of the broader MSE, administered where there may be a concern over cognitive impairment.

In order to clarify the nature of the full Mental State Examination (MSE), candidates are encouraged to review the *Royal Children's Hospital Melbourne* web page at http://www.rch.org.au/clinicalguide/guideline_index/Mental_State_Examination/

Physical Examination:

Candidates had been reminded to bring their own stethoscope. All other equipment required was available and standardised.

Of course, sometimes a patient will present with symptoms or signs which warrant specialised assessment. Candidates are reminded that, should they seek to use a piece of equipment which is unavailable, they need merely comment upon to be assessing examiners.

Poor hand hygiene remains of concern with very few candidates using hand washes/ hand gel before or after handling patients. Candidates **must** adhere to accepted standards of infection control at all times when interacting with the patients.

Candidates are also reminded to remain cognisant of the patients' sensitivities in regard to physical exposure. Judicious use of sheets, drapes and gowns is important.

The standard of the physical examination, although improved over the years, is still not as ideally competent as would be expected for a specialist pain medicine physician. Candidates are directed to start near the presenting complaint. However, they should avoid using the toothpick violently, or retouching the painful part multiple times, especially when the patient exhibits signs of pain.

Neurological examination, in particular, was inadequate in a number of cases. The importance of carrying out a careful neurological examination cannot be stressed enough. Many candidates exhibited poor technique in the performance of the neurological examination of the limbs especially. Testing vibration and position sense were done particularly badly.

A general neurological examination should precede detailed sensory examination. Salient features are:

- Note history given
- Compare sides
- AVOID CAUSING PAIN
- Inspect first. This includes adequate exposure of the limbs and trunk/spine (both for upper and lower limbs). Look for wasting/asymmetry/posture/skin colour/ fasciculation.
- Screen movements and power before "hands on". For example
    - Upper limbs: arms above head/behind back
    - Lower limbs: observe gait/heel stand/stand on balls of feet +/- squat/ single leg stand and squat
- Assess tone, feel sweatiness and temperature at same time
- Assess power of whole of limb
- Have good technique for reflexes (practice shows)
- Coordination
- Then sensation
    - Look for numbness first
    - Identify area of altered sensation (?symmetrical/ ?dermatomal)
    - If time allows and information gleaned indicates, consider more refined pain oriented sensory examination

Leaving inadequate time to perform the physical examination again led to minimal or absent respiratory or cardiovascular examination.

Presentation:

The candidate should aim to demonstrate that they have the ability to be the leader of the Multi-disciplinary Pain Team, and can manage the long case as if the patient were their own.

This year, errors in the Presentation included making assumptions about information that had not been elicited or assuming an answer to a question that had not been asked.

The presentation should include a structured formulation and an objective discussion. There needs to be an emphasis on an **all-round approach** to assessment, diagnosis, formulation, management and prognosis. It should occupy less than **seven** minutes of the total 30 minutes allowed for the viva voce section.

Viva:

Topics relevant to the patient's pain condition, identified by the Examiners from the observed history and examination, form the basis of the viva.

Candidates are encouraged to critically appraise the patient's current management and outline what they may recommend that is different from the plan the patient has described.

Questions may include some of the following:

- The main pathophysiological processes
- What to do if pain progresses.
- Key management issues.
- Indications for medication use, mechanisms of action and complications.
- Expectations and treatment outcomes for modalities previously used or recommended.

4. STRUCTURED VIVA SECTION PASS RATE 33/42 79 %

The viva section consists of three structured vivas and the investigation station. There were two rounds of scenarios and investigations examined so that quarantine times for candidates could be kept to a minimum.

General information:

- Issues that were covered in one or both vivas included:
  - Patient assessment.
  - Nature of the lesion.
  - Anatomy.
  - Investigations to confirm the diagnosis.
  - Possible therapies for current pain.
  - Crisis management.

The introductions to the structured vivas were as follows:

4.1 ACUTE SCENARIOS PASS RATE 36/42 86 %

Introductory scenario 1

You are called to review the management of pain in a 17 year old girl with burns after a Medical Emergency Team for excessive sedation. The sedation occurred 6 hours after admission (0630

hours). She woke after administration of naloxone. Her parents have been present throughout. She sustained 20% burns over her trunk after her dress caught fire at a party, there are no indicators of facial burns or smoke inhalation. What further information would you like?

Introductory scenario 2

An 80 year old man was admitted to the emergency department after a car accident in which he was the driver. There is bruising on his chest which appears to have been caused by his seat belt. He is comfortable when lying still but complains of severe left-sided chest pain when moving. A chest x-ray was taken. What are the major findings?

4.2 CHRONIC SCENARIOS **PASS RATE 30/42** **71 %**

***Introductory scenario 1*:**

You are seeing a 55-year-old man who underwent spinal surgery one year ago. He has a foot drop and pain in his left leg. How would you determine the cause of his problem?

Introductory Scenario 2

You are seeing a 45-year-old woman who is being treated for neuropathic pain in her legs with fentanyl patch 100mcg/hr, topiramate 200mg bd, and celecoxib 100mg bd. She spends most of her day in bed, smoking 2 packets a day. Examination findings include a weight of 39kg and bilateral lower limb oedema. Outline your approach in assessing this patient.

4.3 CANCER SCENARIOS **PASS RATE 30/42** **71 %**

Introductory scenario 1:

A 68 year old man presents with a three month history of persistent pain in his right shoulder and upper arm. The pain radiates to the wrist and dorsum of the hand and is exacerbated by movement. Examination reveals a thin elderly looking man, holding his arm close to his body. He is alert and orientated.
Past history:

- Renal cell carcinoma- nephrectomy 3 years ago
- Pathological fracture neck of humerus – prophylactic intramedullary nail inserted 3 months ago, then radiotherapy

Medication:

- Slow release oxycodone 80mg every 8 hours
- paracetamol 1g 6 hourly
- pazopanib, (a selective multi-targeted receptor tyrosine kinase inhibitor used for renal cell carcinoma) 800mg per day

He requests 2 months' supply of his medications so he can visit relatives in the USA.

Introductory scenario 2:

You received the following referral which states:

"Thank you for seeing this severely distressed 32 year old lady with a 6 month history of local recurrence of her carcinoma of the cervix and invading the right pelvic wall. She presents with very severe pain radiating down the right leg. She is unable to lie on her back, unable to sleep, becoming withdrawn and constantly moans with the pain.
Her current regimen includes – sustained release oxycodone 120mg tds, hydromorphone 6mg SC Q2 Hr prn (averaging 60mg/day SC), pregabalin 300mg bd, amitriptyline 150mg nocte, clonazepam 1mg bd, haloperidol 1mg bd, dexamethasone 2mg tds, paracetamol 1g QID, celecoxib 200mg bd, pantoprazole 20mg nocte and SC ketamine 8mg/hr through syringe driver (slightly dizzy with this). However these do not help much with her pain. She had finished her course of chemo and radiotherapy 2 months ago.
The latest MRI scan showed an unchanged mass extending from the cervix to the lateral pelvic wall.
We would be grateful for any help you can give with regards to helping this lady with her pain. Thank you.
Palliative Care team"

4.4 INVESTIGATION STATIONS **PASS RATE 33/42** **79 %**

A brief scenario was presented to put each investigation into a context. If there was an obvious diagnosis, candidates could mention it as soon as possible and move to the next investigation. If the diagnosis was not immediately clear, candidates were able to explain the key features they were looking for and, hence, draw a conclusion.

The investigations included:

- Radiology including plain X-rays, CT, MR and PET scans
- Neurophysiology findings
- Abnormal biochemical profiles
- ECGs
- Blood drug levels

The instructions provided to the candidates were as follows:

- ***Each investigation or image requires interpretation.***
- ***Consider the normal anatomy and any variation.***
- ***Discuss the features of the investigation that are evident to you.***
- ***If the answer is not obvious, you may derive an accurate interpretation by discussing the features that you can see.***
- ***If you are unsure, ask to move to the next case.*** *(The examiners will not permit you to linger when you cannot see the abnormality.)*
- ***This station awards marks for reasoning and discussion. The number of cases you proceed through is not as important as your discussion of each case.***

As in previous years, the range of investigations was good, and overall, the Investigations Station was done reasonably well. However, many candidates were unable to identify an obvious odontoid fracture and only one could identify the cavernous sinus in a very obvious tumour scan. Some had difficulty identifying a clearly prolonged QT interval.

5. SHORT CASES: **PASS RATE 31/42 74 %**

General Information:

The Short Case section involved each candidate having a brief exposure to 3 patients with acute, chronic and cancer pain plus the Communication station. This section is a test of physical examination techniques or communication skills.

- The duration of each station was 10 minutes.
- Information was provided outside each door regarding the station.
- For each short case, candidates were directed to a specific area to examine then present their findings and answer questions concerning that patient.

Examiners' comments:

Candidates should focus on reading the instructions carefully and addressing the specific issues raised.

Candidates need to be particularly cognisant of some patients having pain and examine the patients accordingly, especially if examining the patient after several previous candidates. Examiners may modify the instructions regarding the examination if a patient becomes distressed.

Similar points were made as for the long case, including the following:

- Inspection is an important element of examination. Appropriate disrobing is important. (Yes, we make this comment every year!)
- Proper examination rarely requires the patient to remain seated throughout. Candidates are reminded that assessment of gait is often required and patients can be moved from chair to bed.
- When examining limbs, comparison with the other side must be made.
- No need to repeatedly perform the same examination task.

5.1 ACUTE STATION **PASS RATE 30/42 71 %**

The clinical conditions presented varied according to the availability of patients who were willing and suitable to be utilised on the day of the assessments.

5.2 CHRONIC STATION **PASS RATE 32/42 76 %**

The clinical conditions presented varied according to the availability of patients who were willing and suitable to be utilised on the day of the assessments.

5.3 CANCER STATION **PASS RATE 32/42 76 %**

The clinical conditions presented varied according to the availability of patients who were willing and suitable to be utilised on the day of the assessments.

5.4 COMMUNICATION STATION: **PASS RATE 31/42 74 %**

This station employs actors to present the candidadte with an opportunity to demobstrate interviewing and communication skilss.

Introduction to Scenario - Letter of Referral

Dear Doctor,

Re: Mr or Mrs Irene (Ian) Palmer

Thank you for reviewing this 72-year-old grandmother, who is was seen at your clinic a number of years ago for PHN.

She has knee pain which for which she is on the list for a knee replacement. The perioperative department are concerned about her operation because of the OSA for which she has a CPAP machine.
Good quality medical assessment has demonstrated:

- *hypertension*
- *Diet controlled diabetes.*
- *OA of left hip and both knees*

I am concerned about the medications she is currently on. She has been depressed in the past and had suicidal ideation. This does not appear to be a problem at the moment but a friend of hers moved into a nursing home recently and she is a bit isolated. A family member calls round to do the gardening.

She was on 40 mgs of oxycontin per day which, she got from her previous GP. I think he has retired.
I have commenced a 50 mcg Duragesic patch but she is still taking the oxycontin 30 mgs once per day. She required an extra script this month. She also gets some 2 mgs dilaudid tablet but only for breakthrough.

I appreciate you have limited time but could you please particularly advise her about her use of analgesic medication.

Yours sincerely,
Dr I Care

Introductory Scenario / Script:

Good morning Mrs/ Mr……

I got a letter from your general practitioner.

(Patient interrupts) I'm not quite sure why I'm here. What you actually do here anyway.

Your doctor is somewhat concerned about the amount of medication that you were on.

But for my knee its for the pain that I have all the time. Actually I think the other knee is just as bad. It's really terrible. I cant sleep I can go do work out in the garden. My nephew comes round to do the lawn and a bit of sweeping up. He has a bad back too. I absolutely need of medication. Is this because I ran out and had to get an script early. That was because my nephew borrowed some of my medication because of a pain that he had. He and his friend help me with the garden and cutting the grass and he has a bad back.

You don't understand Dr I've had this pain for a long time. The surgeon says he wants to replace the knees but the anaesthetist say because of the sleep snoring machine it is more dangerous.

The doctor I had was fine he didn't give me any grief about the tablets but he retired about two months ago and the new doctor gets all concerned about the medication. Something about the government or department or something. She gave me this patch but I don't think it is of any use. It doesn't stay on anyway.
I take the osteo panadols but they are like lollies. The dilids help a bit and the oxys but the doctor decreased them.

I'm beside myself with the pain. Its hard to get to the supermarket. It really gets me down....sometimes I think what's the point……..Sadie has just gone into a nursing home and I just don't want to do that.

6. OVERALL EXAMINATION COMMENTS:

For the second time, this year the written examination was held before the vivas, three weeks prior on this occasion. Feedback in relation to this was again essentially (but not unconditionally) positive. Otherwise, this examination essentially followed the structure of previous examinations. Written questions were chosen from across the curriculum, and the vivas were carefully designed to ensure, as much as was possible, a uniformity of the material examined.

The above statistics speak for themselves; certain areas continue to be poorly addressed by the cohort, and this will no doubt be considered carefully in the planning of future assessments, both for those remaining under the old training scheme and those trainees commencing under of the new curriculum.

The Committee for Examinations remains concerned that the standard demonstrated in the examination is, in many regards, little better than that demonstrated by medical undergraduates. Certainly, the feedback from observers consistently reflects an apparent generosity on the part of the FPM examiners to candidates who do not appear to always be functioning at fellowship standards. Fortunately for some, the current structure often allows for one to redeem a poor performance through better function in other sections of the examination. There is a potential for the new curriculum and assessment process to be more appropriately exacting.

Once again, many candidates did not conduct adequate physical examinations. Practice of an organised approach to physical examination is essential both for success in the examination but more importantly for clinical practice. We would also encourage practice formulating the case and presenting it in an organised fashion as well as talking about a wide range of relevant topics to improve overall performance in the viva.

I am most grateful to Dr Penelope Briscoe, her colleagues, and the staff at Royal Adelaide Hospital for their tremendous effort in the organising of the patients and the exam venue.

The Court of Examiners gratefully acknowledges all of the patients who willingly and enthusiastically participated in the Examinations despite their pain.

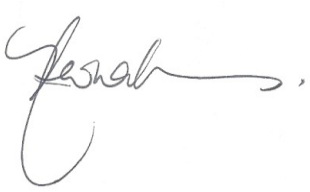
The Court of Examiners also acknowledges the Observers for 2014, Dr Patrick Farrell and Dr James Bartley. Both provided valuable reflection on the examination processes.

I wish to thank all of my colleagues on the Committee for Examinations, and those other members of the Court of Examiners, for so generously giving of their time to assist in the design and conduct of the examinations. My thanks also go to the FPM staff who have provided wonderful support.

7. THE BARBARA WALKER PRIZE / CERTIFICATES OF MERIT

The Barbara Walker prize was awarded to Dr Martine O'Neill FANZCA from NSW.

Certificates of Merit were awarded to Drs Suzanne Cartwright FANZCA (Vic); James Jarman FANZCA (WA) and Wei Chung Tong (Vic).



NEWMAN L. HARRIS

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Court of Examiners

GRETA M. PALMER

Deputy Chair

April 2015

Distribution:

Faculty Board	Supervisors of Training	Examination Committee
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APPENDIX A
2014 FPM WRITTEN EXAMINATION

Question 1 – Compulsory

List the clinical signs and/or medical conditions for referral to a Spinal Surgeon for consideration of spinal surgery in a patient with (either acute or chronic) low back pain and leg pain.

1. History of progressive leg weakness or onset of cauda equina symptoms. This requires urgent referral

2. Clinical signs of upper motor neurone lesion.

3. Acute radiculopathy not responding to conservative management with stable neurological signs and investigations confirming diagnosis.

4. History of acute trauma with investigations revealing potential unstable fractures.

5. History of malignancy with evidence of spinal involvement with primary or secondary malignancy.

6. Evidence of infective process such as osteomyelitis or discitis.

7. Spinal canal stenosis with claudicant leg pain.

8. Spondylolisthesis, particularly in the young adult or where there is evidence of progression of defect.

9. Presence of syringomyelia on investigations.

10. Single level intervertebral disc disease with failed appropriate conservative management.

Question 2 - Compulsory

In a patient with renal or hepatic impairment, the changes may affect the choice and/or dose of medications prescribed. Summarise the key changes and indicate how they might influence your choice and/or the dose of medications used for pain management in these patients. Include both opioid and non-opioid drugs in your answer.

A. Key changes:

1) Renal impairment can result in:
   a) Decreased excretion of parent drugs that are cleared by the kidney
   b) Decreased excretion of active metabolites
   c) Further impairment of renal function by some drugs

Most of the significant alterations in drug choice or dose (for drugs used in pain management) result from renal impairment.

2) Hepatic impairment causes fewer problems but:
   a) Some drugs may have increased oral bio-availability due less first pass metabolism (although limited specific information known in most cases)
   b) Some metabolised for clearance less rapidly → prolonged effects
   c) Further impairment of hepatic function by some drugs

B. influence on choice of drug/dose

	Renal impairment	**Hepatic impairment**
Opioids and tramadol	*No dose adjustment required as none, minimal amount, or only weakly active metabolites* • alfentanil • fentanyl (good choice if severe renal impairment) • buprenorphine (unchanged PK as mainly biliary excretion of metabolites) • oxycodone (in most patients)[a] • methadone (unless impairment severe) *Active metabolites so dose adjustment suggested or, sometimes in preference, use alternative agent:* • codeine • hydromorphone • morphine • tramadol *Avoid:* • pethidine • dextropropoxyphene	*No dose adjustment required*: [b] • alfentanil • buprenorphine • fentanyl • morphine • oxycodone *Dose adjustment may be needed if impairment severe:* • methadone • tramadol *Avoid:* • pethidine
Paracetamol	Safe to use in most patients, may need to reduce dose if severe renal failure	Short-term use at therapeutic dose is reasonable in patients with chronic liver disease; reduce dose to 2-3 g/day for long-term use; preferred to NSAIDs
NSAIDs including non-selective and coxibs	Use with extreme caution if renal impairment and avoid if severe	Reduced doses suggested
Tricyclic antidepressants (TCAs)	Metabolite accumulation may occur but limited evidence about need for dose reductions	Reduced doses suggested if severe hepatic impairment
Gabapentin, pregabalin	Dose adjustment suggested based on creatinine clearance	Suitable for use – nonhepatic metabolism
Older anticonvulsants		Avoid carbamazepine and valproate if severe impairment
Ketamine	Limited data but probably no dose adjustment needed	Limited information
Local anesthetic drugs	No significant difference in plasma concentrations unless renal impairment is severe	Dose adjustment may be required with repeated or prolonged use as clearance may be significantly impaired

[a] significant ↑ in half-life especially in end-stage renal disease and may need dose reduction or alternative agent

[b] possible increased risk of opioid toxicity in patients with hypoalbuminaemia

Question 3 – Compulsory

Mr Smith has attended your clinic for six years due to severe CRPS (Type 1) of the left foot. He has had every possible pain clinic intervention. He applied himself diligently to a multidisciplinary pain management program.

He remains distressed and unable to weight bear. The affected foot is an obstacle for him. He now seeks your advice about amputation.

Please summarise and explain your response.

CRPS type 1 is a condition characterised by pain disproportionate to the inciting event together with sensory, vasomotor, sudomotor and motor / trophic changes. The diagnosis is primarily and clinical one based on the Budapest Criteria.(two or more signs or 3 symptoms, with out any other condition that could explain the problem better.)

CRPS treatment involves the full gamut of available tools for the pain specialist. Education, pharmacological. Multidisciplinary rehabilitation along pain management lines and various interventional options. In this mans case treatment has been resistant.

Treatment resistant CRPS type one is not uncommon with a high prevalence of patients suffering despite having had timely expert care as outline above.

Mr Smith has had CRPS of the left foot for 6 years and he can not weight bear through the affected limb. He seeks advice about an amputation of the affected limb.

I would discuss with him the two options left. To stay as he is as option 1 and to look at amputation which is option 2.

Option 1 is not attractive for him. He has a dysfunctional limb that is an obstacle to him. He is confined to using mobility aids whether crutches with or with out wheelchairs and or scooters. The issue for him are amongst others wear and tear of affected good joints eg wrist and shoulders and his sound limb joints. He can not exercise and so has become unfit and this is not appealing. He can not sleep well as even the sheets on the bed disturb him. Over all option one is not attractive .

Option 2 is the amputation one. I would like to give him a realistic opinion about the risks for him of amputation.

If the CRPS is localised to the left foot and ankle with little evidence of proximal spread then the risk would be lower of CRSP affected the stump.

He needs to be advised that this is still far higher in a CRPS patient than some one with out CRPS.

One study from the Netherlands looked at retrospective study of amputation of 22 patients with CRSP and noted a 24% recurrence rate. There is little evidence to which to base any decision on and so each case needs to be looked at in order to make the right decision.

I would fully assess all the joints involved with him (as above) to determine that he could potentially be a prosthetic user.

The level of amputation would be a transtibial and he would need to be referred to the amputee clinic in order to be given advice about prostheses and what was realistic for him if post operatively he could tolerate wearing a prosthesis.

I would explore with him how he was coping emotionally and exclude any factors that would mitigate against amputation eg severe untreated depression. If appropriate I would establish psychological supports to help him during the process.

In the perioperative period I would advise him to start Vitamin C and Pregabalin as both had been shown to be helpful in alleviating the onset of CPRS and post operative neurpathic pains respectively.

I would advise him that an amputation in his case could be a positive experience and support his decision not with standing the caveats out line above.

The treatment of Mr Smith represents an example of chronic disease management with life long support from his treating pain specialist.

Question 4 – Compulsory

Describe the risk factors for persistent post-surgical pain (PSP), and detail the steps you would follow to reduce the risk of this outcome.

Persistent (chronic) post-surgical pain (PSP) is common to almost every type of surgery. Reported incidence varies between 10% - 80% with up to 10% described as severe. While there is no consensus on the diagnostic criteria, the working definition proposed by Macrae et al (1999) would be:

- Pain developed after a surgical procedure
- The pain is of at least 2 months duration
- Other causes for the pain have been excluded
- The possibility that the pain is continuing from a pre-existing problem should be explored and exclusion attempted

Risk factors

Surgical associations

- Open surgical approach / specific stitching methods (e.g.: pericostal stitching)
- Intraoperative nerve damage
- Long operation time
- Operations performed in low volume units
- Reoperations

Patient / disease risk factors

- High pre-operative pain intensity as the strongest association with PSP
- High post-operative acute pain intensity
- Postoperative chemotherapy / radiotherapy
- Female
- Younger age
- Genetic polymorphism is associated with other chronic pains or in animal studies, while none was reported specifically for PSP in humans.

Negative psychological / social associations with PSP and disability

- Preoperative anxiety and depression, including post-traumatic stress disorder (PTSD)
- Fear of surgery
- Introverted personality
- Passive coping skills
- Solicitous responding to significant others

Positive psychosocial associations

- Presence of social support

Catastrophizing is associated with increased acute postop pain yet has variable effect on PSP (not as a regression towards mean)

Notice that many of these risk factors are statistical associations while causation is yet to be established. Most studies were retrospective, and there was very little long term (> 1 year) data.

Prevention or reduction of severity / impact

Multiple strategies could be made to prevent or minimize PSP including:

Regional anaesthesia / analgesia

- Neuraxial local anaesthetics +/- opioids blocks & perioperative local anaesthetic infusions: conflicting evidence, as they reduce post-prostatectomy PSP, post-thoracotomy PSP, post-GI surgery PSP, but minimal impact on PSP and phantom pains post-amputations.

Systemic analgesics

- Gabapentinoids: significant reduction in PSP but not phantom. Pregabalin reduce post-knee replacement PSP
- Mexilitine: less effective than Gabapentin but still reduce burning sensations
- NMDA antagonists: Memantine reduce post amputation phantom and analgesic requirement. Conflicting results with low dose Ketamine
- Lignocaine infusion: may reduce PSP post-mastectomy
- Venlaflaxine: may reduce PSP post-mastectomy

Multimodal analgesia

- Presurgical paravertebral block & COX-2 inhibitor reduce post-mastectomy PSP
- Presurgical Gabapentin & EMLA together with surgical injection of local anaesthetics reduce post-mastectomy PSP

The variation of efficacy of the above measures might be due to:

- Presurgical pain causing significant central sensitization
- Unknown or untreatable underlying causative factors e.g.: genetics
- Treatment of associations rather than causative factors e.g.: acute postoperative pain
- Use of pre-emptive rather than preventive analgesia
- Interpersonal variations in nociception and analgesic responses
- Statistics, such as studies on subjects with unknown / variable pre-surgical pain, psychological profile, etc.

While methods of predicting severe post-operative pain have been developed (e.g.: psychophysical screening, cold-pressor test), they have yet been adopted as a routine for risk stratification. It is unknown if these methods would lead to reduction in PSP severity or disability.

It is also not known if psychotherapy would reduce the incidence or severity of PSP.

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Question 5 – Compulsory

Discuss the diagnosis and management of Major Depressive Disorder in a patient with persistent pain.

It is essential to recognise that while persistent pain can lead to depression, more than 50 per cent of patients with the primary diagnosis of a depressive disorder also complain of pain that does not have an objectively demonstrable organic basis.

It is therefore essential that all patients presenting with pain have a comprehensive assessment both to ascertain whether or not there is a physical lesion that is consistent and congruent with the pain symptoms and clinical findings on physical examination, and also to determine whether or not a depressive disorder is present and to establish a 'baseline' of relevant psychiatric symptoms.

"Depression" is a descriptive term used to refer to a fundamental change in mood characterised by the presence of sad or irritable mood, together with somatic and cognitive changes that significantly adversely affect the individual's capacity to function.

Individuals might use a variety of terms to describe their sad or low mood (sad, blue, unhappy, down in the dumps, miserable), which in Major Depression is associated with loss of interest or pleasure in usual activities (everyone experiences some of these symptoms from time to time i.e., over short periods, and it may not necessarily mean a person is depressed; not every person who is experiencing depression will have all of these symptoms.).

In clinically significant depressive episodes the individual experiences depressed mood, loss of interest and enjoyment, and reduced energy leading to tiredness and diminished activity. The complaint of marked tiredness after only slight effort is frequent.

Other common symptoms are:

Reduced concentration and attention;
Reduced self-esteem and self-confidence;
Ideas of guilt and unworthiness;
Bleak and pessimistic view of the future;
Ideas or acts of self-harm or suicide;
Disturbed sleep;
Change in appetite.

Management:

- Manage/treat both pain & major depressive disorder
- Suicide risk assessment
- Is this person distressed and/or depressed?
- How severe is the depression?
- How long and how much of the time have the symptoms been present?
- Before starting treatment, exclude other causes and evaluate history. Seek an explanation for the distress (e.g. grief) and exclude treatable causes such as an alcohol or drug problem, other psychiatric conditions including anxiety or bipolar disorder, adverse effects of medication or active medical conditions (e.g., hypothyroidism)
- Stepped care: Use a stepped approach to match the severity of depression with the response to intervention

- Non-drug treatments are useful in mild depression but antidepressant medication is required in the treatment of Major Depressive Disorder.
-Offer comfort and reassurance for patients reacting to grief: Offer comfort, listening and reassurance, counsel and mobilise social support and promote problem-solving for grief reactions including mild–moderate–severe mood disturbance that is understandable as a reaction to the loss.
- Patient education including exercise
- Consider psychotherapy: Always consider supportive psychotherapy as it augments antidepressant treatment
- Refer to specialist care in cases of severe depression where the risk of self-harm is high.
- Antidepressant medications: various e.g., TCA, SSRIs, SNRIs (especially duloxetine), MAOIs
- Antidepressant medication should be considered for moderate to severe MDD.
- There is no single best agent, and selection depends on the patient's antidepressant treatment history, potential drug interactions, and desired side-effect profile
- chronic pain from peripheral neuropathy: duloxetine
- neuro modulation e.g., ?Electroconvulsive therapy (ECT), ??Transcranial magnetic stimulation (TCM), ???Deep brain stimulation (DBS).

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Question 6 – Non compulsory

Provide notes on the management of pain related Irritable Bowel Syndrome (IBS).

EXAMINERS COMMENTS

To be made at the time of marking. Note: candidates are asked to provide notes, not list. The focus of this question is on management, not assessment.

ANSWER RESOURCE

Introductory comments

Irritable bowel syndrome is one of the most common gastrointestinal (GI) complaints encountered in primary or secondary care. It is more common in women 1:1.67 and in their younger years. It has a variable presentation and incidence being most common in South America 21%, northern Europe and North America with 12 % and least common in South East Asia 7%.[i]

There is *no single etiological explanation* and it is thought to be a combination of genetic predisposition, immunological and environmental factors including conditioning. It is not associated with increased mortality.

Diagnosis should be based on symptom based clinical criteria rather than excluding red flags and organic diseases by investigations. A consensus statement on the diagnosis of IBS has been developed called the ROME criteria (current version = ROME III)[ii].

Rome criteria:

- History of abdominal pain for three days per month in the previous 3 months.
- Symptoms should have occurred at lease six months prior to diagnosis
- Symptoms include three or more of abdominal pain or discomfort associated with relief with defecation, change in frequency of stool and change in form of stool.

IBS is considered to be a functional gastrointestinal disorders. A medical condition is considered functional when it impairs the normal function of a bodily process, but no abnormality can be detected by available testing.

As with other functional disorders there is no single management intervention. Reassuring the patient that the physician acknowledges the symptoms and that the condition is not life-threatening should be the first step in supporting the patient. Though no unifying pathophysiology can be identified treatment or management might be directed towards possible alterations in gastrointestinal flora, abnormal colonic transit time, and visceral hyperactivity. Attention to all these elements is vital, as targeting the pain in isolation is likely to result in persistence of symptoms and further complications.

Management

1. Diet and Lifestyle intervention.

High fibre

Advice to increase dietary fibre has conflicting evidence. Studies in secondary and tertiary care suggest no benefit however metanalysis in primary care showed significant benefit over placebo.

Food elimination

Patients with symptoms associated with specific foods may benefit from eliminating fermentable saccharides and polyols (FODMAPS).

Gluten free

The evidence for a gluten free diet is persuasive. [iii]

2. Management of comorbid conditions[iv]

Comorbid somatic intestinal and/or extraintestinal comorbidities (see below) are twice as common in IBS than controls. The occurrence of one or more comorbidity is correlated with higher health seeking, worse prognosis, and higher rates of anxiety and depression.

Comorbidity	Condition
1. Intestinal	
Functional GI disorders	• Functional dyspepsia • Gastroesophageal reflux • Functional constipation • Anal incontinence
Other GI diseases	• Carbohydrate malabsorption • Inflammatory bowel disease
2. Extraintestinal	
Pain syndromes	• Cerebral: migraine, other headaches • Musculoskeletal: fibromyalgia, Temporo-mandibular joint pain, back pain • Urogenital: chronic pelvic pain
Urogenital syndromes	• Dysuria, intersititial cystitis, disturbed sexual function, dysmenorrhea, premenstrual syndrome
Bronchopulmonary syndromes	• Asthma, bronchial hyperactivity
Cardiac syndromes	• Palpitations
Other	• Sleep difficulties, chronic fatigue

Coexisting chronic fatigue and fibromyalgia symptoms of IBS respond to increased physical exercise particularly when this is conducted as a group activity.

Though beneficial for many aspects of chronic pain the evidence for specific sleep hygiene interventions is unclear. Melatonin may be of some benefit in women than placebo.[v] Pain and symptom related education, again in a group setting was related to higher rates of symptoms resolution than non-attendees.

3. Medications

Many medications have been studied for the management of IBS. It is important however, to note that the reported placebo response is high. An open label placebo study of 80 patients reported a 50% response.

- Because *bloating and 'spasm'* are frequent symptoms drugs that compete with Acetylcholine in the parasympathetic nervous have been studied. Many agents such as hyoscine, dicycloverine and cimetropium, have been examined both in combination and individually. The response is more effective than placebo but associated with adverse effects, usually minor. Peppermint oil however has an NNT of 2.5 with few adverse events.

- *Anxiety and depression* are more common in patients with IBS. Tricyclics with an NNT of 4 and SSRI (NNT of 3.5) are more effective than placebo.
- Serotonin receptor antagonists have the potential to aid in smooth muscle spasm and abdominal pain. However, studies show alosetron has a NNT of 8 with the potential of significant harm and several cases of ischemic colitis.
- In attempting to *alter the flora of the large intestine*, rifaximin (non absorbable antibiotic) has been subjected to study. Treatment for two weeks resulted in a significant improvement in symptoms at 4 and importantly 12 weeks. There were no cases of adverse reactions including *c.difficile*. Probiotic (live or attenuated bacteria) preparations may have an anti-inflammatory effect. Many preparations are available however it appears that bifidobacteria show a trend towards benefit.[vi]
- Little or no evidence exist for the *use of opioids* in the long-term management of this condition. Like other functional pain disorders the use of opioids exposes the patient to many side effects and dangers without contributing to a reduction in pain, improvement in function or quality of life.

4. Psychological and behavioral intervention.

- Cognitive behavioral therapy, dynamic psychotherapy and hypnotherapy have been subjected to randomized trials. Pooled data suggest that the outcomes are only marginally greater than placebo.[vii] The type of psychological intervention appears to be important. CBT, hypnotherapy and multicomponant psychological therapy being more effective than relaxation therapy and self-administered CBT. The method in these studies was also questioned. [viii] More recently hypnotherapy and mindfulness based CBT were found to be effective up to three months.

5. Complementary, alternative and novel future therapies

Any benefit of herbal therapy either singly or in combination or for acupuncture (outside Chinese studies) remain unclear.

Emerging therapies such as agents to alter the secretion of mucosal chloride thereby increasing luminal fluid and accelerate transit time show some promise. Bile acid transporters and pancreatic enzyme supplements are also under investigation.

Conclusion

Clearly no single modality will manage the pain distress and other symptoms associated with IBS. In order to obtain the best management a broad approach addressing pain and comorbid conditions is required. Equally treating the pain in isolation to the other aspects of the syndrome will almost certainly result in a suboptimal result.

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Question 7 – non compulsory

Describe the differences in nociception in the immature and mature nervous system.

PERIPHERAL DIFFERENCES

Nociceptors

The number of ion channels on peripheral nociceptors available for transduction and activation of sensory neurones is reduced in neonates. This may lead to reduced nociceptor transduction in the immature nervous system.

Sensory Neurone

Conduction velocity in immature sensory neurones is slower and at a reduced frequency due to incomplete myelination and immature ion channels.

Nociceptor sensitisation

Peripheral sensitization occurs in both the mature and immature systems, however the increased sensitivity to noxious mechanical, but not a thermal stimuli resolves more quickly in the immature system. The levels of nerve growth factor are particularly high in the immature nervous system particularly following skin injury, possibly leading to increased hyperalgesia.

Protective reflexes to nociceptive impulses are more pronounced in immaturity. This is a generalized flexion response away from the source of the injury. It is greater in amplitude and duration than in the mature system but not as specific. These reflexes may be evoked by low intensity stimuli, in the immature system. The threshold for these non-specific reflexes increases with gestational age.

CENTRAL DIFFERENCES

Central connections

A-beta mechanoreceptor fibres terminate more diffusely in the immature system, with a significant number overlapping with C-fibre termination in laminas I and II. The clear separation of the two sensory systems with the A-beta fibres terminating in layers III and IV of the spinal cord, is not complete until well into the post natal phase. Activation of A-beta fibres in the immature animal, thus cause an activation process similar to that typical of activation of a combination A-delta and C-fibre activity in maturity.

Excitatory dorsal horn neurons have wide receptive fields at birth which decreases with age. These neurons have lower thresholds and fire for longer than in adulthood. Their latency of response is also longer and more variable, due to the slower synaptic transmission and immature ion channel development.

Inhibitory interneurones in the dorsal horn mature more slowly than the excitatory neurons. They respond to the inhibitory neurotransmitter gamma aminobutyric acid (GABA) in a similar fashion to adults, but the receptive fields for excitation and inhibition are not matched in neonates as they are

in adults. This leads to the heightened excitability and lack of selectivity and spatial organization of protective reflexes seen in neonates.

Central sensitisation

The *N*-methylp D aspartate (NMDA) receptor is activated by glutamate. This system is fully functional from birth. Despite this the immature nervous system does not develop neuropathic hypersensitivity following nerve injury, Central sensitization spreads to involve glial cells and neuroimmune pathways within the spinal cord and the immature nervous system has different immune responses with lack of activation of microglia and T-cells.

The rostroventral medulla (RVM) is the major control site for descending inhibition and release of endogenous opioids causing inhibition of central nociceptive transmission in the peri-adolescent stage. Prior to maturation this area is involved in facilitation of spinal nociception.

PROCESSING DIFFERENCES

Processing of Nociception

Although infants receive nocicetvive input to the spinal cord, brainstem and subcortical midbrain structures, which is sufficient to generate reflex behaviors and hormonal responses, this is not sufficient to support pain awareness. This requires maturation of the “pain matrix”.
“The pain matrix” in itself is not a defined entity, having varied inputs from cognition, mood, injury and previous experience all of which influence the final perception of pain. This produces a complex multi-factorial, subjective and individual experience. Little is known about the maturation of the pain matrix over infancy. Recent studies show that nociceptive activity is processed at a cortical level in infants, as evidenced by. EEG responses in pre term infants.

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Question 8 – non compulsory

Discuss the role of cognitive-behavioural therapy (CBT) in the pain clinic. Include reference to specific patterns of cognition.

This question pertains to fundamental principles of pain assessment and intervention. It is a broad question for which it is difficult to provide an ideal, model answer.

The candidate was expected to discuss the pivotal role of psychological process in the genesis and maintenance of cognitive and behavioural responses to pain, which in turn fuel the persistent pain states which present to our clinics.

Some reference to the formal definition of pain was expected, highlighting the link with psycho/emotional elements of the phenomena. Failure to do so could be adequately compensated by an appropriate, broader explanation of the interrelating and co-causal links.

It was expected that the "role" of psychobehavioural intervention would be discussed in terms of several angles, which might include:

- Minimisation of unhelpful adherence to, and growing entrenchment in, the sick-role e.g. cure seeking,
- Prevention of passage to greater deconditioning (physical, social, vocational etc.)
- Promotion of functional restoration
- Addressing of comorbid psychiatric disorders eg depression, anxiety, substance misuse

The more sophisticated candidate was able to observe that psychobehavioural interventions provide pain clinics with a potentially effective intervention for patients whose condition may not prove amenable to traditional, medical model treatment. This not only assists the patients but also the practitioners, who might otherwise be prone to perpetuate the sick role through inadvertent reinforcement of that status, and who may themselves develop their own despondency.

In asking that specific reference bed made to at least one style of cognitive error, it was anticipated that most candidates would chose to discuss catastrophistation. An initial definition (preferably referenced) was expected:

> The term was coined by Albert Ellis and developed by Aaron Beck to describe a mal-adaptive emotional style involving an irrational, negative interpretation of conditions and a similarly bleak outlook for the future. In the context of pain, catastrophising relates to a set of extreme, exaggerated, and unrealistic interpretations of the pain stimulus and related matters. Quartana et al have defined it as : **"**a negative cognitive–affective response to anticipated or actual pain and has been associated with a number of important pain-related outcomes".

There are a number of theoretical mechanisms of catastrophisation:

- appraisal theory,
- attention bias/information processing,
- communal coping,
- CNS pain processing mechanisms,
- psychophysiological pathways and neural pathways.

The sophisticated candidate would have made reference to some of these along with a basic expansion**.**

This phenomenon may relate to a premorbid negative cognitive style (ie personality based, or trait dependent), or may be consequent upon the combination of prevailing influences brought to bear during the period of pain (ie state dependent). The candidate was expected to acknowledge this distinction in regard to whichever cognitive error was being discussed, and to discuss the relationship between comorbid mood/anxiety issues in state determined dysfunction.

The literature indicates associations between catastrophizing and pain severity, activity interference, disability, depression / anxiety and pain / illness behaviour.

Ref Quartana et al

Question 9 – Non compulsory

In a typical adolescent chronic pain clinic, 80% of patients are female.

1. **What factors have been proposed in the literature to explain this?**
2. **What implications for pain experience and function in adult life does having a chronic pain syndrome in adolescence have?**

1. Literature

PAIN CLINIC PREVALENCE DATA:

Reported higher prevalence of chronic pain states in adolescent girls in pain clinics and in community/school cohorts (see below). Prepuberty equal numbers of boys and girls.

With puberty hormonal changes, diseases appear in teenagers that occur in adult females or with adult female preponderance –
~1.5-4:1 migraine /CDH, IBS, FM, CRPS, juvenile inflammatory arthritis
dysmenorrhoea/endometriosis/Chronic pelvic pain

(Community data (Hoftun, Romundstad et al. 2011, Kroner-Herwig, Gassmann et al. 2011, Hoftun, Romundstad et al. 2012, Rathleff, Roos et al. 2013))

DIFFERENCES IN PAIN PHYSIOLOGY AND PAIN PERCEPTION

Rodent studies:

- Females are more sensitive than males to noxious stimuli and have lower levels of stress-induced analgesia.
- Female rodents (pain models) generally have lower analgesic response to mu-opioid receptor agonists than males; with a lower level of activity in the endogenous analgesic system of normal females compared with normal males (Wiesenfeld-Hallin 2005). The response to kappa-opioids, mediated by the melanocortin-1 receptor gene in both mice and humans, is different for each sex. MCR1 mutant female mice have increased tolerance to noxious heat and reduced pain post formalin inflammatory insult, with delayed onset hyperalgesia and allodynia (Delaney, Keighren et al.).
- Compared with male rats, female rats post neurological injury have more hypersensitivity following spinal cord injury (Dominguez, Strom et al. 2012) and more profound and prolonged facial hypersensitivity following infraorbital nerve injury (with no difference post sciatic nerve injury) (Dominguez, Kouya et al.).
- Oestrogen receptor knock-out mice developed same level (not greater) mechanical hypersensitivity to carrageenan inflammatory stimulus (Li, Fan et al.)

Human laboratory studies:

Many articles have described sex differences in pain sensitivity. Women experience greater clinical pain (more intense/more frequent), suffer greater pain-related distress (different emotional regulation), and show heightened sensitivity to experimentally induced pain e.g. increased response to noxious stimuli such as heat (including being better able to discriminate variation) and less exercise-induced hypoalgesia.

Women and adolescents girls with chronic pain have more tender points than men/boys.

Mu-receptors in the healthy female brain are activated differently from those in the healthy male brain – females require more morphine (30% for same effect (Cepeda and Carr 2003)) and have reduced placebo response vs males (Loyd and Murphy 2014) (Aslaksen, Bystad et al. 2011). Males demonstrate larger activation of endogenous opioid systems during pain: suggesting more efficient pain inhibition (Zubieta, Smith et al. 2002).

Effect of oestrogen/ Menses cycle variation –humans (and female rodents) experience more pain / have lowered thresholds when oestrogen is low (menses/luteal phase). Morphine is more effective in follicular phase but with more side effects (Ribeiro-Dasilva, Shinal et al. 2011). Women have greater diffuse noxious inhibitory control in follicular phase.

Girls with irritable bowel syndrome have impaired endogenous inhibition of somatic pain (Williams, Heitkemper et al. 2013)**.** JIA patients have increased pain hypersensitivity (Cornelissen, Donado et al. 2014). No sex differences observed in healthy children (Tsao, Seidman et al. 2013)

Imaging studies of the brain have shown differences between men and women in the spatial pattern and intensity of response to acute pain.

DIFFERENCES IN COPING STRATEGIES/ LEARNING STYLES –

- Girls more likely to
    - display symptoms
    - to seek help from people (relatives-peers), including medical help
    - to talk/express themselves and to work with others for solution
    - report recurrent pain and multiple pain sites (Kroner-Herwig, Gassmann et al. 2011) (Rathleff, Roos et al. 2013)
    - be catastrophisers/have anxiety which influences pain related disability and school attendance

SOCIAL CONDITIOING/PSYCHO-SOCIAL FACTORS

Different parenting styles/rearing of normal children (gender neutral vs blue/pink ; dolls/trucks; give sympathy vs toughen up)- social conditioning starts early: playground injury- adults attend to girls and give sympathy vs boys encouraged to get up and move on.

Differences in parental/extended family response to child/teenager with chronic pain – overprotective, distressed, cure seeking on their behalf

Modelling – one or both parents with chronic pain, resilience/active vs passive coping

Patient / family - healthcare provider relationships

PHYSICAL PREDISPOSITION:

Reduced muscle bulk and higher rates of hypermobility

– sports, dancing - overuse injury

FAMILIAL CLUSTERING OF FUNCTIONAL PAIN SYNDROMES –

Psychosocial/environmental influences vs shared genetic factors (rodent and twin studies) underly chronic pain syndromes (Vehof, Zavos et al. 2014)

PSYCHIATRIC-MENTAL HEALTH ISSUES

Victims of childhood sexual and physical abuse/emotional neglect/attachment disorder - 2-3 times higher incidence in girls in Australia, 5 times in US (mean age 9; Sedlak 2010 Congress report)

Girls are possibly more sensitive to early life adversity – parental separation/divorce, separation from parent (maternal) including death

Female preponderance of psychiatric disorders: Somatiform disorder/Anxiety/Depression – chronic pain as a symptom cluster; also borderline personality disorder/ self-harm 3 times more common in girls or is that the cutting is seen

Bullying: emotional by teenage girl peers- peer group interactions

2. **Implications IN LATER LIFE**

INCREASED INCIDENCE OF

Same chronic pain syndrome

- Fibromyalgia associated with fibromyalgia/Chronic widespread pain [CWP] in later life (Shelby, Shirkey et al.)
- Functional abdominal pain/diarrhoea in childhood and adult IBS (Marugan-Miguelsanz, Ontoria et al.) (Horst, Shelby et al.)
- Childhood headaches associated with 2.2x likelihood of adult headache (Fearon and Hotopf 2001)
- Musculoskeletal pains in childhood and CWP in adulthood (increased if low parental educational level) (Flato, Aasland et al.)
- Regional pain as child more likely to develop another site, odds increased if multisite at baseline

Different type of chronic pain syndrome

- Abdominal pain, migraine and non-migraine headaches associated with chronic widespread pain later in life (Jones, Silman et al. 2007) (Marcus 2005)
- Headache and back pain associated with pain complaints in earl adulthood (Brattberg)
- Childhood abuse resulting in PTSD associated with CWP in adulthood (Raphael and Widom)
- Abdominal pain and later hospital admissions for 'unexplained medical symptoms' 2001

Psychological consequences or mental health disorder (e.g. depression, anxiety)

- Functional abdominal pain patients more likely to be anxious as adults (even if abdominal symptoms resolve, with increased psychological disorder over their lifetime (Shelby, Shirkey et al.)
- Migraine is associated with later development of major depressive episodes (Modgill, Jette et al.) Headache and Various psychological symptoms (Fearon and Hotopf)
- Abuse leading to persistent PTSD, anxiety, hypervigilance, hypersensitivity, distrust
- PHx abuse increases pain related disability and depression in a chronic pelvic pain population (As-Sanie, Clevenger et al. 2014)
- Fibromyalgia patients who had juvenile onset more likely to be married (Kashikar-Zuck, Cunningham et al.) (does this reflect dependent personalities/disorder)

Impaired Physical function—

- Fibromyalgia patients who had juvenile onset more disabled as adults (Kashikar-Zuck, Cunningham et al.)
- Relapsing/Spreading CRPS
- Headache as child associated with various physical symptoms (Fearon and Hotopf 2001)

Impaired Social function

- Missed schooling/Lower educational attainment
    - Fibromyalgia patients who had juvenile onset (Kashikar-Zuck, Cunningham et al.) less likely to have attended college
- Missed employment opportunities/Financial impact/Disability support pension
- Impact on future relationships/spouse/dependency
- Impaired interaction with own children

Having offspring with chronic pain condition (as child or adult)

- Heritable component (Lier, Nilsen et al.) as chances higher if both parents have chronic musculoskeletal pain
- Vs environmental factors:

- Influence of adult with chronic pain modelling for own children – learned pain behaviour -interpretation of symptoms, choice of coping strategies – family pain model increases community's chronic pain burden
- Parental chronic pain associated with chronic non-specific pain and especially chronic multisite pain (Hoftun, Romundstad et al. 2013)
- Shared socioeconomic, income, education variables
- Similar expectations
- Psychological impact of parent with anxiety/depression, withdrawn/distressed

Suggested reading for exam report:

Sex differences

(Loyd and Murphy)

(Paller, Campbell et al.)

(Wiesenfeld-Hallin 2005)

Impact

Question 10 – Non compulsory

Discuss the assessment, differential diagnosis and management of unilateral facial pain.

Summary and key points.

The assessment and management of the patient with facial pain is challenging and requires a **detailed history** and **focussed physical and particularly neurological examination** to formulate a differential diagnosis so that specific treatment can be provided.

As with any neurological disorder one should always consider the potential for a serious or potentially life threatening cause (**red flags**) of the patients craniofacial symptoms particularly if neurological symptoms are present e.g. tumour, aneurysm and urgent investigation e.g.MRI should be considered. Another reason for difficulty is the poor understanding of the pathophysiology of a number of conditions that cause orofacial pain and limited training and expertise of doctors in this area.

A broad differential diagnosis for facial pain exists because of the **wide convergence of craniofacial afferent inputs into the brainstem**. Thus **pain may be referred, from regional disease or secondary to systemic disease**. Therefore input from a number of specialists e.g ENT, neurosurgeon, dentist may be necessary

A number of taxonomies provide classification systems that may aid differential diagnosis however these are constantly evolving and tend to focus either on the general or the specific e.g IASP (general), Burchiel facial pain questionnaire (trigeminal neuralgia), IHS (headache), TMD classification systems.

When the pain is acute and can be confined to a region, diagnosis is usually straightforward and management can be focussed and is usually has a good chance of satisfactorily managing the patient's pain. For example trigeminal neuralgia may be readily diagnosed and satisfactorily treated provided a good history and awareness of the condition is available.

Pain medications for facial pain should be specific for the pain phenotype and putative source e.g. nociceptive or neuropathic. The principles and effectiveness are similar to pain syndromes elsewhere except that for trigeminal neuralgia carbamazepine has a NNT of 1.7 for trigeminal neuralgia.

Procedures for facial pain include system specific (e.g. ENT/dental) or ones directed specifically toward the nervous system including lesional or neuromodulatory. Evidence for such procedures is limited with the exception of lesion procedures for trigeminal neuralgia particularly radiofrequency.

When the pain is chronic and difficult to localise, diagnosis is more difficult and management is based upon pain phenotype, is empiric and needs to incorporate psychosocial strategies to achieve the best outcomes. A further difficulty in management is that, in contrast to other locations, **pain in the craniofacial area may have special biological, emotional and psychological meaning to the patient**. In this scenario involvement of a multidisciplinary pain clinic is most effective.

History. The history needs to be detailed and should focus on the origin and evolution of the pain symptom which will direct further enquiry e.g. What were the very first symptoms ? Location of symptoms ? Onset of symptoms ? Details of nature of pain, exacerbating and relieving factors ? Other symptoms e.g. neurological ? What treatment ? Response to treatment ? As is other medical problems the history will contribute most to the differential diagnosis. In neurological disease the classical approach is to direct the history toward both the anatomical localisation of the problem and also the process/pathology responsible suggested by the time course of evolution of symptoms. As with any neurological disorder one should always consider the potential for a serious or potentially life threatening cause of the patients craniofacial symptoms particularly if neurological symptoms are present e.g. tumour, aneurysm (**red flags**)

Past medical, dental, pain and family history may be relevant.

Developmental, psychological and social history will be important when a clear localisation is difficult or the presentation complex.

Examination. General, neurological, regional and specialised (refer to colleagues). The presence of neurological signs is especially important as it may signal a red flag as well as a valuable localising tool.

Investigations. Will be tailored to the site of symptoms but a low threshold should exist for MRI brain and cervical spine which will rule out serious or life threatening diagnoses promptly and will allow for some reassurance for the patient.

Differential diagnosis can be formulated by a number of approaches including

A classical neurological approach – location and process.

Taxonomy approach (IASP) – neuralgias of the head and face

- craniofacial pain of musculoskeletal origin
- lesions of ear, nose and oral cavity
- primary headache – also consider HIS classification
- psychological causes
- suboccipital causes
- visceral pain in the neck

Systemic or regional causes

Specialised facial pain questionnaires –e.g. Burchiel classification are looking for trigeminal neuralgia

Each of these approaches may be valid depending upon the likely diagnosis and have individual strengths and weakness.

Subspeciality referral may be necessary to clarify the regional process responsible for the pain syndrome.

A psychosocial formulation is also important particularly in chronic pain states.

Management. Derives from the differential diagnosis and the psychosocial milieu.

If a treatable cause can be identified and treated this is clearly the optimum outcome with the best chance of immediate and longer term pain relief. This may require specialist intervention e.g. neurosurgeon for trigeminal neuralgia.

If a treatable cause cannot be clearly identified then treatment necessarily becomes symptomatic and multidisciplinary. This does not necessarily mean a treatable cause may never be identified and should still be considered particularly if a complete work up including imaging was not originally performed. Repeat subspeciality referral or second opinion may be helpful

Question 11 – non compulsory

Outline the metabolism of codeine and tramadol. Discuss how genetic differences may alter both the effect and incidence of adverse effects associated with each of these drugs.

A. Metabolism of codeine

- Metabolised in the liver
- Converted to morphine (active metabolite) by CYP2D6 cytochrome P450 isoenzyme and then to M6G and M3G (both renally exctreted)
- Converted to norcodeine (inactive metabolite) by CYP3A4

B. Metabolism of tramadol

- Also metabolised in the liver
- Converted to O-desmethyltramadol (M1) – main active metabolite – by CYP2D6
- M1 is excreted by the kidney

C. Effects of genetic differences

a) General

- Genetic variability in CYP2D6 is common and results in significant differences in enzyme activity
- Over 100 allelic variants of CYP2D6 have been identified, resulting in wide variability in enzyme activity; the result is 4 phenotypes PM, IM, EM and UM

CYP2D6 variants	
Poor metabolisers	▪ no functionally active alleles ▪ minimal or no enzyme activity
Intermediate metabolisers	▪ heterozygotes with two variant alleles (least 1 reduced functional allele) ▪ decreased enzymatic capacity
Extensive metabolisers	▪ two wild-type alleles ▪ normal enzyme activity
Ultra-rapid metabolisers	▪ carriers of CYP2D6 gene duplication (>2 copies of functional alleles) ▪ significantly higher levels metabolites

- There are differences in the proportions of each phenotype in different populations

CYP2D6 variants		
Poor metabolisers	▪ Caucasians ▪ Asians	▪ 7 – 11% ▪ 1 – 2%
Ultra-rapid metabolisers	▪ Caucasians ▪ Asians ▪ Mediterranean ▪ Ethiopia ▪ Saudi Arabia	▪ 7% ▪ 0.5% ▪ 10-12% ▪ 21% ▪ 29%

b) Codeine

- Its analgesic action depends on the metabolism in the liver of about 10% of the dose given to morphine – this accounts for all the analgesic effect of codeine, as the drug itself has a very low affinity for opioid receptors. It should therefore be regarded as an ineffective prodrug of morphine

Codeine	
Poor metabolisers*	▪ low/ undetectable plasma morphine levels ▪ greatly decreased analgesia
Intermediate metabolisers	▪ reduced morphine consumption (not much clinical data)
Extensive metabolisers	▪ normal morphine production
Ultra-rapid metabolisers*	▪ 50% ↑ morphine & glucuronides ▪ has led to ↑ side effects including respiratory depression with usual recommended doses ▪ implicated in deaths in paediatric patients and therefore not recommended for use ▪ implicated in deaths of neonates of breastfeeding mothers who were ultrarapid metabolisers

* = key points

c) Tramadol

- M1 is a 200 x more potent μ-opioid receptor agonist than tramadol itself, thereby contributing significantly to its analgesic efficacy

Tramadol	
Poor metabolisers*	▪ significantly lower plasma concentrations of M1 ▪ less effective analgesia
Ultra-rapid metabolisers*	▪ greater analgesic effect ▪ has led to respiratory depression

* = key points

References (attached as well as see APMSe 3e)

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Question 12 – Non compulsory

Discuss pain in the patient with a spinal cord injury.

evalence of pain in SCI:
- Pain is a common complication after spinal cord injury (SCI)
- Reported prevalence of pain after SCI varies widely between 34-90%, due to differences in study design, and definitions of pain types and severity
- ~ 2/3 of people with spinal cord injury suffer from chronic pain with ~1/3 of these people reporting severe pain

Impact of pain in SCI:
- Pain can significantly impact upon a person's functional ability and independence, psychological well-being, ability to return to work
- Chronic pain is one of the most frequently reported reasons for reduced quality of life

Classification of pain in SCI:

- A number of different types of pain that are commonly seen in persons with a spinal cord injury.
- Pain taxonomies for SCI classify pain as neuropathic or nociceptive, and according to level of injury
- Classification of these pain types has always been somewhat problematic due to considerable uncertainty about the underlying mechanisms and systems involved and a wide variety of terms have been used in describing the same type of pain.
- International Association for the Study of Pain proposed a classification system
- first divided into nociceptive (pain arising from somatic or visceral structures) and neuropathic (pain arising from nerve structures including the spinal cord and brain).
- The system then identifies 5 common types of pain seen following SCI, including:
1. Musculoskeletal pain arising from bones, joints, ligaments and muscles either in the acute post-injury phase or with chronic overuse;
2. Visceral pain arising from disturbances to bladder, bowel or other visceral function;
3. At-level neuropathic pain, sometimes described as endzone or borderzone, which is a band of burning, electric or shooting pain and hypersensitivity in the dermatomes close to the level of injury;
4. Below-level neuropathic pain, referring to pain with the same burning, shooting, electric qualities as the previous type of pain but it is located diffusely below the level of injury usually bilaterally in the buttocks and legs.
5. Above-level neuropathic pain is not exclusive to spinal cord injury but includes several types of neuropathic pain that are commonly seen, such as complex regional pain syndromes and compressive neuropathies (eg. carpal tunnel syndrome).
- This classification attempts to identify most of the pain types commonly seen with the aim of providing direction for treatment.

Mechanisms of pain in SCI:

-understanding of the pathogenesis and biological mechanisms underlying pain after SCI still remains quite limited
- While psychosocial factors seldom give rise to pain in isolation (so-called “psychogenic” pain), they universally influence the pain perception and behaviour and are important contributors to the pain experience.
- the mechanisms underlying musculoskeletal pain and visceral pain are better understood and in common with other types of nociceptive pain are basically due to increased inputs arising from damaged and inflamed structures.
- the mechanisms underlying neuropathic pain are poorly understood. Broadly, neuropathic pain arises from abnormal activity in pain pathways. This means that spontaneous activity of neurons may give rise to pain or increased responsiveness of neurons may give rise to hypersensitivity to touch or other stimuli. However, the site of abnormal activity may vary with different neuropathic pain types.
- Neuropathic pain arising from peripheral structures, as occurs with compressive neuropathies, is similar to other types of peripheral neuropathic pain and is thought to be due to abnormal impulses arising from damaged nerve structures.
- At-level and below-level neuropathic pains are more specifically related to spinal cord injury and a number of possible mechanisms have been proposed to account for these pain types. These include abnormal activity of neurons within the spinal cord close to the site of damage, as well as more rostral changes in the thalamus and cortex that seem to occur as a secondary consequence of spinal cord damage

Pathophysiological changes include the following:
1. direct damage to the spinal cord
- This results in activation of inflammatory mediators, glia and neuronal second messengers. Activation of these mediators and second messengers may in turn induce receptor changes (increased expression or enhanced responsiveness) and changes in the release of neurotransmitters that may transmit pain. All of these local changes may give rise to an “irritated focus” in the spinal cord near the level of injury. This “irritated focus” may generate pain spontaneously or may serve as a damaged amplifier that distorts and amplifies incoming messages from the periphery.
2. damage to inhibitory mechanisms
- Damage to the spinal cord also results in damage to the normal inhibitory mechanisms that serve to block out pain. This can occur following local damage to inhibitory neurons or with interruption to inhibitory pathways descending from the brain to the spinal cord. It has also been suggested that under normal circumstances there is a balance of inhibitory and excitatory inputs and that preferential damage to inputs that dampen pain (for example, light touch transmitted through the dorsal columns) may result in an “imbalance” and the generation of pain.
3. network changes with damage and loss of inputs
- Damage to the spinal cord also leads to attempts to reorganise nerve pathways. Although limited, attempts to reconnect nerves at a spinal level may result in faulty “rewiring” that gives rise to pain. In this scenario, messages that normally travel along touch pathways are “hotwired” into pain pathways so that touch is felt as pain. Even if physical “rewiring” does not occur, the nervous system may attempt to compensate for lost inputs from damaged areas. This may involve activation of

latent pathways in the spinal cord that again may tap into pain perception. It has also been demonstrated that brain changes occur with abnormal activity in the thalamus and reorganisation of the somatosensory cortex as the brain attempts to adapt to the loss of inputs

Clinical presentation of pain in SCI:

- Clinical presentation of pain associated with SCI is highly complex in that different pain types are often present simultaneously
- Neuropathic pains are usually associated with evoked pain, such as allodynia or hyperalgesia
- the refractory nature of pain following SCI and the associated psychosocial distress emphasize the need for a greater understanding of not only pathophysiological but also psychosocial mechanisms in the generation and maintenance of SCI-related pain and pain-related suffering.

Assessment & diagnosis of pain in SCI:
- Like other types of chronic pain, pain following SCI is best considered within a biopsychosocial framework.
- Given the broad range of mechanisms that impact upon the development and maintenance of pain following SCI, a multidimensional approach to assessment and diagnosis is required.
- Assessment and treatment take into account these various factors that may be contributing to the person's pain, including biological (such as level and extent of neurological impairment), psychological (including mood and cognition) and environmental factors (such as responses of significant others
- Assessment of pain severity, physical and emotional functioning would best capture the multidimensional nature of pain
- After SCI, a decrease in physical function may be more related to the physical impairments of SCI rather than to pain; therefore, a decrease in function due to pain, i.e., pain interference should be assessed
- It is recommended that a full review of the patient with a SCI is conducted if one has not otherwise been conducted in the past twelve months. This will help to identify general issues, for example, in relation to seating or equipment, which may
be contributing to the pain problems.
- Identifying the type of pain provides a basis for further assessment, investigation and treatment.

- Clinical assessment of pain following SCI involves:
1. History
-A detailed history should be taken describing the pain type, onset and distribution, exacerbating and relieving factors, including relationship to posture and functional activity, such as transfers, etc.
2. Examination
-Performing a comprehensive physical examination is essential and should include sensory, motor and reflex testing to classify the level and degree of neurological lesion using ASIA standards
- As indicated by the proposed pain classification system, the first step is to determine whether the pain is nociceptive or neuropathic in nature. This is largely dependent on pain description (nociceptive: dull, cramping, aching, worse with movement or related to visceral function, localised tenderness, located in the region of sensory preservation; neuropathic: shooting, electric, burning, unrelated to activity, numbness or hypersensitivity to touch, located in the region of sensory

disturbance).
- The pain can then be classified more specifically as visceral or musculoskeletal or above-, at- or below-level neuropathic in type.
- Neuropathic pain may be indicative of new spinal pathology with development of a syrinx (ie. cystic cavitation of the spinal cord extending above and/or below lesion). This should be considered in someone with the onset or progression of neuropathic pain and deterioration in motor and/or sensory function
3. Psychosocial Assessment
- Screening of psychosocial factors contributing to disability and distress is important at this stage.
- Ideally, a full assessment should be undertaken by a suitably trained psychosocial professional, although some screening of mood may be done through the use of screening questionnaires, eg DASS,
• Adjustment to the injury and pain including tendency to catastrophic thinking
• Self-efficacy for both the pain and injury
• Behavioural responses to the injury and pain including adaptive and maladaptive behaviours
• Mood including depression, anxiety or PTSD symptoms
• Impaired cognition
• Co-morbid psychological disorders including psychiatric conditions or drug and alcohol dependency
• Social factors including social support, significant others responses to pain and injury

- A careful history and clinical examination provides the basis for subsequent focussed investigations, including imaging and/or electrodiagnostic testing or other special procedures

Management:

- Successful management of pain depends on the accurate identification of factors that may be generating or modifying pain perception and using strategies that effectively target these factors.
- Management of chronic pain syndromes following SCI proves very difficult and unfortunately is often only partially effective.
- when treating chronic pain, it is essential to comprehensively evaluate the type/s of pain and psychosocial factors contributing with emphasis on functional capabilities, behavioural responses to pain, adjustment to disability and degree of motivation.
- This is important when selecting an appropriate combination of pharmacological, physical, psychological and other treatment approaches.
- Treatable underlying pathology, such as local nerve root compression or post-traumatic syringomyelia (with expanding syrinx formation) must be excluded.
- Rehabilitation principles should underpin any pain management programme, with the overall objective being to increase self-efficacy and promote greater activity and participation.
- Goals for treatment should be developed collaboratively with the individual prior to making management recommendations, in order to ensure the most effective multi-modal approach.
- Goals may be set in a range of domains, including (but not exclusively) activities of daily living, physical fitness and endurance, vocational activities, recreational activities, relationships and family, and mood.
- Musculoskeletal pain may respond quite well to simple analgesics and opioids. Non-steroidal anti-

inflammatory medications may also be used, but caution needs to be exercised, as gastric symptoms may be masked in people with higher SCI lesions. Changes in posture, exercises, adjustments to wheelchairs and seating, hydrotherapy programs and other forms of physical treatment modalities may be helpful in treating pain that is arising from a mechanical source.
- Visceral pain requires specific attention to the presumed source of pain. Urinary tract infections and calculi need to be treated appropriately. Bowel related pain may respond to simple measures such as change in diet or bowel regime, but may also require further assistance from a spinal specialist

- Ideally, an effective treatment strategy should be tailored to specific pain-generating mechanisms in each individual; because of insufficient knowledge about the precise clinical symptoms and signs associated with a specific mechanism, this is not currently possible
- complementary measures should be added when appropriate for specific pain populations.
- Neuropathic pain responds poorly to most available treatments including opioids. - - The drugs that have been demonstrated to be most effective are the anticonvulsants and tricyclic antidepressants.
- Anticonvulsants work by dampening abnormal neuronal activity in peripheral nerves and the central nervous system.
- Tricyclic antidepressants are thought to work by increasing the available amounts of the inhibitory transmitters serotonin and noradrenaline

Pharmacological Management
(1) Nociceptive
- simple analgesics, NSAIDs and opioids are more likely to be effective in the treatment of musculoskeletal pain
- Visceral pain may be treated with analgesics although only after investigation for pathology that may be amenable to other treatment (renal calculi, bladder infection, bowel dysfunction, etc).
- Opioids are generally considered when simple measures fail to relieve the pain, although problems with constipation as a major side effect in patients with SCI, who already have slow colonic motility, as well as potential for developing physical dependence and tolerance, must be carefully considered.
- As a general principle, short acting and injectable opioids should be avoided. If long term treatment is being considered patients should be placed on a slow release formulation to reduce dose escalation and to provide more stable analgesia. This may be done after titration with a standard immediate release preparation of the same drug to determine analgesic requirements.
(2) Neuropathic
- analgesics are usually insufficient to control neuropathic pain and should be used in conjunction with adjuvant medications (ie. anticonvulsant, tricylic antidepressant; see dosing schedules below).
- For both chronic at-level (radicular or segmental) and below-level types of neuropathic pain, first-line treatment with either gabapentin or pregabalin is now recommended.
- Commonly used older anticonvulsants such as sodium valproate have been shown to be no more effective than placebo in randomised controlled trials. Carbamazepine has been shown effective in combination with amitriptyline in treatment of other types of neuropathic pain, but has not been studied in SCI pain. - - Because of their different modes of action, it may be more effective to combine a tricyclic antidepressant, such as amitriptyline or nortriptyline, or alternatively a weak opioid, such as tramadol, with an anticonvulsant.
- Due to concerns about serotonergic syndrome, the combination of a tricyclic antidepressant and

tramadol should be avoided.
- Although there are no studies specifically looking at spinal cord injury pain, the selective serotonin reuptake inhibitors (SSRIs) are generally less effective in treating neuropathic pain than the tricyclic antidepressants.
- Use of strong opioids, such as oxycodone, morphine, fentanyl and methadone may provide some benefit in treatment of SCI neuropathic pain but is still somewhat controversial because of the generally poor long term response and problems with dose escalation, dependence and side effects.
- Of all types of SCI pain, neuropathic pain remains the least well understood and provides the greatest challenge to treatment, with only one in three people experiencing greater than fifty per cent reduction in pain with biomedical interventions.

Opioids
- A number of opioids are available as slow release preparation.
- Tramadol may be considered as a first step if simple analgesics fail to provide sufficient relief.
- A number of other slow release opioids such as buprenorphine, morphine, oxycodone, methadone and fentanyl patches are also available and can be considered depending on the individual situation.
- Tolerance and dose escalation and side effects such as constipation are always a concern in the patient using long-term opioids.
- Referral to a Pain Clinic may be desirable if long term administration of opioids is being considered. This needs to be discussed early with the patient and clear limits need to be established and agreed upon when prescribing. Addressing psychological factors is also important so that opioids are being used for analgesia rather than the treatment of distress

As a guide the following analgesic “ladder” (Table) may be used for treatment of pain following spinal cord injury

Other treatments
- A number of other drugs and techniques have been used with varying degrees of success
- Subcutaneous or intravenous infusion of local anaesthetics such as lignocaine may be helpful for the treatment of neuropathic pain in the acute setting or as a diagnostic procedure. If successful, however, a satisfactory oral equivalent does not exist. Medications such as oral mexiletine may be tried because of its relationship to the local anaesthetics, but unfortunately is often not very effective. Like the anticonvulsants, local anaesthetics probably act by dampening central aberrant neuronal activity.
- Intrathecal administration of baclofen is effective in patients with poorly controlled spasms and spasm-related pain. Intrathecal administration of clonidine and morphine via an implanted pump has been demonstrated to be effective in some people with neuropathic pain following SCI. Intrathecal drug administration may be an alternative if patients have severe pain or spasm that fails to respond to other approaches, but only after psychosocial factors have been fully assessed and appropriately managed.
- Ablative surgical procedures generally have a very limited role in management, apart from DREZ lesions (performed by radio frequency or laser coagulation) for treatment of at-level neuropathic pain.

Physical Management
- Physiotherapy and occupational therapy interventions may be necessary to improve fitness,

posture and overuse syndromes, in particular. Physical treatments including exercise and hydrotherapy programs, postural re-education, retraining activities of daily living such as transfers and mobility, wheelchair and seating adjustments, modifying lifestyle and possible other physical modalities are often helpful in managing pain resulting from a mechanical cause. Assessment by an experienced physiotherapist and/or occupational therapist is recommended as part of a team approach.

Psychosocial Management

- Significant psychological co-morbidities (such as psychiatric diagnosis, traumatic brain injury or drug and alcohol dependence) are likely to interfere with the optimal management of pain and therefore require separate assessment and management, and must be included in the treatment plan. Detection of general adjustment issues relating to the injury, rather than exclusively to the pain, may indicate that the outcome of pain treatment will be compromised if this is not addressed. Thus, referral to appropriate support services for further assessment and management is warranted in this situation. Social factors may also need to be addressed. This may include education of significant others regarding management of the pain or referral for relationship work. Specific cognitive behavioural pain management interventions have been shown to offer benefit to those participating in relation to mood, interference with daily activities and catastrophic thinking (Nicholson Perry et al., submitted; Norrbrink Budh et al., 006). Although there has been no investigation of these interventions delivered on an individual basis, they could be adapted for use in the context of individual treatment (refer to table 4).

Self-management Strategies

- Strategies that health professionals can suggest to clients that will contribute to reducing their pain-related disability and distress include:

• Maintaining a regular pattern of activity despite the pain, rather than falling into a 'boom and bust' cycle which depends on pain levels

• Breaking activities into manageable chunks and planning ahead for regular rest breaks, rather than pushing on until pain becomes unbearable

• Planning ahead and prioritising activities so that the person with SCI can achieve the things that are most valuable to them – not forgetting to prioritise enjoyable activities!

• Establishing a regular pattern of medication use, rather than only taking it when pain levels become high

• Developing a plan for dealing with days when the pain is worse, which can be shared with family and carers so that they can remind the person with SCI about what they were planning to do

• Trying not to panic! In contrast to acute pain conditions, most persistent pain in the context of a spinal cord injury is NOT an indication that there is anything wrong, but is more likely to reflect central nervous system changes

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Question 13 – non compulsory

Bone pain is common in patients with breast cancer. Discuss the management of this.

Causes of bone pain in breast cancer

1) Bony metastases causing bone pain
2) Pathologic fractures of long bones
3) Pathologic fractures of vertebrae
4) Osteoporosis related fractures exacerbated by steroid use and co-existing loss of nutritional state
5) GCSF can cause a temporary bone pain syndrome in some patients
6) Exacerbating factors
    a. Hypercalcaemia
    b. Opioid tolerance
    c. Psychosocial factors and coping style
    d. Inactivity causing bone mineral loss
    e. Radiotherapy causing mineral loss

Prognosis

In breast cancer with bony metastases median survival is 12 months. It is longer in patients with hormonally responsive disease and with disease confined to pelvis and lumbar spine.

Metastatic bone pain pathophysiology

Caused by osteolytic and osteoblastic overactivity and loss of bone architecture. This can lead to pathological fractures. Nociceptive pain and neuropathic pain caused by inflammatory activity due to macrophage activation. Pain experienced secondary to tumor infiltration may be caused by the destruction of normal afferent sensory fibers and responds to gabapentin in animal models. NGF antibodies show promise in animal models for the same reason.

Practical issues

General pain assessment including psychosocial factors

Cancer staging and prognosis assessment and liason between specialities re planned treatment.

Imaging for assessment – all the following have a role in assessment of bone disease. PET scanning is the most sensitive test for metastatic disease overall but MRI is more sensitive and specific for bone metastases than nuclear medicine techniques.

1) X ray
2) Bone Scan
3) CT
4) MRI

5) PET

Treatment should be directed at cancer regression, relief of symptoms, and preservation of function. Impaired neurologic function and pathological fractures can complicate the presentation and the pain type.

General

Supportive – eg nutrition and general pain management measures eg treat co-existing depression. Hydration in hypercalcaemia.

Physiotherapy and Psychology

Encourage mobilization if safe to do so and reduce fear avoidance and catastrophizing. Co –existing end of life problems need addressing.

Pharmacological

COX -2 selective NSAIDS – some evidence for reduction of tumour activity in animals and probable short term advantages in terms of side effect risk over usual NSAIDS – example celecoxib 200mg per day

Corticosteroids – inhibition of cytokine activity with additional possible suppression of hormone dependent cancer activity – EG Dexamethasone 12-24 mg per day

Bisphosphonates – good evidence for their use to reduce skeletal events. Evidence for zolidronic acid over pamidronate. Also used for hypercalcaemia . Flu like symptoms are common side effects and renal impaired patients need dose reduction

Calcitonin can help although evidence is conflicting

Opioids are a standard treatment but dose should be minimized by other techniques to reduce side effects and hyperalgesic phenomena which may be myoclonus or possibly clinical tolerance .

There is also animal data which suggests bone metastases could actually progress due to opioid use.

Adjuvant anticonvulsants – bone invasion strongly correlates with peripheral nerve fibre involvement and pain is likely to respond to these agents.

Ketamine is a standard analgesic and has been studied in animal models with demonstrated efficacy.

Hormonal therapy

Oestrogen and oestrogen analogues control pain in hormone dependant breast cancer with bone metastases.

Radiation therapy

Strontium 89 Used in patients with widespread bony metastases with multifocal pain.

Radiotherapy – usually approximately 30Gy over 2weeks but varies due to prior history and other factors eg general condition and life expectancy. Majority of patients will obtain pain relief if

indicated both for single site and multifocal disease. Important to realize that only 20-30 % of response is seen within the first 4 weeks and maximum response is seen after 12 weeks.

Surgery and procedural

Vertebroplasty for prevention of fracture and treatment of fracture related pain.

Intralesional injection of steroids ,for example in rib mets has been shown to help some patients

Surgical stabilization : Prevention of pain from fractures by prophylactic stabilization and splinting

Chemotherapy

Bone disease more common in elderly women with hormonally responsive tumours. Younger women with non hormonal responsive disease have more response to chemotherapy but less likely to have bone disease . However in widespread disease a palliative effect has been demonstrated.

Question 14 – non compulsory

Discuss the differential diagnosis and management of Medication Overuse Headache (MOH).

BACKGROUND

Synonyms

analgesic abuse headache, medication misuse headache, drug-induced head- ache; rebound headache

Definition

Medication overuse headache (MOH) is the escalation and worsening of primary headaches associated with an increased use of drugs intended to provide symptomatic relief.

Epidemiology

Global Burden of Disease: tension-type headache and migraine are the second and third most prevalent disabling conditions.

MOH has become a chronic headache of major and growing worldwide importance and perhaps the most important cause of chronic daily headache (CDH). Approximately half of people with headache on 15 or more days per month for more than 3 months have MOH.

Population-based studies show that 11% to 70% of people with frequent headache take acute symptomatic medications almost daily. Medication overuse contributes to chronification of headache by paradoxically aggravating pain (MOH) and a cycle of intensified analgesic use, worsening pain, and long-term disability.

About 50% of people with headache are estimated to be primarily self-treating, without contact with health professionals. Of those who access medical care the most common diagnoses are migraine, tension-type headche or some combination of these. (Other primary headaches, such as 3.3 Chronic cluster headache or 4.10 New daily persistent headache seem less likely to lead to this situation.) In primary and neurology care about 6% and 9% respectively of headache patients present with MOH. Some MOH is related to secondary headaches.

Aetiology

Medication-overuse headache is an interaction between a therapeutic agent used excessively and a susceptible patient.

MOH can be caused by most, if not all, acute headache drug therapies. Takes time to develop: the frequency of drug use is more potent in developing MOH than absolute amount of analgesic taken.

Smoking and inactivity more than doubled the risk of MOH but not CDH without medication overuse.

Prospective study: Triptans: 1.7y to develop MOH ; ergot: 2.7y; analgesics: 4.8y

Doses: triptans 18/month; ergot 37/month; analgesics 114/month

Diagnosis (from ICHD-3)

8.2 Medication overuse headache

Patients with a pre-existing primary headache who, in association with medication overuse, develop a new type of headache or a marked worsening of their pre- existing headache that, in either case, meets the criteria for 8.2 Medication-overuse headache (or one of its sub- types), should be given both this diagnosis and the diagnosis of the pre-existing headache.

Description:

Headache occurring on 15 or more days per month developing as a consequence of regular overuse of acute or symptomatic headache medication (on 10 or more, or 15 or more days per month, depending on the medication) for more than 3 months.

Diagnostic criteria:

A. Headache occurring on 15 days per month in a patient with a pre-existing headache disorder

B. Regular overuse for >3 months of one or more drugs that can be taken for acute and/or symptomatic treatment of headache1

C. Not better accounted for by another ICHD-3 diagnosis.

[Subdivided into:
8.2.1 Ergotamine-overuse headache
8.2.2 Triptan-overuse headache
8.2.3 Simple analgesic-overuse headache
  8.2.3.1 Paracetamol (acetaminophen)-overuse headache
  8.2.3.2 Acetylsalicylic acid-overuse headache
  8.2.3.3 Other non-steroidal anti-inflammatory drug (NSAID)-overuse headache
8.2.4 Opioid-overuse headache
8.2.5 Combination-analgesic-overuse headache
8.2.6 Medication-overuse headache attributed to multiple drug classes not individually overused
8.2.7 Medication-overuse headache attributed to unverified overuse of multiple drug classes
8.2.8 Medication-overuse headache attributed to other medication]

Differential diagnosis

- Primary headaches:
  - 1. Migraine 2. Tension-type headache 3. Trigeminal autonomic cephalalgias 4. Other primary headache disorders
  - NB Dual diagnosis usual, eg migraine *plus* MOH
- Headache attributed to occasional use of non-headache medication (8.1.10)
- Headache attributed to long-term use of non-headache medication (8.1.11)
- Headache attributed to substance withdrawal (8.3)
  - 8.3.1 Caffeine-withdrawal headache
    - Headache developing within 24 hours after regular consumption of caffeine in excess of 200 mg/day for more than 2 weeks, which has been interrupted. It resolves spontaneously within 7 days in the absence of further consumption.
  - 8.3.2 Opioid-withdrawal headache

- Headache developing within 24 hours after daily consumption of opioid(s) for more than 3 months, which has been interrupted. It resolves spontaneously within 7 days in the absence of further consumption.

Management

- **First consider the diagnosis in order to best manage it**
- Majority of patients with MOH improve after discontinuation of the over-used medication
- Simple explanation of causes and consequences of MOH is an essential part of its management. An explanatory brochure is often all that is necessary to prevent or discontinue medication overuse.
- *Complete withdrawal of the responsible drug is needed*: most specialists believe it should be done acutely but with covering medication to ameliorate withdrawal symptoms.
- Duration and severity of headache and accompanying symptoms on withdrawal depend on the type of overused headache drug and appears to be shorter in patients overusing triptans than in those overusing ergots or analgesics.
- [Inpatient detoxification may be advisable for patients who use tranquillisers, opioids or barbiturates, especially in large daily doses]
- Relapse occurs within months or years in a relatively high proportion of patients. Relapse rate is lower for individuals overusing triptans rather than analgesics.
- Having controlled the headache, address associated factors including physical inactivity (physical reconditioning and recreational exercice such as Yoga tai Chi, walking, hydrotherapy), and underlying psychological and behavioural factors, (including cognitive behavioural therapies) to reduce relapse.

Prevention

- Targeted restriction of analgesic use for acute therapy for headache (long-term use of analgesics on more than 2 days a week and no more than 12 triptan doses a month
- Prevention is especially important in patients prone to frequent headache.
- Responsiveness to preventative treatment improves after discontinuation of overused medication.
- For migraineurs who develop MOH the general belief is that migraine prophylactic drugs do not work in an overuse situation.
- Migraineurs should avoid codeine and caffeine medication (or combination analgesics) when possible.
- Some recent data suggest that topiramate at least is of some benefit.

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Question 15 – non compulsory

Describe the assessment and management of painful persistent peripheral neuropathy.

Definitions

- Neuropathy: A disturbance of function or pathological change in a nerve: in one nerve, mononeuropathy; in several nerves, mononeuropathy multiplex; if diffuse and bilateral, polyneuropathy.
- Peripheral neuropathic pain: Pain caused by a lesion or disease of the peripheral somatosensory nervous system.

Introductory comments

Peripheral neuropathies:

- Are common (2-3% community) and increase with age
- Challenging to assess and manage
- Most common underlying disease is Diabetes Mellitus (30%)
- Are idiopathic in 30 – 50%
- Broadly classified as acquired or hereditary
- Potential causes of a ***painful*** peripheral neuropathy include
    - Metabolic e.g. diabetes mellitus
    - Idiopathic (most common)
    - Infective e.g. HIV
    - Inherited e.g. Hereditary sensory neuropathy, Fabry's disease
    - Toxic e.g. antiretrovirals, antineoplastic agents, alcohol
    - Injury e.g. compression from intervertebral disc
    - Autoimmune e.g. Sjogren's disease, Lupus, Vasculitic neuropathy
    - Other e.g. Amyloidosis

Any persistent pain can broadly disturb a person's function therefore an assessment needs to encompass physical, psychological, social and existential domains.

1. Assessment

Due the large list of potential causes the laboratory workup is best guided by the clinical pattern. Involvement of a neurologist in this process should be considered.

History - Key priorities include:

i. Consider current criteria for diagnosis of neuropathic pain
ii. Establish clinical pattern
    - Process acute, subacute or chronic
    - Affects motor, sensory, autonomic or combination
    - Purely sensory, only small fibres, large fibres or both
    - Disease predominantly distal or proximal
    - Symmetric or asymmetric
    - Purely lower motor neuron or upper motor neurone findings
iii. Attempt to identify underlying causative disease or lesion
iv. Assess the functional limitations that result from pain.
v. Assess physical, psychological, social and existential impact on the person

Examination

A clinical examination is necessary to guide more detailed testing.

i. Examine the pattern (e.g. mono, poly, symmetrical neuropathy) and features associated with the neuropathy (small fibre, large fibre, motor involvement, sensory loss and gain).
ii. Relevant examination for other potential causes
iii. For unilateral neuropathies compare with contralateral area.

Of note, allodynia can be found also in nociceptive pain, and so the diagnosis of neuropathic pain cannot be made purely on the basis of allodynia.

Investigations (based on clinical pattern)

i. Questionnaires e.g. Pain Detect, LANNS, DN4, NPQ, ID Pain.
ii. Neurophysiological testing e.g. small-fibres (QST, autonomic testing), large-fibres (NCS, EMG)
iii. Blood tests based on suspected diseases e.g. Glucose testing (Diabetes), Vitamin levels B_{12}, viral markers (HIV, Hep B & C) serum α-galactosidase level (Fabry's), tumour markers (paraneoplastic syndrome), autoimmune markers etc
iv. Other e.g. radiological imaging (CXR – paraneoplastic, MRI spine in radiculopathies), CSF analysis, sural nerve biopsy, intra-epidermal nerve fibre density (if available) etc

2. Management

This should include a 'whole person' approach. Education about the condition should be one of the primary goals. It is important to focus on strategies and treatments aimed at achieving long-term patient-relevant outcomes. For the majority of patients no one intervention is likely to achieve satisfactory long-term outcomes.

Social considerations (social support network, work & litigation)

Are there any specific interventions that could be considered? For example, workplace modification, referral to marital therapy or assisted housing.

Psychological considerations

Does the history and mental state examination indicate any psychological or psychiatric contributors to the patient's pain, disability or distress? If so, these should be managed based on assessment. Management may include pharmacological therapy (antidepressant therapy), arousal reduction strategies, psychological therapy including self-management training e.g. cognitive behavioural therapy or more formal mental health management.

Biological considerations

i. *Disease specific treatment* or *management* is a key component of any plan e.g. stabilisation of diabetes, cessation of alcohol, dietary supplementation (vit B12 deficiency), management of cancer etc
ii. Medications:
    - Numerous reviews of this area have been published (e.g. (5, 6)).
    - The choice of the medication will depend on the condition being treated. Any medication trial needs to be monitored and assessed.
    - Several medication classes may be considered including: anticonvulsants (e.g gabenoids), antidepressants (e.g. tricyclic antidepressants, SSRI, SNRI), Tramadol, Tapentadol and other medications with lower evidence including topical capsaicin and lignocaine.
iii. Interventions: Play a limited role in this patient group. Peripheral nerve injections with local anaesthesia and steroid may assist with diagnosis but are short-lived in their therapeutic

effect, destructive lesioning should be avoided neuromodulation is typically less effective in the presence of neural sensory loss.

iv. Movement therapy: This may include an exercise plan and graded upgrading with the assistance of a physiotherapist. Care and regular inspection of insensate skin.

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