

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

EXAMINATION HELD ON 28th to 30th NOVEMBER 2007

AT THE Geelong Hospital, Geelong, Victoria.

THIS REPORT IS PREPARED TO PROVIDE CANDIDATES AND SUPERVISORS OF TRAINING WITH INFORMATION ABOUT THIS EXAMINATION AND TO ASSIST WITH PREPARATION FOR FUTURE EXAMINATIONS. ANSWERS PROVIDED ARE NOT MODEL ANSWERS BUT GUIDES TO WHAT MIGHT BE COVERED. SOME ANSWERS CONTAIN MORE INFORMATION THAN COULD BE COVERED IN THE FIFTEEN MINUTES, BUT HAVE BEEN INCLUDED AS A TEACHING AID. THE ANSWERS PROVIDED ARE CONSIDERED CURRENT, BUT MAY BE SUBJECT TO CHANGE IN THE FUTURE.

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

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

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

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

In 2007, seventeen candidates presented for the examination and all were successful.

EXAMINATION

PASS RATE 100%

WRITTEN SECTION (See Appendix A for guides to question answers)

The following are the questions. The first five questions were compulsory.

WRITTEN SECTION

PASS RATE 88%

General information:

As always candidates need to:

1. Plan their answer so that it flows and they appear to have an organised approach.
2. *Answer the question.*
3. Give succinct answers and not repeat yourselves.
4. Use headings and dot points if asked to discuss the answers briefly.
5. Give definitions if asked to discuss some aspect. (e.g. personality and personality disorders or breakthrough analgesia) Do not assume examiners know what you understand by a term.
6. Apply more commonsense thinking when answering the questions.
7. Start answer with "I would do..." if asked to "outline your approach".

1. A 45-year-old man on a methadone maintenance program for addiction who is still smoking has peripheral vascular disease proven at angiography. He has longstanding ischaemic pain with an acute exacerbation and ulceration of the 4th and 5th toes of his right foot. He is Hepatitis B and C positive and is on 60 mgs Methadone a day.

Discuss his pain management.

Compulsory

PASS RATE 82 %

Substance abuse question –Core material/ broad question with many issues for discussion. Generally answered well. Some major areas were omitted in discussion. Some candidates did not involve appropriate medical services. A few answers were generic.

2. You are considering commencing long-term opioids for a patient with chronic non-malignant pain. What should you discuss with them about potential negative effects?

Compulsory

PASS RATE 100 %

Non-malignant pain opioid question in 2 parts.

3. There has been an ongoing controversy whether “migraine” and “tension headache” are distinct disease entities, or are due to the same underlying disease process. Discuss your view of the usefulness, or otherwise, of a clinical distinction between these two headache syndromes, with special reference to their diagnostic criteria and currently accepted treatment methods.

Compulsory

PASS RATE 76%

Tension type headache vs. migraine. Core material, reasonably well answered. Answers were generally in table form which was quite suitable for this question.

4. Compare and contrast use of isotope bone scan, MRI and CT scan in the investigation of a patient with persistent lower back pain three weeks after an uneventful disc surgery in the absence of abnormal physical findings.

Compulsory

PASS RATE 65 %

Persisting pain three months after discectomy. Most gave a reasonable differential diagnosis and listed investigations. Most candidates didn't compare DD with investigations. Note importance of ESR as first investigation, then CRP with MRI helpful later.

5. Discuss the effects of involvement in a compensation system on the patient with persistent pain after injury.

Compulsory

PASS RATE 76 %

Failed candidates gave generic answers and didn't focus on individual patient issues. Limited focus on ways of minimising perpetuation of pain.

Some candidates had memorised model answer of previous year's *similar* (but not identical) question.

6. A patient presents with a persistent headache six weeks after spinal anaesthesia for a Caesarean section. Discuss the differential diagnosis, evaluation and management.

Attempted by 13 candidates

PASS RATE 46 %

Generally, poorly done >50% passed.

Most candidates hadn't really thought about the problem. Red flags were not considered. Most candidates simply provided a DD of headache. The model answer is fairly extensive. Recommend this question to be reset.

7. Has functional brain imaging contributed to our understanding of the pathogenesis of pain? Discuss the evidence.

Attempted by 0 candidates

This is an up-to-date question whether the various considerable research information. Candidates may have been turned off by "evidence". Should be reset.

8. What is the evidence that concurrent use of pain medicines with different modes of action is more effective than single therapies for use in management of persistent pain?

Attempted by 4 candidates

PASS RATE 75 %

Few candidates addressed targets in the CNS
Few candidates addressed NNT.
Difficult to assemble a model answer because evidence is difficult to interpret. This is a basic and discriminatory question.

9. Is Quantitative Sensory Testing useful in the diagnostic work up of a patient with neuropathic pain?

Attempted by 7 candidates

PASS RATE 86 %

Some candidates got lost in detail.
This question required a debate about the place of QST in diagnosis and required some discussion of general diagnostic work up.

10. Critically discuss the current controversy concerning the diagnostic criteria for CRPS.

Attempted by 8 candidates

PASS RATE 100 %

This question addresses the Budapest CRPS criteria. Overall, impressively done. This question aimed to highlight clinical diagnostic criteria and to include the causative injury, to assess symptoms as well as signs and to add in motor signs.

11. Discuss the prevention and treatment of post herpetic neuralgia.

Attempted by 17 candidates

PASS RATE 94 %

This is core material. All candidates did this reasonably well, therefore not a discriminatory question.

12. Discuss the role of ion channel abnormalities in pain pathogenesis.

Attempted by 2 candidates

PASS RATE 50 %

The candidate who failed only discussed sodium channels.
Recommend representing this question as compulsory.

13. Outline your assessment of a patient with persistent pain following multiple back surgeries for whom you are considering neuromodulation.

Attempted by 15 candidates

PASS RATE 87 %

Considered a relatively easy question.
This was not a discriminatory question. Most candidates had a generic approach. Few mentioned that intrathecal therapy is also neuro modulatory.

14. Discuss the management of pain for a 6 year old girl with mucositis following chemotherapy for leukaemia.

Attempted by 9 candidates

PASS RATE 56%

None of the candidates appreciated the overall course of disease.
Many offered dangerous and inappropriate therapies including NSAID & rectal therapies. There is no appreciation of how sick these patients really are.

15. Discuss the use of bisphosphonates in pain management.

Attempted by 10 candidates

PASS RATE 70 %

This was a straightforward question & was discriminatory. Many didn't mention the use of biphosphates in Paget's disease and CRPS.

General comments:

Answers were generally better than in previous years. However some answers were difficult to read. There was a degree of generic answering & some repetitiveness in answers. It is important to remind candidates to carefully read the question and answer the same question.

LONG CASES

General comments and observations:

Marks are given equally for History, Examination, Presentation of findings in a logical manner, and a Management plan.

The candidates and the patients are both advised to ignore the examiners. Aim is to establish rapport with the patient.

Supervisors of Training are reminded they need to sign off that candidates have done *five observed long cases under exam conditions* prior to presenting to the examination.

As the long case mirrors a first consultation within a pain clinic, we believe the Long Case should be retained as part of the Examination.

An outline of "How to take a Pain History" is available in the NHMRC booklet, Acute Pain Management: Scientific Evidence.

Candidates all have access to the Pain Orientated Physical Examination (POPE) DVD.

Candidates need to practice long cases under exam conditions as time management is essential.

Start with open-ended questions, ensuring that history taking is patient centred.

In the long case (and also at the communication station), when candidates ask closed questions, they become too focused too early.

Listen to the patient. Patients give important clues, which at times are missed by the candidates.

Demonstrate empathy and sensitivity. (Recall this interaction is being observed.) Attention to the psychosocial history was good in some cases.

Candidates should ensure that they give appropriate time to examining the main area / systems affected by the pain, and consider examining this first.

Candidates should spend a couple of minutes only on the examination on the systems NOT involved in the main area of symptoms.

Candidates need to assess pain, function, co morbidity and underlying disease. Remember pain may not be the major issue, but more disability or psychological dysfunction.

Presentation should be structured and the discussion objective. In presenting their conclusions candidates should consider:

- An initial brief summary of the most pertinent data.

- Their analysis of this, reflecting their judgement regarding the priorities and relevance of issues.
- Considered in relation to predisposing, precipitating, perpetuating and aggravating factors.
- Management in accordance with the above.
- A sophisticated experienced candidate will present this information in less than seven minutes, even with a complex scenario, making use of the concepts outlined above, without needing to use the above terms.
- It is acceptable to indicate in your summary that there was particular information that you would have liked to obtain but did not. (Remember in real life we all may forget, and obtain the information at subsequent consultations.)

Examiners need evidence that the candidate has the ability to be the leader of the Pain Team, and to manage the long case as if they were their own patient.

Candidates are expected to finish their summary with a (biopsychosocial) diagnostic formulation and outline a management plan.

Needs to be an emphasis on an **all round approach** to assessment, diagnosis, formulation, management and prognosis.

Remember the patients who agree to be involved in the exam will be a reasonably select group. They will, as a rule, be "more than willing to please". Candidates should use this advantage, and follow up on any clues given.

Candidates should expect questions on:

- Mechanisms.
- What to do if pain progresses.
- What are the main pathophysiological issues?
- What are the main patient related issues?
- What are the main management issues?

Candidates need to look beyond the current management and ask what else could be offered. Do not assume because the patient has been to a Pain Clinic all that is possible has been done. Also do not assume the treatment that has been done so far is "best practice". Be prepared to critically discuss the patients' current management and what you may do that is different from the plan patient has described.

This year, long case patients were well chosen:-

Mr WM - 68 year old with CRPS Left upper limb, carcinoma of the lung, post-thoracotomy pain and chemotherapy induced painful peripheral neuropathy and exacerbation CRPS. Intrathecal pump in-situ.

Mr RS - 26 year old male with post-traumatic amputation left lower limb resulting in both stump and phantom limb pain. Heroin addict.

Ms TH - 46 year old woman with "failed back surgery syndrome" (x 3) and ongoing low back and radicular pain. Recent implantation SCS.

Ms SN - 46 year old woman with neuropathic pain associated with an A -V malformation affecting the right foot. Multiple surgery.

Ms LP - 59 year old woman with post herpetic neuralgia. Acute herpes ophthalmicus complicated by episodic asystole

Ms MM - 43 year old woman with brittle diabetes mellitus complicated by chronic pancreatitis, below - knee amputation left lower limb with phantom pain and mechanical low back pain

Mr JM - 45 year old male with neuropathic pain post herniotomy secondary to excision of the genito-femoral nerve

Mr TC - 60 year old male with acute pain secondary to laparotomy

Ms JA - 45 year old woman with Ehlers-Danlos syndrome and ongoing pain affecting both shoulders following surgical stabilisation. Previously participated in PMP.

Mr DP - 40 year old male with discogenic low back pain and radicular pain. Being considered for SCS.

Overall, candidates used the interview and examination hour reasonably well.

There is a need to stress detail in physical examination.

Some candidates waited until 10 minutes warning before commencing their physical examination.

*The pain physical examination needs to be **thorough and comprehensive**. This year only 1 candidate did a focused examination like a short case.*

Candidates should outline a differential diagnosis where appropriate.

PASS RATE 100%

SHORT CASES:

The Short Case section involved six patients - candidates are exposed to three.

They included:

Acute:

Mr DA 43 – R) T6, 7, 8 ribs recovery complicated by haemopneumothorax drained by intercostal catheter – empyema treated by thoracotomy and decortication 6 days ago – continuing severe pain.

Ms EC 55 – Polio since 18/12, several abdominal surgeries, PEG for 25 kgm wt loss, PEG resited 1 week ago, rib pain, PCA Fentanyl 20mcg bolus, no background infusion.

Cancer:

Ms PM - 56 year old woman with metastatic adenocarcinoma of the lung with cervical lymphadenopathy and an acute thrombosis left brachial vein.

Ms PD - 54 year old woman with metastatic adenocarcinoma of the breast and fractures through the pubic rami

Chronic:

Mr BJ - 46 year old male with CRPS II secondary to traumatic division of the sciatic nerve.

Mr DM - 69 year old male with long-standing radicular pain secondary to an acute L5/S1 disc compression. Recent ilio-femoral endarterectomy.

PASS RATE 82%

Short Cases with Patients:

General information

- At each station information was provided outside the station door.
- Candidates have 10 minutes and were directed to a specific area to examine or to impart information.
- This section is a test of physical examination techniques or communication skills.
- Candidates need exposure to neurologists and Rehab specialists as part of their training.

This year, candidates tended not to read instructions carefully or to address the specific issues. Some candidates were prepared to prescribe medication without knowing the prior medication history.

Some candidates didn't tailor treatment to the particular patient.

All candidates had areas of strength and some weaknesses. Standards were generally better than previous years.

Communication Station:

General information

- This station involves an actor and is looking at communication skills. The candidate should not delve too much into the history. The history given should be all that is required.
- The aim of this station is to encourage a generalised discussion of why suggested options are preferable. Informed consent may be required.
- Details of the history, and the treatment plans are not necessary.

This year the objective of this station was for the doctor (candidate) to see a patient (actor), with chronic pain who was usually seen by the "boss", a well known professor, currently overseas. The diagnosis was well established as CRPS and had not responded to a rigorous trial of usual therapies. SCS has been considered, but it was not an option.

The Clinical Nurse advised the patient was highly distressed, having recently been told that no further active medical interventions would be offered. The patient has requested the appointment seeking help with the doctor's next appointment in ten minutes.

It was felt this station was handled well this year

PASS RATE 59%

STRUCTURED VIVA SECTION

The viva section consisted of three structured vivas and the investigation station.

General information

- Candidates should expect questions on:
 - Nature of the lesion.
 - Anatomy.
 - Possible therapies for current pain.
 - Investigations to confirm your diagnosis.

STRUCTURED VIVA PASS RATE 100%

The introductions to the structured vivas were as follows:

Chronic scenario

Case details: The patient is a 68-year-old woman who works part-time in interior design. Her husband, aged 73, is in a nursing home with dementia.

She has a history of decades of persistent diffuse lumbo-sacral pain. Currently, diffuse back pain extending to pelvic region and buttocks is intolerable. Complaints include insomnia, lethargy, headaches, and constipation.

Recent general medical assessment and other past medical history are unremarkable.

History includes:

- 1969 Lumbar myelography
- 1970, 2004 L4/5 S1 decompression and fusion

Pain history includes:

- Poor response to medications
 - Opioid
 - Anti-inflammatory
 - Anti-epileptic
 - Antidepressant
- “Bad experience” with cognitive behavioural therapy & physiotherapy
- Self – adjusts methadone between 20 and 80 mg daily depending on the amount of pain.
- 2003 delirium

First question: What might be contributing to the current pain patterns?

In the *Chronic scenario* candidates tended to dismiss some aspects rather than confronting the issues. We do not expect candidates to say in the exam that they would just refer everything over to another specialist, the psychiatrist and the psychologist. What do these different individuals do?

Cancer scenario

Case details: A 65 year old man has had a lobectomy for adenocarcinoma 8 weeks previously. You are called by the oncology resident who is seeing him in the chemotherapy ward as he is complaining of extreme thoracotomy wound pain and sensitivity. This also wakes him from sleep despite MSContin 100mg bd, Paracetamol 3gm/day and Diclofenac 75mg bd. The patient is exhausted and vomiting.

First question: What phone advice would you initially provide?

In the *Cancer scenario* there is lack of a comprehensive care plan. Need to clarify what cancer treatments have already been given. Candidates did not focus on the anatomy the cancer type or the age of the patient. They tended not to answer the question.

Candidates needed to:

- Take notice of who referred the patient.
- **Take responsibility for pain management.**
- Fully elucidate the biopsychosocial approach rather than just paying lip service to it.
- Be careful as to which side the tumour is on and details of the history given.

Acute scenario

Issue: candidates need to carefully read the question and accurately address what the instructions require.

Case details: A 43 year old woman is having an aortic valve repair in two days' time. She has an implanted intrathecal catheter for chronic back pain. She receives 24mg of morphine per 24 hours.

Other medications:

- amiodarone
- carvedilol,
- morphine syrup
- temazepam at night

You are asked to review this patient preoperatively and recommend a perioperative pain management strategy.

First question: What are the relevant issues to consider in planning her perioperative pain management?

The *Acute Scenario* asked candidates to cope with a patient in distress. Candidates need to be able to explain to the examiners, as they would to patients, what they mean by the terms they use.

Investigations Station

General information

- Candidates need to attend regular X-ray meetings. (e.g. weekly)
- Candidates should use general knowledge.
- If there is an obvious diagnosis, mention it as soon as possible.

Specific examples:

Bone scans with metastatic disease
MRI with epidural abscess
Cervical scans with neurofibromata
CT scan far lateral disc
Abnormal biochemical profile.

*This year, most candidates had confidence in interpreting X-rays etc
Experienced candidates moved quickly through this section and did extremely well.
The section was generally well done.*

PASS RATE 82%

OVERALL COMMENTS

The standard of the candidates is improving.

Candidates need to show empathy and understanding.

The pain physician is part of a team, but the Pain Specialist needs to be able to lead the team.

Be aware of what other members of the team can do and critically discuss their roles.

Candidates may irritate painful areas in pain patients. This is recognised by examiners and adjustments are made.

Marks were also given for candidates recognising patients' sensitivities.

Candidates need to know when it is appropriate to probe and when not to, in both history and physical examination.

Patients are usually asked what they felt about the exam. This year they were generally impressed with all aspects of the examination process.

Special thanks must be given to Dr Melissa Viney for her efforts in organising the patients and the exam venue.



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December 2007

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APPENDIX A

QUESTION 1

COMPULSORY

A 45-year-old man on a methadone maintenance program for addiction who is still smoking has peripheral vascular disease proven at angiography.

He has longstanding ischaemic pain with an acute exacerbation and ulceration of the 4th and 5th toes of his right foot. He is Hepatitis B and C positive and is on 60 mgs Methadone a day.

Discuss his pain management.

The 'model' answer provided was very comprehensive and it was not expected that candidates would produce a plan of this detail, but rather this is to be regarded as a learning tool.

The examiners considered the following to be important aspects of a good answer:

- Identification of the major issues (Substance Abuse Disorder – SAD, and associated psychosocial and behavioural considerations including possibility of polysubstance abuse and risk of withdrawal, social support, other psychiatric disorders; opioid tolerance; vascular pathology, current smoking as an index of SAD and vascular impact; medical co-morbidities of SAD and vascular disease)
- Consideration of whether the patient should be managed in an inpatient or outpatient setting
- Recognition of the importance of collaboration for both assessment and management, including involvement of a surgeon, drug and alcohol services, usual methadone prescriber (including to confirm dose)
- Assessment of SAD (including behavioural issues, psychosocial support and other factors, medical consequences of risk-taking behaviour, polysubstance abuse and possibility of withdrawal syndromes), vascular pathology (including examination and investigation to exclude red flags such as critical ischaemia & infection)
- Management:
 1. Disease specific (vascular, ongoing smoking and need to address this)
 2. Pharmacological
 - a. management of methadone
 - b. requirement for additional opioid analgesia and the impact of opioid-tolerance
 - c. role of adjuvants and regional blocks including sympathectomy or epidural to improve blood flow (recognising anticoagulants may be part of ischaemia management)
 - d. identification, prevention and management of withdrawal
 3. Non-pharmacologic including need to address psychological, social and behavioural issues (e.g. contract)
- Recognition of the possible paths the situation may take (amputation, requirement for repeated dressings)

No marks were gained for a generic list of treatment options that did not relate to the management of *this* patient. While a brief introductory outline may be useful, this should not lead to repetition in the body of the answer. Weaning methadone was not considered an appropriate approach given that this was an acute on chronic pain problem.

References:

1. Australian and New Zealand College of Anaesthetists and Faculty of Pain Medicine, *Acute Pain Management: Scientific Evidence*, 2nd ed. Australian and New Zealand, College of Anaesthetists, Melbourne, Australia, 2005.

QUESTION 2

COMPULSORY

Describe the information you provide to patients concerning potential benefits and adverse effects of opioid medication when used long term for chronic non-malignant pain. Describe what factors you would take into consideration, before recommending opioid medication long term for non-malignant pain.

Prior to commencing opioids the practitioner should be comfortable:

- There has been adequate assessment, diagnosis and treatment of physical disorders causing the pain.
- All other appropriate medications and therapies for the management of the pain have been explored.
- The patient is psychologically and socially stable, employing active rather passive coping strategies and responsible.
- The patient is well known to the practitioner who is providing ongoing supervision. (*This may be the GP and the Pain Specialist may be only seeing the person for a brief interval to provide an opinion and perhaps initial stabilisation*)
- The clinician has good reason to believe that the judicious use of opioids may improve the patient's quality of life.
- There is no evidence that the patient will abuse or misuse the medication. Evidence that the medication is not being used appropriately, or is being misused will result in a review of therapy, and possibly cessation.
- Discussion should occur about the possible outcomes of therapy including:
 - Complete removal of pain – unlikely.
 - Partial improvement - there may only be a 30% reduction in pain scores.
 - Opioids have been shown to have short-term efficacy in chronic pain. A recent epidemiological study failed to demonstrate any long-term benefit in any of the key treatment goals of pain relief, improved quality of life or function.
 - No response – non-opioid responsive pain.
 - Unacceptable side effects.
 - Worsening of the pain – progression of the disease, deconditioning and secondary loss of muscular tone, opioid induced hypersensitivity.
- To continue therapy long term after a trial period and review, improvement in function and quality of life with no dangerous or disabling adverse effects should be demonstrated.
- Patients need to accept responsibility for the security of their medication, and the increased risk to them; by ensuring the safe keeping and making sure the drugs do not fall into the wrong hands.

The practitioner needs to discuss potential side effects including both short and long term physical and psychological effects. Opioids can impact on the

CNS

CVS

Respiratory

Dermatological.

GITMusculoskeletal

Urinary systems.

Psychological effects:

See long term effects, as although signs of physical dependence including tolerance may develop rapidly (with ultra-short-acting opioids), and withdrawal syndromes maybe seen as early as one week, these concerns are more of an issue with long term use.

LONG TERM EFFECTS**Physical effects:**

See above although tolerance develops to most side effects except constipation.

Opioid withdrawal syndrome:

Physiological response to lack of drug. It is not addiction.

References:

1. Ballantyne J: Pain: Editorial: Opioids for chronic pain: Taking stock: 2006: 125:3-4.
2. McNicol E: Opioid side effects: Pain Clinical Updates: IASP: April2007: Vol XV:2.
3. Therapeutic guidelines: Analgesic: 2007: Version 5.

QUESTION 3**COMPULSORY**

There has been an ongoing controversy whether “migraine” and “tension headache” are distinct disease entities, or are due to the same underlying disease process.

Discuss your view of the usefulness, or otherwise, of a clinical distinction between these two headache syndromes, with special reference to their diagnostic criteria and currently accepted treatment methods.

Most candidates acquitted themselves well with factual knowledge of distinct diagnostic criteria and treatments for the two conditions. To get a high mark however candidates had to address the actual question asked as to their own view of the usefulness of distinguishing between them or not. Few tackled it that way.

Candidates could have argued for the utility in making the distinction, not only because they are distinguishable by diagnostic features and MPQ responses¹, but because therapeutic directions are different.

Distinguishing features include:

Migraine	CTTH
Pulsating	pressing
Unilateral	bilateral
With HA, at least on of : N&V Photophobia or phonophobia	no N&V NOT BOTH photophobia and phonophobia
Aggravation by activity e.g. stairs	Not aggravated by activity
Specific pharmacologic treatments: Sumatriptan, HT3 antagonists	Non-specific strategies: Tricyclics Relaxation training Maladaptive coping prevalent Mirtazepine? ³

Some notes on Migraine:

Migraine pathophysiology involves the trigeminovascular system and central nervous system modulation of the pain producing structures on the cranium. The degree, to which head pain results from the activation of the nociceptors or pain-producing intracranial structures, or to the facilitation or lack of inhibition of afferent signals, is not clear at this time.

It has also been proposed that individuals prone to migraine have a genetic migraine threshold that renders them susceptible to a migraine attack upon exposure to some or any of a range of patient-specific trigger-factors. Hormonal influences, environmental and physiologic stressors, low blood sugar, and fatigue are all thought to determine this threshold. Once the threshold is exceeded, trigemino-vascular discharge is thought to be responsible for inducing a migraine.

Some notes on CTTH:

One of the main molecules involved is serotonin. Evidence for this theory comes from the observation that CTTH may be successfully treated with tricyclic agents. However, the analgesic effect of amitriptyline in CTTH is not solely due to serotonin reuptake inhibition.

Recent studies of nitric oxide (NO) mechanisms suggest that NO may play a key role in the pathophysiology of CTTH.

Sensitization of pain pathways may be caused by or associated with activation of nitric oxide synthase (NOS) and the generation of NO. Patients with CTTH have increased muscle and skin pain sensitivity, demonstrated by low mechanical, thermal and electrical pain thresholds. Hyperexcitability of central nociceptive neurons (in trigeminal spinal nuclei, thalamus, and cerebral cortex) is involved in the pathophysiology of CTTH⁴. Recent evidence for generalized increased pain sensitivity or hyperalgesia in CTTH strongly suggests that pain processing in the central nervous system is abnormal in this primary headache disorder. Moreover, a dysfunction in pain inhibitory systems may also play a role in the pathophysiology of chronic tension-type headache.⁵

References:

1. Mongini F, Deregibus A, Raviola F, Mongini T. Confirmation of the distinction between chronic migraine and chronic tension-type headache by the McGill Pain Questionnaire. *Headache*. 2003 Sep; 43(8):867-77.
2. The International Classification of Headache Disorders: 2nd edition. *Cephalalgia* 2004, 24 Suppl 1:9-160.
3. Bendtsen L, Jensen R. Mirtazapine is effective in the prophylactic treatment of chronic tension-type headache. *Neurology*. 2004 May 25; 62(10):1706-11.
4. Ashina S, Bendtsen L, Ashina M. Pathophysiology of tension-type headache. *Curr Pain Headache Rep*, 2005 Dec; 9:415-22.
5. Pielsticker A, Haag G, Zaudig M, Lautenbacher S. Impairment of pain inhibition in chronic tension-type headache. *Pain*. 2005 Nov; 118:215-23.
6. Fernandez-de-las-Penas C, Alonso-Blanco C, San-Roman J, Miangolarra-Page JC. Methodological quality of randomized controlled trials of spinal manipulation and mobilization in tension-type headache, migraine, and cervicogenic headache. *J Orthop Sports Phys Ther*. 2006 Mar; 36(3):160-9.
7. Rasmussen BK, Jensen R, Schroll M, Olesen J. Epidemiology of headache in a general population--a prevalence study. *J Clin Epidemiol*. 1991;44(11):1147-57.

QUESTION 4

COMPULSORY

Discuss the investigation of a patient with persistent lower back pain three weeks after uneventful L4/5 discectomy. On examination there is restricted “straight-leg raising” but no frank signs of lumbar radiculopathy.

Discussion points in answer:

- Indication for surgery.
- Differential diagnosis of present problem
 - Infection
 - Sequestered fragments
 - Epidural haematoma
 - “Arachnoiditis”
 - New development of neural impingement
 - Mechanical stiffness post-op
 - Fear/apprehension.
- With respect to imaging in general, define
 - Sensitivity
 - Specificity
 - Likelihood ratio.
- With respect to each of BS, CT and MRI, discussion of
 - Basic mechanism of test (e.g. bone scan reveals increased blood flow or increased osteoblastic activity)
 - *Relative* probability of identifying cause in each scenario discussed in DD.

MRI

Use with and without Gadolinium (relative safety of Gad over iodinated contrast media)
No ionising radiation

Bone Scan

role at this stage

Labelled WC scan

If difficult to identify infection (from other parameters e.g. CRP, ESR, leucocytosis)

CT

Possible no role at this stage. Use of these imaging techniques only if MRI contraindicated (metallic foreign bodies in eye or brain, pacemakers etc)

References:

1. Jarvik JG, Deyo RA. Diagnostic evaluation of low back pain with emphasis on imaging. *Ann Int Med* 2002; 137:586-597.
2. Saal JS. General principles of diagnostic testing as related to painful lumbar spine disorders: A critical appraisal of current diagnostic techniques. *Spine* 2002; 27: 2538–2545.
3. Van Goethem JW, Parizel PM, Jinkins JR. MRI of the postoperative lumbar spine. *Neuroradiology*. 2002; 44(9):723-39.
4. Babar S, Saifuddin A. MRI of the post-discectomy lumbar spine. *Clin Radiol*. 2002;

57(11):969-81.

5. Grane P. The postoperative lumbar spine. A radiological investigation of the lumbar spine after discectomy using MR imaging and CT. *Acta Radiol Suppl.* 1998; 414:1-23.

QUESTION 5**COMPULSORY**

Discuss the effects of involvement in a compensation system on the patient with persistent pain after injury.

The adverse effects of compensation on the individual patient with persistent pain can lead to:

- prolongation of period of work incapacity.
- exaggeration of pain severity.

It has also been recognised that in jurisdictions/countries/states where compensation is available, or litigation is allowed following accidental injury, there is a larger proportion of patients who claim disability benefits than in jurisdictions where no compensation or litigation is available or allowed.

The evidence for the adverse effects of compensation on pain patients was exemplified by the 'RSI epidemic' in Australia in the 1980s, and by studies of whiplash in Canada, Lithuania, New Zealand, and Australia.

A recent study in Victoria showed that among patients with orthopaedic trauma those covered by the no-fault compensation system for transport-related injuries had worse outcomes than non-compensable patients.

The suggested reasons proposed for the poorer outcomes of compensable patients include:

- psychosocial environment of the patient prior to injury.
- the traumatic nature of the injury event.
- greater severity of injury.
- psychosocial environment of the patient following injury.
- the patient's experience within the compensation system.
- illness behaviour directed towards secondary gain.

Occupational factors that can influence the outcome of compensable injuries include job dissatisfaction, work stress, type of work performed, and employer/supervisor attitude.

Societal factors that influence outcome following a compensable injury include expectations concerning prognosis, availability of compensation, and acceptance of the complaint as work or injury related.

The provision of specific treatment protocols has been shown to reduce chronic symptoms and promote return to work.

Research has suggested that screening of patients with work-related low back pain can identify those at risk of chronicity, either using the so-called psychosocial 'yellow flags' or a questionnaire (Orebro Musculoskeletal Pain Questionnaire) and that appropriate early intervention can reduce the risk of pain chronicity and long term disability from low back pain in this group of patients. Similarly, early intervention and rehabilitation of patients with soft tissue injuries can reduce pain chronicity and prolonged disability.

References:

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QUESTION 6**NON COMPULSORY**

A patient presents with a persistent headache six weeks after spinal anaesthesia for a Caesarean section. Discuss the differential diagnosis, evaluation and management.

Candidates are expected to have a working knowledge of the acute and chronic painful conditions that may affect women as a result of the major physiological and psychosocial changes that occur during pregnancy and the postpartum period including the potential obstetric and anaesthesia-related complications causing pain that may occur as a result of labour and delivery, including instrumental delivery and Caesarean section.

Important points include:

- Definition of postpartum period- first 6 weeks after delivery- note return to normal from the hypercoagulable state of late pregnancy by the end of this time period so thrombotic events less likely
- Appreciation of the major psychosocial changes occurring during the postpartum period and their importance in this situation
- Incidence of the various headache diagnoses in the postpartum period
- Differential diagnosis
 - Indicating more likely causes appreciating the time frame
 - a) Primary headache – tension headache/migraine/cervicogenic and cluster headache most common.
 - b) Postdural puncture headache – 2% PDPH still present at 6 weeks after dural puncture although much less likely with fine spinal needle.
 - Indication of life-threatening diagnoses requiring urgent investigation and treatment:
 - a) Tumour.
 - b) Haemorrhage/thrombosis -Subdural haematoma/Subarachnoid. haemorrhage/Cortical Vein/Venous Sinus Thrombosis (usually earlier, strong association with PIH)/Ruptured aneurysm.
 - c) Cerebral infarction/ischaemia/vasculitis/angiopathy.
 - d) Pseudotumour Cerebri/Benign Intracranial Hypertension.
 - e) Infection-Sinusitis/Meningitis.
- Evaluation
 - Pertinent points in history including PH headache, social stressors/examination/relevant investigations.
 - Importance of multidisciplinary approach- consult colleagues i.e. obstetrician, neurologist, neurosurgeon, radiologist, psychological medicine team.
- Management
 - Primary headache.
 - Postdural puncture headache.
 - Other secondary headaches.
 - Psychosocial support.

References:

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QUESTION 7**NON COMPULSORY**

Has functional brain imaging contributed to our understanding of the pathogenesis of pain? Discuss the evidence.

It must be stated initially that pain is a perception; we don't yet understand the basis of any perception even though we may know something about the anatomic and physiologic constructs underlying them.

Functional imaging is undertaken using a number of techniques. The most used are functional MRI scanning and PET scanning. Most of the current data pertains to fMRI. This technique allows for the measurement of blood flow changes in small voxels of several tens of cubic millimetres in size and so represents the average activity of many millions of neurons whose function, either stimulatory or inhibitory, is tightly correlated with local blood flow. Furthermore, the activity measured is the net difference in blood flow between a neutral state and an evoked pain state lasting a few seconds. This is easily understood in terms of changes in experimental pain states, but is more difficult to interpret when applied to clinical pain states.

Despite these caveats, functional brain imaging has contributed to our understanding of the pathogenesis of pain and is expected to do so for the foreseeable future.

Firstly, studies in experimental pain confirmed and extended theoretical considerations on the regions of brain involved in assessing the pain experience. These include the thalamus, sensory-motor areas of cortex, cingulum, prefrontal cortex and insula, thus supporting the concept that pain has sensory-discriminative, affective-motivational and cognitive-interpretive components. There is direct support for inhibitory control systems through the periaqueductal grey, and studies are likely on lower brainstem and spinal cord mechanisms as techniques improve.

Clinical pain patients have also been examined. Observations have included turning off of thalamic centres with continuous nociceptive input, amplification of most centres involved in the brain in patients with generalized pain complaint such as fibromyalgia, bilateral brain representation in many pain states, and support for a number of psychological constructs such as catastrophising and the effects of attention, mood disturbance and distraction on the pain experience, as well as the effect of pain on cognitive function. Information on brain mechanisms involved in visceral pain are also being reported. These observations are leading to more detailed hypotheses on pain modulation, such as more posterior activation of the cingulum in clinical as opposed to experimental pain, the involvement of the basal ganglia and amygdale, and the effects of placebos on brain function. They are already being used to implement strategies such as bio-feedback using fMRI directly.

It can be anticipated that in the near future these techniques will be used as surrogates for improving drug discovery and drug effectiveness in individuals as well as gauging the likely benefit of a cognitive behavioural program for a particular individual. Our understanding of genetic influences on the pain experience is also likely to benefit as well as our understanding of specific pain causing diseases that affect the nervous system.

Perhaps the over-riding benefit of brain functional imaging is to refocus attention on the role of the brain in producing a pain experience after so many years attention on spinal cord mechanisms brought about by the advances in neurophysiology of the 1950s and 1960s.

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QUESTION 8

NON COMPULSORY

What is the evidence that concurrent use of pain medicines with different modes of action is more effective than single therapies for use in management of persistent pain?

The efficacy of pain medicines in treatment is measured by the numbers needed to treat (NNT).

There are two published League of Tables on the scientific evidence of the efficiency of these pain drugs:

- The Oxford League by Bandolier;
- Finnerup et al in 2003.

These allow comparisons of single drugs and multi-modal therapy based on NNT.

The drugs to be compared are grouped as:-

- Non-opioids;
- Opioids; and
- Adjuvant drugs.

The non-opioids include paracetamol and non-steroidal anti-inflammatory drugs. The opioids include codeine, morphine, Oxycodone and methadone. The adjuvant drugs are the anti-convulsants such as Phenytoin, Vigabatrin, Topiramate, Valproate and Carbamazepine. The anti-depressant drugs include the tricyclics e.g. Amitriptyline and the SSRI'S and, lastly, there are the corticosteroid drugs.

The scientific evidence has identified multiple target sites that are involved in the pain pathways. These include peripheral cyclooxygenase, voltage-gated sodium channels, voltage-gated calcium channels, opioid receptors MU and Kappa, M.D.A. receptors, GABA activation, glutamate inhibition systems, serotonin and Norepinephrine descending inhibition.

The voltage-gated sodium channels are blocked by Lignocaine, Carbamazepine, Phenytoin, Mexiletine and Lamotrigine.

The voltage-gated calcium channels are blocked by Gabapentin, Pregabalin and Topiramate.

Tricyclic and SSRI drugs effect the descending inhibition effects of serotonin and Norepinephrine.

The glutamate system is inhibited by Carbamazepine, Lamotrigine, Phenytoin, Valproate and Gabapentin.

The GABA activation system is effected by Baclofen, Valproate and Vigabatrin.

There is scientific evidence that the use of drugs targeted to different physiological points in the nervous system at the level of the peripheral nerve, the dorsal horn and the brain itself can, in combination, provide an enhancement of the pain relief in a patient.

The W.H.O. (1986) Analgesic Ladder provides an example of the combined effects of the multi-modal therapy.

There is Level 1 evidence that paracetamol and non-steroidal anti-inflammatory drugs have increased efficiency in comparison to each drug acting alone (Ramsing et al 2002).

There is Level 1 evidence that paracetamol and Oxycodone reduce the opioid requirements for a patient by 20-30% (Ramsing et al 2002).

Kehlet (1997) identified that N.S.A.I.D.S. enhance multi-modal therapy.

There is scientific evidence that anti-epileptics combined with tricyclic anti-depressive drugs have a combined increase efficiency in the management of post-herpetic neuralgia and diabetic neuropathy.

There is evidence that Ketamine combined with analgesics and opioids decrease the requirements for other analgesic drugs (Hocking & Cousins 2004).

Lastly, there is some support for the use of combined intrathecal drug therapy in persistent pain syndromes, e.g. intrathecal opioids may be enhanced with the use of Ketamine or Clonidine.

In addition, there is Level 1 evidence that Dextropropoxyphene combined with Paracetamol is more effective than the single therapy.

References:

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2. Bandolier, W; Journal Oxford Wil.
3. *Acute Pain Management Scientific Evidence*, Second Edition, 2005; NH&MRC.
4. WHO 1986 Report *Cancer Pain Relief*.
5. Wall, P. & Melzac, R. *Textbook of Pain*, 4th Edition.
6. *The Journal of Pain*, Vol. 7, Number 1, 2006 supplementary.

QUESTION 9

NON COMPULSORY

Is Quantitative Sensory Testing useful in the diagnostic work up of a patient with neuropathic pain?

The diagnostic work up of a patient with neuropathic pain (NP) uses clinical (diagnostic) criteria including

- pain history,
- clinical examination , including traditional neurological examination, and a simplified form of bedside QST
- results of specialized investigations including
 - medical imaging
 - conventional electrophysiological testing: testing larger, myelinated A fibre function
 - Quantitative Sensory Testing (QST).

In theory neuropathic pains should be easy to distinguish from other conditions, but in practice they are difficult to identify and treat, for a variety of reasons

- Rarely one diagnostic test +/- or -/ve
- Perception of pain is subjective
- The borderland bw definite /probable /possible NP is often unclear
- Pain sensory function is dynamic and it changes/ in chronic pain signs and symptoms change
- No agreement on use of a restrictive or broad definitions of NP

According to the IASP definition of NP these disorders are characterised by a lesion or dysfunction of the system that under normal conditions transmits noxious information to the CNS. A core finding in neuropathic pain is a loss of sensory input complete or partial. Loss of sensory function results in NEGATIVE sensory symptoms and signs

The result of reduced sensory input as a result of deafferentation results in regeneration/ disinhibition and secondary hypersensitivity resulting in various POSITIVE sensory signs.

Conventional neurophysiological testing of sensory nerve conduction only tests larger fibre (myelinated A alpha and A beta fibre) function. Hence a deficit of small afferent fibres (C fibres- , involved in conduction of pain,) might escape conventional assessment.

(Skin punch biopsy or laser evoked potentials are an emerging possible standard.)

QST might bridge the gap in assessment

QST is a prescribed assessment of sensory signs. QST is reported to be a reliable psychophysical test of large and small fibre sensory modalities

QST allows researchers to study details about sensory abnormalities that are crucial to understand mechanisms underlying pain. Researchers use strictly defined criteria for assessment eg. threshold measurements, Von Frey hairs.

Clinicians at the bedside hope QST can yield useful diagnostic information, as well as a means to evaluate treatment response. Modalities that can be assessed at the bedside include:

- Pinprick
- Touch- gently applied cotton wool
- Pressure- deep pain by gentle pressure

- Heat and Cold “Thermal testing”-Cold -specific cold or warm thermal stimulus eg menthol or acetone for cold. Thermal rollers.
- Vibration- tuning fork
- Windup or aftersensations

The results can be compared with opposite side or a proximal site and graded as “ normal, decreased or increased”, to determine if they are positive or negative phenomena.

Positive sensory symptoms and signs include

- *Spontaneous symptoms*
- Evoked symptoms and signs

Current screening tools for neuropathic pain with defined criteria included the Leeds Assessment of Neuropathic Symptoms and Signs (LANSS), Neuropathic Pain Questionnaire (NPQ), DN4, Pain DETECT and ID-Pain

The European Journal of Pain (2006) published a standardized protocol for clinical trials of 13 parameters in seven test trials A-G namely:

- Thermal testing comprises detection and pain thresholds for cold, warm, or hot stimuli (C- and A-delta fibre mediated): cold detection threshold (CDT); warm detection threshold (WDT); number of paradoxical heat sensations (PHS) during the thermal sensory limen procedure (TSL) for alternating warm and cold stimuli; cold pain threshold (CPT); heat pain threshold (HPT).
- Mechanical detection threshold (MDT) tests for A-beta fibre function using von Frey-filaments.
- Mechanical pain threshold (MPT) tests for A-delta fibre mediated hyper- or hypoalgesia to pinprick stimuli.
- Stimulus–response-functions: mechanical pain sensitivity (MPS) for pinprick stimuli, and dynamic mechanical allodynia (ALL) assess A-delta mediated sensitivity to sharp stimuli (pinprick), and also A-beta fibre mediated pain sensitivity to stroking light touch (CW = cotton wisp; QT = cotton wool tip; BR = brush).
- Wind-up ratio (WUR) compares the numerical ratings within five trains of a single pinprick stimulus (a) with a series (b) of 10 repetitive pinprick stimuli to calculate WUR as the ratio: b/a .
- Vibration detection threshold (VDT) tests for A-beta fibre function using a Rydel–Seiffer 64 Hz tuning fork.
- Pressure pain threshold (PPT) is the only test for deep pain sensitivity, most probably mediated by muscle C- and A-delta fibres.

For QST to be useful in the diagnostic workup, the challenge is:

- to meaningfully classify patients in terms of mechanisms using simple bedside equipment.
- to correlate pain symptoms with QST findings
- to have specific appropriate treatments, based on assessment findings

d) to be able to validate objective results to challenge scepticism as to the validity of the patients symptoms, when conventional findings are normal.

e) to avoid diagnosing abnormalities when non exist, by developing normative data.

References:

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QUESTION 10

NON COMPULSORY

Compare and contrast the Budapest clinical diagnostic criteria for Complex Regional Pain Syndrome (CRPS) with the previous International Association for the Study of Pain (IASP) taxonomy.

Key points

- Mention CRPS subtypes I and II (nerve injury)
- Table or list IASP criteria with reasonable detail
- Table or list Budapest criteria with reasonable detail
 - Highlight the similarity of both being a clinical diagnosis
 - Highlight the contrasts where the Budapest criteria:
 - Must incorporate an inciting event
 - The increase in elements with splitting of the oedema, skin and sympathetic symptom and sign elements
 - The addition of the motor symptoms/signs
- Mention of why the Budapest criteria are superior with resultant revision of the IASP taxonomy
- Extra points were received if one or more of the following were discussed:
 - The sporadic use of the IASP taxonomy
 - The lower specificity of the IASP taxonomy
 - Related to its grouping of multiple elements
 - Improved specificity of the Budapest criteria
 - Further improvement in specificity with splitting and increased number of signs/ symptom elements
 - And the more inclusive definition for research vs clinical criteria.

Bonus if the following correctly stated:

Clinical: 2+ Sign and 3+ Symptom Categories ⇒ Sensitivity 0.85; Specificity 0.69

Research :2+ Sign and 4+ Symptom Categories ⇒ Sensitivity 0.70; Specificity 0.94

- The impact of improved more specific criteria upon diagnosis, treatment and/or research

References:

1. Reinders MF, Geertzen JH, Dijkstra PU. Complex regional pain syndrome type I: Use of the International Association for the Study of Pain diagnostic criteria defined in 1994. *Clin J Pain* 2002; 18:207–15.
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QUESTION 11

NON COMPULSORY

Discuss the prevention and treatment of post herpetic neuralgia.

1) Prevention

Use of immunization

- a. On childhood varicella
long term effect on population risk
- b. On Herpes zoster (Zostavax)
Reduction of incidence, severity of HZ and PHN

2) Early Identification of HZ

- a. Risks of developing PHN
- b. Aggressive early pain control and reassessment
- c. Role of steroids and Nerve blocks
- d. Immunocompetent v immunosuppressed

3) Management of PHN

- a. Incidence
- b. Paradigm of pharmacological treatment options include dosages appropriate for age of patient
- c. Non pharmacological management
- d. Role of interventions

References:

1. Melzack and Wall, Handbook of Pain Management 2003.
2. A.Pasqualucci et al Acta Anaest. Scand 2000,44,910.
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QUESTION 12**NON COMPULSORY****Discuss the role of ion channel abnormalities in pain pathogenesis.**

Ion channels implicated in pain pathogenesis includes voltage-gated sodium channels (VGSC), voltage gated calcium channels (VGCC), potassium channels, transient receptor potential vanilloid (TRPV) channels, P2X receptors and ASIC channels.

VGSC:

- Normally pacemaker activity is restricted to distal component of sensory nerves
- accumulation in nerve injuries such as neuromas, demyelination or axonopathy and taking on increased pacemaker activities and driving central sensitization.
- Different types involved such as Nav1.7, Nav1.3, Nav1.8&1.9
- Nav1.7 associated with inflammatory pain and mutations associated with erythromelalgia Congenital absence – inability to feel pain
- Nav1.8&1.9 translocated in nerve injury and inflammation
- Can be blocked by membrane stabilizers such as lignocaine, mexiletine, carbamazepine, Lamotrigine, amitriptyline.

VGCC:

- mediate increase in intracellular calcium leading to depolarising of membrane currents and transmitter release
- different types involved e.g L,N,P/Q, R-T type and implicated in different conditions
- N type located in dorsal horn and DRG which is upregulated after nerve section and contribute to increased spontaneous activity. Blocked by ziconotide which is shown to reduce allodynia
- Subunit of $\alpha 2\delta$ on VGCC upregulated in nerve injury leading to increasing Ca^{+} influx and neurotransmitter release. Blocked by gabapentin and pregabalin.

TRPV:

- Normally located in nociceptive neurons located in DRG, trigeminal ganglia nodose sensory ganglia
- phosphorylation by inflammatory mediators leading to increasing pain sensitivity
- blocked by capsaicin

K Channels

- Increased expression in DRG in nerve injury
- The M-Type Kv Channel involved in regulation of nociceptive sensory activity
- Blocked by retigabine
- Kir channels – 7 types – 2 involved with nociception (Gprotein-regulated and ATP sensitive
 - Implicated in the effect of morphine, baclofen, clonidine, nicotine

References:

1. Devor, M: *Sodium Channels and Mechanisms of Neuropathic Pain* J of Pain Jan 06 Vol 7 Supplement.
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6. Cummins TR et al *The roles of sodium channels in nociception: Implications for mechanics of pain* *Pain* 131 (2007) 243-257.
7. Cao You-Qing *Voltage-gated calcium channels and pain*:*Pain* 126 (2006) 5-9.

QUESTION 13

NON COMPULSORY

Outline your assessment of a patient with persistent pain following multiple back surgeries for whom you are considering neuromodulation.

Comprehensive multidisciplinary assessment addressing all aspects of the biopsychosocial components of the pain presentation including psychological assessments.

Determining diagnosis:

- nociception / neuropathy, physical, psychological, psychiatric and social contributions.

Determine treatment strategies that can be implemented in each of the domains identified such as pain, physical conditioning and psychological function before neuromodulation is considered.

Criteria that should be satisfied:

- a well characterized pain syndrome that is amenable to neuromodulation.
- exhausted conservative therapies.
- no medical contraindications e.g. coagulopathies, infection etc.
- no drug abuse.
- no major psychopathologies e.g. major depression, psychosis etc.
- realistic expectations of therapy, not looking for cure, understanding of modality, good psychosocial support with ability to attend follow-up treatments, minimal secondary gain issues. Assessed as suitable by psychologist.
- Informed consent.

Consideration for a percutaneous trial and satisfy criteria for successful trial e.g. > 50% pain relief accompanied by improvement in function and decrease in use of analgesic medications.

Neuromodulation options that can be considered include spinal cord stimulation and intrathecal analgesic administration.

- general principles of spinal cord stimulation e.g. for predominantly neuropathic radicular pain and paraesthesia must cover pain area etc.
- general principles of intrathecal analgesic therapy e.g. for both nociceptive and neuropathic pain such as both axial and radicular pain etc.

References:

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2. Beltrutti D, Lamberto A, Barolat G et al: The psychological assessment of candidates for spinal cord stimulation for chronic pain management. *Pain Pract* 2004; 4(3):204-221.
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QUESTION 14**NON COMPULSORY**

Discuss the management of pain for a 6 year old girl with mucositis following chemotherapy for leukaemia.

Major points expected included:

- Mention of presentation and course of mucositis to indicate that the candidates have been referred/ involved with a patient with mucositis e.g.
 - Patient has chemotherapy induced bone marrow suppression with low WCC/febrile neutropenia and low platelets and anaemia.
 - Pain and ulceration occurs involving any or all of the GIT and is severe when WCC/neutrophil count is low.
 - These patients are nil by mouth, often unable to swallow saliva or talk, on IV fluids and TPN.
 - These patients also routinely have definitive central access (for their chemotherapy).
- Implications of paediatric case aged 6 with cancer
 - Frequent flyers—distress, fear/anxiety (procedural/treatment) of patient and parents
 - Cooperation with all aspects of management
 - Age/developmentally appropriate pain scores VAS or VRS in this age group with acknowledgement that she may regress with illness/distress and need Faces scale or other – option of and validity for parental vs medical staff pain scoring
 - PCA –discussion of age cut off for use 5-7+y
 - May not swallow tablets with condition or with age: syrup preparations for step down
- Pharmacological agents (including doses):
 - Topical/oral hygiene therapies (naming at least one)
 - IV opioids are the mainstay and frequently required. Pain services are frequently involved when breaking through on this therapy and will institute:
 - IV opioid via PCA and/or rotate to another opioid
 - IV paracetamol (depending on LFT derangement with TPN/chemo)
 - IV Cox-2s (depending on hydration status/renal function)
 - IV ketamine infusion
 - Other antineuropathics such as gabapentin/TCAD if NG route available
 - For anxiolysis: IV clonidine, IV BDZ
 - Bonus points were given if scientific evidence for treatments was provided
- Non-pharmacological interventions such as distraction techniques available in an isolation room – books, videos, dvds, music and family
- Mention of holistic approach to management including of the parents (anxiety etc)

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QUESTION 15**NON COMPULSORY****Discuss the use of bisphosphonates in pain management.****Areas of use in pain**

Metastatic cancer deposits in bone -Breast
-Prostate

Myeloma

Paget's disease of bone

Osteoporosis -Primary: post menopausal/senile
-Secondary: steroid induced, dietary/malabsorption, metabolic

Complex regional pain syndrome

Bisphosphonates (BPs) are second line drugs for the treatment of bone pain in cancer and are not as potent as narcotics or radiotherapy which should be used first line therapy, except in Paget's Disease. Although a number of small studies have shown efficacy in the treatment of pain with CRPS, BPs should be regarded as adjunctive therapy at present.

Mechanism of action

BPs are potent inhibitors of osteoclast-mediated bone resorption, by reducing their number and activity. As such can stabilize & reduce bone loss in various settings of osteoporosis, and stabilize bone structure in areas of high turnover e.g. Paget's Disease. Tumour cells in bone particularly breast, prostate & myeloma can stimulate osteoclast formation and activity, leading to increased release of growth factors and cytokines, which further stimulate cancer cell growth and their secretion of osteolytic factors. Pain reduction is often obtained by stabilization of bone structure and reduction of associated skeletal events. Although increases in bone mineral density usually occur with BPs therapy, this is not always accompanied by pain relief. Recent evidence in an animal model suggests that BPs may also have anti-inflammatory & anti-nociceptive activity. Their mechanism of action in CRPS is unknown.

Which BPs are more effective in which setting is unknown, as is the dose and duration of therapy required. It is clear that they have an effect in bone many months after their levels in blood can no longer be measured, as such they can be administered intermittently to minimize side effects.

Side effects

Nausea, vomiting, fever, hypocalcaemia, osteonecrosis of the jaw

These drugs are often poorly tolerated, and as such are often discontinued by the patient. Since they are often second line therapy, their side effect/benefit ratio needs to be considered.

Intermittent administration is often better tolerated. Prior to commencement of therapy, dental hygiene needs to be inspected, to avoid the complication of osteonecrosis of the jaw. Severe hypocalcaemia can occur with treatment particularly of polyostotic Paget's disease and the newer very powerful BPs. In primary osteoporosis they must be supplemented with calcium.

References:

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2. Wong R, Wiffen PJ. Bisphosphonates for the relief of pain secondary to bone metastases. Cochrane Database of Systematic Reviews, 2002, Issue 2.
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