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

FELLOWSHIP EXAMINATION HELD ON 13th then 28th NOVEMBER 2015

at regional centres (13 Nov 2015) then at the Pullman Hotel, Albert Park, Victoria

THIS REPORT IS PREPARED TO PROVIDE CANDIDATES AND SUPERVISORS OF TRAINING WITH INFORMATION ABOUT THIS FELLOWSHIP 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 SUPERVISORS SO THAT THEY MAY PREPARE APPROPRIATELY FOR FUTURE EXAMINATIONS.

The fellowship examination is an integral part of the pain medicine training program, leading to the award of Fellowship of the Faculty of Pain Medicine.

The 'Pain Medicine Training Program 2015 Curriculum' details the range of content which may be assessed.

The fellowship examination consists of written and clinical sections and covers the theory and practice of pain medicine.

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 then made to reduce the likelihood of a candidate being examined by an examiner to whom he/she is known.

The new initiative to use a function centre instead of a hospital as the venue for the clinical examinations presented some logistical challenges to the organisers. Of course, every effort was made to see that the 2015 fellowship examination was conducted to the standard well-established over the 16 years of Faculty of Pain Medicine examinations. This new process will continue to be developed in 2016 as the Faculty continues to strive for excellence in its conduct of the fellowship examinations.

This was the first year in which the long case assessment was not an integral component of the fellowship examination, having been devolved to regional centres as a part of the redesign of the curriculum and assessment processes. The clinical component now takes the form of 4 structured oral viva voce (SOV) as in the past, as well as 4 observed structured clinical examinations (OSCE). Further details of these is available in the 'Structured Oral Viva Voce (SOV) and Observed Structured Clinical Examination (OSCE) guidelines' document prepared by the Examinations Committee during 2015. The traditional, patient-based short cases have been removed, although actors were used as subjects for both communication and physical assessment in the clinical examination.

In order to be successful, a candidate was required to attain a mark of 50% or greater **in each** of the written and clinical sections of the examination.

The 2015 FPM fellowship examination was formally observed by external observer Dr Lisa Lampe, Chairman of Examinations, RANZCP.

1. EXAMINATION OVERALL PASS RATE 75%

This year, 28 candidates presented for the examination and 21 were successful.

2. WRITTEN SECTION PASS RATE 22/28 78.5%

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…"

For the first time in 2015, candidates were required to answer all ten compulsory questions.

General comments from the examiners:

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 expected to be specialists in the area and know more than the referrer. **It remains apparent that trainees are not digesting the content of recent journals.**

Familiarity with the subject areas detailed in the published 'Pain Medicine Training Program 2015 Curriculum' proved advantageous to the candidates.

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 **PASS RATE 26/28 92.8%**

What is the rationale, benefits and risks of exercise for chronic pain patients?

Majority of the candidates did well on this question. Half of the candidates managed to address all important facts on the question, i.e. chronic pain, exercise and the subdivided titles (rationale, benefits and risks).

It is an easy to pass question if the candidate showed good explanation of the facts with succinct points. An introduction does help. Good candidates are those who gave an introduction on how chronic pain and exercise are linked. Only one candidate defined chronic pain. Most people forgot about chronic pain until they quoted fibromyalgia etc. Good candidates also showed good understanding on their answers. The less desirable answers only included points without explanation; this generic approach did not show proper understanding of the question.

Question 2 **PASS RATE 18/28 64.2%**

The presence of renal or hepatic impairment in a patient may affect the choice and/or dose of medications prescribed.

Summarise how renal or hepatic impairment might influence your choice and/or the dose of medications used for pain management. Include both opioid and non-opioid drugs in your answer.

1. Answers are generally adequate. Good answers are based on sound knowledge, rational pharmacological principles, and attention to detail.
2. The question asked 'how renal or hepatic impairment might influence your choice ……' and yet the 'how' was often not answered. It is inadequate to simply give a list of drugs together with comment made that dose needed to be reduced, without saying how/why. There is also a tendency to use generic phrases such as "caution", "risk-benefit balance" without context-specific details.
3. Common mistakes: Metabolite levels of drugs do not increase with liver impairment. Pregabalin and Gabapentin are renally excreted. With renal impairment doses are adjusted according to the patient's GFR. Tramadol and Codeine are often missed in the discussion altogether.

Candidates are advised to be focused in answering the question. Common irrelevant points include pharmacogenetic variations in P450 cytochromes, pathophysiology of hepatic and renal failure

Question 3 **PASS RATE 19/28 67.8%**

How would you assess pain in a patient with significant dementia?

Detail the management issues.

Overall, this question was answered poorly. Many candidates did not mention that self-report of the pain scores remains the gold standard and is possible in patients, depending on the degree of impairment. There was generally inadequate discussion of the pain scales and the various components. Many answers also included irrelevant details which take up precious time.

Question 4 **PASS RATE 25/28 89.2%**

A 14 year old girl presents with a 2 year history of daily headaches not relieved by paracetamol or ibuprofen. She is missing at least 10 days a month from school and her academic grades are falling. Her father died 3 years ago from a brain tumour.

a. **How would you differentiate the possible causes?**
b. **What treatment modalities would you recommend, indicating the advantages and disadvantages of each for a patient of this age?**

This question is about a common presentation to pain clinics (adult and child/adolescent) with a patient with a multifactorial chronic daily headache (CDH) with significant psychosocial issues and functional impairment. The marks awarded varied from 2 to 8/10. A couple of candidates answered the first part only with limited detail provided for the second component of the question. A couple of candidates presented an answer relevant to a generic (adult) patient without highlighting the issues pertaining to an adolescent. Candidates did not demonstrate an understanding of the evidence base of treatments for CDH particularly the benefit of psychological interventions and some neglected to mention any advantages or disadvantages of therapies.

The following achieved marks within the 2 parts of the question

Part A: Establishing chronic (2 year) daily headache differential diagnosis through history, examination, investigation and therapy review. Identifying and ruling out red flags even though long history. Obtaining collateral history – mother and school. Family history and associations with headache. Acknowledgment of psychosocial contribution and particularly paternal history/death/post-traumatic stress and current impacts on child/mother and extended family and if coping. Associated psychopathology and Risk. If abuse or neglect (including through grief) are present. Identifying important issues specific to a teenager (vs an adult presentation) – school (bullying) - social-academic – sport – maternal attachment/interaction/coping/modelling – impact on siblings.

Part B: Combination intervention desirable (either as single practitioners vs multidisciplinary). Simple interventions relevant to a teenager e.g. lifestyle modification/sleep hygiene/graded return to school/ headache diary. Presentation of the multiple effective non-pharmacological interventions that are available. Various pharmacological interventions and reasoning behind choice for this child including comment on Advantages and Disadvantages (Table or column presentation ideal). Bonuses for evidence comment (acknowledging adult vs adolescent data).

Question 5 **PASS RATE 21/28 75%**

Discuss the role of immunocompetent cells underpinning chronic pain mechanisms.

There were some very good answers with quite a few candidates showing a very strong grasp of the area. However, many candidates were not able to integrate the mechanisms of glial responses to the development of persistent pain and many relied on the usual general neurobiological mechanisms involving NMDA receptors and central sensitisation without much detail regarding the specifics of glial cell mechanisms.

There was a reasonable understanding of the types and function of glial cells and immune mechanisms in opioid tolerance and neuropathic pain. Potential therapeutic strategies were only glossed over for most candidates. Several candidates made little reference to glial cells at all suggesting they had missed the thrust of the question entirely.

Many of the candidates' writing were illegible and it was difficult to award marks in this situation.

Question 6 **PASS RATE 24/28 85.7%**

A 67 year old man presents to your clinic with pain in his feet in a stocking distribution. He has a BMI of 34.

a. **What is your differential diagnosis?**
b. **Detail your approach to his management.**

This is the third year in a row that this question has been asked. Peripheral neuropathy is core knowledge about which a good understanding is required.

Most people did not read the question correctly. It required a differential diagnosis of the pain then a differential diagnosis of peripheral neuropathy (the leading DDx of the pain). Most did one or other not both.

As it asked for management, candidates spent too much time detailing the history and examination findings they would expect or look for. Little marks were allocated for this.

Comprehensive multimodal management was done well by a subset. Detailed pharmacotherapy also done well by a subset.

Obscure detail like confocal corneal microscopy does not gain you marks when important basics are left out instead.

It was clear that many candidates had no extensive experience of actually treating these patients longitudinally.

Overall this question was done better than in past years.

Question 7 **PASS RATE 19/28 67.8%**

Describe the rationale for intrathecal analgesia in the management of pain in the patient with cancer. Briefly discuss your clinical approach to its use.

It was expected that a broad perspective could be demonstrated, as this was specifically a question about cancer pain and referred directly to ETA 6. Those who made reference to chronic pain received no marks and one person mentioned acute pain but unfortunately failed to recognize that intrathecal techniques may and are, employed to manage acute pain in the patient with cancer, particularly after major surgery. Candidates tended to produce a list of contraindications as if this were a patient undergoing a spinal anaesthetic and many spent too much time in the detail of consent. For those who talked at length about coagulopathy being a contraindication to the utilization of intrathecal analgesia techniques in the patient with cancer pain – this is relative. It is also assumed that all invasive procedures will be undertaken in an appropriate environment, using aspetic technique and by individuals who have adequate experience and skill in their conduct. Candidates should take care when converting opioid doses and I would suggest down-loading the FPM opioid calculator to your smart-phone as a constant reference source.

It was expected that candidates would understand and comment upon the rationale for intrathecal analgesia in the cancer patient being to alleviate pain usually, where more conservative approaches to analgesia have failed; to mitigate against intolerable side-effects including opioid-induced hyperalgesia which may be complicating the patient's analgesic needs from the administration of systemic opioids/morphine; to provide better analgesia at both rest and with movement (to allow for nursing care, especially in the terminal phases) and quality of life (or as one candidate put it, "quality of death"). No candidate mentioned improvement in life expectancy or that aggressive anti-tumour therapy may be better tolerated. Many candidates eluded to cost and spent significant time discussing the roles of exteriorized versus implanted systems for drug delivery in patients with differing prognoses, but none discussed that this could be cost-saving by allowing for patient discharge to the community.

A few candidates discussed intrathecal neurolysis and one candidate talked about intracerebroventricular drug delivery. Some candidates mentioned education of patients / carers / nursing / medical staff in the care of the various systems employed. Most candidates talked about different drugs and combinations that could be used and one even provided a detailed list of combinations but failed to say that these combinations are not recommended in implanted systems due to uncertainty about the effects on the pump mechanism.

The marking reflected the lack of appreciation of a number of candidates to come to grips with the fact that a sociopsychobiomedical approach to the patient was required. This involves consideration of the social milieu of the patient, the patient's psychological state – a couple of candidates wrote that mental illness precludes this technique in the patient with cancer related pain - and, the cancer itself. There are many cancers and multiple phases of the disease process (diagnosis, treatment, palliation and terminal phases +/- survivorship) and no candidate managed to describe these and the different issues that influence the consideration of intrathecal analgesic techniques at the different times.

Question 8 **PASS RATE 20/28 71.4%**

Discuss the evidence based techniques available to minimise the transition of acute to persistent post-surgical pain.

Most candidates offered a definition of persistent post-surgical pain and a list of risk factors although these were not specifically required. Poor answers provided limited or no discussion of preoperative assessment and management of psychosocial factors, surgical techniques or optimal management of preoperative pain. Central neuraxial blockade was well covered but discussion of peripheral nerve blockade and catheter techniques as well as wound infiltration was limited or omitted. The use of various medications formed the majority of answers; however, the number of named medications was limited mostly to vitamin C, pregabalin and ketamine. Mechanisms of action of each were not required. The discussion of the use of opioids, the mainstay of acute pain management, was surprisingly limited. A few answers included "multimodal analgesia" without defining the term or mentioning which medications would be included. Better answers included discussion of the evidence base for each technique.

Question 9 **PASS RATE 15/28 53.5%**

The current DSM criteria (DSM-5, 2013) for substance-related and addictive disorders no longer use the terms dependence and abuse. Discuss the criteria required for the new term *substance use disorder* and relate this to patients in a pain clinic.

This topic is covered in ETA 3.4 and in a Pain: Clinical Update, 2013 (see reference list). In addition, several learning objectives from ETA 3.4 directly relate to this question (see below). In addition, the quiz contained in ETA 3.4 directly assesses elements of this question.

Many candidates provided generic detail rather than specifically answering the question. The heart of the question (discuss how DSM V relates to patients with pain) was not addressed well overall.
Selected learning outcomes from ETA 3.4

3.4.2 Critically discuss the differences in understanding and use of these terms (tolerance, physical dependence, psychological dependence, problematic substance use & addiction/dependence) between the disciplines of pain medicine and addiction medicine

3.4.4 Describe the impact of the following non-prescription substances on health and pain experience: caffeine, nicotine, alcohol, cannabis, methamphetamine and other stimulants

3.4.6 Discuss the current DSM criteria for diagnosis of substance use disorder

3.4.8 Recognise the different forms of substance abuse that may be co-morbid with the experience of chronic pain

One other comment I think worth making is that most candidates did not distinguish between "discuss" and "describe". That is a very basic examination requirement. There were some very good answers listing the criteria of DSM V for SUD (i.e. describing), but they did not answer the question "discuss changes from DSM 4". Need to read carefully, and answer the actual question.

Question 10 **PASS RATE 19/28 67.8%**

Outline the presentation and pathogenesis of cancer-related mucositis.

Describe your approach to managing the pain of this condition.

The question was answered poorly by many candidates, with a lack of structure and core knowledge. Many answers gave the impression that candidates had not been exposed to these patients in their training.

There was a limited knowledge of analgesia options including the use of opioids as mainstay and mucosal topical agents.

Most candidates did not comment that children and adolescents are a special group affected by this condition.

We suggest candidates revise the acute-cancer pain and cancer-related mucositis sections in the upcoming *Acute Pain Management Scientific Evidence 4th edition,* and gain exposure to managing patients with mucositis via an acute pain service or haematology/oncology ward.

3. VIVA VOCE SECTION PASS RATE 25/28 89.2%

The oral examination now takes the form of 4 structured oral viva voce (SOV) assessments as in the past, as well as 4 observed structured clinical examinations (OSCE). Further detail of these is available in the 'Structured Oral Viva Voce (SOV) and Observed Structured Clinical Examination (OSCE) guidelines' document prepared by the Examinations Committee during 2015.

General information:

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

3.1 STRUCTURED ORAL VIVA VOCE (SOV) **PASS RATE 26/28 92.8 %**

SOV 1 **PASS RATE 27/28 96.4%**

Mr G is a 39 year old single man who experienced a left middle cerebral artery infarct 6 months ago. This was thrombolised. He has had good motor recovery but has a persistent expressive dysphasia. He has pain on the right side of his face, his right arm and right leg. This is your first consultation with him.

The structure of the station was logical and flowed well during the exam. Several candidates seemed to struggle with some core knowledge but most were able to gain solid marks quoting the Finnerup et al paper from the Lancet as one would expect from such an authoritative source.

SOV 2 **PASS RATE 23/28 82.1%**

A 12-year-old boy, the oldest son in a close-knit Greek family, fell from a tree at his friend's house and suffered a mid-shaft fracture of his right forearm.

He underwent an open reduction and internal fixation of the fracture which was complicated by compartment syndrome and intense postoperative pain.

Three months later, he was unable to move his hand and forearm due to severe pain and stiffness.

Question: What is your differential diagnosis?

This question was reasonably well done. Most candidates were able to generate a reasonable differential diagnosis and discuss possible pre-disposing factors for CRPS. The management of the difficult communication situation presented a challenge for some. Discussion of the role of regional blockade in CRPS was quite variable with only a few candidates providing a comprehensive risk-benefit analysis addressing this particular setting. Very few candidates were able to approach the medico-legal situation succinctly. Those who understood the basic requirements of the process scored very well. This is an area that is not well covered in many basic medical or specialist training programs; however, it is an important skill for specialist pain medicine physicians practicing in the Australian environment. Examiners were cognisant of different practices in other jurisdictions. Candidates are directed to e-Learning module 8 (CRPS) for an introduction to this topic. Candidates who scored highly were able to target their answers to the specific situation, indicating their understanding of the differences between adult and adolescent presentations of CRPS.

SOV 3 **PASS RATE 24/28 85.7%**

The public has access without prescription to low dose codeine and paracetamol / NSAID analgesic combinations. You agree to give a talk about these medicines.

Before you begin work on your talk what further information would you like?

Candidates generally provided a pleasing approach to the scenario overall, yet it was concerning that there was still an inadequate knowledge of the specific issues pertaining to the OTC availability of an idiosyncratic substance. Particularly as this is matter of current socio-political focus, that aspect would have been expected to have been understood better by many candidates.

SOV 4 **PASS RATE 23/28 82.1%**

James is a 62 year old teacher with carcinoma of the prostate.

He had radical prostatectomy followed by radiotherapy 2 years ago.

Since the surgery he's had severe pelvic pain requiring morphine Slow Release 100 mg twice daily.

Over the past 2 weeks he's developed left-sided back, hip and flank pain, nausea and drowsiness.

He's been admitted to hospital and you are asked to help.

Many candidates did not tailor their answers to the scenario presented, and instead provided a generic treatment plan for acute-cancer pain.

Many seemed to confuse the concept of morphine toxicity in renal failure with opioid induced hyperalgesia/tolerance.

Some candidates failed to recognise basic dangers in prescribing certain analgesics in a drowsy patient with renal failure.

Some forgot hypercalcaemia as a cause of nausea and drowsiness.

Most candidates did not manage the patient from the standpoint of safety first— ABC, 02, consciousness, monitoring-this is basic medical student teaching.

Any discussion of patient treatment should start with consideration of safety.

Some candidates discussed providing multidisciplinary input such as acceptance and commitment therapy and psychology for acute cancer pain, but not ABC, oxygen etc.!

Most candidates discussed issues surrounding euthanasia and end of life *quite well.*

We suggest candidates revise the acute cancer pain sections in the upcoming *Acute Pain Management Scientific Evidence 4th edition,* and gain exposure to managing patients with acute-cancer pain in a ward or palliative care situation (including cancer-related mucositis, bone metastatic pain).

3.2 OBSERVED STRUCTURED CLINICAL EXAMINATIONS (OSCE) PASS RATE 26/28 92.8 %

OSCE 1 **PASS RATE 28/28 100%**

Ms. Michelle Longford (alternatively Mr Michael Langford) is a senior journalist from the large regional newspaper 'The West Victoria Courier Gazette'.

They have made an appointment with you to get some background information and comment on the government's decision to make marijuana available to patients.

The story is due to be run in the next week and distributed widely in regional newspapers in other jurisdictions. It will also be carried on the associated websites facebook pages and on twitter.

The journalist would like to know whether you will be providing it for the patients at the pain management unit. They also want to clarify some details around how effective it is and how it might be used.

A photo opportunity may be available also.

This station has been part of the exam sentence since it's over 15 years ago. The issue of aberrant opioids prescribing, consumption, demands etc has been a recurring theme and certainly one that any specialist pain medicine physician should feel comfortable with and skilful in dealing with.

More recently we have tried to expand the experiences with which a specialist pain medicine physician might be faced with. In the last three years we have had professional or at least semi-professional actors who are skilled in role-playing.

Three actors (two female one male) participated in this year's examination.

The issue of marijuana and its use in medicine has gained widespread attention in all forms of broadcast electronic and newsprint media. Various different professional medical and legal organisations have published position statements. The faculty of pain medicine has also formulated a position statement on this issue. Marijuana and its putative use in medicine has been the basis for altering the legislation around its use in many jurisdictions most recently in the United States and in Austria. The Victorian government here in Australia are currently drafting legislation around the provision of marijuana for medical purposes. The federal government have also indicated that it would probably move in this direction. Similar moves have been made in New Zealand and in other European countries.

It is not unusual for patients to ask us as specialist pain medicine physicians argue of the consumption of marijuana to treat the pain and suffering.

The structure of this year's actor station/communication station was for the candidate to be interviewed by a journalist from the local regional newspaper on this subject. It aimed to cover for specific areas.

1. An opening statement around the prevalence and epidemiology of persistent pain. This was an opportunity for the candidates to highlight the problem of persistent pain and its under-appreciation
2. The evidence for using marijuana for medicinal purposes in persistent pain, multiple sclerosis, cancer pain, and paediatric neurology (epilepsy). This aspect also covered the pharmacology delivery systems doses interactions etc. There was an expectation that the candidates might quote some of the recent literature from the International Association for the study of pain, *Nature etc*
3. The public health and perception aspect. The candidates were challenged by the journalist in suggesting that the side-effects were either overplayed or at worst very mild. Mention of the faculty's statement on medical use of marijuana was expected.
4. An overall impression of how a candidate (specialist pain medicine physician) might to deal with being asked probing questions by a journalist who may record the conversation. It was hoped that the candidates would be aware that there words and phrases could be manipulated to underpin a counterargument. The actor journalist did highlight the fact that the story would be distributed across a network of regional newspapers into regional broadcasting services and possibly picked up by social media. Finally a photograph opportunity to have the candidate in the same frame as a 'marijuana' plant was put up as a challenge.

This station was handled reasonably well.

OSCE 2 **PASS RATE 18/28 64.2%**

Bill is a 68 year old retired farmer. He has a longstanding history of mild hypertension and hyperlipidaemia, but no history of IHD or peripheral vascular disease. He is not on any regular medications. He has never smoked and drinks 40 standard drinks a week. He lives with his wife of 43 years and has 3 adult sons. Two of the sons live nearby in town, but his oldest son is still on the family farm. At the time of his initial presentation with pain, he was playing competition lawn bowls twice a week and walking his wife's terrier around 3km most days.

He consulted his GP with vague lumbar pain of moderate severity and short-lived but frequent radiating pain into his left leg below the knee but not into his foot. For twelve months prior to the consultation, he had found the back and leg pain was increasingly limiting his ability to socialise and play bowls.

The GP obtained some imaging and referred him to see you with a request to advise on management in the light of the findings. Bill has always been averse to any discussion of regular medication use.

It was surprising how many candidates declined to give an opinion on surgical suitability even when the question was repeated. The better candidates got right through the question and were able to be taken back to sections where they did not get full marks and they picked up some extra marks in most cases. Poor candidates missed marks by not giving specific enough answers to management questions or not reporting obvious x-ray findings such as the loss of lumbar lordosis or presence of scoliosis. The materials given to the candidates seemed to be fair and easily read.

OSCE 3 **PASS RATE 23/28 82.1%**

Musculoskeletal Examination of the Shoulder.

Please report your findings as you go.

We were impressed that most candidates had a good system of examination to run through. However some candidates seemed less certain of what they could find/ interpret when we interrupted to ask them what they were testing for? , and what would a certain reduction of range mean? , or what did a special test mean?

One question that was challenging was asking how to determine inconsistent findings, for example when a patient was fearful of movements, and guarding movement, (or potentially exaggerating restricted movement), could they suggest ways to evaluate shoulder range of movement?

The normal shoulder exam subjects worked well for the shoulder OSCE.

OSCE 4 **PASS RATE 24/28 85.7%**

These arterial blood gases were taken from a 69 year old male with chronic obstructive pulmonary disease who was receiving PCA fentanyl 16 hours after his laparotomy.
His respiratory rate was 16 breaths/min.
He was getting 4L/min supplemental oxygen via nasal specs.

pH 7.2
pCO2 63 mmHg
HCO3 24 mmol/L
PO2 120 mmHg

Question: What do you think of these results and the patient's respiratory status?

It is a reasonable topic and still not done very well by some. Many still struggle with basic blood gases as most still thought the initial results were, in part at least, due to the patient having chronic COPD. The results showed acute respiratory depression only and there was quite clearly no chronic component to the CO_2 retention.

4. OVERALL EXAMINATION COMMENTS

The written examination was again held before the vivas, three weeks prior on this occasion. Feedback in relation to this was essentially positive. Written questions were chosen from across the curriculum, and the vivas were carefully designed to ensure, as much as was possible, a conformity and diversity 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 the revised curriculum.

The Examinations Committee 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 a regard for the diligence of our process, and there was less feedback than in previous years to prompt concern regarding possible leniency on the part of the examiners in their assessment of candidates who are functioning sub optimally. Fortunately for some, the current structure often allows candidates the opportunity to redeem a poor performance through better function in other tasks within that same section of the examination.

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.

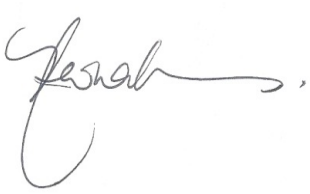
The Court of Examiners acknowledges the Observers for 2015: external observer Dr Lisa Lampe FRANZCP, and provisional examiners A/Prof Steven Faux, Dr Geoffrey Speldewinde, Dr Frank Thomas, Dr Jane Thomas, and Dr Aston Wan. Their valuable reflections on the examination processes has been very helpful and will assist in further improving our process.

I wish to thank all of our colleagues on the Examinations Committee, 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.

5. THE BARBARA WALKER PRIZE / CERTIFICATES OF MERIT

The Barbara Walker Prize for Excellence in the Pain Medicine Examination was awarded to Dr Charlotte Hill, FANZCA from NSW/NZ.

A Certificate of Merit was awarded to Dr Jacquelyn Nash, FANZCA (Vic).



NEWMAN L. HARRIS

Chairman
Court of Examiners

GRETA M. PALMER

Deputy Chair

April 2016

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APPENDIX A
2015 FPM WRITTEN EXAMINATION

Question 1

What is the rationale, benefits and risks of exercise for chronic pain patients?

RATIONALE

Most patients with chronic pain have excessive disability. The pain spiral is one in which many patients become increasing disabled with time. Patients become deconditioned quickly and joints muscles stiffen up and permanent stiffness results. Balance can also be adversely affected with falls being a common sequelae.

- Exercise can reduce pain and disability for a variety of chronic pain conditions (back pain, fibromyalgia, neck pain, osteoarthritis, etc.)
- Mechanism of exercise induced hypoalgesia (EIH) or exercised induced analgesia (EIA):
    - Neurochemical: implied mediators: endorphins, growth factors
    - direct stimulation of supraspinal inhibitory centres
    - exercise induced pain (see below) which in turn inhibit pain (diffuse noxious inhibitory control)
    - exercise induced hypertension with baroreceptor-analgesic reflexes
- Large amount of exercises can induce pain in normal subjects with raised intramuscular pressure, muscle damage and activation of stress response
- Some chronic pain conditions (e.g.: fibromyalgia / chronic fatigue / migraine) have abnormal endorphin regulations and benefit less from the EIH/ EIA, and sometimes have more pain after exercise and reduce their compliance with rehabilitation
- No one form of exercise, including aquatic, is consistently more effective than other. There is no evidence on the frequency, the intensity nor the duration of exercise. Thai Chi compared with Pilates for instance.
- While aerobic exercise (at > 70% of maximal aerobic capacity), and resistance exercise to a lesser extent, is associated with greater EIH, there is no “dose dependent” relationship
- Other benefits of exercise: weight loss, muscle strengthening, cardiopulmonary function with aerobic exercises, mood elevation (Depression and Anxiety)

Reference

Daenen L, Varkey E, Kellmann M, Nijs J. Exercise, not to exercise or how to exercise in patients with chronic pain? Applying science to practice. Clin. J. Pain. 2014 March.

EXERCISE

- Types of exercise – Active versus passive exercise
- Generalised aerobic or regional specific
- Regional specific exercises :
    - Isometric (against resistance)
    - eccentric or isometric (Isotonic)
- Exercise with or without supervision
- Land versus water based exercise
- Exercise prescription is based on individual’s capacity
    - Age and medical background
    - Disability and frailty
    - Ability in learning new techniques (e.g pelvic tilt)
    - Type of condition; eg mirror box therapy for phantom pain or CRSP
    - Accessibility

PAIN

Definition of Chronic pain
Types of Pain: Nociceptive or/and neuropathic

Different painful conditions would benefit from different types of exercise.
Example:

- Osteoarthritis of the knee and hip – weight offloading exercise would be more preferred, such as hydrotherapy
- Phantom limb pain – imaginary exercise with Mirror Box therapy
- Non-specific LBP – core muscle strengthening
- Generalised muscle deconditioning – simple walking exercise
- Geriatrics with poor balance and mobility issue – simple sit to stand exercise

RATIONALE

Pharmacological approach is not always beneficial in chronic pain management as there are other contributors to the pain. Secondary muscle deconditioning is not an uncommon contributor; together with sedentary lifestyle in some cases are highly associated with chronic pain presentation.

Exercise is a broad terminology. There are different types of exercises as aforementioned.

Exercise is generally cheap and non-drug. Exercise in a group settling has the advantage of socialisation to this marginalised group.

BENEFITS

Neck pain exercises from Cochrane

No evidence found in acute neck pain control.

For chronic pain, specific strengthening exercises as a part of routine practice for chronic neck pain, cervicogenic headache and radiculopathy may be beneficial. Strengthening and endurance exercises for the cervico-scapulothoracic and shoulder may be beneficial in reducing pain and function.

Chronic MSK pain from Cochrane

Supervised or individualized exercise therapy and self-management techniques may enhance exercise adherence.

A wide range of exercises - general aerobic, specific body-region exercises for strengthening and flexibility, continuing normal physical activities and increasing physical activity levels

Exercise for OA of the knee from Cochrane

There were 44 high quality trials showing “exercise reduced pain” immediately after treatment, 13 trials showing “exercise improved quality of life immediately after treatment”.

Pain reduction: the scale used was measured from 0 to 100; exercise reduced pain by an equivalent of 12 points. There high quality evidence showed land based exercise offers sustainable short term

knee pain reduction and improvement in QOL among people with knee OA for at least 2 to 6 months after cessation of formal treatment.

QOL improvement: the scale used was measured from 0 to 100; exercise improved QOL by an equivalent of 4 points.
There were 8 studies reported increased knee or LBP attributable to the exercise intervention, not actual serious adverse effects.

Individual delivered programs gave better result in pain reduction and improvement of physical function, compared to the clas-based programs or home based programs.

RISKS

- Patient subtypes do not get EIH / EIA. Patients with Whiplash and associated disorders who have PTSD get an aggravation of their pain, compared with WAD who do not have PTSD.
- Patients who have high fear avoidance or catastrophisers also can have an aggravation of their pain unless this is adequately addressed/ treated beforehand.
  Wrong prescription of exercise – for instance, individual with poor respiratory function should not be for hydrotherapy as it can increase risk of cardiac overload.
- Lack of pacing may result in unwanted anxiety development which may lower an individual's confidence in continuation of exercise. In people with significant comorbidities such as poor cardiac and respiratory function, lack of pacing may result in unwanted exacerbated medical event.
- Over enthusiastic therapist or inexperienced therapist who give the wrong intensity/ type of exercise.
- When establishing an ongoing exercise regime, the type chosen must be achievable and enjoyed by the patient.
- Importance of warm up and cool down exercises to prevent exercise induced injury.
  Balancing exercise in unsteady individuals needs close supervision.
  Cognitive impairment which may impair the understanding of the techniques of exercises and prevent learning of the techniques.
- Hyponatremia in health subjects – one study

References

1. Gross A, Kay TM, Paquin JP, Blanchette S, Lalonde P, Christie T, Dupont G, Graham N, Burnie SJ, Gelley G, Goldsmith CH, Forget M, Hoving JL, Brønfort G, Santaguida PL, Cervical Overview Group. Exercises for mechanical neck disorders. *Cochrane Database of Systematic Reviews* 2015, Issue 1. Art. No.: CD004250. DOI: 10.1002/14651858.CD004250.pub5.
2. Bronfort G, Aker PD, Evans R, Goldsmith CH, Nelson B, Vernon H. A randomized controlled clinical trial of rehabilitative exercise and chiropractic spinal manipulation for chronic neck pain. *Spine* 2001;**26**(7):788–99.
3. Chiu T, Huiu-Chan C, Cheing G. A randomized clinical trial of TENS and exercise for patients with chronic neck pain. *Clinical Rehabilitation* 2005;**19**:850–60.
4. Jordan JL, HoldenMA, Mason EEJ, Foster NE. Interventions to improve adherence to exercise for chronic musculoskeletal pain in adults.*CochraneDatabase of SystematicReviews* 2010, Issue 1.Art.No.:CD005956.DOI: 10.1002/14651858.CD005956.pub2.
5. Fransen M, McConnell S, Harmer AR, Van der Esch M, Simic M, Bennell KL. Exercise for osteoarthritis of the knee. *Cochrane Database of Systematic Reviews* 2015, Issue 1. Art. No.: CD004376. DOI: 10.1002/14651858.CD004376.pub3.
6. Ettinger WH, Burns R, Messier SP, Applegate W, Rejeski WJ, Morgan T, et al.A randomized trial comparing aerobic exercise and resistance exercise with a health education program in

older adults with knee osteoarthritis. The Fitness Arthritis and Seniors Trial (FAST). *JAMA* 1997; **277**:25–31.
7. Jenkinson CM, Doherty M, Avery AJ, Read A, Taylor MA, Sach TH, et al.Effects of dietary intervention and quadriceps strengthening exercises on pain and function in overweight people with knee pain: randomised controlled trial. *BMJ* 2009;**339**:b317

Question 2

The presence of renal or hepatic impairment in a patient may affect the choice and/or dose of medications prescribed.

Summarise how renal or hepatic impairment might influence your choice and/or the dose of medications used for pain management. Include both opioid and non-opioid drugs in your answer.

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 2) Hepatic impairment causes fewer problems but: a) Some drugs may have increased oral bio-availability due less first pass metabolism b) Some metabolised for clearance less rapidly $\rightarrow$ prolonged effects
Subtotal

Influence on drug/dose		
	Renal impairment	**Hepatic impairment**
Opioids, tramadol and tapentadol	*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) • methadone (unless impairment severe) • oxycodone (OK if mild-moderate renal impairment)[a] • tapentadol (OK if mild-moderate renal impairment) *Active metabolites so dose adjustment suggested or, sometimes in preference, use alternative agent:* • codeine (morphine) • hydromorphone (H3G) • morphine (M6G) • tramadol (M1) *Avoid:* • pethidine (nor-pethidine) • d-propoxyphene (nor-d-propoxyphene)	*No dose adjustment generally required*: [b] • alfentanil • fentanyl • morphine (decrease if oral administration) • buprenorphine • oxycodone *Dose adjustment may be needed if impairment severe:* • methadone • hydromorphone • tramadol, tapentadol *Avoid:* • pethidine
Paracetamol	Safe to use in most patients	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. Limited renal reserve. Same for coxibs	Reduced doses of some suggested

Gabapentin, pregabalin	Dose adjustment suggested based on creatinine clearance	Suitable for use – non-hepatic metabolism
Tricyclic antidepressants (TCAs)	Metabolite accumulation may occur but limited evidence about need for dose reductions	Reduced doses suggested if severe hepatic impairment
Older anticonvulsants		Avoid carbamazepine and valproate if severe impairment
Ketamine[c]	Limited data but probably no dose adjustment needed	Limited information
Local anesthetic drugs [c]	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 [b]

Question 3

How would you assess pain in a patient with significant dementia?

Detail the management issues.

Patients with dementia are likely to be older adults. As such they are also at risk of multiple sources and types of pain. There is no consistent evidence that patients with dementia experience less pain. Pain in cognitively intact patients is usually assessed by self-reporting scales. This can include:

1. Verbal descriptor scale
2. Numeric scores
3. Graphic picture scales
4. Multidimensional scale

Use of such scales involves ability to give a verbal response.

Studies have indicated that demented patients with an MMSE of between 11 and 16, 83% can complete at least one scale such as:

a. Verbal reporting scale
b. VAS
c. Facial Pain Scale

(Pesonen et al in Acta Anaesthesia scanned at 2009).

With an MMSE of less than 10, 60% can complete a Visual Rating Scale but only 20-30% other scales. Hence the right scale needs to be used for the level of dementia. If patient is unable to do one scale, then try another.

The ability to undertake scales may be related to the past cognitive ability such as a numeric scale with an Accountant and verbal descriptor with a Journalist.

If verbal communication skills are absent, then behaviour coding scales need to be used.

1. Facial Action Coding System. There are 44 muscle actions in the face which can be coded, 9 action units of the face are related to pain. Such an assessment can be done electronically using a Smart Phone Application and a Warning Scale.

The American Geriatric Society has 6 behaviour domains including:

1. Facial features
2. Vocalisation
3. Body manoeuvres
4. Behaviour change
5. Physiological change
6. Physical change

This can detect and quantify pain.

2. Several pain behaviour coding scales have been developed. These include the PAINAD which uses the domains of breathing, negative vocalisation, facial expression, body congruence and consolability.

Other such pain behaviour coding scales include ABBEY, DS-DAT, Doloplus-2, NOPAIN and PACSLAC.

Pain in the presence of cognitive impairment results in increased incidence of depression by 2.6 times (Achtenburg et al 2010). Using self-reporting cognitive intact patients reported 32.4%

depression, mild cognitive impairment 53.3% and severe cognitive impairment 54.5% (Leong and Nuo 2007). That is cognitively impaired patients with pain are more susceptible to depression. Depression can be assessed using the Geriatric Depression Scale.

Patients with cognitive impairment and pain have increased psychiatric disturbance, particularly increased delusions, increased abnormal thought processes and increased aggression, increased verbal agitation. Pain in cognitively impaired patients leads to increased social inappropriate behaviour, increased resistance to care and tends towards increased wandering. Hence assessment of both pain and depression in patients with significant dementia is required.

Six main pain behaviours.

- Facial expression: frown, sad, frightening face, grimacing.
- Verbalisations, vocalisations: sighing, moaning, groaning, grunting, chanting.
- Body movements: rigidity, tense, guarding, rocking.
- Changes in interpersonal interactions: aggressive, combative, resisting care.
- Changes in activity patterns or routines: refusing food, appetite changes, sleep pattern changes.
- Mental status changes: crying, tears, depression, delusions, abnormal thought processes, increased aggression or increased verbal agitation.

Ask family or usual caregivers about changes in behaviour may indicate pain.

Pain expression in older adults with dementia may take on less obvious forms such as confusion, social withdrawal, aggression or subtle changes in behaviour which are not typical pain manifestations. Always ask the patient about their pain, some can respond to simple questions.

Proper assessment of pain in the demented patient will hopefully lead to better pain management which will translate to better quality of life and function and reduced carer burden. Professional caregivers, both Doctors and Nurses tend to underestimate the intensity of pain while family caregivers tend to overestimate the intensity of pain.

Treatment

Treatment of the patient with severe dementia must be individualised and may require an interdisciplinary approach. Underlying comorbidities need to be assessed as these will impact on pain and depression. Bowel, bladder and skin status all have an impact on intensity of pain. Sleep disturbance contributes to pain. There are impacts of ageing on the use of medications such as possible changes to oral absorption, distribution of drugs with increased fat mass in elderly demented patients resulting in reduced serum concentrations of lipofilling drugs and increased concentrations of hydrofilling drugs. Reduced serum albumin leads to increased free drug levels. All these factors need to be considered with medication prescription in the elderly patient with significant dementia.

Trials of appropriate analgesics can be undertaken, using a Pain Scale to assess the benefit. Appropriate investigations need to be undertaken, particularly for musculoskeletal pain which is the commonest cause of pain in elderly demented patients. Interventions may be appropriate such as shoulder glenohumeral or subacromial bursa injections, medial branch injections for facet joint pain and adjuvant medications for neuropathic pain. Adverse side effects need to be monitored.

Keela Herr et al. Journal of Pain and Symptom Management 2006; 31 (2): 170-192
Herr K, Decker S.Ann Long Term Care 2004; 12:46-52

Question 4

A 14 year old girl presents with a 2 year history of daily headaches not relieved by paracetamol or ibuprofen. She is missing at least 10 days a month from school and her academic grades are falling. Her father died 3 years ago from a brain tumour.

a. **How would you differentiate the possible causes?**
b. **What treatment modalities would you recommend, indicating the advantages and disadvantages of each for a patient of this age?**

The possible differential diagnoses in this teenage girl are many- **top 4 indicated by *.** They are as classified by the International Headache Society, divided into primary and secondary headaches [1] and include:

Primary Headache

1.3 Chronic Migraine*
Headache occurring on 15 or more days per month for more than 3 months, which has the features of migraine headache on at least 8 days per month (sharp throbbing, unilateral , photophobia, phonophobia , nausea and vomiting, with or without aura and localised neurological symptoms such as facial or limb paraesthesias). It is a diagnostic challenge to differentiate chronic migraine from chronic tension type headache as it can be bilateral and not necessarily associated with aura**.**

2.3 Chronic Tension Type headache*
A disorder evolving from frequent episodic tension type headache, with daily or very frequent episodes of headache, typically bilateral, pressing or tightening in quality and of mild to moderate intensity, lasting hours to days, or unremitting. The pain does not worsen with routine physical activity, but may be associated with mild nausea, photophobia or phonophobia.

4.10 New daily persistent headache*
Persistent headache, daily from its onset, which is clearly remembered. The pain lacks characteristic features, and may be migraine-like or tension-type-like, or have elements of both.

Notably if the criteria for this diagnosis is met then this is the default diagnosis even if there are features of chronic migraine or tension type headache present.

Episodic primary headache: Exertional, Menstrual

Secondary headache

Medication overuse*
(which may be complicating the original headache presentation). Subclassified according to the type of drug overused (eg opioids or barbiturates, combination analgesics, NSAIDS and triptans). Likely if medication is being used more than 15 days/ month.

Chronic sinus infection

Vision impairment –

Excessive screen use

TMJ-teeth grinding

Sleep deprivation

Post-traumatic

Brain tumour

Raised intracranial pressure –Benign cyst/ intracranial hypertension

Arnold-Chiari malformation

Vascular abnormality – AVM, aneurysm

Neuralgia – eg occipital

12.1 Headache attributed to somatization disorder - Headache occurring as part of the symptomatic presentation of a somatization disorder.

To differentiate the diagnosis, a thorough history and examination is required and further assessment: medical and clinical (child) psychologist (if available) and questionnaires. Identification/exclusion of red flags is imperative (especially with the likely anxiety stemming from the paternal history/death although inheritance not a likely factor) and potential musculoskeletal contributors. To satisfy somatisation disorder there are strict criteria including a history of multiple medically unexplained symptoms and pain from 4 or more sites.

Headache history

Presentation – sudden onset or gradually evolved from episodic pattern
Frequency- daily stated: chronic= 15+ days per month (for more than 3 mths)
Location: unilateral (migraine) vs specific site– over eye /over occipital groove (neuralgia)
vs bilateral: bitemporal (classic for tension type) or global (chronic TT or migraine)
Quality: sharp (migraine/neuralgia) vs pulsing throbbing (migraine) vs tight-pressure (tension type)
Onset/offset: – time of day (present in morning on waking and improves -raised ICP) vs increases afternoon to evening (tension type) vs triggered.
Associations

1. Red flags
    a. Headache different or pattern changed; worsened or worsening
    b. Systemic signs: fever, weight loss, neck stiffness
    c. Neurological signs – loss of cranial nerve function – eyesight, eyelid drooping or limb function eg weakness
2. Aura and triggered by food precipitators and nausea and vomiting, light noise sensitivity (migraine),
3. Triggered by reading-studying/computer use/school days, exercise, perimenstrual
4. Sympathetic symptoms

Medications trialled – actual pattern of analgesic use and intake? Is it worsened by paracetamol, nsNSAIDs?; other agents used and response eg triptans
Response: What patient does when has a headache – withdraws to room, stays in bed, distracts self
Study habits: posture, desk-chair ergonomics
Other interventions trialled CAMs – acupuncture/osteo- psychological, exercise
Impact: concentration, school attendance poor – functional – previous grades and now, academic goals; physical-sport; social; mood/psychological; sleep
Family history

1. of headache/migraine & other associations (IBS/FM/Anxiety-Depression-Suicide) – what is usual response and what has worked not worked for family members
2. Father's headache history prior to diagnosis and course including death and impact on teenager/family (including financial and on mother-sibling or extended family members)

Psychosocial: stress with dad's diagnosis/death, family's reaction and coping, anxiety, depression, overachiever, other stressors reasons for poor school attendance: bullying, friendships, academic pressure –aspiration –matched to capacity – learning disability – social dysfunction (aspergers-autism spectrum). The psychological assessment should include risk assessment. Various issues may be identified within the family and further mood disturbance and grief-related issues may not have been recognised and addressed.

Adolescent HEADSS screen (Home and Environment, Education and Employment, Activity, Drugs (includes illicit/ caffeine/smoking), Sexuality, Suicide and Depression)

Associated medical conditions: chronic pain in other areas, fatigue, GI or menstrual disturbance.

Examination: to exclude any neurological signs including cranial nerves, eye check vision/fundoscopy and peripheral nervous system examination, scalp or facial allodynia, neural groove palpation and triggering, sinus percussion signs, primary or secondary musculoskeletal problems. BMI. Documentation of affect, concentration, localised tenderness in the scalp or elsewhere (suggestive of central sensitisation).

Depending upon the level of anxiety, the investigations done previously and the clinical assessment/suspicion, **further investigation** may be required. Over-investigation has risks in this population (particularly with CTs and radiation). Also includes re-enforcing medical model and increasing anxiety levels by constant focus on investigation and positive re-inforcement of health related anxiety. Minimum investigations would include MRI brain vs CT brain and facial bones best for sinuses, LP for pressure assessment, and blood tests for signs of an inflammatory disorder (full blood count/C-Reactive Protein or disturbance of thyroid function).

4B. Depends on working diagnosis and examination findings and if further workup required

May require adjustment depending on

- rapport built (Note strong placebo response to interventions -particularly known for RCTs of migraine therapy)
- acceptance of explanation and education by teenager and family
- investigations done and if further reassurance required
- and patient engagement with strategies pharmacological and non-pharmacological

Aim to foster treatment adherence without encouraging non evidence based treatments.

Headache diary may be a positive in that gives patient a job and can be a negative in increasing focus on headache

Simple general interventions (without disadvantages) include
Gain Collateral history – mother, other relative, school, prior learning assessment
Further assessments required

?Eye test ? glasses
?Dental splint

?Neurologist assessment (needed and accessible?)
?ENT assessment –vs intranasal steroid/Abc trial

Education about headache contributors and what patient can improve (no disadvantages)

Healthy living/behavioural modification

Routine/Sleep hygiene

Environmental

Identify and avoid triggers/ Diet modification

Reduce stressors/anxiety where able:
Mindfulness/Grounding/Relaxation

Pacing/work-study plan diary

Regular exercise in moderation - yoga

Training in parenting/ Boundary setting ("tough love")

Modification of Screen time

Reward system – social outings

Step-up/flare management plan

Counselling (school/other) – grieving for father's illness, needs someone to talk to, career advice
Psychology referral for assessment – treatment -CBT based approaches (positive evidence) and /or family therapy.
School attendance (graded return) and social functioning need to be addressed.

Specific intervention

Adult dosing if adult sized 14 year-old (vs reduce maximum dosing by 25-30%)

Combinations of the below may be preferable depending on the individual patient

Treatment modalities	**Advantages**	**Disadvantages**
Menstrual headache/migraine		
OCP trial Continuous	Treat acne Benefits if sexually active	Weight gain Acceptability in this age group by parents Nausea
Medication overuse		
Wean from excessively used medications eg simple analgesics or opioids using other simple analgesics / triptans/ prophylaxis/ antineuropathics/ inpatient management if required	Diagnostic	May not engage Stressful and anxiety provoking Resource intensive
Episodic migraine		
Trial of early environmental modification vs abortive/analgesic/antiemetic intervention nsNSAIDs vs triptans for migraine Stratify intervention/step up plan	NNTs 6 to 12	May worsen pharmacological reliance and if medication overuse NB High placebo response rate Cost Risk of medication overuse headache
Episodic or chronic tension type		
Exercise	Harnesses endorphins Non-pharmacological Gets patient out of house	May be challenged by this if socially withdrawn May feel guilty about re-embracing life if father terminal
Trial of migraine prophylaxis		
Propranolol 20-80mg bd	Evidence in adult episodic migraine NNT 4.7 Positive and negative data in adolescents Benefits anxiety/panic attacks	Side effects of Weight gain Sedation/lethargy/fatigue impair physical (including sexual) performance
Topiramate 25 to 50mg bd	Evidence in adult episodic migraine Cochrane [2] NNT 4 Weight loss if obese	Concentration Weight loss (as disadvantage if thin over achieving anorexia nervosa tendency)

	Openlabel study in adolescent migraine	
Na Valproate 100-500mg bd	Evidence in adults episodic migraine; and RCT in adolescents Cochrane [3] NNT 4 Well tolerated	
Candesartan (angiotensin 2 receptor antagonist)	Equal to propranolol and superior to placebo in 1 RCT [4]	No evidence in adolescents
Calcium antagonists	Used in migraine and cluster HA in adults – 2nd line to B-Blockers	Trial data in children ~placebo
Trial of antineuropathic medication		
TCAD Amitriptyline/ Nortriptyline 10-75mg	Evidence for and against in CDH: TTH NNT=3-4 Cost effective vs other ADt Sedation may assist sleep initiation May assist coexistent anxiety,depression Benefit in adolescent trials	Hangover effect Weight gain Postural hypotension May not assist comorbid depression[5] RISK in OD (if suicide risk)
GBP/PGB	Evidence in TTH?? Treat coexistent anxiety	Impair concentration Sedation Weight gain Negative evidence for GBP in episodic migraine vs placebo [6]
SNRI = duloxetine	Treat coexistent anxiety/depression Sleep initiation	Evidence poor Some patients don't tolerate well – nausea, neuropsychiatric symptoms
SSRI eg fluoxetine, venlafaxine	Evidence positive for venlafaxine [5] Treat coexistent anxiety/depression	Evidende poor for other SSRIs Some patients don't tolerate well – nausea, neuropsychiatric Symptoms
Non-pharmacological Intervention		
Acupuncture	Few adverse effects vs pharmacological Rx Recommended by [7] (UK) and Cochrane reviewed for tension type [8] and migraine [9] ? ≥50% in 70% of adolescents vs 30% of waitlist controls [10]. Treatment success is maintained for at least 1 year, although comparative efficacy	May not have acceptance as non-pharm/non-mainstream therapy Cost Needle phobia

	with pharmacological treatments has not been investigated. NB also positive response to sham treatments used prophylactically Sham treatments superior to oral placebo for migraine prophylaxis [11] RCT true point acupuncture superior to sham for acute treatment [12]	
Distraction eg music, art	Often very acceptable to this age group (and parents)	
Biofeedback Relaxation strategies (group and individual) Progressive muscle relaxation	Good evidence in adolescents with migraine episodic/chronic [10]	
CBT	Evidence positive [13] and [10]	May not engage Cost Time off school Time to take effect Availability and proximity of appropriate services
Psychological/grief/relationship counselling/ Family therapy	Dealing with existensial issues can reduce stress and improve headache	As above
Botox A	Decreased HA frequency [14,15]	Not superior to placebo injections Intervention reliance Cost Access
Blocks eg occipital/supraorbital	Cycle breaking vs placebo-positive treatment effect	Anecdotal evidence Cost Reliance on intervention
Neurostimulation		As above Married to interventionalist

References

1. Headache Classification Committee IHS. The International Classification of Headache Disorders, 3rd edition (beta version). Cephalalgia : an international journal of headache 2013;33:629-808.
2. Linde M, Mulleners WM, Chronicle EP, McCrory DC. Topiramate for the prophylaxis of episodic migraine in adults. Cochrane Database Syst Rev 2013;6:CD010610.
3. Linde M, Mulleners WM, Chronicle EP, McCrory DC. Valproate (valproic acid or sodium valproate or a combination of the two) for the prophylaxis of episodic migraine in adults. Cochrane Database Syst Rev 2013;6:CD010611.
4. Stovner LJ, Linde M, Gravdahl GB, et al. A comparative study of candesartan versus propranolol for migraine prophylaxis: A randomised, triple-blind, placebo-controlled, double cross-over study. Cephalalgia : an international journal of headache 2013;34:523-32.
5. Smitherman TA, Walters AB, Maizels M, Penzien DB. The use of antidepressants for headache prophylaxis. CNS neuroscience & therapeutics 2011;17:462-9.
6. Linde M, Mulleners WM, Chronicle EP, McCrory DC. Gabapentin or pregabalin for the prophylaxis of episodic migraine in adults. Cochrane Database Syst Rev 2013;6:CD010609.
7. Headaches: Diagnosis and management of headaches in young people and adults. 2012. (Accessed 9 September 2015, at https://www.nice.org.uk/guidance/cg150.)
8. Linde K, Allais G, Brinkhaus B, Manheimer E, Vickers A, White AR. Acupuncture for tension-type headache. Cochrane Database Syst Rev 2009:CD007587.
9. Linde K, Allais G, Brinkhaus B, Manheimer E, Vickers A, White AR. Acupuncture for migraine prophylaxis. Cochrane Database Syst Rev 2009:CD001218.
10. Trautmann E, Lackschewitz H, Kroner-Herwig B. Psychological treatment of recurrent headache in children and adolescents--a meta-analysis. Cephalalgia : an international journal of headache 2006;26:1411-26.
11. Meissner K, Fassler M, Rucker G, et al. Differential effectiveness of placebo treatments: a systematic review of migraine prophylaxis. JAMA internal medicine 2013;173:1941-51.
12. Wang LP, Zhang XZ, Guo J, et al. Efficacy of acupuncture for acute migraine attack: a multicenter single blinded, randomized controlled trial. Pain Med 2012;13:623-30.
13. Carod-Artal FJ. Tackling chronic migraine: current perspectives. Journal of pain research 2014;7:185-94.
14. Cady R, Schreiber C. Botulinum toxin type A as migraine preventive treatment in patients previously failing oral prophylactic treatment due to compliance issues. Headache 2008;48:900-13.
15. Diener HC, Dodick DW, Aurora SK, et al. OnabotulinumtoxinA for treatment of chronic migraine: results from the double-blind, randomized, placebo-controlled phase of the PREEMPT 2 trial. Cephalalgia 2010;30:804-14.

Question 5

Discuss the role of immunocompetent cells underpinning chronic pain mechanisms.

Key points (Key facts in bold, Higher level facts not bold)

- **Identifies different types of glial cells and their location**
  - **microglia, astrocytes, oligodendrocytes in CNS**
  - endothelial cells, perivascular macrophages, infiltrating T cells
  - satellite glial cells and Schwann cells in PNS
- **Normal function of glial cells**
  - **historically regarded as support cells**
  - **microglia interact with brain synapses to modulate structure and function**
  - necessary for normal brain development
  - involved in synaptic pruning
  - facilitate intercellular calcium signalling
  - contacts with blood vessels mediate blood flow increases evoked by synaptic activity
  - regulate external chemical environment during synaptic transmission
  - are active components involved in synaptic transmission ("tripartite synapse" theory)
- **Describes triggers for glial activation**
  - **activity in primary afferents, neuronal firing, neurotransmitter release**
  - **nerve damage**
- **Describes mechanisms and effects of glial response**
  - **Broad mechanisms of glial activation (kinases, signalling pathways)**
  - **Describes involvement of specific receptors eg Toll like receptor**
  - **Glial mediators, eg release of proinflammatory cytokines, excitatory amino acids, etc**
  - **involvement in central sensitisation**
  - form coupled networks mediated by gap junctions
- **Describes involvement/relevance for clinical pain conditions,**
  - **involved in pain conditions, peripheral nerve injury, SCI, diabetic neuropathy, surgical incision**
  - **involved in opioid tolerance**
  - active role in acute and chronic neuronal disease, eg seizure, stroke, ischaemia
- **Mentions potential therapeutic agents**
  - **eg minocycline, amitriptyline**

References

- Grace PM, Hutchinson MR, Maier SF, Watkins LR. Pathological pain and the neuroimmune interface. Nat Rev Immunol 2014; 14: 217-231.
- Ji RR, Berta T, Nedergaard M. Glia and pain: is chronic pain a gliopathy? Pain 2013; 154 Suppl 1: S10-28.

Other background info

- Microglia
    - necessary for normal brain development
    - microglia interact with brain synapses to modulate structure and function
    - involved in synaptic pruning
    - microglia activated and undergo rapid proliferation after nerve injury
    - critical role in the development of neuropathic, inflammatory and postoperative pain
- Astrocytes
    - most abundant cells in CNS
    - historically regarded as support cells
    - active role in acute and chronic neuronal disease, eg seizure, stroke, ischaemia
    - form coupled networks mediated by gap junctions
    - facilitate intercellular calcium signalling
    - contacts with blood vessels mediate blood flow increases evoked by synaptic activity
    - regulate external chemical environment during synaptic transmission
    - are active components involved in synaptic transmission ("tripartite synapse" theory)
    - some questions about tripartite theory
    - important for the induction and maintenance of inflammatory and neuropathic pain
- Satellite glial cells
    - prominent in PNS
    - derived from neural crest cells
    - activated after painful injuries
    - enhanced coupling in persistent and inflammatory pain
    - play an active role in the development of persistent pain
- Different activation states
    - Causes of glial activation (peripheral nerve injury, SCI, diabetic neuropathy, surgical incision, opioid exposure)
    - Broad mechanisms of glial activation (kinases, signalling pathways)
    - Detailed description of receptors, channels etc
    - Involvement of toll-like receptors, opioid tolerance
    - Glial mediators, eg release of proinflammatory cytokines
- Neuronal-glial, Glial glial interactions
    - Glia "listen" and "talk to" neurons
    - activity in primary afferents induces glial activation (microglia and astrocytes)
    - signalling pathways involved in interactions
    - interaction between microglia and astrocytes
    - satellite cell activation requires activity and inflammation
- Glial modulation of synaptic transmission
    - Modulate excitatory and inhibitory transmission

Question 6

A 67 year old man presents to your clinic with pain in his feet in a stocking distribution. He has a BMI of 34.

a. **What is your differential diagnosis**
b. **Detail your approach to his management**

Definitions (1)

- 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 (2):

- 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
    - He may have peripheral vascular disease with ischaemia of the distal nerves.

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

Management:

1. 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. What are the supports he can call on eg family or friends? Is access to these supports available? Can he afford to take new therapies? Does he have the ability to research his illness on line? Can he access literature about pain?

Psychological considerations

With a BMI of 34 it would be worth enquiring about his long-term health goals. Does he recognise that his weight will contribute to the problem? Try to identify the barriers to losing weight and what helped him to lose weight in the past. Continue with motivational interviewing techniques. 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. He should be referred to a dietician and an obesity clinic.

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. To lose weight he will require an increase in muscle mass so a whole body exercise such as pilates guided by a physiotherapist would be helpful. Care and regular inspection of insensate skin.

References

1. IASP Taxonomy Working Group. IASP Taxonomy. IASP website: IASP; 2012. http://www.iasp-pain.org/Education/Content.aspx?ItemNumber=1698. Accessed 7 October 2014.
2. Levine TD, Saperstein DS. Laboratory evaluation of peripheral neuropathy. Neurol Clin. 2013 May;31(2):363-76.
3. Haanpää M, Treede R-D. Diagnosis and Classification of Neuropathic Pain. Pain: Clinical Updates. 2010;XVIII(7).
4. Treede RD, Jensen TS, Campbell JN, Cruccu G, Dostrovsky JO, Griffin JW, et al. Neuropathic pain: redefinition and a grading system for clinical and research purposes. Neurology. 2008 Apr 29;70(18):1630-5.
5. Attal N, Cruccu G, Baron R, Haanpää M, Hansson P, Jensen TS, et al. EFNS guidelines on the pharmacological treatment of neuropathic pain: 2010 revision. Eur J Neurol. 2010;17(9):1113-e88.
6. Finnerup NB, Sindrup SrH, Jensen TS. The evidence for pharmacological treatment of neuropathic pain. Pain. 2010;150(3):573-81.

Question 7

Describe the rationale for intrathecal analgesia in the management of pain in the patient with cancer. Briefly discuss your clinical approach to its use.

Aim is to explore the understanding by the candidate of the role of intrathecal analgesia strategies, principally infusions of opioid and adjuvants. Neurolytic approaches using alcohol and phenol could also be included, although were not the focus of the question.

Rationale and clinical approach is based on a **multidimensional assessment from a bio-psycho-social perspective.**

Intrathecal analgesia can be considered in those with pain, both nociceptive and neuropathic mechanisms, with significant impact on physical, psychological and social function. By providing analgesic and anti-hyperalgesic agents direct to spinal cord or providing partial denervation at nerve root level, less nociceptive activity will reach and sensitise the central nervous system, with subsequent reduction in pain experience

Rationale for intraspinal analgesia is thus:
1) Improve quality of analgesia: lower pain score at rest and movement
2) Lessen impact of pain: improved mobility cognitive, bowel function, psychological
3) Lessen toxicity of systemic analgesia including cognitive and bowel dysfunction. This is primarily achieved via significant opioid dose reduction, intrathecal dose being typically 1/100-1/300th oral morphine equivalents; and reduction of systemic adjuvant dosage.
4) Improve quality of life. Studies have demonstrated increased QOL with intrathecal infusions when compared to systemic analgesia. This includes improved ability to mobilise and improved cognitive function.
5) Cost. Intraspinal analgesia in cancer related pain might be cost effective, specifically via reduced in-hospital costs if intraspinal analgesia promotes discharge to community.
5) Improve life expectancy. Prospective studies have suggested increased life expectancy in association with intrathecal analgesia systems, although the mechanisms remain unclear; possibly via less sedation, improved cognitive function, metabolic effects, less immunosuppression due to lower opioid dosages.
6) One prospective study suggested aggressive anti-cancer therapy could be better tolerated, and so maintained for longer, if patient receives intraspinal analgesia in comparison to systemic analgesia.
8) A specific role exists to reduce central sensitisation, via reducing nociceptive input, with subsequent improved analgesia and systemic opioid dose reduction In this process, using low dose IT opioid plus local anaesthetics, clonidine, and potentially ketamine or midazolam, for a short period (days to weeks), may lead to sustained benefit and resumption at lower dose systemic analgesia on completion of a period of intraspinal analgesia.

Clinical Approach
Who is person: age, sex, and psychosocial state. Distress levels, health literacy, support processes including palliative care/cancer services, home situation: in hospital vs. home. Community services, pain service ability to provide support/skill set
What are the mechanisms of pain: cancer diagnosis and management, site and severity of pain reports, nociceptive vs. neuropathic. Does this patient have neuropathy due to cancer or its treatment?
What is the Impact: Impact of pain and its current treatment on cognitive, visceral (bowels) and somatic (muscle) function, sleep, mood, global functioning including mobility, and social interaction. Could these be improved/impaired by intraspinal (intrathecal) analgesia?

Risk/benefit analysis and treatment team discussion required incorporating oncologists/palliative care physician, nursing support services including community, patient and their family. Goals of care should be considered, including potential for intrathecal to achieve benefits. Expected prognosis with and without intraspinal therapy should be considered, including potential for long term survival. Patient, family and treating team consent.

Of the multiple considerations, attention needs to be given to whether pain complaints can be responsive to intraspinal analgesia, whether opioid, local anaesthetics or adjuvants. Widespread nature of pain complaints would suggest a hydrophilic opioid would be required, whereas localised pain could be amenable to more regional techniques such as hydrophilic opioids or neurolysis (e.g. intrathecal phenol/glycerol in anorectal cancer). Adjuvant agents that should be considered including local anaesthetic (specifically bupivacaine, due to antibacterial affects) and clonidine. The ability of patient to tolerate procedure and potential morbidity should be considered.

A clinical approach may include temporary catheter at appropriate site, to assess pain relief and bio-psycho-social response. Decision on technique required: i.e “temporary” intrathecal catheter with in-hospital care vs subcutaneous port and community care via external pump. If prognosis reasonable and/or life expectancy >6 month, then an implantable pump could be considered although this will be opioid predominant technique due to volume constraints.

Question 8

Discuss the evidence based techniques available to minimise the transition of acute to persistent post-surgical pain.

Background information:

PPSP Incidence: affects 10-60% of patients following surgery with severe disabling pain in 4-10%

- Varies with surgical type
- Previous injury or surgery explains up to 40% of persistent pain complaints in patients attending chronic pain clinics (Crombie, Davies and Macrae 1998; Johansen et al 2012)

PPSP frequently demonstrates features of neuropathic pain, tending to have the highest incidence when major nerves are either transected or can be easily injured.

Underlying mechanisms most likely involve peripheral and central sensitisation. A wide range of factors may contribute to these mechanisms. However, neither all of the variables, nor the relative extent to which the known factors trigger postoperative pain and maintain it in chronicity, are known but may include:

- patient factors such as genetic predisposition, gender, younger age, psychological vulnerability (catastrophising, anxiety, depression), environmental variables (e.g. expectations, cultural, dietary, worker's compensation, low income & education etc.) and repeat surgery
- preoperative noxious inputs and pain
- intraoperative nociceptive discharges brought about by cutting tissues- peripheral sensitisation; wound retraction; manipulation of organs; chemical irritation by sterilizing substances and by substances (e.g. nerve growth factor) released from injured tissues; impaired nociceptive inhibitory modulation and enhanced nociceptive facilitatory modulation (Gilron and Kehlet 2014)
- postoperative afferent inputs from regenerating wounded tissues including the inflammatory response and neuropathic activity from regenerating afferents nerves.

The anaesthesia literature tends to focus on outcome measures of pain and analgesic use, and the psychological literature to focus on measures of pain disability or pain interference making cross-study comparisons difficult, and investigation of ways to modulate the development of PPSP challenging. Although there is some correlation between pain intensity and the aversive-affective aspects of the pain, this correlation is not very strong and using it as a sole indicator of the development of PPSP is unlikely to assist in identifying ways to modulate its development.

Measures available to minimise transition from acute to PSPP:

The following classification could be applied to the concept of prevention of PPSP:

- *Primary prevention*- e.g. avoid surgery whenever possible/modify surgical procedure
- *Secondary prevention* – e.g. early intra- or postsurgical interventions to prevent transition to PPSP
- *Tertiary prevention*- treat PPSP once it has developed

Primary prevention has limited value as most surgery would be deemed necessary but modification of surgical technique could have some impact. Tertiary prevention is similarly challenging in that chronic pain is very difficult to treat. Most opportunity is in secondary prevention and is where most research is focused. (See Gilron and Kehlet 2014)

Psychological/psychiatric (depression, anxiety, catastrophising, beliefs and expectations of recovery), genetic (high-risk pain responders related to polymorphisms e.g. in COMT) and social factors involved in pain perception may have potential for intervention but, so far, have not been well addressed.

Patient education is important in the management psychosocial risk factors for PPSP which include:

A systematic review and meta-analysis by Theunissen et al (2012) demonstrated evidence that anxiety plays a role in the occurrence of persistent pain after surgery. Fifty-five percent of studies reported a positive association between preoperative anxiety or pain catastrophizing and PPSP and no studies reported a negative association. The meta-analysis on a subset of studies yielded pooled ORs in the range of 1.55 to 2.10 for the association between anxiety/catastrophising and CPSP. The results also suggest that the predictive value of pain catastrophizing is more consistent compared with general anxiety and pain-related fear, similar to the study by Khan et al. No other interventions reducing preoperative anxiety have been identified to be effective in the prevention of PPSP as yet.

Surgical procedure-related measures focus on:

- Surgical approach including the extent and nature of tissue damage
- Minimising risk of nerve damage or specific nerve-sparing approaches: may include nerve identification, dissection, avoidance of involvement in sutures, mesh inflammation. There is little high quality research supporting this although clinical data demonstrate nerve injury as one of the most important factors for chronification of pain (Kehlet , H, Edwards, RR, Buvanendran, A 2012)
- Duration as short as possible
- Choice of surgical unit by volume (low vs high volume surgical unit implying relative expertise)

Perioperative analgesia measures focus on:

Preoperative pain management:
Pre-operative screening to assess risk and prioritise intervention has been investigated- e.g. development of a score to predict severity of post-op pain; Anxiety score, and Catastrophising score; or neurophysiologic tests e.g. nociceptive responses/ ice / DNIC. What aspect of preoperative pain is predictive or whether it is a causal risk factor is not known although moderate to severe preoperative pain of more than 1 month's duration is considered to be a predictive factor in the development of acute pain and PPSP.

Preventive analgesia:
The concept of "preventive analgesia" evolved with the aim to minimize sensitization induced by noxious perioperative stimuli. A preventive analgesic effect is demonstrated when postoperative pain and/or analgesic consumption are reduced relative to another treatment, a placebo treatment, or to no treatment, as long as the effect is observed at a point in time that exceeds the clinical duration of action of the target drug. The accepted criterion is 5.5 half-lives of the target drug as a cut-off for determining when the drug is no longer pharmacologically active. The onset of treatment may be during the procedure or even after surgery. The typical explanation for the prolonged effect is that the drug prevented or minimised peripheral and/or central sensitization and thereby reduced long-term pain. However, there really is very little good clinical evidence that this is the case because there are not any accurate measures of sensitization in humans.

Currently available techniques used as preventive analgesia include regional blockade and parenteral/enteral drugs:

Regional blockade:
Early studies examining the incidence of PPSP after amputation in the setting of perioperative epidural analgesia resulted in some encouraging findings. However, the results of more recent randomized prospective
studies were less promising. A study in 2010 attempted to determine which of five analgesic regimens, including epidural analgesia in three of the five regimens, reduced chronic phantom limb pain after amputation concluded that epidural analgesia throughout the perioperative period was no different at reducing the prevalence of chronic phantom limb pain than patient controlled opioid analgesia.
Studies of peripheral nerve blockade as a method for preventing PPSP showed similar results- early promise but prospective studies showed much less. Recently, a study of preoperative percutaneously placed peripheral nerve catheters in patients about to undergo amputation. Local anaesthetic infusion was not started preoperatively, but the infusion was continued postoperatively for a median duration of 30 days. The incidence of phantom limb pain at 12 months was 16%, much lower than the background incidence of phantom limb pain noted in other studies. Although encouraging, this was not a randomized trial. A recent Cochrane systematic review assessed outcomes 6 or 12 months post surgery (Andreae and Andreae 2012). The studies were diverse but the authors were able to pool data for epidurals in thoracotomy showing a significantly lower report of persistent pain at 6 months with epidural analgesia compared with placebo; paravertebral block in breast cancer surgery with significantly lower incidence of pain at 6 months but the studies had limitations.
These conclusions are applicable across multiple surgeries including thoracotomy, as neither epidural analgesia nor paravertebral nerve blockade has been proven to reduce the incidence of severe, chronic postthoracotomy pain. Regional anaesthetic techniques, however, remain a vital part of the effort to reduce pain perioperatively, as perioperative pain severity is strongly associated with the development of PPSP.

Parenteral/enteral drugs:
Aim is to prevent long term potentiation (LTP) of synaptic strength induced by noxious stimuli and considered to be a spinal 'pain amplification' mechanism. Dysfunction of LTP is suspected to underlie the development of PPSP.

Opioids:
Intraoperative opioid administration reduces the intensity of acute postoperative pain Recent studies suggest opioids prevent LTP but only when low doses are used. However,

NMDA receptor antagonists:
Ketamine in sub-anaesthetic doses is a non-competitive blocker of the NMDA receptor. There is level 1 evidence that ketamine has a preventive effect but conflicting results for effects of prevention of PPSP (positive results in laparotomy, thoracotomy and hip arthroplasty but not amputation, knee arthroplasty, radical prostatectomy and hysterectomy).
Co-administration of opioids and low dose NMDA antagonists or low-dose opioid antagonists in rodents has been found to interfere with the development of acute opioid tolerance and opioid-induced hyperalgesia.

Alpha-2 receptor agonists:
Act on pre and postsynaptic neurons in both peripheral and central nervous system. Act by increasing descending nociceptive inhibitory control. Also extend the duration of central neuraxial blockade.

Include clonidine and dexmedetomidine which activate G1-protein-gated potassium channels in neurons resulting in membrane hyperpolarisation plus reduce calcium conductance via G0-protein-coupled N-type voltage-gated calcium channels so prevent neuronal firing and local signal propagation.
Theoretically should be useful but currently no evidence for preventive role in PPSP.

Alpha-2-delta-1 presynaptic calcium channel blockers:
Gabapentinoids -pregabalin and gabapentin (level 1 evidence for preventive role in PPSP)
Act on presynaptic calcium channels inhibiting calcium influx which results in decreased release of excitatory neurotransmitters (glutamate, substance P, CRGP) from primary afferent nerve.
Improve analgesia at rest and on movement, decrease opioid consumption by 20-60% in first 24 hrs postop.
Recent systematic review and meta-analysis demonstrated moderate to large reduction in PPSP (although possible reporting bias suggested).

Intravenous local anaesthetics:
Lignocaine
The analgesic, anti-inflammatory (intrinsic and through attenuation of neurogenic inflammation) and antihyperalgesic effects are mediated through multiple mechanisms including inhibition of sodium channels, NMDA receptors and g-protein-coupled receptors.
No consensus on dosing regimen- usually 1.5-2 mg/kg half an hour before incision followed by infusion of 1.3-3mg/kg/hr, probably best for 24 hours.
It has been used in a variety of surgeries but only demonstrated to be of benefit in abdominal surgery – decreased anaesthetic and opioid requirements, decreased postop pain (rest and movement), PONV and duration of postop ileus. Preventative effect is shown for up to 72hrs after abdominal surgery. Preventative role also demonstrated for breast cancer surgery in a RCT.

Antidepressants:
TCAs and SNRIs most commonly used to treat chronic neuropathic pain but evidence for their use in acute neuropathic pain is sparse and tends to be extrapolated from use in chronic pain states. There is Level II evidence for TCAs reducing the incidence of postherpetic neuralgia at 6 months but evidence for a role in modulating the transition of acute to chronic pain remains sparse.

Glia-modifying drugs:
Those that have demonstrated or potential efficacy in reducing neuropathic pain include

a) minocycline, a second-generation tetracycline that inhibits proinflammatory cytokine production
b) IL-1b antagonist (anakinra)
c) TNF-α inhibitor (etanercept, thalidomide, propentofylline)
d) cannabinoid agonists (CB2)
e) anti-inflammatory cytokines such as IL-10. E.g. AV411 (ibudilast) and propentofylline (currently under investigation in humans for the treatment of neuropathic pain)

These glial modulators also potentiate the analgesic effects of acute opioid administration and reduce
signs of opioid dependence and tolerance.
Placebo-controlled trials suggest efficacy of most medications is not high and is probably around 30%, even for gabapentin and duloxetine, the most commonly used agents. Neurostimulation and neuromodulatory approaches have been trialled but yet to be proven efficacious and their role in clinical practice has not been determined.
Early identification of acute neuropathic pain may provide an opportunity to introduce non-opioid analgesia early. Classically this is identified by ineffective pain relief in the acute post-surgical period

despite increasing doses of opioid and/or high pain scores reported by the patient despite significant sedation. (see Ch 12 in Macintyre & Schug, Acute Pain Management 2015)

Avoidance of amplifiers

Repeat surgery is identified as a risk factor for PPSP so avoiding additional surgery unless absolutely necessary would seem sensible. Similarly, procedures to minimise the impact of chemotherapy and radiotherapy, through skilled calculation of treatment regimen (e.g. timing of treatment with respect to timing of surgery, minimising drug choice/ doses/ treatment duration, minimising associated or secondary infection). In addition, perioperative administration of opioid analgesics under certain circumstances, may contribute to the establishment of acute opioid tolerance and opioid-induced hyperalgesia. Therefore, it is important to avoid the use of large doses of opioids without adjuvants because they increase LTP.

Since the severity and duration of postoperative pain is a risk factor for the development of PPSP, adequate and long-term monitoring of patients' pain in the postoperative period should be mandatory. However, how long this should be continued is not known yet (regional block after amputation).

Recognition of fatigue, confusion, concentration disturbances, depressed mood, and cachexia as indicators of the extent of the other inflammatory manifestations could provide further opportunities for modulation of the development of PPSP. However, there is little evidence that monitoring of these parameters is done and no evidence concerning any possible interventions that may modulate these factors.

Useful references:

Andreae MH, Andreae DA. Local anaesthetics and regional anesthesia for preventing chronic pain after surgery. Cochrane Database Syst Rev 2012; 10: CD007105

Chaparro L, Smith, S Moore RA, Wiffen PJ, Gilron I. Pharmacotherapy for the prevention of chronic pain after surgery in adults. Cochrane Database Syst Rev 2013; 7: CD008307

Crombie IK, Davies, HT, Macrae, WA. Cut and thrust: antecedent surgery and trauma among patients attending a chronic pain clinic. Pain 1998;76:167-71

Gilron I, Kehlet H. Prevention of chronic pain after surgery: new insights for future research and patient care Editorial Can J Anesth 2014; 61:101-111

Johansen A, Romundstad L, Nielsen CS, Schirmer H, Stubhaug A. Persistent postsurgical pain in a general population: prevalence and predictors in the Tromso study Pain 2012;153:1390-6

Macintyre P and Schug S. Acute neuropathic and persistent postacute pain. Ch12 in Acute Pain Management (4th Ed.) 2015

For a comprehensive review of cellular, molecular and clinical aspects of the topic see

Deumens R, Steyaert A, Forget P, Schubert M, Lavand'homme, Hermans E, De Kock M. Prevention of chronic postoperative pain: Cellular, molecular, and clinical insights for mechanism-based treatment approaches Progress in Neurobiology 2013; 104:1-37

Question 9

The current DSM criteria (DSM-5, 2013) for substance-related and addictive disorders no longer use the terms dependence and abuse. Discuss the criteria required for the new term *substance use disorder* and relate this to patients in a pain clinic.

Intro

Longstanding controversy has surrounded how to best define (and manage) problematic drug use. Significant changes to the diagnostic criteria have occurred with the transition from DSM (Diagnostic and Statistical Manual of Mental Disorders) IV to 5.

In contrast to the National Institute of Drug Addiction, the American Psychiatric Association (APA) in its diagnostic manual (DSM-5) does not use the term *addiction* in its description of problematic drug use (1). The more neutral term *substance use disorder* is used to describe the wide range of the disorder, from a mild form to a severe state of chronically relapsing, compulsive drug taking. The word *addiction* has not been used because of its uncertain definition and potentially negative connotations (2).

Criteria for substance use disorder (DSM-5)

Due to the lack of a clear boundary between the DSM-IV categories of substance abuse and substance dependence DSM-5 now uses a single term *substance use disorder* (3). In DSM-5 problematic drug use is characterised as a continuum.

DSM-5 defines a *substance use disorder* as a problematic pattern of substance use leading to clinically significant impairment or distress, as manifested by at least 2 of 11 features, occurring within a 12-month period. The features are clustered into 4 groups (4):

1. *Impaired control*
    1.1. Taking more for longer than intended.
    1.2. Unsuccessful efforts to stop or cut down.
    1.3. Spending a great deal of time obtaining, using or recovering from use.
    1.4. Craving for substance.
2. *Social impairment*
    2.1. Failure to fulfil major obligations due to use.
    2.2. Continued use despite problems caused or exacerbated by use.
    2.3. Important activities given up because of substance use.
3. *Risky use*
    3.1. Recurrent use in hazardous situations.
    3.2. Continued use despite physical or psychological problem caused or exacerbated by substance use.
4. *Pharmacological dependence*
    4.1. Tolerance to effect of substance.
    4.2. Withdrawal symptoms when not using or using less.

The severity of the problem is stratified by the number of criteria met :

mild (2-3)
moderate (4-5)
severe (6 or more).

Each substance is treated separately (i.e. alcohol use disorder, stimulant use disorder, opioid use disorder etc.) but the same criteria are used.

Relate *substance use disorder* (DSM-5) to patients in a pain clinic.

There are significant differences between illicit drug users and patients in a pain clinic. There was however little consensus about what constitutes dependence or addiction in opioid-treated pain patients (5). This is in the situation where prescribed medications intended for analgesia and/or for anxiety are amongst some of the most dependency forming categories (i.e. opioids, anxiolytics).

- As a result the term *iatrogenic addiction* has been coined (5).

Confusion has occurred when the word substance dependence has been used for patients regularly prescribed drugs prone to dependence. This is because these patients consistently experience neurobiological adaptations resulting in withdrawal and tolerance yet do not necessarily fulfil the criteria usually associated with addiction (compulsively seeking drugs) (5). In the case of improved QOL from their use, "constructive dependence" is a non-official but accurate term.

- In view of this, in DSM-5 tolerance and withdrawal cannot be used as criteria to signify *substance use disorder* during appropriate medical treatment.

Many of the behaviours used to indicate problematic drug use in *non-iatrogenic addiction* can also be attributed to the effects of pain e.g. failure to fulfil major role obligations, continued use in hazardous situations (e.g. driving), social or interpersonal problems etc.

- This has led to the development of lists of behaviours specific to indicate compulsive use the pain setting e.g. multiple prescribers, frequent ED visits, multiple drug intolerances, frequent escalations (self-escalation), early scripts, frequent clinic calls, focus on opioids and prescription loss (5).

The DSM V definition uses a 12 month time frame, however Pain Medicine specialists need to be aware that problematic substance use may occur in a much shorter time.

SUD also can be present from illicit substance use and prescribers in pain services may well be targeted i.e. patients may spend much time in a consultation trying to obtain the substance for use which is harmful, or not contributing to improved QOL. Specialist Pain Medicine Physicians (SPMP) and other professionals in such services need to be able to recognise this and respond appropriately.

Finally, non-prescription substances often taken by persons with long term pain can also cause harm as described in the 4 categories and 11 specific behaviours of SUD: e.g. nicotine, alcohol, cannabis and caffeine (though caffeine use disorder has not been defined).

References:

1. Anon. The Science of Drug Abuse and Addiction: The Basics. 2014. In: The National Institute for Drug Abuse Media Guide [Internet]. https://www.drugabuse.gov/publications/media-guide/science-drug-abuse-addiction-basics: National Institute for Drug Abuse.
2. Substance-Related and Addictive Disorders. Substance Use Disorders: Recording Procedures for Substance Use Disorders. Diagnostic and Statistical Manual of Mental Disorders-5: American Psychiatry Association; 2013.
3. Rastegar DA. Diagnostic and Statistical Manual of Mental Disorders: DSM-5 Replaces DSM-IV http://www.bu.edu/aodhealth/issues/issue_sept13/rastegar_dsm5.html2013 [Accessed 17 October 2015].
4. Facult of Pain Medicine of the Australian and New Zealand College of Anaesthetists. Problematic Substance Use. FPM Essential Topic Area 3.4. Learning module. 2015.
5. Ballantyne JC, Stannard C. New Addiction Criteria: Diagnostic Challenges Persist in Treating Pain With Opioids. Pain: Clinical Updates. 2013;21(5).

Examiners comments

This topic is covered in ETA 3.4 and in a Pain: Clinical Update, 2013 (see reference list). In addition, several learning objectives from ETA 3.4 directly relate to this question (see below). In addition, the quiz contained in ETA 3.4 directly assesses elements of this question.

Selected learning outcomes from ETA 3.4

3.4.2 Critically discuss the differences in understanding and use of these terms (tolerance, physical dependence, psychological dependence, problematic substance use & addiction/dependence) between the disciplines of pain medicine and addiction medicine

3.4.4 Describe the impact of the following non-prescription substances on health and pain experience: caffeine, nicotine, alcohol, cannabis, methamphetamine and other stimulants

3.4.6 Discuss the current DSM criteria for diagnosis of substance use disorder

3.4.8 Recognise the different forms of substance abuse that may be co-morbid with the experience of chronic pain

Question 10

Outline the presentation and pathogenesis of cancer-related mucositis.

Describe your approach to pain management.

Definition

- Inflammation and dysfunction of mucous membranes due to toxic effects of cancer therapy, usually chemo/radiotherapy.
- Mucous membrane: mucous producing epithelium usually exposed to air or gas interface.
- Face-gut-perineum.

Pathogenesis

- Toxicity of 'high turnover' epithelial cells in mucous membranes due to toxins such as chemo, radiotherapy.
- Usually chemotherapy for haematopoietic cancers (high turnover cancer cells) rather than solid tumours), localised radiotherapy (eg, head, neck, pelvis). Common with 5 FU.
- Decreased saliva & mucous, inflammation, necrosis, ulcers, blisters, fissures, pseudo-membranes, fistulae.
- Secondary inflammation & disruption of epithelium: trauma, bleeding, (thrombocytopenia), infection, candidiasis, herpes, ischemia, oedema, nutritional.
- **Linked to low neutrophil count**; recovery when neutrophil count increases.
- Causes loss of normal function of affected mucous membranes.
  -Oro pharyngeal: swallowing, taste, hydration, nutrition, oral drug administration.
  -Gut failure: lower gut: diarrhoea, nutrition, fluid balance, normal gut flora, gram negative sepsis, immune integrity, GIT haemorrhage.
  -Eyes, nose, sinuses (infections).

Presentation

- 10-40% of cancer patients. Children more at risk (higher cell turnover rates).
- Oropharyngeal cavity most often affected.
- Haematopoietic cancers > solid tumours.
- Stem cell bone marrow transplants.
- Head, neck (oropharyngeal) radiation.
- Pelvic (colorectal) radiation.
- Paediatrics (leukaemia).
- Usually starts day 5-10, peaks 1-2 weeks, last 2-4 weeks.
- Inflammation and dysfunction of mucous membranes affected.

Oro-pharynx-larynx

- Dry mouth, sticky membranes (hypo-salivation).
- Inflammation (red, swollen, painful).
- Psuedo-membranes.
- Sores, blisters, fissures, ulcers, candidiasis, herpes, varicella.
- Swallowing.
- Phonation.
- Taste.
- Dental.
- Nutrition.
- Fluid balance.

Lower gut, rectum, vagina.

Eyes.

Pain

- Pain can be combination of nociceptive/inflammatory, neuropathic, visceral pain.
- Remember to manage other pain eg. chemo & radiotherapy neuropathy, visceral pain, other cancer pain, dressings, mouth or rectal care, NGTs.
- Remember opioid hyperalgesia, tolerance.

Management

- Pain: background, breakthrough, incident.
- Mucous membrane dysfunction.
- Secondary effects (immune, infection, hydration, nutrition, nausea, vomiting).
- Nausea and vomiting.
- Whole person: Anxiety, depression, family, parents, spiritual etc.
- Paediatric cancer pain issues.

Prevention

- Prevention of cancer.
- Prevention of mucositis & secondary effects (dental care, nutrition, topical agents).
- Prevention of psychological sequelae; anticipatory anxiety (frequent flyers).

Team management

- Oncology, palliative care, nursing, family, dietician, dental etc.
- Protocols, evidence based regimes

Bio-psycho-social approach

Bio

General care

- Monitor mucous membranes: Oro-pharyngeal, nasal, sinuses, eyes, gut, rectum, vagina.
- Avoid mucosal irritants; smoking, alcohol etc.
- Hydration, nutrition.
- IV access.
- Substitute oral pharmacotherapy (absorption).
- Oral hygiene, thrush prevention.
- Gut care: PPI, gut flora (probiotics), diarrhoea, nausea & vomiting management.
- Ulcer & fistula care.
- Continence & stoma care.
- Analgesia: background and incidental (eg painful mouth care).

Topical mucous membrane care (also helps pain)

- Many different regimes of antibiotics, antiseptics etc.
- Laser light therapy.
- Keratinocyte growth factor.
- Chlorhexidine.
- Caphosol (phosphate, calcium, fluoride ion) mouthwash.
- Mu guard.
- Neutrasel.
- Episil.

- Honey.
- Artificial saliva.
- Sucralfate.

Topical mucous membrane analgesia

- Ice chips.
- Lignocaine gel.
- Topical doxepin.
- Topical clonazepam.
- Topical ketamine.

Systemic analgesia

- IV opioids are the mainstay and frequently required.
- Is there opioid hyperalgesia?
- Substitution of oral baseline opioids if nil by mouth?
- Acute Pain services frequently involved.
- Multimodal analgesia.
- PCA opioid (Cochrane).
- IV paracetamol (depending on LFTs).
- IV ketamine infusion.
- Pregabalin if NG route.
- Avoid TCAs (dry mouth?).
- Avoid NSAIDs/coxibs (gut, renal, bleeding).
- *Anxiolysis*: IV clonidine, IV BDZ.

Psychosocial

- Manage depression, anxiety, sleep.
- Prevent post traumatic stress & future anticipatory anxiety, pain (frequent flyers).
- Room facilities (often in isolation).
- Relaxation, distraction, virtual reality, hypnosis.
- Attend to family.
- Special issues regarding paediatric patients, parents.
- Spiritual.