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

EXAMINATION HELD ON 25th to 27th NOVEMBER 2011

at Royal Brisbane and Women's Hospital Brisbane, Queensland

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.

EXAMINATION **PASS RATE 82.4%**

In 2011, 28 candidates presented for the examination and 23 were successful.

WRITTEN SECTION **PASS RATE 16/28 57.1%**

See Appendix A for comprehensive educational information and references for the questions.

General information:
Always, candidates need to:

1. Plan their answer so that it flows and they appear to have an organised approach.
2. *Answer the question.* Key information may be highlighted.
3. Give succinct answers and not repeat themselves. *Do not repeat the question in your answer. You may assume the examiners know which question is being answered and it wastes precious time.*
4. Use headings and dot points if asked to discuss the answers briefly.
5. Give definitions of terms used in your answer (e.g. neuropathic pain, personality and personality disorders or breakthrough analgesia). Do not assume examiners know what you understand by a term.
6. Be careful with spelling *e.g. adolescence instead of adolescents.* Acronyms or abbreviations must be defined when first used.
7. Apply more common-sense thinking when answering the questions.
8. Start answer with "I would do…" if asked to "outline your approach to…"

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

Question 1 – Compulsory PASS RATE 15/28 53.6%

Define neuropathic pain and outline the clinical tools used to make the diagnosis. What are the limitations of these tools in clinical practice?

Examiners' comments:

The question was to see if the candidates were aware of the recent changes to the definition of neuropathic pain and how this may change the treatment given to patients. We were interested in the "bedside clinical tools" used in the assessment of neuropathic pain more so than the electrophysiological or QST evaluation of the process. Those that launched directly into electrical/QS evaluation without any mention of history, descriptors, physical examination and screening questionnaires, and did not define neuropathic pain or used only the old definition did not perform well. The overall pass rate of 53.6% was quite disappointing considering such a very basic question.

Question 2 - Compulsory PASS RATE 11/28 39.3%

A patient who is taking 80mg controlled-release oxycodone 3 times a day for his persistent lumbar spinal pain is said to be "addicted".

1. **Define addiction.**
2. **How do you assess whether his use of this drug is appropriate?**
3. **What features would cause you to be concerned about the potential development of addiction in this patient?**

Examiners' comments:

This is a very basic and common encounter in the practice of pain medicine. Unfortunately majority of the candidates were unable to define fully what "addiction" is. Even those who attempt to use the DSM 4 criteria were unable to define the set of criteria. The difficulty with the current definition to pain medicine is recognized. A brief recognition of this being a chronic relapsing neurobiological disease and characterized by the 4 C's is thus accepted.

Many candidates used this question to show off their familiarity with the ORT (Opioid Risk Tool) which is not what it is about. It merely asks the basic question of appropriate use of opioids in chronic non cancer pain. Future candidates are urged to read the excellent paper written by Graziotti & Goucke (MJA 1997) many years ago which is still applicable today. The final part deals with the range of behaviours which would cause the pain medicine physician to look out for in patients on opioid therapy who may be losing control in its use and on the way to developing addiction.

This question highlights contemporary thinking on the interface of pain and addiction medicine and pain specialist should be fully informed and be competent in dealing with emergent issues arising from this.

Question 3 – Compulsory PASS RATE 22/28 78.6%

What do you tell your patients about the adverse effects of long-term opioid therapy?

Examiners' comments:

It is important to note that this question only requires attention to the adverse effects, and does not require any information about the classification, efficacy, or mechanisms of action of opioid analgesic medication.

There are a very large number of adverse reactions, and a very large amount of information available about these and the management of them. Therefore a good answer would indicate to the examiner the author's knowledge of the broad range of effects, the presenting symptoms, the significance of these effects (which would vary with the patient's actual predicament e.g. terminal care vs operating machinery and driving) and their influence on management.

This question is presented in such a manner that the answer requires the author to attend to how to effectively communicate these concepts in terms that are readily understood by the patients, with recommendations that they can readily implement. This requires use of a literacy level equivalent to Grade 6 primary school.

Question 4 - Compulsory **PASS RATE 18/28 64.3%**

You are preparing a presentation for GPs on opioid prescribing in the management of patients with pain. A pharmaceutical representative has recommended that patients taking 8 (eight) paracetamol 500mg/codeine 30mg tablets per day can be switched to transdermal fentanyl 12 microgram/hour.

a) **Discuss this recommendation with reference to relevant pharmacological information.**
b) **What advice would you provide GPs about the appropriate use of fentanyl patches?**

Examiners' comments:

Many candidates tried to do complicated mathematics to work out the equi-analgesic dosing of the patch to oral morphine. In an exam situation (where stress is involved) that turned out to be problematic. Candidates should have an idea of approximate equi-analgesic doses for all opioids readily available on the market.

This question should have been core knowledge, and candidates should have the knowledge to lecture/give advice to GP's and also be able to guide Pharmaceutical representatives who may well give "skewed" information.

Question 5 – Compulsory **PASS RATE 18/28 64.3 %**

Prescription medications used in the management of pain may influence a patient's ability to drive a motor vehicle.

a) **Outline how this can be assessed.**
b) **How can this situation be managed?**

Examiners' comments:

The major fault in answering this question was to give a long list of offending medications and their effects. This lost candidates a lot of time because these were not needed to answer the question.

Question 6 - Non Compulsory PASS RATE 13/17 76.5%

Discuss the recognition, management and prognosis of Serotonin Syndrome.

Examiners' comments:

This should have been a straightforward question and yet some candidates performed surprisingly poorly.

Failure to define the condition was a common omission. The majority of candidates indicated that Serotonin Syndrome was caused by a combination of serotoninergic drugs, ignoring the potential for serotonin syndrome to arise from just one such substance.

Contributing substances were covered inadequately, even with regards to common pain medicine drugs. Also lacking was the inclusion of common over-the-counter and recreational drugs.

Few candidates provided a useful list of differential diagnoses, and an understanding of the treatment of Serotonin Syndrome was in many cases poor, and whilst this was not specifically requested, the examiners considered this to be a valid facet of the recognition / management of Serotonin Syndrome, and therefore worthy of inclusion.

Question 7 – Non Compulsory PASS RATE 5/7 71.4%

Survivors of childhood cancer are experiencing delayed effects of disease and treatment, including chronic pain.

Discuss the psychological impediments to management of pain in a 25-year-old survivor of childhood cancer.

Examiners' comments:

This question was answered reasonably well – with three candidates scoring very well. It was not expected that many candidates would have had significant experience managing pain in this group of patients. However, the long term sequelae of childhood cancers and its treatment is becoming a significant burden for many. Hence it is expected that candidates will read broadly and develop an appreciation for all patient groups who experience pain.

Haematological malignancies remain the highest proportion of all childhood cancers with the greatest rate of cure. Some candidates focused on brain tumours and sarcomas requiring amputation. The statement that "many of these children are on opioids for many years" is incorrect.

Question 8 – Non Compulsory PASS RATE 3/11 27.3%

You have taken on the position of Director of Acute Pain Services in an *adult* hospital

and are concerned that there is no clinical practice guideline for the use of intravenous and subcutaneous ketamine as an adjunct in acute pain management.

a) Describe the essential elements of such a guideline
b) Discuss controversies associated with the use of ketamine in acute pain
c) Outline the approach you would take in developing such a guideline

Examiners' comments:

Candidates commonly gave very generic answers. Specific detail would have greatly improved the quality of the responses.

There was insufficient information specific to ketamine in many answers.

Question 9 - Non Compulsory PASS RATE 14/22 63.6%

A 45 year old single mother and supermarket 'checkout' operator is referred by her GP with "whiplash". She reports severe neck pain and headaches since the day of her motor vehicle accident 6 months previously, and is suing the other driver. She has not driven a car or returned to work since.

a) Outline possible factors associated with acute-to-chronic pain transition <u>in this case</u>.
b) Discuss your assessment and management of this patient.

Examiners' comments:

This question was reasonably answered. The candidates often failed to <u>read the question</u> i.e. risk factors for acute-to chronic pain transition <u>in this case</u>- most answered generically and wasted time with unnecessary information.

Most candidates understood the definition of whiplash injury and knew the diagnostic features. Most considered the factors in failure to recover in general terms but few knew the factors in acute to chronic pain transformation in more than general terms.

Most identified "red" and ''yellow" flag situations but few stated ongoing litigation as a significant factor. Evaluation and management was not as well answered. Evidence for the efficacy of treatment was rarely mentioned.

Question 10 – Non Compulsory PASS RATE 5/6 83.3%

a) Define the concept of "catastrophising" in patients with pain.

b) Discuss the role of this trait in patients with pain.

Examiners' comments:

Several candidates provided disappointingly brief responses in regard to a simple question pertaining to core knowledge.

The examiners expected a breadth of knowledge which was not demonstrated by many of the candidates who attempted this question. Many answers were way too brief. All reflected

a limited knowledge of anything beyond the intuitively obvious.

Question 11 - Non Compulsory PASS RATE 11/16 68.8%

A 65 year old man wakes up following oesophagectomy with loss of sensation and motor function in his lower body. His operation was performed under general anaesthesia and appeared straightforward. An epidural catheter was inserted prior to induction but only a test dose was administered.

Please outline:

a) The potential causes for this unexpected neurological deficit

b) Your postoperative management plan.

Examiners' comments:

This question was a clinical follow-on from a previous poorly answered question about the blood supply of the spinal cord.

Candidates were expected to

- *Demonstrate a clinical understanding of the most likely surgical and anaesthetic causes and through this demonstrate an understanding of the relevant anatomy*
- *Identify this as a potentially catastrophic event that requires urgent assessment and management*

Failure to state they would undertake urgent assessment/imaging resulted in a significant reduction in the marks allocated.

Candidates seem to confuse the anatomy (which was a point being re-tested) by using the following terms interchangeably which is incorrect:
intraspinal vs. intrathecal/subarachnoid/spinal catheter vs. subdural vs. extradural/epidural

The candidates who focused on psychopathology as a cause and psychiatric intervention in the management section in this anaesthetic/potential neurosurgical emergency, lost points. Some candidates focussed on infection, while others correctly attributed this appropriately to being unlikely Injury caused by neck movements (by the anaesthetist during intubation) was also mentioned with candidates failing to mention surgical positioning during thoracotomy as a possible cause. A few mentioned correction of surgically induced coagulopathy (associated with intraoperative blood loss); others focussed on epidural haematoma with coagulopathy induced by preoperative medications (such as warfarin) without stating that this is a contraindication to epidural insertion.

One candidate mentioned discussion at morbidity/mortality meeting. No candidates mentioned medical defence liaison.

Question 12 - Non Compulsory PASS RATE 14/17 82.4%

Outline an introductory lecture to be given to patients commencing a two week multidisciplinary outpatient pain management program.

Examiners' comments:

This question was generally well answered for a core knowledge topic.

Question 13 – Non Compulsory **PASS RATE 12/21 57.1%**

A 28 year old woman had a lumbar epidural catheter inserted during labour. Twelve hours after delivery and removal of the catheter, she complains of back pain.

a) Outline the key features of your assessment of this patient.
b) List your differential diagnosis.
c) What advice would you give the hospital risk manager regarding a system for early identification of epidural complications?

Examiners' comments:

This question highlights the rare complications of epidural analgesia in labour and the importance of the Acute Pain Service (or the responsible Anaesthetist) reviewing these patients within 24 hours of removal of the epidural catheter in early detection of these complications. Candidates who did not indicate that they understood that this could be a medical emergency or that MRI was the only investigation required urgently did not score well.

The final part of the question reflects the types of advice that a Specialist in Pain Medicine could be asked to provide in the development of hospital guidelines and protocols that ensure patient safety. These would include the provision of appropriate recommendations along with the evidence base to support them. Details specific to this situation were required to score high marks.

Question 14 - Non Compulsory **PASS RATE 3/14 21.4%**

a) Briefly outline the changes that may occur in the older person in perception and report of pain.

b) In the older person, pharmacokinetic and pharmacodynamic changes may affect choice and/or dose of medication prescribed. Summarise these changes and indicate how they would influence choice and/or dose of opioid and non-opioid medications used for pain management in the older person.

Examiners' comments:

*This question was based on core knowledge expected of a pain medicine specialist. Candidates were expected to describe the applied pharmacokinetic and pharmacodynamic principles in prescribing both **opioid** and **non-opioid** analgesics to their elderly patients. Candidates should have summarized the pharmacokinetic and pharmacodynamic changes that occur in the elderly and how their **choice** of analgesic would be influenced and how **dosing** may be modified in the elderly as a result of these changes.*

*The answer required acknowledgment of the fact that the elderly (i.e. those aged >60 years) are a heterogeneous group and include individuals who have **normal** physiological decline secondary to the ageing process per se and, those who have additional co-morbidities which contribute to their physiological decline and hence, their abilities to tolerate the same doses of drugs as their younger counterparts. Only one candidate recognized this.*

Only 2 candidates mentioned delirium as a cause for poor reporting of pain, and as being of significantly greater occurrence in drug dosing in the elderly. The anatomical and physiological changes in the ageing nervous system were very poorly completed. Motherhood statements e.g. there is a decrease in renal function with ageing, did not earn marks. This part of the question leant itself well to a table but unfortunately no candidate attempted this.

There is an expectation that at this level of seniority that candidates will be able to discuss the risk assessment in dosing analgesics and analgesic adjuvants used in the treatment of pain, in the elderly. Blanket statements such as "I would avoid opioids" are unreasonable in a pain medicine specialist and did not gain marks. Only one candidate discussed tramadol in the elderly, no-one discussed local anaesthetics, and most candidates confined their discussions to opioids with a few talking superficially about NSAIDs and gabapentin. Incorrect statements such as norfentanyl is an active metabolite or that opioid patches are preferred because diminished blood supply to the GIT reduced oral drug absorption were very disappointing at this level.

Question 15 – Non Compulsory **PASS RATE 8/9 88.9%**

a) **Describe the effects on the clinician of dealing with "difficult" pain patients and the implications for patient management.**

b) **What are the strategies you might use to minimise such effects?**

Examiners' comments:

Not answering the question was still the greatest fault – one person wrote totally about the patient, not the doctor. There was only one really good answer.

General comments on written paper:

Poor legibility of writing was once again a detractor for some candidates.
Yet again, candidates are reminded to read the question carefully.
There was a degree of generic answering, "Motherhood" statements and some repetitiveness in answers.
Abbreviations always, and specific medical terms generally, require definition when used for the first time.

LONG CASES **PASS RATE 22/28 78.6%**

General comments and observations:

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.

The Long Case is an important part of the Examination because it aims to mirror a first consultation within a pain clinic, core business for a Specialist Pain Medicine Physician.

Marks are given equally for History, Examination, Presentation of findings in a logical manner with a management plan, and the Viva discussion of issues highlighted by the case.

History:

An outline of "How to take a Pain History" is available in *Acute Pain Management: Scientific Evidence.* 3rd Edition, 2010 Macintyre et al., (Eds), ANZCA available at www.anzca.edu.au/resources/college-publications

Candidates need to practice long cases under exam conditions as time management is essential. Establishing rapport with the patient should be an early aim. The candidates and the patients are both advised to ignore the examiners.

Candidates should:

- Routinely ask all patients about *pertinent negatives*-red/yellow flag safety issues and *present these as part of the long case summary*.
- Start with open-ended questions, ensuring that history taking is patient centred. Then focus in on issues relevant to ensuring a comprehensive history is obtained. Some candidates became focussed too early by asking closed questions and missed key points.
- **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.)

Physical Examination:

Candidates often do not perform this aspect of the Long Case very well, despite it being worth equivalent marks to the History.

Candidates should bring their own stethoscope. All other equipment required is available and standardised.

Candidates **must** adhere to accepted standards of infection control at all times when interacting with the patients.

Candidates should ensure that they give appropriate time to examining the main area / systems affected by the pain, and consider examining this first. However, other systems involved in pain must also be examined. Remember, patients often have multiple pain complaints. A couple of minutes only should be spent on examination of the systems NOT involved in the main area of symptoms.

Candidates all have access to the Pain Orientated Physical Examination (POPE) DVD which may help to provide a structured approach. It is not essential to follow this format. However, candidates should demonstrate that they have an organised, structured approach.

The ASIA (American Spinal Injury Association) assessment criteria are recommended for examining spinal cord injury. It would be helpful if Supervisors of Training could assist candidates with this.

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:

The presentation should include a structured formulation and an objective discussion. There needs to be an emphasis on an **all-round approach** to assessment, diagnosis, formulation, management and prognosis.

Candidates should consider the following format:

- An initial brief summary of the most pertinent data.
- Their analysis of this, reflecting their judgement regarding the priorities and relevance of issues, including predisposing, precipitating, perpetuating and aggravating factors.
- Outline a differential diagnosis where appropriate.
- Critical review of the patient's management to date in accordance with the above.
- Finish the summary with a (bio psychosocial) diagnostic formulation and outline a management plan.
- 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.)

The Candidate should aim to present this information in less than **seven** minutes, even with a complex scenario, making use of the concepts outlined above.

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

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 to their advantage, and follow up on any clues given.

Viva:

Issues relevant to this patient's pain condition are identified by the Examiners during the 20 minutes following the interview with the patient. These topics form the basis of the viva.

Candidates can expect questions on:

- Pain mechanisms.
- What to do if pain progresses.
- The main pathophysiological issues.
- The main patient related issues.
- The main management issues.
- Medications including indications for use, mechanisms of action and complications.
- Expectations of treatment modalities previously used or recommended.

Candidates should look beyond the current management and ask what else could be offered. Do not assume because the patient has been to a Pain Clinic that 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 the patient has described.

***This year, long case patients included*:-**

- 49 year old male with long history of back pain, radicular leg pain and foot drop. .

- 47 year old male with multiple neurofibromata on sciatic nerve with persistent neuropathic pain.
- 47 year old male who had brachial plexus injury at birth; persistent neck pain.
  39 year old male with neuropathic pain of his left arm after it was twisted when his glove was caught in a machine at work.
- 39 year old male with brachial plexus avulsion injury amongst other traumas after a scooter accident in Vietnam.
- 28 year old woman with Klippel-Trenaunay Syndrome of left leg requiring amputation. Ongoing venous malformations in stump with pain of a vascular nature.
- 41 year old male. Neurosurgery patient with acute C8 radiculopathy.
- 56 year old male with phantom limb pain following traumatic amputation and Trigeminal Neuralgia.
- 41 year old man with PHN.
- 35 year old male who has pain, ongoing gait problems and secondary large joint and spine problems following an operation (wrong operation) on his foot that exposed the metatarsal heads to pressure.
- 60 year old woman with CRPS of the dominant arm. Spinal cord stimulator.
- 52 year old man with upper limb phantom pain post brachial plexopathy from MBA.
- 26 year old man with mononeuropathy, CRPS, and compartment syndrome after spider bite.
- 42 year old woman with SAPHO Syndrome (Synovitis Acne Pustulosis Hyperostosis Osteitis) and widespread pain.
- 26 year old occupational therapist with CRPS of leg post ankle sprain
- 32 yr old with compartment syndromes, acute on chronic neuropathic pain
- 33 year old male with mechanical LBP and bilateral leg pain.

Overall, candidates used the interview and examination hour reasonably well. Many missed asking a systematic review and a pain history i.e. one candidate missed details of chronic daily headache/migraine and atypical chest pain because they simply didn't take a history of any other pain problems. Candidates are reminded to take a short forensic history.

There is a need to stress detail in ***physical examination.*** *Some candidates waited until the 10 minute warning before commencing their physical examination. In particular, <u>neurological examination</u> seemed poorly done or unpractised in many cases.*

Mental State Examination:
A mental state examination should be part of the examination when the case warranted assessment. Few attempted any form of MSE. It is usually necessary even in a brief form.

''Pertinent negatives'' (important safety issues): *A number of Candidates failed to ask these and other important screening questions despite this being highlighted in the 2010 examination report. "Pertinent negatives" include: weight loss, fever, night sweats, cancer history, renal function, peptic ulcer disease, seizure history, sleep apnoea, driving/occupational safety and self-harm risk. Also ask about allergies, seizures, renal and liver disease, sleep apnoea, drug and alcohol history.*

As well as comparing sides (in a systematic fashion) and mapping out areas of allodynia on examination, Candidates should look for important ***'clinical clues'*** *such as:*

- *<u>medical alert bracelets</u>*
- *packs of cigarettes and asthma puffers in pockets*
- *hearing aids*
- *magnetic necklaces*
- *scars*

- *IVDU track marks*
- *hand dominance*

STRUCTURED VIVA SECTION **PASS RATE 21/28 75%**

The viva section consists 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.

The introductions to the structured vivas were as follows:

Acute scenario **PASS RATE 20/28 71.4%**

Introductory scenario:

You are asked for advice about a 64-year-old woman who has a long history of neck and low back pain following extensive scoliosis surgery when a teenager. She has an implanted intrathecal morphine pump which provides adequate analgesia for her low back pain. She also uses a transdermal fentanyl patch to control pain in her neck. Her other medications include aspirin, atenolol (for hypertension) and Lipitor (atorvastatin). She is otherwise reasonably well and keeps very active.

She presented to her cardiologist with new dyspnoea and angina on modest effort. Coronary angiography identified severe triple-vessel coronary artery disease and she has been scheduled for coronary artery bypass surgery the next day.

You are asked to provide advice regarding:

(a) Pain management immediately prior to surgery and

(b) Pain management strategies in the postoperative period after she has been extubated

Question 1: What is your advice?

Comments from the Examiners included the following:

A pain medicine specialist should be able to provide advice re management of an intrathecal pump when patients are to undergo surgery and whether or not a fentanyl patch is removed (and the reasons for either choice), to assess a patient's pain before surgery as well as try to allay anxiety regarding previous bad experiences with postoperative pain. This was generally not done well.

Postoperative pain management was management of an opioid-tolerant patient – a very common scenario and one where management (provision of good pain relief, attenuation of tolerance and prevention of withdrawal) should have been straightforward for any pain medicine specialist. However, as in previous exams, it seemed that a number of candidates may not have had much exposure to acute pain patients or participated in Acute Pain Service wards rounds and many struggled with basic concepts.

It was also disappointing to see that a number of candidates struggled with opioid conversions (equianalgesic doses), which should be second-nature to pain medicine specialists. Some wasted half the viva in doing complicated calculations. The equivalent of 100 µg /hr fentanyl patch is 100 µg/hr parenteral (IV, IM, SC) fentanyl.

It was disappointing to have options such as epidural analgesia suggested (in a patient with an IT pump and who has had past scoliosis surgery). Even though epidural analgesia has been used in some patients undergoing CABG, it was not an appropriate choice in this patient. Similarly, candidates at this level should be able to discuss the pros and cons of using non-selective and COX2-selective NSAIDs in this setting. Most could not and chose to ignore the possibility of using these agents, even if the contraindications were not present. Many had limited knowledge about the acute management of neuropathic pain. This was particularly so with reference to the neuropathic pain states that may accompany coronary artery grafting.

All in all, the viva was managed poorly. Those with experience in the area clearly stood out.

Chronic scenario **PASS RATE 25/28 89.3%**

***Introductory scenario*:**

A general practitioner wants to discuss with you a patient reporting increasingly severe back pain. The patient is a 50-year-old man, a former labourer who is on a disability pension because of a 5 year history of low back pain. The GP reports the patient has severe pain despite the patient taking

- **oxycodone** CR 40 mg tds with
- **oxycodone** IR 5 mg tds prn
- **alprazolam** 2 mg twice daily which the GP prescribed "for muscle spasm"

The patient wants to increase these medications.

The GP recently organised a second series of x-rays and CT scan of the lumbar spine within two years. The imaging shows no acute or interval change and there are no features of neural impingement or other significant pathology.

The GP is requesting an urgent appointment for you to assess his patient.

The GP is also asking you to telephone him with advice about how he can properly assist the patient now, before the appointment.

Question 1: Describe how you would respond to the GP's requests.

Comments from the Examiners included the following:

The underlying issue in the scenario was identifying the distress of the GP and that he had lost confidence in managing the patient and therefore the therapeutic relationship. The role of the consultant was seen to give advice to the GP on how to re-establish this relationship and continue managing this patient (and future patients). Many candidates attempted to take over the management of the patient to the exclusion of the GP. Few pain clinics would be able to see all such patients the next day, thus other strategies are required. Nearly all candidates excluded the issue of "red flags" but some were confused as to the relevance of

further investigations. Some candidates failed to explore the issues surrounding the patient's activity level, supports, recent stressors and expectations. Most enquired of the GP the risk of substance abuse.

The question managing the request to increase the dose was done poorly in general with a few exceptions. This included the potential of rotating opiates which required giving exact instructions on dosages and time frames to the GP and revision periods and inviting further telephone consultations with the GP

Giving advice to the GP on how to manage the patient's distress was only done well by half the candidates.

A few candidates were unsure on how to advise the GP on the patient's use of alprazolam.

Cancer scenario **PASS RATE 22/28 78.6%**

Introductory scenario:

A 54 year old male patient has metastatic adenocarcinoma involving the pelvis, sacrum and invading the lumbosacral plexus.

He is currently on maximum conservative drug therapy with

- subcutaneous hydromorphone and ketamine
- optimised doses of oral medication including
    - duloxetine
    - pregabalin
    - paracetamol
    - celecoxib
    - dexamethasone

His pain is intractable involving the right sided pelvis buttock and leg.

Question 1: What are appropriate management measures in this situation?

Comments from the Examiners included the following:

The candidates broadly performed well. Most candidates seemed to know the basics. There were several weak candidates who did score poorly however.

Nerves and style of presentation seemed a big issue for some candidates.

Knowledge of specific intrathecal techniques versus epidural was lacking in some candidates. However, candidates were not marked down heavily on this if they clearly did not have an anesthetic background.

Investigation Station **PASS RATE 20/28 71.4%**

The investigations may include:

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

- Blood drug levels

General information:
A brief scenario was presented to put the investigation into a context.

- Candidates should use general knowledge.
- If there is an obvious diagnosis, mention it as soon as possible.

Comments from the Examiners included the following:

Most candidates knew the answers but were slow to progress through findings to diagnosis.

Pain management specific to the radiological investigation abnormality required a focus on the progressing of the problem and the need for mechanical stabilisation.

Nearly all candidates recognised long QT but few could discuss the implications thoroughly.

Most missed the absent kidney in the abdominal scan.

Again, the majority passed but most struggled to demonstrate familiarity with common or classical pain problem investigations.

SHORT CASES: **PASS RATE 25/28 89.3%**

General Information:

The Short Case section involved each candidate having a brief exposure to 3 patients with acute, chronic and cancer pain plus the Communication station.

- Candidates had 10 minutes and were directed to a specific area to examine and discuss or to impart information.
- Information was provided outside each door regarding the station.
- This section is a test of physical examination techniques or communication skills.
- Candidates benefit from exposure to neurologists and rehabilitation specialists as part of their training in the performance of this section.

This year's cases included:

ACUTE

- 27 year old male with burning dysaesthesia from electrical burn to left fingers, arm and scalp.
- 41 year old male. Neurosurgical patient with C8 radiculopathy.
- 34 year old woman, IVDU with injection of amphetamine into post tibial artery with acute cellulitis and ischaemia. Suboxone ceased 3 days prior to admission.

CHRONIC

- 33 year old male with post-MBA brachial plexopathy, sensorimotor signs.
- 39 year old woman with a moderate L5/S1 disc extrusion with compression of left L5 nerve root. L5 radiculopathy.
- 50 year old male with neuropathic pain experienced in legs after SCI.
- 51 year old woman with at-level and LL pain following incomplete mid-thoracic SCI.

CANCER

- 52 year old spray painter with SCC of left vocal cord. Resolving mucositis and chest wall radiation burns.
- 63 year old man with head and neck malignancy and radiation necrosis.
- 67 year old woman with metastatic breast cancer, neurofibromatosis, neuropathic pain secondary to leptomeningeal disease.
- 48 year old male with chemotherapy induced peripheral neuropathy.

The examiners considered that the short case patients were well matched for signs and examination specific to the particular station. Short case patients are often brought to the exam at short notice with incompletely settled pain. Candidates need to be particularly cognisant of this and examine the patients accordingly, especially if examining the patient after several previous candidates. Examiners may modify the instructions regarding the examination if a patient becomes distressed.

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

Candidates may talk with the patient but do not need to take a comprehensive history. The focus should be on the targeted examination as indicated in the instructions.

Common discussion points included:

- Identification of specific anatomy
- Underlying pain mechanisms
- Targeted treatment for that patient's pain problem
- Prescription of medication

In general, this section was well done.

Communication Station: **PASS RATE 25/28 89.3%**

General information:

- This station involves an actor and addresses communication skills.
- 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's situation details:

An Interview with a Newspaper Reporter

Door information:
"Mr Ray Porter, who works for The Daily Mail, a national newspaper, has arranged an interview with you about the use of Opioid medications in the management of chronic pain.

This request was prompted by previous reports of concerns about the increasing rate of opioid prescribing, and of complications including deaths.

Mr Porter has been told that you can spare him only 10 minutes."

Expectations for the management of this interview included:

- *Clarification of the reasons for the request, at this point in time*
- *Clarification of the expectations of the reporter*
- *Other sources (MJA, Pain Medicine Suppl. Vol 12, 2011. No other interviewees, yet)*
- *Clarification of issues re publication, such as anonymity, attribution, editorial control, timing, intended purpose*
- *Identification of key issues*
- *Allowing time during interview to clarify issues with the reporter*
- *Offer opportunity for follow-up for further clarification*

OVERALL EXAMINATION COMMENTS:

This examination is structured to sample the pain medicine curriculum comprehensively and to provide multiple opportunities for candidates to display their knowledge in a variety of formats. As a result, it can be an arduous examination process. Successful candidates can be proud indeed of their achievement.

There is considerable strength in the assessment process partly due to the wide-ranging sampling and also from having two examiners at each examination point including marking the written questions. In addition, every endeavour is made to arrange minimal exposure of candidates to examiners from their own training institution, thus maximising examiner impartiality. However, time constraints implicit in multiple sampling instil some limitations, especially the brevity of both the structured vivas and the short cases. Hence there is considerable merit in practicing examination techniques specific to each section of the examination so that time is not wasted on irrelevant details.

This year, the written paper was relatively poorly done, particularly questions on addiction medicine, opioid pharmacology and the use of pain medications in the elderly, all areas a Specialist Pain Medicine Physician is expected to be expert in. There was an emphasis on opioid use in various settings once again. Detailed knowledge of these medications is essential in the practice of pain medicine and testing of that knowledge will be undertaken in every examination.

In the long cases, candidates performed the history taking section very well but fell down in the physical examination and viva sections. Again, having an organised approach to physical examination reduces the likelihood of omissions. Practice formulating the case, presenting it in an organised fashion and talking about a wide range of topics pertinent to the individual pain patient would improve performance in the viva.

Special thanks must be given to Professor Julia Fleming and her staff at the Royal Brisbane and Women's Hospital for their efforts in organising the patients and the exam venue. This year's large cohort of 28 candidates added an extra layer of complexity in identifying and coordinating approximately 40 patients as well as the candidates and examiners.

The Court of Examiners gratefully acknowledges all of the patients who willingly participated in the Examinations despite their pain. As in previous years, the patients were impressed with the examination process and the skills and professionalism of the participants.

The Court of Examiners also acknowledges the Observers for 2011, especially the International Observers, Dr Kate Grady, Vice Dean, Faculty of Pain Medicine, Royal College of Anaesthetists, United Kingdom, and Dr Donal Hearney, Chairman of Examinations,

Faculty of Pain Medicine, College of Anaesthetists of Ireland in addition to the two Observers from the Faculty who provided valuable commentary on the examination processes.

THE BARBARA WALKER PRIZE

The Barbara Walker prize was awarded to Dr Roderick Grant.

Merit Certificates were awarded to Drs Simon Cohen, Corry De Neef and James Yu.

MEREDITH CRAIGIE

Chairman
Court of Examiners

PENELOPE BRISCOE

Deputy Chair

31 December 2011

Distribution:

Faculty Board	Supervisors of Training	Examination Committee
Court of Examiners	Registered Trainees	ANZCA Director of Education

APPENDIX A

Candidates are reminded that the information published in this Appendix regarding the questions in the Written Paper is quite extensive in many cases and is intended as an **educational resource**. **All** of the information is **not required** to meet the criteria for a pass.

Question 1 – Compulsory

Define neuropathic pain and outline the clinical tools used to make the diagnosis.

What are the limitations of these tools in clinical practice?

Definitions

Old: Pain initiated or caused by a primary lesion or dysfunction of the nervous system.
New: Pain caused by a lesion or disease of the somatosensory nervous system.

Neuropathic pain is a clinical description - not a diagnosis*. Under the new definition, this description requires a demonstrable lesion or a disease that satisfies establishes neurological criteria. The term "lesion" is commonly used when diagnostic investigations reveal an abnormality or where there was obvious trauma. The term "disease" is commonly used where the underlying cause of the lesion is known (eg, stroke, vasculitis, diabetes mellitus, and genetic abnormality).

Neuropathic pain is generally characterised by being associated with a combination of positive and negative phenomena (i.e. evoked paraesthesia and/or dysaethesia, and/or sensory deficits) in an anatomically plausible neurological distribution. The presence of symptoms or signs (eg touch-evoked pain) alone does not justify the use of the term "neuropathic". The lack of operational and validated diagnostic criteria remains a difficulty in this field, probably accounting for the poor recognition of neuropathic pain and the under-treatment or inappropriate treatment of the condition.

Some disease entities (eg trigeminal neuralgia) are currently defined by their clinical presentation rather than by objective sensory testing. Other diagnoses such as post-herpetic neuralgia are normally based on the history.

It is common when investigating neuropathic pain that diagnostic testing may yield inconclusive or even inconsistent data. In such instances clinical judgement is required when allocating a putative diagnosis or group of diagnoses.

Extra comment: the removal of the term "dysfunction" from the definition of the neuropathic pain descriptor leaves *in limbo* a number of conditions thought to have a "neuropathic" component, such as CRPS type 1 and "fibromyalgia". Other conditions where both "nociceptive" and "(old) neuropathic" descriptors may be appropriate, such as post-surgical spinal pain or chronic arthropathy, where there may be signs of altered nociception but not of a neurological lesion, are also excluded from the new "neuropathic" descriptor. At this time a third descriptor to accommodate these conditions does not exist – but is needed.

Clinical tools

Sensory descriptors used in patients with neuropathic pain include:

- burning
- squeezing
- painful cold
- electric shocks
- lancinating
- tingling
- pins and needles
- itching and numbness

Features present on physical examination:

- Allodynia: pain or unpleasant sensations with light stroking/brushing (dynamic mechanical allodynia)
- Hypoaesthesia: decreased threshold to touch and/or pinprick
- Hyperpathia: abnormally painful reaction to a stimulus, especially a repetitive stimulus, as well as an increased threshold

Screening tools

Over the past decade, 5 screening tools have been developed and validated for the identification of neuropathic pain. However it must be noted that the "validation" was based on the "old" definition.

All these tools rely principally on verbal reports of pain qualities (i.e. pain descriptors).

- Leeds assessment of neuropathic symptoms and signs (LANSS)
- Douleur Neuropathique en 4 questions (DN4).

These rely upon questionnaires (symptoms) *and* physical sensory examination (signs)

- Neuropathic pain questionnaire (NPQ),
- ID Pain,
- painDETECT

These three are self-administered questionnaires.

The ability of these questionnaires to detect neuropathic pain is very good to excellent, with sensitivity ranging from 67% to 85% and specificity from 74% to 90%.

Limitations of Screening tools

- Screening tools for neuropathic pain have been validated *only* in patients with pain in a single location. These tools have difficulty assessing patients with pain in multiple sites and should not be used in patients with widespread pain.
- The screening tools fail to pick up 10-20% of patients with clinician-diagnosed neuropathic pain and cannot replace clinical judgement and general assessment of the patient.
- The questionnaires provide no information as to the cause of the neuropathic pain condition which may require a full clinical examination, imaging, laboratory and possibly electrophysiological studies.
- The screening tools are also not suitable for assessment of treatment response.

References

- Bouhassira D, Attal N. Diagnosis and assessment of neuropathic pain: The saga of clinical tools. Pain 2011;152:S74-S82
- Jensen TS et al. Commentary: A new definition of neuropathic pain. Pain 2011;152:2204-2205
- IASP Taxonomy and Pain Terms 2011. http://www.iasp-pain.org/AM/Template.cfm?Section=Pain_Defi...isplay.cfm&ContentID=1728

Question 2 – Compulsory

A patient who is taking 80mg controlled-release oxycodone 3 times a day for his persistent lumbar spinal pain is said to be "addicted".

1. **Define addiction.**
2. **How do you assess whether his use of this drug is appropriate?**
3. **What features would cause you to be concerned about the potential development of addiction in this patient?**

1. Addiction is a primary neurobiological disorder, or brain disease with genetic, psychosocial and environmental factors. It is characterised by one or more of the following:

(I) Impaired control over drug use
(II) Compulsive use
(III) Continued use despite harm
(IV) Craving

2. Assessment of appropriate use of the drug:

(i) The patient has objective evidence of organic disease or dysfunction causing nociception and pain. The demonstration of pathology is commensurate with degree of pain behaviour.
(ii) Use of the drug has been demonstrated to improve analgesia as well as aspects of function deemed compromised by the pain. TDS regime may cover some aspect of breakthrough pain and around-the-clock analgesia better. This higher dose range may lead to higher risks of tolerance & physical dependence.
(iii) The patient is psychologically/psychiatrically stable and in a stable social environment with no history of alcoholism and/or other substance abuse.
(iv) Patient should be reliable, articulate and have health-support systems and a mood appropriate to the extent of pain and dysfunction.
(v) Ideally, other conservative therapy should have been tried before consideration to the strong opioid.
(vi) Continued use contingent on continued Analgesia, no Aberrancy of drug use, stable Affect, manageable Adverse-effects and improvement in Activities of daily living provided by the analgesic. $\pm$ opioid contract.

3. Potential factors causing concern about development of addiction are when maladaptive and problematic drug seeking behaviours are manifest and patients deviate from the prescribed opioid treatment program. These include:

(i) The patient displays an overwhelming focus on opioid issues which impedes progress in dealing with other issues related to the patient's pain.
(ii) Evidence of escalating drug use and a persistent pattern (>3) of early refills in the absence of an acute change in the pathology
(iii) Persistent requests (e.g. multiple phone calls, turning up in clinics without appointments) to request more opioids and early refills.

(iv) Evidence of a pattern of prescription problems for a variety of reasons, e.g. lost medications, spilled medications or stolen medications.

(v) The patient has secured supplemental sources of opioids from multiple providers, emergency rooms or illegal sources.

References:

Graziotti PJ, Goucke CR. The use of oral opioids in patients with chronic non-cancer pain. MJA 1997; 167: 30-34.

Ballantyne J, LaForge KS. Opioid dependence and addiction during opioid treatment of chronic pain. Pain 2007; 129: 235-255.

Bailey JA, Hurley RW, Gold MS. Crossroads of pain and addiction. Pain Medicine 2010; 11: 1803-1818.

Question 3 - Compulsory

What do you tell your patients about the adverse effects of long-term opioid therapy?

It is important to note that this question only requires attention to the adverse effects, and does not require any information about the classification, efficacy, or mechanisms of action of opioid analgesic medication.

There are a very large number of adverse reactions, and a very large amount of information available about these and the management of them. Therefore a good answer would indicate to the examiner the author's knowledge of the broad range of effects, the presenting symptoms, and the significance of these effects (which would vary with the patient's actual predicament eg terminal care vs operating machinery and driving) and the influence of these on management.

This question is presented in such a manner that the answer requires the author to attend to how to effectively communicate these concepts in terms that are readily understood by the patients, with recommendations that they can readily implement. This requires use of a literacy level equivalent to Grade 6 primary school.

The adverse reactions can be grouped into those affecting particular systems.

- Conscious State
- Sedation
- Cognitive impairment
- Sleep disturbances including sleep apnoea
- Impaired psychomotor performance (notably driving)
- Loss of initiative, volition
- Respiratory Depression
- Gastrointestinal effects
- Gastroesophageal reflux
- Gastroparesis
- Nausea and vomiting
- Constipation
- Weight gain (with a contribution from inactivity and de-conditioning)
- HPA suppression, with testosterone insufficiency and sexual dysfunction
- Impairment of immunity
- Inappropriate Use
    - Abuse
    - Dependence
        - Psychological
        - Physical
            - Tolerance
            - Withdrawal
    - Addiction

1. Opioid Induced Hyperalgesia (OIH)
2. Urinary retention

3. Myoclonus
4. Cardiovascular effects

1. Conscious State

- Sedation
- Cognitive impairment
- Sleep disturbances including sleep apnoea
- Impaired psychomotor performance (notably driving)
- Loss of initiative, volition

Opioids may induce drowsiness, particularly worse when starting and in the elderly, though with significant potential for all users. Sedation is thought to be caused by the anticholinergic activity of opioids. There is some evidence that reaction decreases with time, and this is particularly a problem when changing to an increased dose for perhaps one week after that increase in dose.

Sedation will not always be apparent during a clinical interview especially if this is brief. When attending for an interview, patients will be more attentive than when not in such a stimulated context, especially when over more than a short period. More accurate assessments can be obtained from other observers, such as a partner or children.

Very mild levels of sedation may be reflected in the lower level of activity, when the patient may be less enthusiastic and less inclined (less volition) when they have a choice to be active or not. This can then lead on to reduced activity, de-conditioning, reduced intellectual stimulation and reduced social interaction.

Opioid-induced sleep disturbances: Opioids increase the number of shifts in sleep waking states and decrease total sleep time, delta sleep, and rapid-eye-movement (REM) sleep.

Advice about driving or using heavy machinery should be given. The negative effects of opioid medications on **psychomotor or driving performance** in opioid-naive patients are controversial. Many studies support that, for many patients, it is reasonable to drive when on a stable dose of opioids. There is an obligation on Medical Practitioners to report to Licensing Authorities those patients who continue to drive when at significant risk of an accident.

The suggested treatments are dose reduction, and/or opioid rotation.

2. Respiratory Depression

This can be lethal, especially in an opioid naive patient, or for a patient resuming their previous dose after a withdrawal and period of abstention. During long term use of opioid medication, in common with all sedative medication, Sleep Apnoea can be aggravated with all the associated risks, including death. An increased death rate in those using long term opioid medication has been attributed to this as one of the causes. Respiratory depression and death, although extremely rare in patients who have pain and who are properly managed, can occur. This is more likely if the opioid dose is increased without medical supervision and or the opioids are used with alcohol and/or Benzodiazepine medications.

3. Gastrointestinal effects

- impaired oral hygiene
- Gastroesophageal reflux
- Gastroparesis

- Nausea and vomiting
- Constipation
- Weight gain (with a contribution from inactivity and de-conditioning)

Virtually all levels of the GIT can be affected by long term use of opioid medications.
This starts with **impaired oral hygiene**, including as the result of reduced salivation, and decreased personal care, consistent with reduced volitional behaviour.

Gastroparesis is not uncommon, predisposing to aggravated Gastro-oesophageal reflux, with the subsequent risk of inhalation, especially if accompanied by reduced cough reflex, and deeper sleep patterns.

Nausea is usually short-lived and can be treated with anti-nausea medication such as Metoclopramide 10mg two to three times a day. Occasionally nausea persists, which should prompt gastroenterology referral and investigation for other causes and complications, such as gastroparesis and peptic ulceration.

Constipation is reported to be a problem in 40 to 95% of patients treated with opioid medications, particularly in the elderly, though not all patients get constipated. It is a significant complication, not only because of the discomfort, but also because of the potential for associated problems (urinary retention, delirium in elderly, poor tolerance of the pain, bowel obstruction, rectal prolapse, megacolon and recurrent constipation and blockage) Some patients require a GA to relieve their constipation.

Advice should be provided about a high fibre diet, increasing fluid intake, being active and the prophylactic use of laxatives, including consideration of prophylactic use, such as Coloxyl with or without Senna depending on the age of the patient.
The use with other medications, notably Amitryptyline, markedly increases the risk of constipation.
Disturbed appetite associated with GIT disturbance and activity levels can contribute to weight loss or weight gain, which accentuates these problems.

4. **HPA suppression,** with testosterone insufficiency and sexual dysfunction

The hormonal effects of chronic opioid therapy can affect both men and women. Opioid medications can cause a decrease in testosterone, oestrogen, luteinizing hormone, and gonadotropin-releasing hormone. Hypothalamic-pituitary axis, in particular testosterone production in males, can be suppressed; this usually requires high dose long-term opioids. A major effect of this is the development of osteoporosis in men and postmenopausal women with a potential for an increase in osteoporotic fractures. There is some limited evidence regarding decreased sexual function in males, which anecdotally is common, though there are many other contributing causes which need to be considered in the differential diagnosis of low libido, failure of arousal and impotence..

5. **Immunity:**

There are some limited animal studies that report depression of immune responses manifesting with increased instances of infections. The effects of this in humans are not clear but this is a potential side effect for long-term high dose opioids. This effect may be related to HPA suppression. Some of the evidence comes from the “Drug Addiction” literature, introducing the possibility of many other causative factors to consider. In clinical practice, different opioids may have different effects on the immune system (e.g. tramadol can enhance NK-cell activity, lymphocyte proliferation, and interleukin-2 release when compared to morphine).

6. **Inappropriate Use**

- Abuse
- Dependence
    - Psychological
    - Physical
        - Tolerance
        - Withdrawal
- Addiction

(*It should be noted that currently there is no universally accepted definitions for these terms, with respect to the precise detail.*
DSM 4 does not use the term 'Addiction' and describes the features used by several other authors to Substance Dependence, and uses this term to only refer to maladaptive patterns of substance use. This does not include examples of changed neurophysiology with tolerance and withdrawal syndromes that occur with appropriate, adaptive therapeutic use.
This is an area awaiting better definition.
The important issue in addressing questions about this is to be aware of the varying definitions used, and to clarify the definitions used at the time. Several definitions at present are deemed acceptable, by different authorities. The disorders continue, as they have during man's history; the issue is how these are to be described now.)

Inappropriate use is a term which can describe all the phenomena below, as well as a style of use of opioid medications that, while not sufficiently severe as to satisfy the criteria for these conditions, may present as a clinical problem because it predisposes the patient to complications without providing the patient with the desired relief and improvement.

A very common cause of inappropriate use by patients is inappropriate prescribing by practitioners.

Another very common cause is inadequate patient education.

Abuse is a term used to describe use which is clearly beyond a use intended for relief of pain, but which employs the opioid medications for another purpose. It is a maladaptive pattern of substance use manifested by recurrent and significant adverse consequences related to the repeated use of the substances. The consequences do not include the development of tolerance and withdrawal symptoms. Commonly this use will be for emotional relief, for insomnia, as well as for recreational purposes. Some people continue a pattern of substance abuse without developing substance dependence, though this remains a likely risk. Substance abuse may be associated with diversion and sale of the prescribed medication in an illegal manner.

Dependence can be both psychological and physical dependence. Identifying the presence of dependence is not sufficient. The issue is not so much whether it is present but whether it is appropriate and how it should be managed. On some occasions the reluctance of a patient to accept a degree of dependence is the problem, and may be so severe as to be pathological.

Some degree of **psychological dependence**, is a necessary part of appropriate Illness Behaviour, and allows a cooperative relationship between doctor and patient to develop so effective treatment and recovery can occur. Psychological dependence is influenced by many factors including the patients's personal style, past experiences, their belief system, their culture and the context in which they live and work. A well informed opinion needs to be developed as to whether the dependence that develops enhances or inhibits progress. Inappropriate dependence is manifest by the patient having formed the belief that they are not able to function without a particular agent (eg a medication, intervention, physiotherapy,

therapy, person) when this is actually not the case. A patient who relies on others to make decisions and carry out tasks that they are capable of themselves is unlikely to get the best result. Such opinions need a good knowledge of the conditions, of the recovery process and rehabilitation and of the patient and their circumstances.

Physical dependence is a physiological effect of opioids on the body, and can be manifest by the development of tolerance and/otr withdrawal symptoms.

Tolerance is the need for increased doses to have the same effect, because the body becomes "used to" the opioid. This may be a pharmacokinetic effect or a pharmacodynamic one. However the requirement for increased doses may not be due to tolerance but due to worsening of the disease, particularly if used for cancer.

When a physical dependence has developed **withdrawal** symptoms will occur if the opioids are stopped suddenly. Withdrawal is a maladaptive change, with physiological, behavioural and cognitive changes, that occurs when tissue levels of a drug decline in a person who has had prolonged use of the substance. The features of the withdrawal vary depending on the person and the substance involved. Weaning opioids slowly will help to minimise these effects as will the use of drugs like Clonidine.

Addiction_is a serious potentially life threatening disorder which develops to involve behavioural and psychological features as well as neurophysiological changes. The features of addiction include loss of control of the drug taking (unable to reduce use despite knowledge of the adverse consequences and attempts to reduce), salience (preoccupation with drug use and associated activities such as getting supplies, and the expected effects/relief of use), craving, loss of usual pattern of activities, relationships and standards.

Addiction to prescription medication is reported to be rare when opioids are prescribed for the management of pain. Some individuals are genetically predisposed, others begin using opioids initially for recreational purposes. Induced neurophysiological changes have been observed to persist for up to two years after last use, and remain predisposed to relapse.

Pain patients who smoke and particularly if they use cannabis, have a high alcohol intake or have had previous experiences with illegal and particularly IV drug use are at a higher risk of developing substance abuse disorders.

7. **Opioid Induced Hyperalgesia** is an enhanced pain response to a noxious stimulus which occurs after prolonged administration of opioid medications at high doses.

8. **Urinary retention**

Urinary retention due to opioid medications can occur in bed-bound patients receiving IV opioids, at times associated with constipation, but not a major problem in mobile patients using oral opioids.

9. **Opioid-induced myoclonus**

Myoclonus may be present in 3- 87% of cancer patients treated with opioids. The mechanisms by which opioids induce myoclonus are not well understood.

10. **Opioid-related cardiovascular effects**

Cardiovascular side effects of chronic opioid therapy are uncommon. However, a syndrome of QT prolongation and torsades des pointes has recently drawn attention because of an increase in the use of methadone for treatment of chronic pain.

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1. Chan BK, Tam LK, Wat CY, Chung YF, Tsui SL, Cheung CW. Opioids in chronic non-cancer pain. Expert Opin Pharmacother 2011;12(5):705-20.

2. Benyamin R, Trescot AM, Datta S, Buenaventura R, Adlaka R, Sehgal N, Glaser SE, Vallejo R. Opioid complications and side effects. Pain Physician 2008;11(2 Suppl):S105-20.

3 Editorial: Stress and Addiction American Journal of Psychiatry V 168 No 6 p566

4 DSM 4 American Psychiatric Association, 2005

Question 4 – compulsory

You are preparing a presentation for GPs on opioid prescribing in the management of patients with pain. A pharmaceutical representative has recommended that patients taking 8 (eight) paracetamol 500mg/codeine 30mg tablets per day can be switched to transdermal fentanyl 12 microgram/hour.

a) Discuss this recommendation with reference to relevant pharmacological information.
b) What advice would you provide GPs about the appropriate use of fentanyl patches?

4 a) Discuss this advice with reference to relevant pharmacological information

Opioid Equivalence:

Calculation is
Oral Morphine to codeine 1:8 so 240 mg codeine per day = 30 mg morphine per day
Fentanyl 12mcg per hour converts to between 30-60mg morphine per day

This large range is because codeine to fentanyl patch is somewhat unpredictable in the chronic setting.

May reflect:

- Incomplete cross tolerance.

- Slow metabolisers (cytochrome p450 - CYP2D6) an issue with some patients getting poor response because of failure of metabolism of codeine to morphine – 7-10% of Caucasians . Therefore they are in fact opioid naïve.

- Ultrarapid CYP2D6 metabolisers on the other hand , although rare , can have a high response to codeine because of high levels of morphine relative to usual, particularly if they have renal impairment. This would create uncertain dose requirement for fentanyl.

Analgesic equivalence – May be similar because opioid dose potentially higher but paracetamol not included. This will vary with individual pain type and individual responses to opioids.

Kinetics
Topical absorption slow and therefore fentanyl patch is more challenging to titrate and initiate. Pain relief during the change of drugs may be problematic because of the 12-18 hour onset time of the patch. (Oral medications work within 1 hour)
Distribution and excretion need to be considered in certain patient groups eg liver and renal disease - fentanyl probably safer in both considering paracetamol.

Side effects
Reduced constipation and reduced sedation are usual on fentanyl patches versus codeine

Safety
Removing paracetamol maybe advantageous in patients at risk of toxicity eg the elderly or renal impairment , hypovolaemic patients etc

Safety concerns with patches
Different level of understanding and cognitive function required for patch:
3 day dosing requires ability to use diary or similar
Avoiding local heat – risk of overdosage
Separate strategy required for incident pain
Care needed with drug disposal and storage

4b) Advice to GP's about appropriate use of fentanyl patches?

1) Consider whether pain is opioid responsive.
2) 30-60mg of morphine equivalence maybe a large dose if the patient is a non metaboliser
3) Advantages and disadvantages of fentanyl patches versus oral opioids

Advantages
Improved compliance in some patients
Reduced side effects (constipation and sedation)
More stable levels
Easier for staff / carers of incapacitated patients
Can be used in non swallowing patients
Safer in renal failure than some other opioids
More individualized that a combination analgesic

Disadvantages
Difficult and slow to titrate
Cutaneous side effects – rash in some patients
Overdose risk with local heat
Not suitable for opioid naive patients
Need to be able to keep diary / remember days for changing
Need care with storage / disposal
Need separate prescription for breakthough pain on PBS

References:

1. Cautions with Codeine: Iedema J: Australian Prescriber: Oct 2011: 34: 5: 133
2. Has the time come to phase out codeine?: MacDonald N: CMAJ: Nov 10: 182 (17): 1825
3. Durogesic Full PI: TGA approval 25/02/2011
4. The Metabolism of Opioid Agents and the Clinical Impact of their Active Metabolites: Smith H: Clin J Pain: Dec 2011:27: 9: 824
5. Codeine Intoxication Associated with Ultrarapid CYP2D6 Metabolism: Gasche Y et al **N Engl J Med 2004; 351:2827-2831December 30, 2004**
6. Pharmacogenetics of morphine poisoning in a breastfed neonate of a codeine-prescribed mother Koren G, Cairns J Chitayat D et al The Lancet, Volume 368, Issue 9536, Page 704, 19 August 2006
7. www.healthnetworks.health.wa.gov.au/cancer/.../Opioid_Conversion...

Question 5 – Compulsory

Prescription medications used in the management of pain may influence a patient's ability to drive a motor vehicle.

a) Outline how this can be assessed.
b) How can this situation be managed?

Driving and pain medication

Influences on driving from pain medication can be both positive and negative. Education of the patient and family should accompany these prescriptions. This is very important as it may impact on the health of the patient, their family and the community. Some evidence suggests that chronic pain itself has a greater impact on driving then the medications taken for it. Most evidence suggests that most patients on stable dosing of pain medication are not driving impaired by that medication.

a) Outline how these influences can be determined.

These influences can be determined both subjectively and objectively.

I. Subjectively
- Ask the patient and family if they have noticed any influences

II. Objectively
- Examination of the patient looking for drowsiness and confusion
- Number of accidents or other incidents on the road
- Formal neuropsychological testing including cognitive testing
- Formal driving assessment – this can be on-road or in a simulated situation

b) How to manage these influences.

Managing these influences can be considered from prevention and reduction aspects

I. Prevention
- Detailed education to patient and family of the risk and warning signs including the following: -
    - If feeling impaired or if there are signs of impairment should be an indicator not to drive.
    - Avoid driving soon after taking breakthrough analgesia
    - Avoid driving when the regular dose is being adjusted

- Avoid driving when in significant pain
- Adhere to the prescribed dose of pain medication
- Don't drive if any alcohol is taken in combination with the usual pain medications

- Trialling a new medication or dose adjustment during a period when the patient does not need to drive.
- Starting on low doses with gradual titration
- Avoiding polypharmacy – especially with agents that may have synergistic effects on concentration and situational awareness.
- Avoiding higher doses in the elderly
- Stop driving a motor vehicle if the drugs are needed for pain control but cause excessive drowsiness

II. Treatment

- Advise patient and family that if they notice impaired function that they should pull the motor vehicle to the side of the road and allow someone else to drive or call for assistance.
- Reduce the dose of the medication
- Change to a different pain medication

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Question 6 – Non Compulsory

Discuss the recognition, management and prognosis of Serotonin Syndrome.

1. Definition / Contextualisation

Serotonin syndrome occurs when you take one or more medications that cause high levels of serotonin to accumulate in your body.

Any drug(s) that directly or indirectly increases serotonin at the postsynaptic 5 Hydroxytryptamine 1A(5-HT1A) or 5HT2A can induce Serotonin syndrome

Serotonin syndrome can occur when you commence or increase the dose of such a drug or add a new drug to your regimen.

Excess serotonin causes an array of symptoms that can range from mild — anxiety, shivering, diarrhoea, sympathetic imbalance — to severe — odd neurological symptoms, muscle rigidity, fever and seizures. Severe serotonin syndrome can be fatal if not treated.

2. Causes

MUST include all of SSRI/SNRI's, TCA's, Tramadol, Triptans, Fentanyl – 1.5 marks

Illicit's (Ecstasy, amphetamine cocaine) – 0.5 mark

OTC's - St. John's wort, ginseng, dextromethorphan – 0.5 mark

MAOI's, lithium, metoclopramide and ondansetron – 0.5 mark

3. Presentation

Symptoms

Presents as clinical triad of altered mental state, autonomic dysfunction anf neuromuscular excitation

- Agitation or restlessness
- Diarrhea
- Fast heart beat
- Hallucinations
- Increased body temperature
- Loss of coordination
- Nausea
- Overactive reflexes
- Rapid changes in blood pressure
- Vomiting

Signs

To be diagnosed with serotonin syndrome, you must have been taking a drug that changes the body's serotonin levels (serotonergic drug) and have at least three of the following signs or symptoms:

- Agitation
- Diarrhea
- Cardiac dysrhythmia
- Hypertension (can be labile)
- Heavy sweating not due to activity
- Fever
- Mental status changes such as confusion or hypomania
- Muscle spasms (myoclonus)
- Overactive reflexes (hyperreflexia)
- Piloerection
- Pupillary dilatation
- Shivering
- Seizures
- Tremor
- Involuntary movements
- Uncoordinated movements (ataxia)
- Loss of consciousness

3. Treatment

- Admit - Patients with serotonin syndrome should stay in the hospital for at least 24 hours for close observation.
- Cease contributing drugs
- Exclude other causes for presentation(DD neuoleptic malignant syndrome or malignant hyperthermia,
- Haemodynamic stabilisation, sedation,hydration and tempraturer control
- Symptomatic / supportive intervention prn
- Cyproheptadine

4. Outlook (Prognosis)

Milder forms of serotonin syndrome may go away within a day of stopping the medications causing symptoms.

Patients may get slowly worse (esp if long half life drugs have been used eg fluoxetine) and can become severely ill if not quickly treated. Untreated serotonin syndrome can be deadly. However, with treatment, symptoms can usually go away in less than 24 hours.

Possible Complications

Uncontrolled muscle spasms can cause severe muscle breakdown. The products produced when the muscles break down are released into your blood and eventually go through the kidneys. This can cause severe kidney damage if not recognized and treated appropriately. With appropriate treatment, the condition is reversible.

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Question 7 – Non compulsory

Survivors of childhood cancer are experiencing delayed effects of disease and treatment, including chronic pain.

Discuss the psychological impediments to management of pain in a 25-year-old survivor of childhood cancer.

This question is deliberately broad. The candidate is expected to develop their answer in an organised way and dot/points note form is acceptable.

<u>The psychological impediments to pain management will be influenced by multiple factors:</u>

<u>Disease factors</u>

Type of tumour e.g. leukaemia as a toddler vs Ewings sarcoma as a teenager

Treatment (surgery, prosthesis, type of chemotherapy, BMTx)

The treatment experience (comfort kids program, procedural sedation services offered vs general anaesthesia) and recall of that

Complications experienced

- multiple admissions for febrile neutropenia, vascular access
- poorly controlled/treated pain
- radiation damage e.g. with brain/spinal irradiation: learning difficulties, gait impairment

Duration of disease free period – will influence medical vigilance/ response to symptomatology/ intervention required e.g. PET scan

Current physical impairment/comorbidity e.g. prosthesis/walking aids, toxic effects of chemotherapy e.g. cardiac impairment, secondary cancers

<u>Patient factors</u>

Age at diagnosis/during treatment

- different impact/ experience-recall of 3 vs. 15 year-old
- non-involvement with consent- existential distress

Patient's psychiatric disease/reaction during the 'cancer journey' and persistence of these –

- anxiety, depression,
- sick role adoption/secondary gain
- maladaptive attachment disorder, dependency

- post-traumatic stress disorder, needle-procedural and hospital phobia
- support group/contact with other survivors and non-survivors
- heightened response to somatic complaints (pain, nausea)
- loss of childhood – missed playing sport/dancing, school
- Cure focused
- Survivor guilt
- New onset psychiatric disorder e.g. shizophrenia

  Transition to adult centre may be resisted, have its own challenges

  Patient's self efficacy/coping strategies – ability to use guided imagery, self distract-meditate, what strategies they were taught and if able to apply to current chronic pain condition

  Pharmacological coping

  Medicalisation, exposure to multiple treatment strategies
- Multiple specialists involved
- May have fixed ideas " done it all before"
- May be 'savvy' – internet resource, support groups
- Cynical, argumentative, disengaged, disenfranchised
- Attached to doctor
- Catastrophisation - increased patient distress, partner solicitousness and poorer outcomes following self-management programs for chronic pain
- Malingering

  Opioid addiction and or divergence are possible

  Excessive vigilance, doctor seeking- concern about relapse/recurrence, needing reassurance, fight/flight/panic with new symptoms, reaction to injury/pain condition will vary and can include denial vs. excessive fear avoidance response e.g. bedrest for low-level sprain

  How patient deals with the normal issues of adolescence young adulthood – emotional separation from parents/ independence/confidence/body image/identification/peer network-support relationships/acting out/maturity vs. immaturity/

<u>Parent/family factors</u>

Birth order

Impact on/reaction of family-siblings

Parental coping/psychopathology/reaction

- anxiety, depression
- pharmacological coping for self and child
- ability to engage with non-pharmacological techniques – distract their child when coping with their own distress
- overly solicitous/protective/distressed/ catastrophisation
- tertiary gain
  - foster/maintain dependent role of young adult
  - opioid addiction/divergence

Family history of psychiatric disorder

Social factors

Missed school, education, employment

Socioeconomic status of parents and self

Financial impact – carer time off work

Literature:

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Multiple publications available from:

“Childhood Cancer Survivor Study”

”Surveillance Epidemiology and End Result database”

Question 8 – Non Compulsory

You have taken on the position of Director of Acute Pain Services in an *adult* hospital and are concerned that there is no clinical practice guideline for the use of intravenous and subcutaneous ketamine as an adjunct in acute pain management.

a) Describe the essential elements of such a guideline
b) Discuss controversies associated with the use of ketamine in acute pain
c) Outline the approach you would take in developing such a guideline

(Outline = give the main features or general principles)

Definition of adjunct = supplementary rather than essential part. This highlights that ketamine is often used to assist/enhance the effect of another drug, usually an opioid. (It may also be used in burns dressings combined with midazolam as a primary analgesic (MacPherson *et al.*, 2008)).

Answer may include the following:

1. Introductory information about Ketamine

- Ketamine in low *subanaesthetic doses* acts as noncompetitive antagonist of the NMDA receptor also binds to other PNS and CNS sites
- As adjunct principal effect is antihyperalgesic/antiallodynic not as primary analgesic per se (Not so in burns dressings)
- Used as an adjunct for pain associated with central sensitisation e.g. severe acute pain, neuropathic pain, and opioid resistant pain
- May attenuate opioid tolerance
- Useful adjunct for pain in opioid tolerant patients to reduce opioid requirements
- In burns patients may be used for incident pain associated with burns dressings to reduce trips to theatre

2. Pharmacokinetics (PK)

- Rapid onset , elimination in 2 to 3 hours
- Caution in renal impairment and hepatic impairment as dose may accumulate

3. Recommended dosing (also provide an example of how to write script and the dilutions used for junior medical and nursing staff).

- 0.1 to 0.3 mg/kg/ hour as subcutaneous or intravenous infusion
- When used in an IV PCA for burns dressings often combined with midazolam to reduce dysphoric effects. Common prescription Midazolam 0.5mg /Ketamine 10mg 5 min lockout.

4. Adverse effects/contraindications (CI)

- Cardiovascular effects: can raise BP, inc. HR and inc. cardiac output
- CNS: Dissociation, euphoria vivid dreams – some patients do not like the effects. Hallucinations can occur and appear dose related. Bolus dosing more likely to cause these hallucination symptoms
- CI: head injuries as it raise intracranial pressure (apart from potentially worsening confusion)
- CI: hypersensitivity

- CI: other

5. Setting/Monitoring

- Apart from its use for anaesthesia in theatre, may be used in Emergency department, ICU and wards though needs supervision by Palliative Care or Pain Services or consultant staff familiar with guidelines outlined here.
- Supplemental oxygen recommended during use
- Routine monitoring guidelines need establishing especially for IV PCA use

6. Storage

Store in drug cupboard and record use due to risk of misuse and diversion

b) Controversies associated with use ketamine as an adjunct for hospital-based acute pain management

1. Opioid sparing effect

- not found in a meta-analysis of paediatric postoperative pain management (Dahmani et al., 2011) - acknowledge question relates to adults
- Cochrane review (Bell *et al.*, 2006) concluded evidence supported a reduction in 24 hour PCA morphine consumption and postoperative nausea or vomiting (PONV) with subanaesthetic ketamine.

2. Prevention of chronic post surgical pain including phantom limb pain

- Yes - expert opinion (Macintyre et al., 2010)
- Does not appear to prevent persistent pain post-thoracotomy (Duale et al., 2009).

3. Reduction of hyperalgesia (Berti *et al.*, 2009)

- Nociception-induced hyperalgesia - likely though limited evidence
- Drug-induced hyperalgesia (e.g. opioid) - likely though limited evidence

Effectiveness and safety.

Benefit of adding Ketamine to i.v. opioid PCA for orthopaedic or abdominal surgery remains unclear (Carstensen *et al.*, 2010). For thoracic surgery the addition of ketamine was associated with reduced pain scores, morphine consumption and postop desaturation (Michelet et al., 2007). As part of a multimodal analgesic regimen, low-dose ketamine infusion may be able to improve postoperative pain status (Suzuki *et al.*, 2009).

c) Approach to developing a clinical guideline (NHMRC, 1998)

(List = catalogue by groups or classes with minimal explanation)

Principles:

1. Determine need and scope for guideline (outcomes based)
    a. Are there existing guidelines?
    b. Reasons for development
    c. Determine desired outcomes from guidelines (?safety etc)
2. *Use best available evidence
    a. Perform literature review
    b. If limited evidence, develop consensus recommendations
3. *Multidisciplinary input (consider consumer involvement)
    a. Pain Medicine, Nursing, Surgical, Pharmacy, Hospital Safety other

    b. Use accepted format (Local/State/National)
4. Flexibility and adaptability
5. Economic impact
6. *Dissemination
    a. Make accessible
    b. Inform target audience available (education, reminder systems, audit …)
7. *Evaluation
    a. Dissemination, change in practice
8. Revision
    a. Schedule review based on evaluation

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Question 9 – non compulsory

A 45 year old single mother and supermarket 'checkout' operator is referred by her GP with "whiplash". She reports severe neck pain and headaches since the day of her motor vehicle accident 6 months previously, and is suing the other driver. She has not driven a car or returned to work since.

a) Outline possible factors associated with acute-to-chronic pain transition <u>in this case</u>.
b) Discuss your assessment and management of this patient.

Definitions and preamble:

- 'Whiplash' is a non-specific, non-medical term; 'metaphor' for a proposed mechanism of neck injury. The term 'whiplash' should not really be used in isolation.*
- The Quebec Taskforce (on Whiplash-Associated Disorders, 1995) term: *'whiplash-associated disorder(s)' (WAD)* is most commonly used.
- Before the invention of cars, whiplash injuries were called "railroad spine" as noted mostly during train collisions. Since the invention of cars, the number of whiplash-related injuries have risen sharply due to an increase in rear-end motor vehicle collisions. The first case of severe neck pain arising from an auto collision was documented around 1919.
- **Definition of WAD(s)** (Quebec Task Force): *"Whiplash* is an acceleration-deceleration mechanism of energy transfer to the neck. It may result from....motor vehicle collisions...The impact may result in bony or soft tissue injuries which in turn may lead to a variety of clinical manifestations including neck pain and associated disorders (whiplash-associated disorders)."*
- The IASP term (Merskey & Bogduk 1994) *'Acceleration-deceleration injury of the neck (cervical sprain)*' is not commonly used.
- A 'syndrome' of neck pain (following a neck flexion-extension mechanism) associated with regional pain (headache, shoulders, back, arms, face), neurological symptoms (eg. dizziness, vertigo, auditory or visual disturbance), regional dysautonomia, widespread pain, anxiety, mood and cognitive changes.

  -acute: <2 weeks

  -sub-acute: 2-12 weeks

  -chronic: >12 weeks (3M)
- WAD is a *clinical diagnosis*; there's no 'gold-standard' test or investigation.
- Secondary headaches are a common component of WAD (IHS 2004 definitions):

  -*'chronic headache attributed to whiplash injury'*

-'cervicogenic headache'

a) Outline possible factors associated with acute-to-chronic pain transition in this case.

Summary of answer

- Severe acute pain
- Significant neck-related disability
- Female gender
- Possible pain-related anxiety (PTSD-not driving)?
- Unrecovered at 6 months
- Headaches
- Adverse work impacts, single mother
- Legal claim (secondary gain, stressor loading?)

"The mechanism of injury is different from the mechanism of chronicity." (Prof. Chris Main)

- WAD is common.

  -the most commonly reported injury after a MVA.

  -total lifetime prevalence: 2.9% (women 2.7% and for men 3.0%) (Norway)

  -annual cumulative incidence for ED attendance: ≈ 300/100 000 (variable)

  -most patients recover within 8 weeks; ≈ 2/3 are recovered at 12M.

  ≈ 1/3 of patients go on to develop chronic pain/disability at 12M.

- WAD is one of the better-studied clinical models of *acute-to chronic pain transition.*

  -consider transition to chronic neck-related disability.

  -Note pain and disability are different outcomes, although inter-dependent.

- **Factors associated with increased risk of acute-to-chronic pain (+/- disability) transition in WAD:**

  Epidemiological:

  - High initial self-reported (acute) pain intensity.
  - High initial self-reported pain-related 'disability'.
  - Psycho-social factors ('load') ('yellow flags'); "*trauma-specific psychological distress"* (NHMRC guidelines 2008); *'anxiety'* (esp. PTSD) *& 'dependency'* (passive coping style, poor self-efficacy, 'helplessness'); *'depression'.*

- Being ‘unrecovered’ at 3M (seems to be a ‘threshold’, after which chances of recovery are reduced significantly).

- **Specific factors associated with ‘failure to recover’ (pain and/or disability) at 3-6M:**

    1. High initial VAS pain score (>7/10).
    2. High initial Neck Disability Index (NDI) score (>40/100).
    3. Poor ‘coping’ (low self-efficacy, ‘helplessness’).
    4. ‘Catastrophising’.
    5. No post-secondary education.
    6. Canadian C spine-WAD grade II or III injury (see below†)?
    7. Early reports of headache (headache at baseline).
    8. Anxiety (especially PTSD).
    9. Female gender.
    10. Increased number of symptoms and self-reported severity of injury.
    11. Regional cold sensitivity (decreased cold threshold).
    12. ‘Unrecovered’ at 3 months.
    13. Older age (children recover quickly).
    14. Reduced range-of-movement of the neck.
    15. History of previous neck pain.
    16. Pre-injury work status adversely affected.
    17. Increased health care utilization in first weeks after injury.
    18. Legal/compensation case? (see below**)

- **Predictors of WAD-related disability & PTSD-both share the same trajectory post injury**. (Sterling M et al. *J Pain* 2011)

    1. Reduced cold pain threshold (OR 26)
    2. High initial pain score
    3. Age

- **Factors not associated with risk of chronic pain or disability:**

1. Severity of injury
2. Canadian C spine-WAD? (conflicting data†)
3. Collision factors
4. Radiological findings

- Effects of compensation and legal proceedings on recovery are conflicting. Recovery may be slower with 'tort' compensation cases *vs* 'no-blame' system. However 'payout' usually does not lead to recovery.** Younger age and being female is associated with increased claims.
- **Biomedical and psychological processes associated with acute-to-chronic pain (disability) transition in WAD:**

1. **Ongoing 'pain generation' in the neck:** Cervical facet joint (capsule, micro-fracture), disc disruption, myofascial trigger points (esp. trapezius), neural structures (greater occipital nerves, sympathetic chain), 'dural tears' (CSF-leak headache).
    i. cervical extensor muscle fat infiltrates on MRI at 3-6M is associated with PTSD.
2. **Development of central sensitization and neuropathic pain:**
    i. increased glutamate and glial activity in spinal cord.
    ii. dysfunction of DNIC?
    iii. development of extra-regional hyperalgesia (eg. shin) & widespread pain (fibromyalgia syndrome).
    iv. changes in hypothalamo-pituitary axis function.
3. **Activation of the cervico-trigeminal complex (headaches).**
4. **Pain-related anxiety:** (especially PTSD, catastrophization, fear-avoidance)
    i. PTSD is strongly associated with post whiplash-related *disability.*
5. **Secondary gain?** Malingering or factious disorders (somatoform disorders).
6. **Genetics?** Pain-prone genotype (COMT) for increased acute pain and distress in the ED after whiplash injury. (McLean S et al. *J Pain* 2011)

b) Discuss your evaluation and management of this patient.

Evaluation (history, examination and special tests):

- Full pain & medical history, history of the accident, determine WAD grade?
- Ask about headaches, regional & widespread pain.
- Exclude *'red flags'* (eg. trauma [fracture], neurological [radiculopathy, occult head injury], inflammation/infection).
- Ask about *'yellow flags'* (eg. anxiety, PTSD, depression, 'coping style', legal & work issues).
- Ask about 'disability.'
- Neck & regional examination (myofascial trigger points, greater occipital neuralgia, hyperalgesia, allodynia, temperature sensitivity [cold pressor], neuro-exam).
- Questionnaires/screening instruments

  VAS, Neck Disability Index (NDI), Self Efficacy Scale (SES) (self efficacy), Coping strategies Questionnaire (CSQ-CAT) (coping & catastrophising), Cold sensitivity (pressor).

  NHMRC/*TRACsa: Trauma and Injury Recovery. Clinical guidelines for best practice management of acute and chronic whiplash-associated disorders. TRACsa, Adelaide: November 2008.*

- Cervical spine & cranial imaging (headaches) if concerned about 'red flags'.

  Imaging is almost always done–usually instead of a proper examination–to have some 'documentation' and for medico-legal purposes. There are often pre-existing 'degenerative changes' on x-ray which may lead to 'unnecessary' procedures being performed (eg. facet joint injections?).
- Controlled diagnostic cervical medial branch blocks with local anaesthetic (?)

Treatment (multimodal, multidisciplinary pain *management* approach) based on;

- Recognising the role of injury *prevention*, early screening & intervention (first 6 weeks) (early video education reduces incidence of chronic pain in WAD). (level II)
- Information, education, reassurance (hurt ≠ harm), provide clear explanatory models for symptoms (level II); set obtainable, yet challenging goals.
- Multimodal analgesia (avoiding opioids where possible).^^ (see below)
- CBT/pain management programmes (esp. if added to injections, analgesia or physiotherapy). (level II)
- Treatment of anxiety (esp. PTSD) & depression.
- Aerobic exercise, functional exercise, neck strengthening. (level II)
- Myofascial trigger point injections (sterile water) (level II); lignocaine. (level II-Cochrane review)

- Great occipital nerve blocks (LA & steroid) (neurotomies?) (cervicogenic headache). (level II)
- 'Diagnostic' medial branch blocks (of mid-low cervical spine for neck pain; C2/3-3rd occipital nerve for occipital headache).
- Percutaneous radiofrequency medial branch neurotomies (level II) or pulsed radiofrequency treatments. (case series)
- Headache treatments (medication-overuse, amitriptyline, cervical blocks).
- Vestibular physiotherapy?
- Blood patch therapy? (case series)
- Surgery if cervical instability, severe radiculopathy.
- *No clear evidence of benefit* with; botulinum toxin injections (level I), collars, rest, surgery, pillows, facet joint injections, nerve root sleeve or epidural injections electrotherapy, melatonin supplements.
- For chronic neck pain including WAD, NSAIDs, muscle relaxants and analgesics-limited evidence and unclear benefits.^^ (level I-Cochrane)
- Consider effects of pain & disability on work, income, legal issues (single mother, unemployed); may need social work input.
- Counsel the patient; associations between prolonged tort legal cases and delayed recovery.

Chronic whiplash (> 12 weeks) treatment recommendations	Grade of Evidence
Treatments that should be routinely provided	
• Advice to 'act as usual' / reassurance	Grade B
• Active exercise (in combination with advice), involving functional exercises, range of motion exercises, strengthening of neck and scapular muscles, specific strengthening of deep neck flexors	Grade B
Treatments that may be undertaken provided there is ongoing evidence of benefit	
• Cognitive behavioural approach	Grade C
• Passive joint mobilisation / manipulation, in combination with active therapy	☑
• Multimodal Therapy	☑
• Vestibular rehabilitation	Grade C
• Radiofrequency neurotomy (in carefully selected cases)	Grade B
• Subcutaneous sterile water injections (in carefully selected cases)	Grade C
Treatments that should not be undertaken	
• Collar immobilisation	☑
• Prescribed rest	☑
• Surgery (other than radiofrequency neurotomy)	☑
• Cervical pillows	☑
• Intrathecal and intra-articular injections	☑
• Botox injections	☑
• Electrotherapy	☑
• Analgesic injections	☑

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Question 10 - Non-compulsory

a) **Define the concept of "catastrophising" in patients with pain.**

b) **Discuss the role of this trait in patients with pain.**

Definition

Substantial inter-individual variability exists in the strategies that are used to self-manage pain, and the most consistently important of these coping-related variables is catastrophizing.(1) Sullivan et al defined catastrophizing as an exaggerated negative orientation toward noxious stimuli.(2) It is the tendency to focus on and magnify pain sensations and to feel helpless in the face of pain.(3)

Catastrophizing can result from past experiences perceived as negative, or from threatening information coming from the environment, causing fear and anxiety.(2) Different theories have been proposed to explain why people catastrophize. Some argue that it can be attributed to negatively biased perception (i.e. cognitive), whereas others believe it is a means of dealing with distress and regulating emotion.(4)

The influence of sensitised arousal mechanisms (secondary to early life or even intrauterine maternal trauma) is thought to predispose to an exaggerated proneness to anxiety driven response sets. So too is the influence of learned response sets which may be consistent with behaviours of a parent, a family or a culture.

Catastrophizing is thought to be a multidimensional cognitive construct with three components:(5)
Magnification - "I am afraid that something serious will happen"

Rumination - "I cannot stop thinking about how much it hurts"

Helplessness - "There is nothing I can do to reduce the intensity of the pain"

To manage these negative emotions, a person pursues safety strategies such as excessive medication / substance use, the pursuit of multiple opinions and interventions, and avoidance of any movement or activity likely to elicit pain. These strategies are invariably inadequate or in other ways unhelpful, and escalate their anxiety. Whilst a maladaptive strategy reduces the anxiety in the short term, negative consequences such as deconditioning (physical, social, vocational), addiction, iatrogenic adversity etc occur in the long-term.(6)

Catastrophizing is considered to have a continuous distribution in the population without a clear cut-off level to distinguish between high and low catastrophizers.(4) Individual differences in pain catastrophization may manifest as early as adolescence.(7)

Measurement

The tools used to measure catastrophizing should contain subscales that can assess rumination, helplessness, and magnification. Two scales have been described in the literature: the Coping Strategies Questionnaire (CSQ) (8) and the Pain Catastrophizing Scale (PCS).(9)

CSQ

The CSQ scale was developed in 1983 and often is used to measure coping in patients with chronic pain.(8) Six cognitive coping strategies have been identified: diverting attention, reinterpreting pain sensations, coping self-statements, ignoring pain sensations, praying/hoping, and catastrophizing. Two behavioural coping strategies also were recognized: increased behavioural activities and increased pain behaviour. Each of the 8 coping strategies is assessed via a subscale of 6 items. Respondents indicate on 7-point scales how often they use each strategy when experiencing pain. The scale also includes 2 items that measure coping beliefs.

PCS

The PCS scale was developed by Sullivan et al in 1995.(9) It is more specific for catastrophizing and encompasses 7 more items than the CSQ. It has been developed for use in both clinical and non-clinical contexts. The items relate to the frequency of thoughts and feelings experienced when participants are in pain. The respondent is required to rate each of the 13 items on a 5-point scale. The PCS has been shown to have adequate internal consistency and construct validity.(10)

Identification of catastrophizers, through various validated measuring tools, can help optimise patient care. Reducing levels of catastrophizing has numerous benefits.

b) Discuss the role of this trait in patients with pain.

Catastrophizing is one cognitive factor that involves an exaggeration or magnification of the perceived threat of pain sensations.(11) Even in normal subjects exposed to experimental pain, catastrophizing has been found to have substantial effects. Exaggerated responses to pain can help catastrophizers obtain greater social support, which may act as a positive reinforcer.(12) However, catastrophizing can affect the quality of life by an indirect influence through other psychological factors. Catastrophizing has been associated with higher affective ratings of pain, depressive symptoms, and general affective distress.(11)

Peri-operative pain

Catastrophizing has been shown to be predictive of the severity of postsurgical pain.(5) Measuring a patient's tendency to catastrophize (using scales such as the PCS) can allow analgesia to be tailored to the patient's needs. More aggressive analgesia may need to be provided to those who catastrophize more. Educational achievement and positive social interactions may protect against some of the deleterious effects of catastrophizing. (13)

In 75 osteoarthritis patients who underwent total knee replacement surgery it was found that presurgical pain and pain catastrophizing were both unique predictors of pain reports at 6-week follow-up.(14)

Peri-operative catastrophization is becoming recognized as a key predictor of the severity of acute post surgical pain and its progression to chronic post surgical pain.(5) Through an appropriate measuring scale, the patients with a tendency to catastrophize can be identified, and the information used to optimise management of post surgical pain.

Rheumatological Pain

Catastrophizing is increasingly implicated as a critical factor shaping individual differences in the pain experience among patients with rheumatic disease.

Among subjects with tempero-mandibular joint disease, it has been shown that catastrophizing to be associated with characteristics of pain intensity, disability, and with the

onset and progression of clinically significant pain.(11) In patients with arthritis, catastrophizing was found to be associated with increased pain severity and psychological distress, and with poorer physical functioning.(14)

Summary
Independent of negative affect, catastrophizing is associated with a greater likelihood of developing chronic pain, higher pain severity (even in cancer pain), and more pain-related disability.(15,16)

Psychological distress
To manage these negative emotions, a person pursues safety strategies such as medication, substances, procedures, or avoids any movement or activity likely to elicit pain and, therefore, anxiety.(6) This maladaptive strategy reduces the anxiety in the short term, however, over time it maintains and cultivates fear, leads to reduced physical fitness, increases functional disability, and generates depressive symptoms.(17,18) Catastrophizing has been found to be associated with increased psychological distress, and with poorer physical functioning. Among pain-related coping skills, catastrophizing has emerged as a predictor of both the presence and severity of suicidal ideation, even after controlling for depression and anxiety.(19) Depression and catastrophizing are consistently associated with the reported severity of pain, sensitivity to pain, physical disability, poor treatment outcomes, and inflammatory disease activity, and potentially with early mortality. A variety of pathways, from cognitive to behavioural to neurophysiological, seem to mediate these deleterious effects. (20) Catastrophizing has its biggest impact on psychological functioning, and perhaps only indirect, and weaker effects, on pain intensity or severity. (21)

Management
Cognitive Behavioural Therapy (CBT)
Catastrophizing often decreases after psychological interventions such as CBT.(22) Particular CBT techniques used to reduce catastrophizing include thought-monitoring and reappraisal.(23) Reduced catastrophizing levels post treatment with CBT are associated with reduced reported pain intensity in studies of chronic lower back pain.(24) and rheumatoid arthritis.(25) Another benefit is fewer physician visits for the pain.(26)

Distraction techniques

Distraction techniques involve patients actively thinking about topics unrelated to their pain so that there is less focus on pain sensations.(5) Hypervigilance is a proposed mechanism for the association between catastrophizing and increased pain severity. Sullivan and Neish (27) found that catastrophizers might find it difficult to suppress thoughts about pain, possibly owing to the activation of pain schemata.

Whilst a useful short-term strategy, successful distraction does nothing to help such a person manage pain that they *do* notice. Successful distraction teaches a person that pain is bearable as long as one can bring him to not notice it.

Desensitisation
Desensitisation involves teaching a person the techniques of achieving physiological arousal reduction, then once competent the patient uses this strategy whilst thinking about the pain, rather than deflecting from it. The resultant effect is that the patient learns (both consciously and automatically) that pain awareness does not by

necessity warrant the level of distress previously experienced in association with the pain.

Other methods

Other methods to reduce catastrophizing include self-instruction techniques, emotional disclosure, public health prevention programs, and the removal of the social reinforcers of catastrophizing.(5) There is relatively little evidence on their effectiveness.

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Question 11 – non compulsory

A 65 year old man wakes up following oesophagectomy with loss of sensation and motor function in his lower body. His operation was performed under general anaesthesia and appeared straightforward. An epidural catheter was inserted prior to induction but only a test dose was administered.

Please outline:

a) The potential causes for this unexpected neurological deficit

b) Your postoperative management plan.

Answer resource:

Candidates are expected to

- Identify this as a potentially catastrophic event that requires urgent assessment and management
- Demonstrate a clinical understanding of the most likely surgical and anaesthetic causes
- Ideally demonstrate an understanding of the relevant anatomy (as this has been a past question answered poorly)

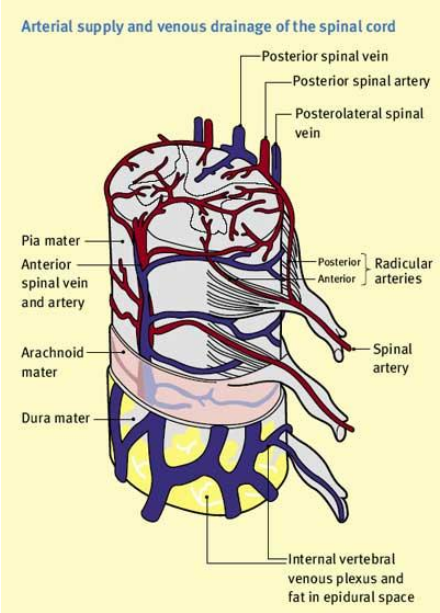
Opening statement

- The patient requires urgent attendance and assessment*
- Assessment will require urgent imaging and a coordinated response with teams involved

Potential causes

1. Drug related
   a) Prolonged drug effect from the epidural test dose - this would be unusual with an uncomplicated thoracic epidural.
   b) Incorrect drug or type of drug used for test dose including: *inadvertent neurotoxic substance injection & vasospasm with adrenaline*
   c) Misplacement of drug e.g. intrathecal/subdural/intracord
2. Ischaemic injury to spinal cord*
   a) ***Surgical*** *interruption/occlusion/surgical* ***division of aortic radicular branches*** by retractor, mobilisation of thoracic aorta, dislodgement of aortic plaques, dissection of lymph nodes, scalpel, diathermy.
   Note: extreme patient positioning may also cause compression of radicular artery passing through intervertebral foramen – older patient may have osteophytes and foraminal narrowing
   b) *intraoperative* ***hypotension***
   - *Operative blood loss/inadequate resuscitation/pharmacologically induced hypotension causing compromise of Artery of Adamkiewicz* supply – watershed of spinal cord blood supply.
   - Patient specific causes also important (see below)

3. Epidural specific
    a) *Spinal cord injury* at the time of *epidural* insertion
    - Needle related
    - Intracord injection
    b) Space-occupying lesion associated with insertion *i.e. epidural haematoma* (potentially reversible if early intervention)*



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4. Patient specific factors

Age, reason for oesphagectomy, comorbidities especially vasculopath e.g. smoker, diabetic, obesity, renal failure may increase chance of the

above occurring.

a) Hypotension:
- AMI/cardiac arrhythmia
- Aortic dissection/Thoracoabdominal aortic aneurysm

b) Extradural compression
- Metastatic lesion (particularly if oesophagectomy for carcinoma) of vertebral body
- Crush fracture
- Acute disc rupture/prolapse/extrusion

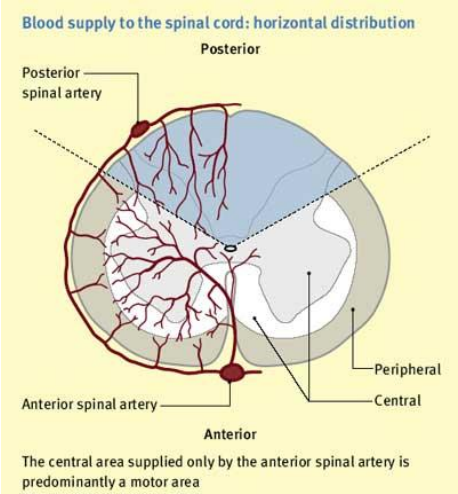
c) Intraspinal lesion least likely – primary neoplasm, AVM, transverse myelitis
d) CVA if cortical aetiology suggested on exam

Postoperative management plan

1. Assessment
    a) Liaison with the involved surgeons and anaesthetist is a priority
    b) Depending on findings urgent involvement of neurology/ neurosurgery (transfer if is not available at your centre)*

c) Urgent investigation (MRI preferable as delineates intracord oedema/infarct and epidural lesion the best, CT next preferred – will exclude extradural cause and show epidural haematoma)*

d) Determine if anterior spinal artery syndrome [supplies 75% of cord] with motor and sensory loss vs. includes dorsal columns as well [posterior spinal artery supply – 25% of cord])



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2. Management

Causes may result in a short-lived neurological deficit (be potentially reversible) or long-term deficit. The first priority initially is to determine the underlying cause and address any reversible factors.*

a) In addition to that listed above:

- Optimise patient's blood pressure, volume and Hb, coagulopathy, temperature
- Consider role of steroids if oedema/infarct (evidence equivocal); liaise with spinal trauma unit
- Urgent neurosurgery for epidural haematoma* (leave catheter in situ especially if coagulopathic)

Notification of medical defence organisation if involved in procedure would be wise.

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Question 12 – Non Compulsory

Outline an introductory lecture to be given to patients commencing a two week multidisciplinary outpatient pain management program.

The aim of the introductory lecture is to outline what the *person-with-pain* can expect from *a multidisciplinary pain management programme*; its structure, aims and expected outcomes, including;

What's involved (see below for details)? Education ('de-mystifying' pain); learning pain management skills, interacting with health care professionals and other persons-with-pain; providing *time* (2 weeks rather than minutes in a doctor's surgery or physio practice).

Who's involved? Explain the 'multidisciplinary' approach: A 'team' of health care professionals ('disciplines') is needed to manage the complexity of a person's pain experience (clinical psychology, physical therapists, dieticians, social workers, nurses other medical specialities); the traditional 'doctor-centric' medical approach is usually not the answer on its own. We (in the 21st C Western World) expect a 'magic medical cure'-a pill, injection or operation that will fix us quickly-this isn't always available in pain management, just as in other diseases like diabetes or cancer.

Team-based disease management programmes are already used with great success in diabetes, mental health, asthma, heart disease, bowel disease, cancer.

Highlight that *you* are *the key player* in the multidisciplinary team, along with family and the work place. Pain programmes are about *self management and empowerment*-doing things for ourselves rather than waiting for someone else to *fix* us.

Reinforce that we are embarking on pain *management* versus pain *treatment* and *cure*.

Set realistic expectations from the start-pain levels may not be reduced, but function and QOL are usually improved.

Explain that it's a *group-based programme &* requires *commitment* by the attendee, also respect for the privacy and viewpoints of others. It will take hard work, there will be good and bad days, and it will take continued effort after the course is over.

Explain that pain management programmes are not for everyone; eg. physical limitations, severe psycho-pathology, substance abuse, language and cultural issues etc (a review and selection process before enrolment).

Briefly highlight important pain concepts that will 'enlighten' the person and their families (who often crave a plausible explanation for their situation) eg. Bio-psycho-social paradigm, concept of pain sensitization, 'pain without evidence of tissue damage or injury', 'you can't *see* pain on x-rays', the 'sickness and stress response' paradigms.

Explain there's high level scientific evidence these programmes improve pain-related outcomes such as QOL, physical endurance, mood, sleep, medication use, work, reduced use of healthcare resources (more money and time to enjoy) and *sometimes* reduced pain levels (this is a bonus if it happens).

Knowledge is power-the programme will make the person-with-pain an expert in their own condition-promoting self efficacy and management, demystifying their situation (lessening anxiety) and allowing them to deal with conflicting information from health care providers and other sources eg. the internet.

Practical issues:

- Introductory lecture would be in the form of a Power-point presentation.
- No longer than 45 minutes: concentration, pain tolerance etc.
- Significant others, carers would be welcome to attend.
- Explain we are very flexible in terms of patient comfort, pain flare ups etc.
- Use lay terms, minimise jargon, use metaphors, 'pain stories', illustrations.
- Keep it simple to start-not too much detail or too many concepts.
- Consider the effects of language eg. pain '*patient' vs person-with-pain*, catastrophic language (neuro-linguistic programming) etc.
- Allow time for questions.
- Attendees are given written information to reinforce the lecture.
- Explain we will collect data for quality control and research-privacy requirements, consent etc.

Conclusion: Pain programmes should include;

- Education and information about pain (which promotes self-management and the ability to make informed choices about pain management strategies; promotes an understanding of pain and its mechanisms, thereby taking away some of the 'mystery', fear and threat associated with pain).
- Psychological techniques (stress management, relaxation, mindfulness, hypnosis).
- Physical therapy techniques (activity pacing, fitness, spinal core stability).
- 'Brain-power training techniques' (mirror box, virtual reality, placebo).
- Lifestyle management (sleep, medication use, workplace and home, relationships).
- Medical treatments (procedures and medications).
- Strategies to deal with pain 'flare-ups' (the bad days).
- Strategies to deal with health care professionals, conflicting advice, 'stigma of pain.'

Example of handout: Patient information introductory lecture

Adopted from STEPS: Fremantle Hospital and Health Service © 2011(provided with permission)

What is chronic pain?

Pain is defined as an **"unpleasant sensory and emotional experience associated with actual or potential tissue damage."** (International Association for the Study of Pain)

Chronic pain is "pain that continues after an injury has healed", or longer than 3 months.

Pain is the body's alarm system, warning us that our 'tissues are under threat', usually by injury or illness (for example, an acute back 'strain' or appendicitis).

The human body and especially the brain and spinal cord have evolved over millions of years to *amplify* pain 'alarm' signals (a bit like a 'hi-fi' system) so we don't ignore pain when we are injured. This is usually a good thing which helps us to survive. People born with the rare condition of pain insensitivity die at a young age because they don't realize they've been injured until it's too late.

Unfortunately, in some people who develop chronic pain, this alarm system 'keeps on ringing' even though the injury (such as a back strain) has well and truly healed. We now have an alarm system that hasn't switched itself off!

This occurs because of changes in the nervous system (called 'pain sensitization') and the whole-body's responses to stress, injury and illness, which 'amplify' (turned up the volume on the pain 'hi fi') and produced a 'memory' of the original pain signals in the brain and spinal cord, even though the injury that caused the pain in the first place has healed.

This explains why some people experience pain even though an x-ray or scan looks "normal", or why a patient with an amputated leg feels pain in his missing limb.

You don't have to have damaged tissues to experience pain; you could be experiencing a memory or 'echo' of the original pain.

This doesn't mean there's anything wrong with brain or spinal cord of patients with chronic pain (there's no problem with the 'hardware'); it's more like a 'glitch' in the 'software programme' that processes pain signals in the body.

Remember no one can see 'pain' on an x-ray or MRI scan! Some people have 'terrible' looking x-rays and yet have no pain and some have x-rays or scans with 'minor changes' but report severe pain.

Chronic pain can also develop as part of the body's response to stress: Exposure to 'stresses' that 'threaten' the body, such as **injuries** (eg. whiplash, broken bones), **illness** (eg. glandular fever virus, cancer, arthritis) or **'life-events'** (eg. depression, anxiety, work or relationships problems) cause changes in the whole-body's nervous, immune and hormone systems, producing a **'sickness response'** (exactly like a dose of the 'flu'), which sometimes doesn't 'switch-off' as expected, but continues over the long-term (especially if the 'stresses' continue).

This 'sickness response' to stress not only produces chronic pain in various parts of the body (like the aching muscles you feel when you're sick in bed with flu), but many of the other symptoms that go hand-in-hand with pain, like chronic fatigue, poor sleep, lack of energy and motivation, difficulty thinking, poor memory, depression, anxiety, sensitivity to various sensations (cold or bright lights), poor appetite and libido. In some people this is called 'fibromyalgia syndrome'.

Pain is a 'whole-person' experience: Pain affects a person's general health, ability to work, income, travel, relationships, all aspects of life. That's why we often need the help of a team of health care professionals such as psychologists or physiotherapists to treat the 'big picture' of someone's pain experience.

Pain is an unpleasant 'emotional' experience: Fifty percent of patients with chronic pain suffer with anxiety or depression.

Pain is associated with increased levels of anxiety and fear. Pain and fear may have developed together during evolution as a means of ensuring survival when primitive man was threatened by injury (such as an attack by a saber-toothed tiger!). Brain scans also show the parts of the brain controlling fear, depression and pain are closely linked together.

Pain, depression and anxiety interact in a 'vicious cycle'. When you experience pain, you may get anxious or depressed, which in turn makes it harder to cope with the pain. This is quite understandable: there are few things as stressful as living with constant pain! That's why we pay a great deal of attention to people's emotions and stress levels when treating chronic pain.

Physiotherapy and exercise are a vital part of pain management: Back pain is a classic example. People who stop exercising their back because of pain become 'de-conditioned', weakening the 'scaffold' of supporting muscles and ligaments, which in turn makes the back pain worse.

Exercise boosts your immune system and produces your body's natural pain killers called 'endorphins': This not only helps to ease pain but also makes a person feel better, just like the 'high' runners describe after jogging. In this way, exercise is also very effective in treating stress, depression, chronic fatigue (fibromyalgia) and a whole host of pain-related problems.

However people with chronic pain can easily 'overdo' their exercises and become exhausted, disheartened, avoid further exercise and get even more unfit; a 'vicious cycle'. **Pacing of physical activities**, by gradually 'building-up' stamina and endurance a little each day, is a key pain management strategy that physical therapists can teach.

Procedures and pills: Although procedures and pain killers play a valuable role in pain management, more often than not, a 'magic' injection, operation or drug that 'cures' chronic pain is not available. Most spinal injections (eg. epidurals, facet joint injections, rhizotomies) and pain killers (eg. morphine, tramadol, paracetamol, antidepressants, gabapentin, anti inflammatory drugs), only work to reduce pain in about **1 in 5 patients**, and they all have potential side effects.

Morphine-like pain killers (called 'opioids') include morphine (eg. MS Contin™, Kapanol™), oxycodone (eg. Oxycontin™, Oxynorm™, Endone™), methadone, hydromorphone, codeine, pethidine or pain 'patches' (eg. Durogenic ™-fentanyl, Norspan™-buprenorphine).

Although some carefully selected patients respond to opioid pain killers without major side effects, a small proportion may become **addicted**; these patients need a sympathetic approach including careful management of their medications and other life-style factors.

Opioid pain killers can actually *increase* pain: The over-use of opioid pain killers inhibits the production of the body's own powerful natural pain killers ('endorphins'), thereby *increasing* pain levels. This is called **opioid induced hyperalgesia**. Sometimes we have to *decrease* the dose (very slowly) or change the type of opioid (what we call **opioid rotation** or 'switching') to overcome this problem.

A 'ceiling' or 'maximum dose' is now being used by many doctors to limit ever-increasing doses of opioids which can do considerable harm. In general, if an opioid pain killer is going to work, it will do so in low-to-moderate doses (of less than 120 mg oral morphine per day or equivalent as oxycodone or patches); **going ever-higher in dose will not reduce the pain any further**. Some people's pain simply won't respond to morphine-like drugs, whatever the dose!

Goals of procedures and pills: Procedures and pain medications are rarely a 'cure' for pain, but may provide a 'window of opportunity' of improved pain control, which allows you to engage in the long-term goals of pain management such as improving self-management, physical function, fitness, mood, coping and reducing stress and medication-use.

Disappointments and frustrations: What many people who live with chronic pain realize is that despite our best attempts with injections, operations or medications, the complex and whole-person experience of chronic pain may not respond to using medical techniques in isolation.

The chronic pain experience can be very frustrating; lots of doctors, therapists, scans and procedures, conflicting information, disappointments and failures, often leading to a loss of faith and hope and even anger. "Why isn't this pain getting better"?

Managing the whole person in pain: To manage chronic pain successfully, the 'whole-person' needs to be 'engaged' in a wide range of therapies, provided by a team of experts, including the person-in-pain!

There is good scientific evidence that pain management programmes *improve* physical functioning, energy levels, fitness, coping-skills, sleep, mood, quality of life (work, social, family), and *reduces* medication and healthcare use and to some extent pain levels, in many people with chronic pain.

Modern pain management focuses on helping **you** take control and using your whole-body's resources to deal with your pain.

Although this may not sound easy, with help and support it **is** possible.

Everyone's pain experience is unique. That is why **you** are the most important person in successfully managing **your** pain.

Question 13 – Non Compulsory

A 28 year old woman had a lumbar epidural catheter inserted during labour. Twelve hours after delivery and removal of the catheter, she complains of back pain.

a) Outline the key features of your assessment of this patient.
b) List your differential diagnosis.
c) **What advice would you give the hospital risk manager regarding a system for early identification of epidural complications?**

Context:

Postpartum back pain is common. The best predictor of postpartum back pain is antepartum back pain.

Postpartum backache occurs in 3- 45% of women who have epidural analgesia for labour and delivery. There is no significant difference in the incidence of postpartum back pain in women who have epidural analgesia compared with those who do not.

a) Key features of assessment

3 essential features (in bold print)

1. Description of **back pain**
    1. Location
    2. **Severity** – 4/10 or more
    3. Features – nociceptive vs neuropathic, radiation ?radiculopathy
    4. History –
        1. Time of onset – **new pain** or continuation of pain from during pregnancy?
        2. Previous back pain/injury/surgery
2. Examination
    1. **Motor signs** – Bromage scale – signs of dense motor block, foot drop
    2. **Sensory signs** – sensory loss in dermatomal distribution
    3. Urinary retention (assessment often complicated by insertion of urinary catheter)
    4. General observations – fever, tachycardia, tachypnoea, hypotension/hypertension, examine the back for haematoma/tenderness on palpation (very non-specific)
3. Review obstetric history
    1. O-P position
    2. Prolonged second stage – head stuck on pelvis
    3. Forceps delivery
    4. ?pre-eclampsia with unidentified thrombocytopaenia
    5. Medication – history of drug misuse/abuse

4. Review Anaesthesia record
    1. Patient assessment – any medical issues e.g. pre-eclampsia
    2. Number of attempts at epidural insertion
    3. Traumatic insertion especially blood via needle or catheter
5. Investigations
    1. **Urgent MRI** for new, increasing pain with motor and/or sensory signs
    2. Nothing else urgent – CBE, blood cultures, coagulation profile

b) Differential diagnosis

1. Exacerbation of pre-existing back pain from pregnancy – antepartum backache predicts the occurrence of postpartum backache, including spinal canal stenosis, lumbar disc protrusion
2. Acute muscular pain from prolonged unnatural positioning during labour masked by epidural analgesia (more likely if high dose epidural local anaesthetic solutions used causing more dense block)
3. Sacro-iliac strain from moving lower limbs before resolution of anaesthesia/analgesia
4. Supra-spinous ligament haematoma
5. Acute disc prolapse
6. Epidural haematoma causing cord or cauda equine compression– 50% occur after catheter removal; most occur within 24hrs of catheter removal
7. Infection – generalised viral, epidural abscess (less likely to be detected in this time frame), meningitis (extremely rare)
8. Unmasking of spinal canal tumour (rare)

c) Advice for hospital risk manager

a) Education program for staff- importance of rare complications, need for urgent assessment
    1. Nurses
    2. Junior doctors
b) Change nursing observations
    1. develop specific guidelines
    2. 3 key obs (anal sphincter assessment intrusive, urinary retention often masked by use of urinary catheter)
        1. Back pain – 4 or more on 0-10 NRS scale, or if increased since last assessment
        2. Motor signs – weakness or abnormal movement
            1. Needs to be something simple – Bromage score too complex, suggest simple hip flexion- lift knee off bed
        3. Sensory signs – numbness or loss of sensation
    3. Notify predetermined designated person i.e.
        1. Acute Pain Service consultant/registrar in hours
        2. On call anaesthetic registrar out of hours
c) Review of all patients by Acute Pain Service within 24 hours of catheter removal/performance of block

d) Patient education
    1. Written and/or verbal warning
    2. Incorporate into pre-epidural procedural information
    3. Reinforcement by Acute Pain Service at review – written and/or verbal

e) Develop guideline for the management of patients who develop neurological complications following epidural analgesia
    1. Access to urgent MRI scan
    2. Access to urgent neurosurgical assessment
    3. If a) and b) not available in hospital, access to urgent transfer to hospital with such services

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Question 14 – Non compulsory

a) Briefly outline the changes that may occur in the older person in perception and report of pain.

b) In the older person, pharmacokinetic and pharmacodynamic changes may affect choice and/or dose of medication prescribed. Summarise these changes and indicate how they would influence choice and/or dose of opioid and non-opioid medications used for pain management in the older person.

Part a)

Older people show the widest heterogeneity in physical, psychological, social and functional characteristics of any age group. Decreased physiological reserves and multisystem dysregulation result in limited capacity to maintain homeostasis and respond to stressors.

a. Nervous system changes include alterations in structure, neurochemistry and function in both peripheral and central nervous systems resulting in altered nociceptive processing

    i. PNS:
        1. decreased density of myelinated and unmyelinated fibres
        2. increased number of fibres with signs of damage and degeneration
        3. slowing of conduction velocity

    ii. CNS:
        1. Dorsal horn – sensory neuron degenerative changes, reductions in neurotransmitters e.g. substance P, CGRP, somatostatin
        2. Impairment of descending inhibitory systems due to decreases in noradrenergic and serotoninergic neurons
        3. Brain- loss of neurons and dendritic connections esp in cerebral cortex including areas involved in nociceptive processing
        4. Neurotransmitter synthesis, axonal transport and receptor binding change
        5. Opioid receptor density decreased in brain but not spinal cord
        6. Decreases in endogenous opioids

b. From experimental pain studies
    i. Higher thresholds for thermal pain, not mechanical or electrical
    ii. Lower threshold for temporal summation
    iii. Reduced ability to endure or tolerate strong pain
    iv. Smaller increases in pain threshold following prolonged noxious stimulation

v. Prolonged recovery from hyperalgesia - reduced capacity to down-regulate after sensitisation

c. Increasing incidence of cognitive impairment
    i. Diminished memory
    ii. Impairment of capacity to report pain
    iii. Episodes of acute delirium more common

Outcome of these changes

1. Pain threshold is elevated
2. Pain does not function as 'early warning system' as well as in younger patients
    a. Pain becomes a less frequent and/or less severe symptom of variety of medical conditions –may be atypical pain presentation or pain complaints may be absent or reported late
        i. Pain absent or atypical in up to 50% of older patients with unstable angina and in 33% with acute MI
        ii. Older patients with acute intra-abdominal pathology report less or no pain compared with younger patients
    b. Delay in diagnosis and treatment
3. Pain intensity after surgery may be less – 10-20% decrease per decade after 60yrs
    a. Inverse relationship between increasing age and pain intensity (lower pain scores) after surgery
4. Prolonged sensitisation after tissue damage which could affect recovery from injury plus increase risk of neuropathic pain
5. Alterations in placebo response
6. Postoperative cognitive dysfunction (POCD) relatively common after non-cardiac surgery

Part b)

Heterogeneity in extent of age-related physiological changes, the presence of comorbidities and concurrent medications plus pharmacokinetic and pharmacodynamic changes influence doses required and effects of analgesic medications. These can lead to serious drug-drug and disease-drug interactions.

Pharmacokinetic consequences - most easily summarised in a table (courtesy Coldrey et al.)

Physiological variable	**Change**	**Potential pK consequence**	**Examples of possible effects on analgesia strategy**
Cardiac output	↓ or ↔	↓ CO = ↑ peak plasma concentrations with i.v. bolus	↓ initial i.v. bolus dose of CNS depressants (e.g. opioids) ↓ speed of injection
Hepatic clearance	↓ liver mass & blood flow	↑ Oral bioavailability ↓ clearance (CL) = ↑ plasma conc. For some, not all, high	Dose reductions necessary with some drugs; limited/no adjustment needed for

	↓ phase I metabolism ↔ phase II metabolism	extraction drugs (e.g. morphine, fentanyl) ↓ CL some low extraction drugs (e.g. ibuprofen)	most analgesic and adjuvant drugs
Renal clearance	↓ size & function of kidneys ↓ renal blood flow ↓ GFR	↓ CL = ↑ plasma conc. of renally cleared drugs & metabolites	Caution with renally cleared drugs (e.g. gabapentinoids, some NSAIDs) or renally cleared active metabolites (e.g. morphine, pethidine, dextropropoxyphene)
Body composition	↑ body fat ↓ body water ↓ muscle mass (range from obese to frail)	↑ Vd & t ½ of lipophilic drugs	Drug-specific – dose based on total body weight (lipophilic drugs) or lean body weight (hydrophilic drugs)
Protein binding	↓ albumin ↑ alpha-1-acid glycoprotein Drug-specific binding changes	Vd changes ↑↓ hepatic CL of low extraction drugs T ½ changes Altered cerebral uptake of drug	Possible change in clinical effect related to altered free drug fraction NSAIDs, many LAs, opioids are highly (>90%) protein bound
Oral and transmucosal absorption	Generally unaffected in absence of disease		No change in absorption
Transdermal absorption	↓ for hydrophilic drugs ↔ for lipophilic drugs	No change in time to peak conc. for lipophilic drugs	No change required for transdermal fentanyl
Intramuscular absorption	Muscle perfusion unchanged	No change	Probably minimal change
Subcutaneous absorption	Skin perfusion unchanged at normal temps	No change	Probably minimal change

Pharmacodynamic changes

Drug	Change	Potential consequence	Examples of possible changes in analgesia strategy
Paracetamol	changes in pK reported; ↓ CL & ↑ AUC with increasing age	Accumulation, possible hepatic toxicity	If no sig. renal impairment, routine dose reduction not needed
NSAIDs	CVS, renal and GI effects effects on renal blood flow Effects on GI tract CVS effects	risks & severity of side effects ↑ associated with long-term Risk of renal complications higher ↑ incidence of GI ulcers & bleeding Conflicting evidence re risk Interfere with actions of some other drugs commonly used in elderly	Short-term post-op use may be reasonable if periop renal function normal Increased incidence of renal failure with • Combination with diuretics, ACE-inhibitors and other nephrotoxic drugs • Pre-existing renal impairment, low serum albumin, hypovolaemia, hypotension Higher risk with • NSAIDs with high degree of inhibition of both COX isozymes (indomethacin, piroxicam, ketorolac, ketoprofen, naproxen) • Longer half-life, higher dose or SR drugs e.g. warfarin (↑ INR), diuretics, ACE inhibitors; ibuprofen (poss Naprosyn) may interfere with cardio-protective effects of low-dose aspirin
Opioids	Large inter-individual variation Reduced ventilatory responses to hypoxia & hypercapnia Cognitive changes	Ageing brain more sensitive to opioids Increased risk of ventilatory failure during high demand states (pneumonia, heart failure) + ↓ respiratory reserve → resp depression Increased risk with tramadol, pethidine (active metabolite, norpethidine – neuroexcitatory)	Age best clinical predictor of requirement 2-4 fold decrease in dose to get same analgesia Dose reduction may be required/frequent sedation monitoring essential Fentanyl may cause less postop confusion Adverse events less likely with fentanyl & buprenorphine (no/minimally active metabolites)

Tramadol	Active metabolite renally excreted Less effect on respiratory centre Less effect on GIT	O-desmethyl-tramadol more potent μ agonist than parent Less respiratory depression Less constipation	Increased risk of POD so may need to avoid or decreased dose May be preferable to other opioids May help to reduce opioid requirement
Local anaesthetics	↓absorption from site of delivery to systemic circulation ↓ CL ↓nerve density, conduction velocity & spinal cord neuron numbers	↓ in peak plasma levels of drug Epidural spread greater resulting in higher block level, more intense motor block	↓ infusion rate with continuous nerve blocks may be needed ↑ duration of blockade ↓ dose required for same clinical effect
Ketamine	↑CNS sensitivity with age NMDA receptor antagonists improve function in AD ↑ risk of delirium		↓ dose suggested May not worsen cognitive impairment in elderly Intraop ketamine may ↓ risk of POD and cognitive dysfunction
Tricyclic anti-depressants		↑ risk of hypotension & other cholinergic side effects with tertiary amine TCAs; less likely with secondary amines	Secondary amines (Nortriptyline) better choice than amitriptyline Start with low dose: ↑ as tolerated
Gabapentin-oids	↓ GFR		Reduce daily dose

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Question 15 – Non Compulsory

a) **Describe the effects on the clinician of dealing with "difficult" pain patients and the implications for patient management.**

b) **What are the strategies you might use to minimise such effects?**

The effect of the patient on the treating clinician is technically referred to as 'countertransference'. It has to be distinguished from the concept of transference, which refers to the patient's response to the clinician by 'transferring' feelings from important persons in their early lives onto the treating health care practitioner.

'Difficult pain patients' are generally those who arouse negative feelings within the clinician, such as frustration, dislike, anxiety, or guilt. Such patients might fail to respond to treatment modalities such as nerve blocks, medications, and physical therapy. They might be non-compliant with treatment, object to or question treatment recommendations, or fail to develop a therapeutic alliance with the clinician.

Groves described 'the hateful patient' as one who arouses strong negative feelings in the clinician, and delineated four such sub-groups of patients, whom he characterised as 'dependent clingers', 'entitled demanders', 'manipulative help-rejecters', and 'self-destructive deniers'.

A subsequent review explored what the authors referred to as 'evidence-based models of the hateful patient', and described patient, clinician and health care system factors that contribute to problems in the clinician-patient relationship. Patient factors include unrecognised personality disorders or psychiatric disorders, subclinical behaviour traits, and being perceived by the clinician as violent, demanding, aggressive, rude, seeking secondary gain, and having non-specific complaints. Unrealistic treatment expectations can also contribute to the patient being seen as 'difficult'. Clinician factors include overwork, poor communication skills, low level of experience, difficulty tolerating uncertainty, and an abrasive personality style. Health care system factors that can contribute to the characterisation of a patient as 'difficult' include efficiency pressures, financing considerations, and challenges to the clinician's authority.

The negative effects of dealing with difficult pain patients can lead to an adverse impact on relationships among pain clinic staff (especially when dealing with patients who have borderline personality traits and use 'splitting' in their dealings with others), low morale, and high staff turnover. Effects on the individual clinician might include an increased level of anxiety, due to concern about patient aggressive behaviour, feelings of helplessness, and hostility that can tend to escalate the problems. In extreme cases clinicians develop doubts about their value as therapists, and this might lead to clinical depression.

Unrecognised negative countertransference can lead to inappropriate treatment, including the failure to recognise psychosocial factors contributing to pain complaints. Inappropriate treatment has been described as 'abnormal treatment behaviour' (by analogy with Pilowsky's concept of 'abnormal illness behaviour'). It might also lead to inappropriate prescribing of ever-stronger analgesic medication, with the ultimate development of opiate abuse, the ordering of excessive and invasive investigations, or performing ill-advised surgery.

One of the consequences of such 'abnormal treatment behaviour' might be prolongation of illness behaviour.

Effective strategies to overcome such negative effects on the clinician require, first of all, that these negative feelings be recognised.

Such self-insight and self-integration have been considered to be particularly useful in enabling the clinician to gain self-understanding, and manage the patient more appropriately. Other factors described in the literature include anxiety management, empathy training, and enhancing interpersonal skills.

In multidisciplinary pain clinics there is the opportunity of discussing the difficulties with a psychiatrist or with a suitably trained and experienced clinical psychologist. Formal peer review and support groups can be utilised to help the clinician deal with 'difficult pain patients', and to recognise his or her negative countertransference.

In cases where a therapeutic impasse has been reached, the best course might be to refer the patient to a colleague. It is imperative that patients not be 'abandoned' and left without the opportunity for ongoing treatment.

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