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

EXAMINATION HELD ON 26th to 28th NOVEMBER 2008

At St Vincent's Hospital
Darlinghurst, New South Wales.

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

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

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

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

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

In 2008, 20 candidates presented for the examination and 14 were successful.

EXAMINATION **PASS RATE 70 %**

WRITTEN SECTION (See Appendix A for guides to question answers)
The following are the questions. The first five questions were compulsory.

WRITTEN SECTION **PASS RATE 40%**

General information:
As always candidates need to:

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

QUESTION 1

A 32-year-old woman who is 28 weeks pregnant is referred to your Pain clinic with severe back pain limiting her ability to walk.

You are asked to review her and advise her on possible management options.

What is your differential diagnosis?
What investigations could you use to confirm a diagnosis?
What treatment options are available?

Compulsory PASS RATE 90%

QUESTION 2

Discuss the role of ion channels in normal and abnormal nociception.

Compulsory PASS RATE 25%

A similar question was set in the previous exam and was re-set because of poor responses.

QUESTION 3

Compare and contrast opioid-associated hyperalgesia and opioid tolerance.

Compulsory PASS RATE 70%

QUESTION 4

Breakthrough analgesia has become a catch phrase for the prescription of more opioids in the clinical setting.

Discuss this issue in different clinical scenarios (Acute, chronic and cancer).

Compulsory PASS RATE 40%

This question was answered with very little pharmacotherapeutic discussion. There was repetition of material in each section of the answer. There was a lack of definition of 'breakthrough'.

QUESTION 5

You are referred a patient diagnosed with chronic pain and 'adjustment disorder'. Discuss the factors that you would elicit in your assessment of this patient, with particular emphasis on the emotional symptoms and signs.

Compulsory PASS RATE 35%

A majority of candidates did not appreciate that "adjustment disorder" was a separate condition from "Post Traumatic Stress Disorder ". Candidates did not differentiate symptoms and signs. Most candidates suggested referral to psychologist or psychiatrist but, in referring patients, the candidate should specify what they want from the consultation.

QUESTION 6

Write short notes on the relationship between childhood victimisation and neglect and chronic pain in adulthood.

Attempted by three candidates PASS RATE 67%

QUESTION 7

What factors would you discuss in obtaining informed consent in a patient who is considering dorsal column stimulation?

Attempted by 18 candidates PASS RATE 67%

This is topical question which has been covered in detail in a recent issue of "Pain Medicine". Most candidates should have had experience in discussing this issue.

QUESTION 8

An elderly patient with mild dementia has severe pain three days after hip surgery for a fractured neck of femur.

Describe your approach to evaluation and management.

Attempted by 19 candidates PASS RATE 63%

This is a basic question.

QUESTION 9

Describe the blood supply of the spinal cord.

Attempted by 2 candidates PASS RATE 50%

This is a basic question – anaesthetists in particular should know this well.

QUESTION 10

Write notes on the gender influences in pain conditions and responses to treatment.

Attempted by 7 candidates PASS RATE 71%

QUESTION 11

GPs are prescribing opioid patches for non malignant chronic pain. You are asked to give a lecture to GPs about this practice.

Give an outline of your lecture entitled "The use of transdermal opioids in non malignant pain".

Attempted by 13 candidates PASS RATE 92%

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

QUESTION 12

A 68 year old woman with metastatic breast cancer has uncontrolled bone pain in spite of regular paracetamol, NSAIDs and M.S. Contin 240 mg bd. She is requiring 50 mg mist morphine 2-4 hourly and continues to experience pain in spite of sedation.

Discuss the role of adjuvant pain therapy options for this patient.

Attempted by 11 candidates PASS RATE 65%

Many candidates discussed opioid rotation -- this is not adjuvant therapy.

QUESTION 13

(a) State the diagnostic criteria of Major Depressive Disorder? (Name the classification used).

(b) What is the differential diagnosis for a person presenting with depressive phenomena?

(c) Why are antidepressant medications used for patients with chronic pain?

Attempted by 6 candidates PASS RATE 83%

Considered a relatively easy question and well answered. Therefore, this was not a discriminatory question.

QUESTION 14

The consumption of opioids in Australian and New Zealand has risen exponentially since 1990. Discuss the factors that have led to this trend.

Attempted by 2 candidates PASS RATE 50%

This was considered to be an important topic. In view of the low response rate, it is likely to be reset in future papers.

QUESTION 15

Discuss the differential diagnosis and predisposing factors in a patient presenting with chronic pelvic pain.

Attempted by 13 candidates PASS RATE 62%

This is a common problem and therefore a straightforward question which was discriminatory.

General comments:

The relatively low pass rate in the written examination was noted. An additional 8 candidates were one mark below the pass standard.

In each question, more careful reading of the question may have provided a more accurate answer.

There was a degree of generic answering & some repetitiveness in answers.

LONG CASES **PASS RATE 65%**

General comments and observations:

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

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

Candidates are recommended to arrange with their supervisors of training to have at least five long cases observed under exam conditions prior to presenting for the examination.

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

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

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

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

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

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

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

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

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

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

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

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

- An initial brief summary of the most pertinent data.
- Their analysis of this, reflecting their judgement regarding the priorities and relevance of issues.
- Their conclusions in relation to predisposing, precipitating, perpetuating and aggravating factors.
- Management in accordance with the above.

A sophisticated experienced candidate will present this information in less than seven minutes, even with a complex scenario, making use of the concepts outlined above, without needing to use the above terms.
It is acceptable to indicate in your summary that there was particular information that you would have liked to obtain but did not. (Remember in real life we all may forget, and obtain the information at subsequent consultations.)

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

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

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

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

Candidates should expect questions on:

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

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

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

Ms MM - 44 year old woman with brittle type 2 diabetes mellitus complicated by painful diabetic lumbar plexopathy. Past history of talipes, knee surgery and multiple pain episodes.

Mr R.B. 63-year-old man with ischaemic left arm pain, past neck and back surgery, HIV-positive. Original subclavian occlusion related to antiviral medications

Mr M.W. 58-year-old man with Staph. Aureus sacro-iliac joint arthritis with piriformis myositis and sciatic nerve irritation.

In this case, appropriate physical examination should appreciate the possibility of SBE. Therefore, examination for a heart murmur and peripheral stigmata was essential

Mr B. G. 61-year-old man with complex regional pain syndrome involving right shoulder more than hand. Also type 2 diabetes and obesity.

Mr G. G. 57-year-old man -- below knee amputation -- phantom limb pain -- failed response to treatments. Pain related stump jerks.

Mr C. M. 73-year-old man with four month history of painful nondiabetic lumbar plexopathy with bilateral hip girdle weakness and foot drop.

Mrs T. K. 65-year-old woman with severe rheumatoid arthritis, oesophageal stricture.

Ms L.D. Low back pain, radiculopathy, right sciatica, eating disorder, OCD.

Mrs M. B. 62-year-old woman with trigeminal neuralgia, post-treatment analgesia dolorosa, well controlled bipolar disorder.

Mr W. I. Painful peripheral neuropathy -- retired waterfront worker with past high alcohol intake

Mr P. D. Complex regional pain syndrome type 2

Overall, candidates used the interview and examination hour reasonably well.
There is a need to stress detail in physical examination.
Some candidates were slow to commence their physical examination and did not complete a pain related general examination.
The pain physical examination needs to be ***thorough and comprehensive****.*
Candidates should outline a differential diagnosis where appropriate. This requires some general medical knowledge which was lacking in some candidates.

Some candidates were in "registrar mode" and not "consultant mode".

STRUCTURED VIVA SECTION **PASS RATE 95%**

General information

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

Candidates need to be able to explain to the examiners, as they would to patients, what they mean by the terms they use.

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

- 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 80%

The Emergency Department Consultant calls you to see a 15-year-old girl. She has presented yet again with acute on chronic abdominal pain. With every period over the last 6 months, her mother brought her to the emergency department. This time, the girl has not been to school for the last 2 days. She has been taking oxycodone slow release 20 mg twelve hourly for the pain. Her mother is demanding intramuscular morphine for the pain.

Questions:

1. What is your understanding of the pathophysiology of dysmenorrhoea?
2. What further assessments would you do? How are you going to manage the patient's immediate pain?
3. How are you going to get this girl to attend the psychologist?
4. What management options would you consider/recommend for future episodes?

KEY POINTS

1. *Pathophysiology of dysmenorrhoea*
2. *Assessment of the adolescent female presenting with abdominal pain*
3. *Psychological aspects of dysmenorrhoea as well as psychological sequelae of chronic pelvic pain*
4. *Communication with adolescent females*
5. *The practical management of pain in dysmenorrhoea*

Chronic Scenario PASS RATE 90%

55 year old female patient who has been told by her referring doctor that she has Fibromyalgia. Early in the interview the patient asks you whether "you believe" in Fibromyalgia.

Questions:

1. How would you reply to this patient's question?
2. What are the essential criteria for a diagnosis of Fibromyalgia?
3. How would you interpret these blood results?
4. Does she have an autoimmune disease?

KEY POINTS

1. *Be aware of the controversies surrounding Fibromyalgia*
2. *Be aware of the diagnostic criteria*
3. *Have a management approach with an understanding of the evidence base*

Cancer Scenario PASS RATE 85%

Female patient with cervical cancer with metastatic disease.

Questions:

1. Management of pain for primary disease

2. Management of pain from treatment modalities
3. Adjuvant therapies
4. Management of pain from metastases
   - Bony metastases
   - Lumbar plexopathies
   - Spinal medications
   - Neurolytic nerve blocks
   - Concomitant subcutaneous infusion

In the *Cancer scenario,* candidates did not focus on the anatomy of the cancer type. There was lack of a comprehensive care plan. There is a need to clarify what cancer treatments have already been given.
Candidates needed to:

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

Investigations Station PASS RATE 85%
Imaging, biochemical studies - cases taken from hospital files and on-line radiology images.

General information

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

Specific examples:

Bone scans with metastatic disease
MRI with spondylolisthesis
Cervical scans with neurofibromata & myelomalacia
Abnormal biochemical profile.

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

SHORT CASES: **PASS RATE 70%**

Short Cases with Patients:

General information

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

This year, some candidates tended not to read instructions carefully or to address the specific issues.

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

They included:

Acute:
Ms B. M. Acute pain/on methadone program / gender reassignment.
Was there another patient in this section?

Cancer:
Mr I. V. Chest pain -- lung cancer
Mr P. K. Back pain -- prostate cancer

Chronic:
Mr I. L. Chronic spinal cord injury: below- level pain
Mr K. H. Brachial plexus pain.

Examiners' comments for short cases were similar to those relating to the long cases.

Communication Station PASS RATE 80%

General information

- This station involves an actor and is looking at communication skills. The candidate should not delve too much into the history. The history given should be all that is required.

- The aim of this station is to encourage a generalised discussion of why suggested options are preferable. Informed consent may be required.

- Details of the history, and the treatment plans are not necessary.

In this scenario you have the role of a Pain Medicine Specialist, the consultant in an inpatient Multidisciplinary Pain Management Team.
The candidate is instructed to interview a member of the multidisciplinary team, a physiotherapist with concern about "burnout".

Concern has been developing about the performance of Peter Brown, a physiotherapist who joined the team 9 months ago. Late yesterday, while you were otherwise occupied, you had your secretary arrange a meeting with Peter who was told "There had been some matters of concern that are important to discuss with him."

Marking was based on:

- Interview manner, firmness, form, rapport
- Understanding and grasp of the issues and priorities (patient care, Peter's wellbeing,)
- Establishing authority, clarifying problems, offering help and remediation.
- Balancing disciplinary, boundary setting, helpful and supportive approaches.
- Practical suggestions.
- Assessing safety, offering help and support particularly with respect to professional support and development within the unit,
- Limiting personal intervention to advice on how to get treatment, not providing personal treatment

It was felt this station was well handled

The structured viva section was generally well performed. The standard in the communication section has improved in the last few years.

OVERALL COMMENTS:

This year, the examiners expressed concern about the overall pass rate of 70%. However, some candidates performed well in all areas and well-prepared candidates stood out.
Some candidates had difficulty in putting forward information and performing examinations.
(*Is this a problem with the individual candidate, the supervision or the pressure of the year of training? One year may not be long enough for training particularly for overseas candidates.)*

The examiners considered that the long and short cases were reasonable, fair and straightforward pain problems. Poorer candidates were easily influenced in the wrong direction. There was a wide range of marks in each section (both written and oral). Some core areas were not well understood by some candidates -- e.g.: the difference between tolerance and addiction; opioid hyperalgesia.

In general, physical examination should be a means of gaining marks. Many candidates did not give themselves adequate time. Supervisors of training should emphasise the need for focusing on physical examination.

The examiners are well aware of the time restrictions of the training programme and are also aware of the vast expansion of information and evidence in the various fields of pain medicine so marking guides in each section take this into account.

The examiners were mindful of the physical restrictions of the examination area and the proximity of some of the stations. Each examination centre has its own set of limitations and restrictions which, while potentially annoying, do not disadvantage candidates.

Part of the Pain Medicine Examination assesses the pain physician's involvement in a team, leadership issues and also the ability to critically evaluate the roles of other team members. A number of candidates called for consultations with other team members in structured viva and long case answers -- the answer generally requires an assessment of what is expected from other team members or consultants.

Candidates may irritate painful areas in pain patients. This is recognised by examiners and adjustments are made.
Marks were also given for candidates recognising patients' sensitivities.
Candidates need to know when it appropriate to probe and when not to, in both history and physical examination.

Patients are usually asked what they felt about the exam. This year they were generally impressed with the examination process and several expressed interest in being called for future examinations.

The role of examination observers is extremely important. External observers bring different perspectives and assist greatly in transparency and quality assurance. New examiners are able to critically evaluate the strengths and possible difficulties with each examination section. We are extremely grateful to Dr Michel Dubois Director, NYU Pain Management Centre and the Dean of American Academy of Pain Medicine for his oversight of this examination. Drs Mark Tadros and Diarmuid McCoy provided helpful observer's comments.

In particular, it is important to provide the examiners and candidates with clear guidelines in relation to the level of rigor required as well as maintaining a transparent process. This relates particularly to marking of written questions, instructing candidates before each section and to provide candidates with a "timeline" to answer questions. It is important for candidates to know when to stop.

Special thanks must be given to Dr Steven Faux for his efforts in organising the patients and the exam venue and to Melinda Gamulin and the staff of the St Vincent's Clinical School for their assistance with the examination rooms.

THE BARBARA WALKER PRIZE

The Barbara Walker prize is awarded to the leading candidate provided the total mark is above 70%. This year the prize was awarded to Dr Charles Kim, Victoria.

A merit award was awarded to Dr Richard Sullivan, Victoria.

RAY GARRICK
Chairman
Court of Examiners

MEREDITH CRAIGIE
Deputy Chair

December 2008

Distribution:

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

APPENDIX A

QUESTION 1 **COMPULSORY**

A 32-year-old woman who is 28 weeks pregnant is referred to your Pain clinic with severe back pain limiting her ability to walk.
You are asked to review her and advise her on possible management options.

What is your differential diagnosis?
What investigations could you use to confirm a diagnosis?
What treatment options are available?

Back pain occurs at some time in every second woman.
35.5% women report at least one episode moderately severe back pain during pregnancy. Increased by younger age, "ill health", unemployed.
> 1/3 interferes work, ADL's.
> 1/3 interferes sleep.
10% can't work.
Occurs usually 12^{th} – 28^{th} weeks.
Often decreases after 30 weeks!
Possible causes include:

1. Obstetric causes.

- Spontaneous abortion / premature labour.
- Antepartum haemorrhage .
- Pre-eclamptic pain.
- Infections.
  - UTI's
  - Osteomyelitis.
- Femoral vein thrombosis.

2. Mechanical causes.

- Weight gain (in pelvis).
- Anterior abdominal protuberance.
- Increased lumbar lordosis.
- Shift body back – center gravity.
  - disc herniation.
  - facet "stress".
- Pelvic insufficiency
  - (pubic symphysis (1/36 deliveries).
  - SI joint subluxation (incidence approx 25%)..
  - Pubic diastasis (gap >10mm) - rare
- Sciatica - herniated disc 1/10,000.
- Lumbar disc pathology (2.5/1000 live births)
- Spondylolisthesis (1/1000 live births).
  - Postural back pain.
  - Thoracic pain (10%)
  - Coccydynia

3. Hormonal causes.

- Total body water increases 8.5 litres
  - Amniotic fluid / placenta / connective tissue.
  - Oestrogens are natriuretic.
  - renin / angiotensin II / aldosterone / ADH.
- Increased laxity joints spine / pelvis.
- Endocrine changes.
  - Increase Relaxin
    - Increasing pain with increasing relaxin levels.
    - Regulates collagen
    - Softens ligaments pelvis / cervix.
    - Decreased substance P / Increased Progesterone.
      - Increased Pain threshold?

4. Not related to pregnancy.

Acute appendicitis (1/5000).
Rheumatoid or osteo - arthritis.
Ankylosing spondylitis.
Neoplasms
Lymphoma
2^{o} breast / ovary / thyroid.
Multiple myeloma
Osteoporosis / osteomalacia
Sickle cell disease.
Torsion ovary.
Chronic Pain Syndromes.
There is a high incidence of abuse during pregnancy?
25%♀ report DV - ↑ pregnancy.

What investigations could you use to confirm a diagnosis?

Blood tests looking for inflammatory markers, infection etc.
X-rays.
Not absolute CI
Safe exposure < 10 rads.
MRI
Revolutionised imaging.
Safe and useful.

What treatment options are available?

Initially aim to use non-pharmacological treatments:

- Reassure patient
- Patient education.
- Physio. General advice back care.
  Advice re lifting etc.
  Maintain normal activity.
  Hydro / gym
- Support/ belts/ crutches/ wheelchair etc
- Trochanteric belt.
- Pillows.
- TENS.
- Avoid smoking. Psychology – relaxation/ hypnotherapy.
- Epidural steroids controversial. Reserve patients new onset signs (unilateral loss deep tendon reflex or sensory / motor changes)

What drugs could you recommend?

1^{st} tenet medical management pregnant patient “minimize use *all* medications”.

Use non- pharmacological therapies whenever possible.
No drug used currently to treat pain, is risk free.
Drugs are classified by the TGA into classes depending on their safety:
Category **A,B1,B2,B3,C,D,X** (See below).
Combination paracetamol, opioids, low dose TCA's or SNRI's aiming to cease prior to delivery, plus Gabapentin would be reasonable.

Paracetamol (A) / Used 41% women
Low dose aspirin (C) / ↓PIH, recurrent abortion, IUGR.
Aspirin (C) – inhibits PG, closes fetal DA, delays labour.
Codeine (A)

NSAID's (C)
Don't effect organ development but alter physiology pregnancy.Delay onset of labour.
Decrease amniotic fluid volume.
Increase risk pulmonary hypertension.Early use: - malformations? / ↑ miscarriage
PDA closure?
mid trimester OK.
Opioids (C) Respiratory depression in neonate / Withdrawal symptoms prolonged use.
No increase congenital defects.

Antidepressants:
Tricyclics (C)Prolonged high dose cause withdrawal symptoms. Wean last weeks.
Mirtazapine B3Venlafaxine B2.
Paroxetine was C → D / 1st trimester↑ CVS malformations.

Anticonvulsants: Mainly (D)
Risks abnormal child ↑ 2x.
Women need pregnancy counselling. Use mono-therapy / lowest dose.
Folic acid from 4 wk before until 12 wk after conception and do a mid –trimester US.
1% ♀ carbamazepine → spina bifida.
But Gabapentin B1Pregablin B3, has not been used enough.
Lamotrigine was B3 → D

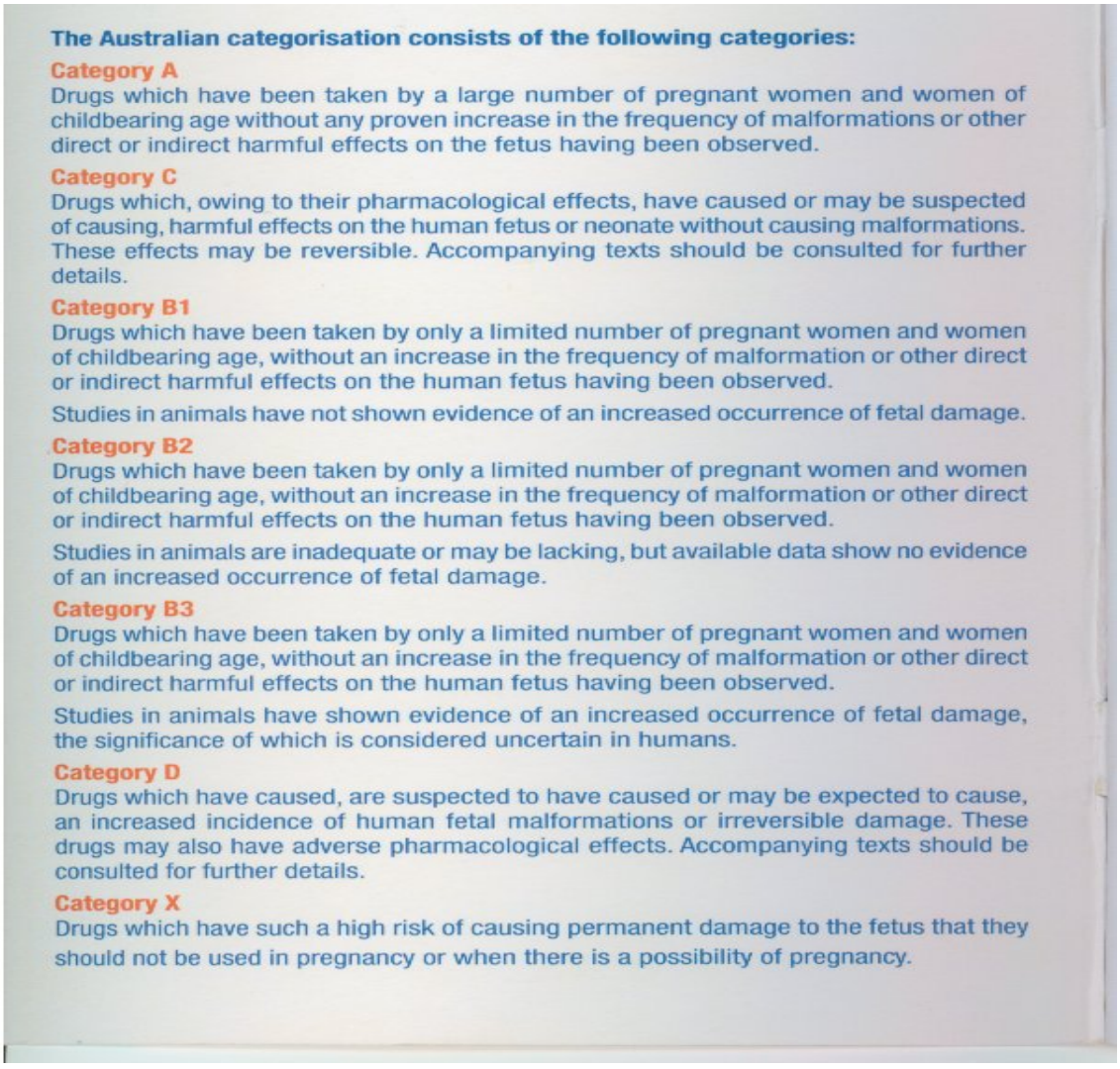
Local Anaesthetics:
Bupivacaine, lignocaine A
Ropivacaine B1
Procaine B2.
Continuous infusions dilute solutions small placental transfer
Where can you go to get this information?
Pharmacists' local Womens and Paediatric hospitals.
ę- Mims / Internet.
Therapeutic Goods Association Drug Evaluation Committee

References

- Back Pain & pregnancy: a review: Mac Evilly; Buggy: Pain 64 (1996) 405.
- Management non-obstetric pain in pregnancy: Rathmell; Viscomi: A&A: 1997:85: 1074
- Back pain, pregnancy &childbirth: Reynolds: BMJ: 1997: 314:1062

- Prescribing medicines in pregnancy: 4th edition: TGA 1999Stapleton: SA survey: ANZJOG: 2002
- Management Non-obstetric pain in pregnancy: Pyke; Shutt: RA&PM: 2003: 28: 54
- Treatment of pain in pregnancy: Wunsch; Stanard: CJP: 2003: 19: 148
- Symphysis pubis dysfunction: a review: Leadbetter; Mawer: J Maternal-Fetal Neonatal Medicine: 2004: 16: 349.

Australian Drug Evaluation Committee Prescribing Guide



The Australian categorisation consists of the following categories:

Category A
Drugs which have been taken by a large number of pregnant women and women of childbearing age without any proven increase in the frequency of malformations or other direct or indirect harmful effects on the fetus having been observed.

Category C
Drugs which, owing to their pharmacological effects, have caused or may be suspected of causing, harmful effects on the human fetus or neonate without causing malformations. These effects may be reversible. Accompanying texts should be consulted for further details.

Category B1
Drugs which have been taken by only a limited number of pregnant women and women of childbearing age, without an increase in the frequency of malformation or other direct or indirect harmful effects on the human fetus having been observed.

Studies in animals have not shown evidence of an increased occurrence of fetal damage.

Category B2
Drugs which have been taken by only a limited number of pregnant women and women of childbearing age, without an increase in the frequency of malformation or other direct or indirect harmful effects on the human fetus having been observed.

Studies in animals are inadequate or may be lacking, but available data show no evidence of an increased occurrence of fetal damage.

Category B3
Drugs which have been taken by only a limited number of pregnant women and women of childbearing age, without an increase in the frequency of malformation or other direct or indirect harmful effects on the human fetus having been observed.

Studies in animals have shown evidence of an increased occurrence of fetal damage, the significance of which is considered uncertain in humans.

Category D
Drugs which have caused, are suspected to have caused or may be expected to cause, an increased incidence of human fetal malformations or irreversible damage. These drugs may also have adverse pharmacological effects. Accompanying texts should be consulted for further details.

Category X
Drugs which have such a high risk of causing permanent damage to the fetus that they should not be used in pregnancy or when there is a possibility of pregnancy.

QUESTION 2 **COMPULSORY**

Discuss the role of ion channels in normal and abnormal nociception.

Ion channels are significantly expressed in sensory nerves as a means of controlling electrical membrane stability, propagation of action potential, and initiating neurotransmitter release

Voltage Gated Sodium Channels (VGSC) – "Na Channels"

- Large transmembrane proteins with voltage-gated central pore selective for Na+ ions
- At Least nine subtypes $Na_v1.1$ to 1.9 divided into two groups
    - TTX sensitive Na_v 1.1-1.4 & 1.6,1.7
    - TTX insensitive $Na_v1.5$,1.8.1.9
- Na_v 1.3 ,1.7,1.8, 1.9 known to be on nociceptive neurons
-
- Critical determinate of axonal electrical excitation with Na+ channels opening to overwhelm K+ efflux to produce action potential
- Pacemaker activity restricted to distal component of sensory nerves
- Sensory Na+ channels have a turnover ½ life of 1-3 days transported from DRG to periphery
- Absence of $Na_v1.7$

- **In Injury**

- Inflammatory and neuropathic pain states are characterised by alterations in VGSC subtype composition and activity in sensory neurons
- Accumulation of Na channels in injured nerves e.g. neuromas, areas of demyelination or axonapathy leading to remodeling & increased excitability to stimuli
- Increase ability to take on pacemaker activity
- Phenotype change in Aß fibres
- Drives central sensitization
- TTX sensitive $Na_v1.7$
    - Congenital absence - inability to feel pain
    - Mutations associated with erythromelalgia
    - Associated with inflammatory pain
- TTX sensitive $Na_v1.3$ up-regulated fastest after injury
- TTX resistant Na_v 1.8 & 1.9 likely to be important as selective for sensory neurons and known to be translocated in nerve injury and by inflammatory mediators
- Anti-neoplastic drugs (Taxans, Vincaalkaloids,platinum –based) cause neuropathy due effect on$Na_v1.3$ and ? $Na_v1.7$
- Drugs effective
    - Lignocaine, bupivicaine, mexilitine
    - Phenytoin, Carbamezapine, lamatrogine (? Clinical effectiveness)
    - Tricyclic antidepressants(some only eg like amitryptilline)
    - Physeptone ? celecoxib

Votage Gated Calcium Channels (VGCCs) "Ca Chanels"

- Increase intracellular Ca+ contribute to depolarising membranes current, initiate transmitter release, initiate increased transcription and initiate phosphorylation of membrane proteins e.g. NMDA receptors
- Within the spinal cord, increases in the activity of calcium channels contribute to wind up and central hypersensitivity
- Depolarisation of membrane leads to opening of voltage-sensitive calcium channels
- Defined as either high or low voltage, structure sub unit classification and their pharmacology –high voltage related to neurotransmitter release
- Classified as high voltage (L,N,P/Q and R –Type) or low voltage (T-type)
- L-Type –located in dorsal horn In astrocytes after activation. In microglia blocked by verapamil
- N-type located in DRG and in superficial dorsal horn including those containing substance P. CRGP and glutamate. Ziconotide (it) shown to reduce allodynia
    - Upregulation occurs after nerve section and may contribute to increased spontaneous activity .
- P/Q type implicated in migraine
- Sub units on VGCC:- Many of the α2δ-1 upregulated in nerve damage increasing Ca+ influx and neurotransmitter release. Blocked by gabapentin and pregabalin
- Upregulation of voltage-gated calcium channel (VGCC) alpha2delta1 subunit (Ca(v)alpha2delta1) in sensory neurons and dorsal spinal cord by peripheral nerve injury has been suggested to contribute to neuropathic pain
-
-
- **K Channels**
- 4 types (Kv,$K_{Ca}K_{2p}$(2))
- Increased expression in DRG in nerve injury
- The M-Type Kv Channel involved in regulation of nociceptive sensory activity
- Blocked by retigabine
- Kir channels – 7 types – 2 involved with nociception (Gprotein-regulated and ATP sensitive
    - Implicated in the effect of morphine, baclofen, clonidine, nicotine

Transient Receptor Potential Vanilloid (TRPV) Channels

- Located in nociceptive neurons located in DRG, trigeminal ganglia nodose sensory ganglia
- Phosphorylation and dephosphorylation markedly alter TPRV 1 ion function altering pain sensitivity .
- Inflammatory mediators phosphorylate TRPV1, increasing pain hypersensitivity

Others

1. Ankkyrin-Repeat TRP 1 Channels
2. P2X Receptors
    - Upregulated in neuropathic pain
3. ASIC3
    - Tissue acidosis – Mechanical hypersensitivity
    - Inflammatory pain
4. Hyperpolarisation-activated Cation Currents (1h or HCN)
    - Related to the voltage-gated K+ channel superfamily
    - Conducts both Na+ & K+ ions

- Involved in hyperexcitability and spontaneous neuronal firing in damaged peripheral nerve and DRG

References:

1. Devor,M: *Sodium Channels and Mechanisms of Neuropathic Pain* J of Pain Jan 06 Vol 7 Supplement
2. Park SY et al: *Celecoxib inhibits Na+ Channels in rat dorsal root ganglion* Brain Research 1148:53-61 2007May7
3. Vink IA, et al: *Lamotragine for the treatment of pain associated diabetic neuropathy: Results of two randomised, double-blind, placebo controlled studies;* Pain 128 (2007) 169-179
4. Yaksh T.L.,Calcium; *Channels as therapeutic targets in neuropathic pain* J of Pain Vol 7 No1Supplement 1 13-s30January 2006 ppS
5. Xie Wenrui; *Ion Channels in Pain Transmission*
6. Cummins TR et al *The roles of sodium channels in nociception: Implications for mechanics of painI* Pain 131 (2007) 243-257
7. Cao You-Qing *Voltage-gated calcium channels and pain*:Pain 126 (2006) 5-9
8. Ekberg J, Adams DJ Neuronal voltage-gated sodium channel subtypes: key roles in inflammatory and neuropathic pain, International Journal of Biochemistry & Cell Biology. 38(12):2005-10, 2006.

QUESTION 3 **COMPULSORY**

Compare and contrast opioid-associated hyperalgesia and opioid tolerance.

Introduction

A patient who describes an increase in pain while on opioid therapy may be experiencing opioid associated hyperalgesia (OAH) or opioid tolerance (OT).

This occurs when opioids are given over a prolonged period to prevent chronic pain. Opioids can activate pronociceptive mechanisms resulting in heightened pain sensitivity.

Tolerance definition:

This is a state of adaption in which exposure to a drug, e.g. opioids, induces changes that result in a diminution in one or more of the drug effects in time. *(Definition, American Academy of Pain Medicine, 2007)*

In both cases, the condition develops over a prolonged period of administration of opioids for chronic pain and also in some acute/subacute settings.

The physiological cause of opioid-associated hyperalgesia includes a sensitisation of the primary afferents, upregulation of the glutamatergic transmission at the spinal level, an increase in endogenous dynorphin and descending facilitation. It is a pronociceptive mechanism. There is a decreased nociceptive threshold as a result of the prolonged opioid administration.

The physiological cause of tolerance is due to a diminished opioid efficiency at the level of the receptors and post-receptors. Clinically, in animal studies, there are changes in the baseline nociceptive threshold which can be assessed by observing changes in the reflex responses in a controlled manner. Similar changes can be seen in humans. Pain sensitivity is increased in individuals with opioid addiction, for example those on methadone maintenance.

Clinically, opioid-associated hyperalgesia is characterised by changes in the spread, the quality and the intensity of the pain. This diffuse hyperalgesia is often a clinical characteristic.

Clinically, tolerance may be associated with physical dependence and drug abuse/dependence but each may occur separately.

"Pseudo-tolerance" may occur in the presence of:-

1. A progression of the underlying pathology;
2. A development of secondary pathologies;
3. Changes in the pharmacokinetics of the drugs;
4. Drug interactions; and
5. Significant psychological issues.

Clinically, with tolerance, there is a worsening of the existing pain state. As a result, an opioid dose escalation is the normal approach to restoring effective opioid analgesia.

The clinician is faced with a decision; is the increasing pain sensitivity an ineffectiveness of the opioid due to tolerance or due to opioid-associated hyperalgesia?

In opioid-induced hyperalgesia, the increase in the dose of the opioid may intensify the clinical hyperalgesia and it may improve after supervised opioid tapering. In contrast, opioid tolerance can be overcome by a trial of an opioid dose escalation.
Compare and contrast opioid associated hyperalgesia and opioid tolerance

Management:
In opioid-associated hyperalgesia, the management is focussed on rotation of the opioids. For example, patients on a dose of 400 mgs of Oxycontin per day might be placed on 50 mgs of Methadone. Drugs that act on the single u-opioid might be replaced by ones that act on a multiplicity of sites. Clinically, in tolerance, the cause of the tolerance needs to be re-examined. A range of issues as noted above under pseudo-tolerance needs to be excluded. Addiction and drug dependence need to be considered. At times, the dose of the opioid will need to be increased as a result of the diminished opioid efficacy at the level of the receptors.

Compare and contrast opioid associated hyperalgesia and opioid tolerance

These effects will be compared in the following table.

	OAH	OT
Definition	An increase in pain response to a noxious stimulus induced by the exposure to an opioid	A reduction in therapeutic effect of an opioid on a stable dose requiring an increased dose to maintain that effect
Mechanism	Multiple mechanisms are likely to be involved including sensitisation of primary afferent neurons, enhanced release &/or reduced uptake of excitatory neurotransmitters in the dorsal horn, sensitisation of second order neurons and neuroplastic changes in the rostral ventromedial medulla. The NMDA receptor is thought to play an important role. Recent research also suggests that glial cell activation may have an important role to play. The accumulation of pro-nociceptive opioid metabolites has also been	Many mechanisms of tolerance. May innate or acquired. Acquired involves pharmacodynamic and pharmacokinetic changes. Pharmacodynamic tolerance – this is due to a change in the response to the opioid due to alterations at a receptor level. This mechanism may involve receptor internalisation but the exact cause remains under investigation. The NMDA receptor may also be involved. Pharmacokinetic tolerance – this is due to alterations in the distribution, metabolism and elimination of the opioid with a reduced delivery to the therapeutic target.

	suggested - particularly morphine-3-glucuronide.	
Animal evidence	High dose opioid causes an increase in tactile allodynia and hyperalgesia.	Repeated opioid dosing shifts dose-response curve to right.
Human evidence	Prolonged secondary hyperalgesia in human volunteers following remifentanil infusion. Studies in subjects on a methadone maintenance program suggest they are more sensitive to cold pressor pain. Intraoperative remifentanil infusion has caused increased post-op pain and peri-wound hyperalgesia. Case series of cancer patients on high dose morphine with widespread pain that reduced with dose reduction or opioid rotation.	Limited clinical evidence. Difficult to measure response to opioid therapy. Some studies have shown intraoperative remifentanil infusion results in increased post-op opioid requirement with no change in pain scores. Tolerance to opioid side-effects occurs with nausea and respiratory depression but not to constipation.
Diagnosis	The differentiation between OAH and OT can be difficult. The pain of OAH is potentially more diffuse and distant from the original pain site. May have a more sensitive response to Quantitative Sensory Testing. Improved pain with dose reduction and/or opioid rotation.	Pain more localised to site of pathology. Responds to a dose increase.
Treatment	Education of patient. Dose reduction and/or opioid rotation. Addition of non-opioid adjuvant agents as indicated. There is weak evidence on the use of perioperative low-dose ketamine suggesting it may modulate OAH. S-ketamine may be a better choice. Utilisation of non-pharmacological pain management strategies.	Education of patient. A careful dose increase with monitoring of effect including function. Addition of non-opioid adjuvant agents and non-pharmacological pain management strategies.
Differential Diagnosis	These two opposing phenomena may coexist or not be present at all. It is important to carefully reassess the patient who describes an increase in pain and consider the following differential diagnoses – • Disease progression • New pathology • Pain that is not responsive to opioids • Neuropathic pain • Psychosocial stressors • Opioid addiction	

	• Opioid physical dependence

References:

- Pain Clinical Updates, International Association for the Study of Pain, Page 1-4, February 2008
- Opioids in Non-Cancer Pain, Kathy Stannard et al, Oxford University Press, 2007.
- Pain; Wall & Malzack, Churchill Livingstone, Page 1187
- Chang G, Chen L, Mao J. Opioid tolerance and hyperalgesia.Med Clin North Am. 2007 Mar;91(2):199-211.
- Chu LF, Angst MS, Clark D. Opioid-induced hyperalgesia in humans: molecular mechanisms and clinical considerations. Clin J Pain. 2008 Jul-Aug;24(6):479-96.
- DuPen A, Shen D, Ersek M. Mechanisms of opioid-induced tolerance and hyperalgesia. Pain Manag Nurs. 2007 Sep;8(3):113

QUESTION 4 **COMPULSORY**

Breakthrough analgesia has become a catch phrase for the prescription of more opioids in the clinical setting.

Discuss this issue in different clinical scenarios (Acute, chronic and cancer).

Breakthrough pain has not been defined by the International Association for the Study of Pain but is an entity that has been identified since the early 1980s. It is to be found in the three broad categories pf pain acute pain, persistent / chronic pain and cancer pain. It can be defined as a flare up of moderate to severe intensity in a patient with background pain considered to be of moderate to severe intensity which is under acceptable control. Breakthrough pain has very different implications in each of the three main pain categories. Pain must always be assessed and treated in a biopsychosocial milieu. The management of breakthrough pain is no different.

Acute Pain

Breakthrough pain in patients suffering acute post operative or trauma pain may be due to a number of factors. Repeated assessment of the patient is required to identify the cause and possible treatment. It is necessary to identify if breakthrough pain is an increased level of the underlying pain, from the presenting pathology or a new pain emerging as a consequence of the medical / surgical intervention.
An increased level of the underlying pain may be associated with deep breathing, movement, personal activities of daily living etc. This is best assessed by functional activity scores rather than isolated pain scores. Tailoring a patient controlled analgesia regimen, regional analgesia infusion technique or oral medication may be then undertaken. An estimation of the patients premorbid analgesic use, pain experience, learned pain behavior substance use misuse or abuse may be useful in influencing the approach adopted. Should there be an increase in pain score following an initial improvement a new pain generator should be sought. Laboratory investigation and imaging should be sought. Examples of this scenario might be anastamotic breakdown and leak, or compartment syndrome. Finally acute neuropathic pain is emerging as an identifiable element in acute pain. This may form part of breakthrough pain and needs to be approached in a different way.
The key to treating acute breakthrough pain is assessment of pain level and activity repeatedly and assessment of the effect of any change introduced. Breakthrough pain in the acute pain setting is common and might be considered predictable

Chronic / Persistent non cancer pain

Breakthrough pain in persistent pain is a complex and may be multifactorial. It is less predictable and more difficult to manage. Chronic pain either neuropathic or nociceptive pain can flare for no identifiable reason. Breakthrough or flare ups in pain can be an amplification of the underlying pain, a manifestation of distress, or an opportunity to report an unrelated health matter. Measures of pain scores, functional activity score, catastrophising scale and pain and disability ratings may be useful especially if historical scores are available for comparison. New symptoms associated with breakthrough pain should be assessed and investigated if necessary. The patients health anxiety should not allow dismissal of symptoms by the physician. Similarly the degree of anxiety may be heightened by unnecessary investigation. Other strategies that can be helpful for breakthrough pain episodes include relaxation training, hypnosis, and cognitive behavioral therapy, although scientifically controlled

studies have not validated these techniques in breakthrough pain. Patient education and family involvement are critical and should be addressed early in treatment. Frequent reports of breakthrough pain may necessitate a harm reduction / mimimisation or containment strategy. This may take to the form of reassurance that hurt does not always accompany harm or infrequent but regular interventions to maintain the best quality of life and optimal function despite pain

Cancer pain

It is probably in the area of cancer pain that breakthrough pain has been best described and studied. Flair ups in cancer pain are to be expected but may be unpredictable. It may be a sign of an inadequate underlying level of analgesia, end or dose failure, or suboptimal analgesic agent. Patients with cancer fear pain from early in the course of their treatment. Modern cancer treatments are successful in treating the primary pathology but may be responsible for introducing additional pain syndromes eg mucositits, radiation burn injury pain associated with opportunistic infection. Peripheral neuropathic pain and some visceral pain syndrome are attributable to chemotherapeutic drugs and radiation therapy. In assessing patients breakthrough pain consideration must be given to possible causes eg progression of the primary tumour, metastatic deposits, structural destruction due to the primary or secondary tumour ie pathological fracture or pathological condition unrelated to the primary cancer eg acute appendicitis, prolapsed intervertebral disc, or trauma.

Pseudoaddiction is a phenomonen characterized by patients reporting an increased level of pain and requesting additional analgesia with behaviour similar to inappropriate opiodi seeking. When the correct level of analgesia and pain relief is reached the behaviour ceases. Incorrect handling of this situation can damage the therapeutic alliance. Though not strictly a breakthrough pain association it may be a consideration

Cancer related breakthrough pain may require multiple modalities and time frames for treatment. An example is employing a regional analgesia technique while radiotherapy / chemotherapy is undertaken or parentral opioids around the time of prophylactic surgery. Analgesic medication especially opioids require constant review but especially after an episode of breakthrough pain. When prescribing opioids a reduction in dose may be appropriate if the pain generator has been removed or minimised. Modern management of cancer includes maintenance chemotherapy sometimes for a considerable period of time. This may make the assessment of breakthrough pain in this setting complex. An approach similar to that taken in persistent non-cancer pain many be employed.

Though initially considered the domain of cancer pain and associated with opioid analgesia, breakthrough pain can be identified as part of acute pain and persistent pain clinical picture. There are numerous formulae designed to aid in managing the entity. Inhaled and transmucosal preparations of opioids make the management of breakthrough pain easier and less uncomfortable in cancer patients. Other agents nonsteroidal anti inflammatory preparations, local anaesthetics and adjuvant pain therapies should also be considered in the management. Not treating breakthrough pain can even add to economic burdens. It is estimated that in patients with chronic pain those who had breakthrough pain incurred costs 5 times higher than patients without breakthrough pain. The greater costs were primarily related to hospitalizations for uncontrolled pain, emergency department visits, and unscheduled office visits

Marking guideline

Introduction: Make some attempt as defining the entity of breakthrough pain. Opening statement indicating that the pain should be assessed with due consideration to biological issues and not omitting important psychosocial aspects

Can divided into the three broad categories of acute pain Persistent pain and cancer pain as outlined in the question.

Acute pain key points

- Common problem
- Frequent assessment
- Functional activity score in addition to pain score
- Emergence of new pain or pain uniquely associated with the surgical procedure
- Premorbid pain experience
- Acute neuropathic pain 30%

Chronic persistent pain

- Common but complex problem
- Social psychological issues important
- May be a new pain that requires investigation
- Pain and psychological measures for comparison
- Education 30%

Cancer pain

- Best described historiacally
- Causes: tumour / 2nd deposits / paraneoplastic / unrelated
- Multimodal treatments
- Reassessment of pain and medications following episode of breakthrough pain
- Complex problem for chronic cancer pain 30%

Closing

- Opioids not the only option
- Significant negative effect of not treating breakthrough pain adequately

10% for layout and logical progression

QUESTION 5 **COMPULSORY**

You are referred a patient diagnosed with chronic pain and 'adjustment disorder'. Discuss the factors that you would elicit in your assessment of this patient, with particular emphasis on the emotional symptoms and signs

My assessment of the patient would be informed by a biopsychosocial approach, preferably within the setting of a multidisciplinary pain management clinic. Consideration would be given to the physical symptoms and signs, psychological symptoms and mental state examination, and a review of the person's social situation.

The assessment would involve the evaluation of possible physical pathology, and the exclusion of so-called "red flag" conditions. The patient's level of functioning would be evaluated, as well as the presence of any specific dysfunction. The assistance of other members of the pain clinic (such as a physiotherapist, occupational therapist and clinical psychologist) would be utilised.

In establishing the diagnosis of an Adjustment Disorder a formal mental state examination would be required, evaluating the patient's appearance, speech, cognition, thinking (form and content), affect, perception and judgment. A detailed history of emotional symptoms, and in particular of any manifestations of depressed mood or loss of interest or pleasure (anhedonia) also would be obtained. Depressed mood might be indicated by subjective report (e.g., feeling sad, feelings of worthlessness or excessive and/or inappropriate guilt, thinking about death or suicidal ideation) or by tearfulness. Other manifestations of clinically significant depression also might be present (sleep disturbance, decrease or increase in appetite, fatigue or loss of energy) but such symptoms need to be differentiated from those that are secondary to pain itself or to analgesic medication.

With specific reference to the diagnosis of "adjustment disorder", this diagnostic category is included in both the International Classification of Diseases, 10th edition, published by the World Health Organization in 1992, and in the Diagnostic and Statistical Manual of Mental Disorders, published by the American Psychiatric Association, currently in its fourth edition (Text Revision – 2000).

The essential features of "adjustment disorder" are the presence of subjective distress and emotional disturbance, leading to interference with social or occupational functioning, which develops in response to an identifiable stressor or series of stressors, including the presence or possibility of a serious physical illness. Thus, chronic pain is considered as a stressor that can give rise to an adjustment disorder.

Subtypes of "adjustment disorders" are recognised on the basis of the predominant symptoms – these might be, for example, those of depression, anxiety, or both.

In making the diagnosis of "adjustment disorder" it is necessary to consider whether or not the person's emotional response and level of distress meet the definition of a mental disorder, or whether they represent an understandable psychological reaction to chronic pain and its sequelae. In other patients the symptoms might be so severe that the diagnosis of Major Depression or of one of the specific Anxiety Disorders is justified.

It is also necessary to differentiate 'adjustment disorder" from demoralisation, which has been described as a not uncommon response to physical illness, including chronic pain.

Other factors to be considered in the assessment of the patient who has been diagnosed as having an "adjustment disorder" include a review of treatment that the patient has had for the psychiatric condition, such as medications and/or supportive psychotherapy.

References

American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders*, Fourth Edition, Text Revision. Washington, DC: APA, 2000.

Casey P, Maracy M, Kelly BD, et al. Can adjustment disorder and depressive episode be distinguished? Results from ODIN. J Affect Disord. 2006; 92:291-7.

Fishbain DA, Goldberg M, Meagher BR, et al. Male and female chronic pain patients categorized by DSM-III psychiatric diagnostic criteria. Pain 1986; 26:181-97.

Merskey H. Pain and psychological medicine. In Wall PD, Melzack R. Textbook of Pain, 3rd edition. Edinburgh: Churchill Livingstone, 1994, pp. 903-920.

O'Keeffe N, Ranjith G. Depression, demoralisation or adjustment disorder? Understanding emotional distress in the severely medically ill. Clin Med. 2007; 7:478-81.

Twillman RK. Mental disorders in chronic pain patients. J Pain Palliat Care Pharmacother. 2007; 21(4):13-9.

World Health Organization. *The ICD-10 classification of mental and behavioural disorders: clinical descriptions and diagnostic guidelines*. Geneva: WHO, 1992.

QUESTION 6 **NON COMPULSORY**

Write short notes on the relationship between childhood victimisation and neglect and chronic pain in adulthood.

A history of childhood abuse is frequently reported by older adolescent and adult patients presenting with a variety of persistent pain syndromes. Candidates require a working knowledge of this issue to enable them to assess the likely impact of such reports on their patients, their treatment and their long-term outcomes.

Points were awarded under the following headings:

1. Definition of childhood victimization
2. A discussion of the incidence of the various types of abuse and incidence in chronic pain patients
3. Factors that put children at risk of victimization
4. Factors that influence the impact of the victimization on the child
5. A discussion of the impact of victimization on child development
6. Comments on the relationship between childhood abuse and chronic pain
7. A discussion of the implications for treatment of patients reporting a history of childhood victimization

Bonus points were awarded for a discussion of gender issues and the relationship between childhood victimization, depression and chronic pain.

The following discussion outlines the key issues.

Definition of childhood victimization

Childhood victimization is a broad term that is categorized under 3 headings:

- Physical abuse - physical violence against a child by a person in authority or someone at least 2 years older than the child
- Sexual abuse - a range of activities involving sexual contact not desired by the "child" recipient
- Childhood neglect - general lack of care and emotional neglect

Epidemiology

Statistics quite variable. In USA National Co-morbidity Survey, 10.6% of people reported childhood abuse: 3.8% physical abuse only, 5.0% sexual abuse only, 1.8% both and 2.89% childhood neglect in the general community. In studies of chronic pain patients, the incidence is much higher e.g. approximately 40% of women. In addition, victims of sexual abuse are at greater risk of becoming depressed or suicidal during adolescence and young adulthood. Abuse victims are also at increased risk of organic disease including lung disease, peptic ulcers, arthritis, cardiac disease, diabetes and autoimmune disorders.

In Australia, the most common type of abuse is neglect, followed by emotional abuse according to the 2004/5 Child Protection Act report.

- 73% of parents had at least 1 problem, especially domestic violence, alcohol or substance abuse or a psychiatric disability
- Girls twice as likely to have been sexually abused, boys physically abused more than girls
- Aboriginal and Torres Strait islanders over represented

Factors that put children at risk of abuse or neglect
Abuse most commonly occurs within the family situation. Risk factors include

- Factors in the child: chronic illness, handicap, difficult temperament
- Dysfunctional child-rearing and family relationships: unpredictability and inadequate family limits
- Parental issues: relationship problems with at least one parent, drug and alcohol addiction, parental death
- Poverty and related stresses in the family

In Australia, children of one-parent families, especially with female sole parent, constitute a high percentage of child abuse cases seen by child protection agencies as sole parents were more likely to have low incomes and be financially stressed, suffer social isolation and have less support from their immediate family.

Factors that may influence the impact of the abuse or neglect on the individual child include

- Child's temperament
- Severity, frequency and duration of the abuse
- Age at time of abuse (i.e. stage of personality development)
- Threats of violence
- Age difference from perpetrator
- Relationship to the perpetrator

Developmental outcomes of childhood abuse or neglect
The younger the child at the time of the abuse, the more serious the outcomes are likely to be. Abused infants and toddlers experience a wide range of developmental issues. Resultant outcome is an increase in children having deficiencies in emotional, behavioural, physiological and social functioning, particularly

- Reduced ability to self-regulate emotion resulting in disturbances of body image, poor modulation of affect and impulse control, uncertainty about the reliability of others leading to distrust
- Impaired neurophysiologic development of the brain, especially in the limbic system, amygdala and prefrontal cortex, essential for long term learning, cognition and self protection.
- Emotional reactions may change the way their bodies function e.g. impaired pain perception
- Learning difficulties due to inattention, difficulties distinguishing between relevant and irrelevant information. Children may have hearing, speech and visual perceptual problems, resulting in reading and writing difficulties
- Increased risk for other mental health disorders
- Higher risk of exposure to relationship violence as an adult

Gender issues
Women presenting to pain management units are more likely than men to report childhood abuse/neglect, especially sexual abuse. Also women, in the general community, presenting with more pain symptoms or physical conditions are likely to report a higher incidence of childhood abuse or neglect.
Women reporting childhood sexual abuse are also more likely to report having chronic painful conditions, including a greater number of painful body areas, more diffuse pain, and have had more hospitalizations, more surgery and more family physician visits.

Relationship between childhood abuse, chronic pain and depression
Many retrospective studies indicate a moderate to strong relationship but some prospective studies and some studies of laboratory pain suggest no or minimal

relationship. Individuals reporting history of childhood abuse report greater incidence of headaches, gynaecologic, gastrointestinal, respiratory, neurological and overall physical problems compared to individuals who do not report such history. They also report greater healthcare utilization, greater symptom severity and more surgeries.

Depression is common in chronic pain patients. Recurring relationships between depression and chronic pain, childhood abuse and depression, and childhood abuse and chronic pain have been demonstrated. Physiologically, victims of childhood abuse have altered flight/fight responses that result in hyper-arousal and regression to earlier stages of development. Compensatory responses include emotional numbing, withdrawal and dissociation, particularly in girls so that depression is very common. Childhood abuse and depression are independent contributors to chronic pain. Changes in neurological processes increasing sensitivity to stress and a negative attributional style are associated with childhood abuse and increased risk of depression. Victims of childhood abuse have less support from family and friends and as such are 3 to 4 times more likely to become depressed and suicidal during adolescence and young adulthood. Sexual abuse carries the greatest risk.

Implications for assessment and treatment of chronic pain

The relationship between childhood abuse and adult chronic pain conditions appears to be significant. The fact that the effect seems to be relatively modest needs to be taken into account during the assessment of the patient. Not every patient will require an in-depth assessment for possible childhood abuse. However, if childhood abuse has been implicated, selective assessment of factors such as the age at which the abuse occurred, the type of abuse, the victim's gender and the victim's current illness and distress will indicate the likely impact of the abuse on the individual.

QUESTION 7 **NON COMPULSORY**

What factors would you discuss in obtaining informed consent in a patient who is considering dorsal column stimulation?

General considerations

1. Assessment and implantation of a neuromodulatory device should take place in a multidisciplinary pain unit
2. Implantation of a dorsal column stimulator (dcs) is not the 1st line of treatment. Implantation should only be offered after other, more conservative therapies have failed ie rehabilitation / pain program, medication other more temporary interventional techniques
3. Only to be used patients with a definitive anatomical / pathological diagnosis

Elements of Informed Consent

1. Is a mutual decision making process on behalf of treating clinician(s) and patient (or surrogate)
2. Delivery of information (in language understandable to the patient), best in multiple different formats eg face to face, written and audiovisual
3. Respect for patients autonomy
4. Implies that the patient had the capacity to understand and make an informed decision

Description of Procedure / Equipment

- outline of procedure in laymans terms including the need for sedative analgesia
- description of mechanism of analgesia and the uncertainty that exists with regards to this (gate theory, alteration in neural conduction, changes in blood flow etc)
- outline of longevity of implant and need for regular replacement of generator
- detail of the process of temporary trial lead. Description of subsequent clinical measurement of pain rankings and function following this as an indication for permanent implantation
- need for ongoing rehabilitation following implantation

Indications and Evidence Base

- Emphasized only as one of the last lines of treatments when all other more conservative options have been exhausted
- Description of current evidence base – largely single blinded randomized trials (level 3 evidence) have shown improvement in pain rankings and function specifically for 1. radicular pain 2. peripheral vascular disease 3. complex regional pain syndrome and 4. ischaemic heart disease
- Explanation of uncertainty in outcome if used for any other indication eg phantom limb pain, brachial plexopathy, idiopathic diagnosis etc

Contraindications

- only relative contraindications need to be discussed and the possibility of uncertain outcomes or increased complications in these circumstances

- immunosuppresion (eg diabetes, patients on long term glucocorticoids etc)
- non intact dorsal columns (eg prior drez lesions, spinal cord injury (trauma, transverse myelitis etc)
- patients with psycholosocial issues that although aren't a an absolute contraindication will increase risk of suboptimal outcome eg unrealistic expectation of outcome, low self efficacy and resilience, poor social supports, lack of access to medical facilities eg those living in remote areas
- those requiring regular magnetic resonance imaging – eg patients with progressive spinal lesions eg tumors, syrinx etc

Complications

- any complication (42%)
- no improvement in pain or function
- infection (1 – 5%)
- stimulation problems - over / understimulation / poor coverage (30%)
- electrode migration / fracture / misplacement (24%)
- generator issues (1%)
- other complications – radicular injury, spinal cord injury, dural puncture and leakage

Patients Responsibility and Duties

- explanation that success of implantation is also the responsibility of the patient and requires:

- knowledge of above
- being proactive in interactions with medical practitioners eg informing doctors of implant, not entering MR scanner
- keeping regular follow up appointments
- maintaining contact with equipment representative and having emergency contacts on person
- consideration of a medialert bracelet

Other treatment offered

- definitive treatment – eg decompression eg foraminotomy, laminectomy, fusion for radicular pain or revascularization for peripheral vascular disease / ischaemic heart disease
- trial of other medication eg 3rd and fourth line agents eg ketamine, mexilitine, carbamazepine, lamotrigine, therapeutic trial of opioids etc
- trial of other procedures eg blocks etc
- trial of further rehabilitation

Capacity

- Assessment for capacity of decision making and use surrogate decision maker (epoa or legal friend) if required.

2nd opinion

- provision of a 2nd expert opinion if required

References:

1. Burchiel (2002) Surgical Management of Pain. 2002 Thieme. New York Carter, M. (2004) Spinal cord stimulation in chronic pain: a review of the evidence. Anaesth Intensive Care 32. 11 - 21
2. Dorsten, B (2006) Psychological consideration in preparing patients for implantation procedures. Pain Medicine 7(S1) 47 – 57
3. Jones, J McCullough, L and Richman, B (2007) A comprehensive primer of surgical informed consent. Surg Clin N Am 87. 903 - 18
4. North, R and Wetzel, F. (2002) Spinal cord stimulation for chronic pain of spinal origin. Spine 27(22): 2584 – 91
5. Oakley, J and Prager, J (2002) Spinal cord stimulation: mechanisms of action. Spine 27(22): 2574 – 83
6. Turner, J Loeser, J Deyo, R. and Sanders, S (2004) Spinal cord stimulation for patients with failed back surgery syndrome or complex regional pain syndrome: a systematic review of effectiveness and complications. Pain 108. 137 – 47

QUESTION 8 **NON COMPULSORY**

An elderly patient with mild dementia has severe pain three days after hip surgery for a fractured neck of femur.

Describe your approach to evaluation and management.

Assess site of pain

- History if possible.
- Examine patient locally and generally.
    - ? True recurrence of hip pain.
    - ? Other possible causes of pain/distress.
        - Local – eg:
            - haematoma
            - Poor position.
            - Musculo-skeletal.
        - General – eg:
            - Urinary retention.
            - Constipation.
            - Neuropathic pain post-operatively.

Barriers to assessment may be:

- Increased dementia from
    - Pain
    - Disorientation.
    - Metabolic or other disease processes.

  Therefore usual assessment techniques may not be helpful, but could be replaced by observation of behaviour, facial expression, and verbalisation, for instance.

Review type of anaesthesia/analgesia, eg:

- Has local anaesthetic catheter just been removed?
- PCA may not be understood.
- Analgesia withheld or prescribed inappropriately.

Review medications that may contribute to confusion/dementia, eg:

- Benzodiazepines.
- Tricyclic analgesics.
- Dementia medication.

Urgent surgical review + imaging of hip

If no surgical cause is identified

Then institution of appropriate analgesia should be done using medication if possible that will contribute minimally to further confusion and/or dementia, eg:

- Local anaesthetic techniques.
- Parenteral Paracetamol/Tramadol.
- Carefully titrated opioids.

    - Other helpful ongoing physical measures, eg TENS.

- Establish previous level of functioning, eg:
    - Past pain experienced.
    - Past behaviour.
    - Previous dementia assessment.
    - Information may be gained from family, General Practitioner, nursing home, and other staff.

- Discuss with staff and family the problems involved of pain assessment in the elderly and demented patients.

- Frequent review may be necessary.

REFERENCES

Acute Pain Management, 3rd Edition, Macintyre/Shug.

Pain in Residential Aged Care Facilities 2005, Australian Pain Society.

IASP Clinical Update, June 2006.

QUESTION 9 **NON COMPULSORY**

Describe the blood supply of the spinal cord.

The arterial supply of the spinal cord is initially derived from the vertebral arteries, and the posterior cerebellar arteries, via the anterior and posterior spinal arteries. These longitudinal arteries are reinforced by arteries which pass through the inter-vertebral foramina from vertebral, ascending cervical, posterior intercostal, lumbar and lateral sacral arteries. These are called radicular arteries. Beginning with the lower cervical segments, the spinal cord depends on the radicular arteries for survival The longitudinal arterial trunks are largest in the cervical and lumbar regions, near the ganglionic enlargements and are much smaller in the thoracic region. The metabolic demands are greater for grey matter and as such the white matter contains fewer capillary networks.

Each vertebral artery gives rise to an anterior spinal artery, caudal to the basilar artery. These two arteries fuse to form a single midline vessel, the anterior spinal artery at a variable level between C1 and C6. This artery lies along the anterior median fissure of the spinal cord. It is the largest of the arterial vessels of the cord and extends from the foramen to filum terminale, supplying the whole of the cord in front of the posterior grey columns. The anterior spinal artery is also supplied by a series of 5-10 unpaired radicular arteries, that originate from vertebral arteries and aorta or its branches. Blood supply to the anterior portion is much more vulnerable than that of the posterior portion, and can be compromised by an occlusion of a large radicular artery or a lesion to the aorta. Seventy five percent of the blood supply to the spinal cord is derived from the anterior spinal artery, including the base of the posterior horn and a variable portion of the lateral corticospinal tract. It is usually a continuous vessel for the length of the spinal cord. It gives rise to hundreds of central and circumferential branches

Each vertebral artery or posterior inferior cerebellar arteries, give rise to a posterior spinal artery, which runs along the line of attachment of the dorsal roots. These paired arteries supply the posterior grey and white columns on either side, and are fed by smaller radicular arteries at almost every spinal level. The posterior arteries are more of a plexiform network of small arteries. They supply the posterior columns, substantia gelatinosa, dorsal root entry zone, and a variable posrtion of the lateral corticospinal tract.

The segmental arterial supply to the cord is maintained by a series of radicular arteries that are derived from intercostals and lumbar arteries. In early embryonic development every segment of the spinal cord receives paired radicular arteries, these disappear leaving one or two cervical, two or three thoracic and one or two lumbar arteries.

These segmental arteries proceed to inter-vertebral foramina appropriate to their level, where they divide into terminal branches. The largest of these radicular arteries is the arteria radicularis magna, also known as the artery of Adamkiewicz. It has a characteristic hairpin bend and arises most commonly around T10 on the left, however may vary from T7 to L4. It arises from the right side in about one fifths of cases. It usually enters a single inter-vertebral foramen at the T9 to T11 level. This artery may be injured or its blood flow compromised during a variety of surgical of other therapeutic procedures, the more common being aortic surgery or trauma, thoracic or lumbar spinal surgery, neurolytic coeliac or lumbar sympathetic blocks and spinal or epidural blocks. The resultant outcome is usually motor paraplegia with variable sensation being maintained.

The venous drainage of the spinal cord is by six irregular plexiform channels. These lie along:

1. The anterior and posterior midlines
2. The line of attachment of the dorsal roots on each side
3. The line of attachment of the ventral roots on each side.

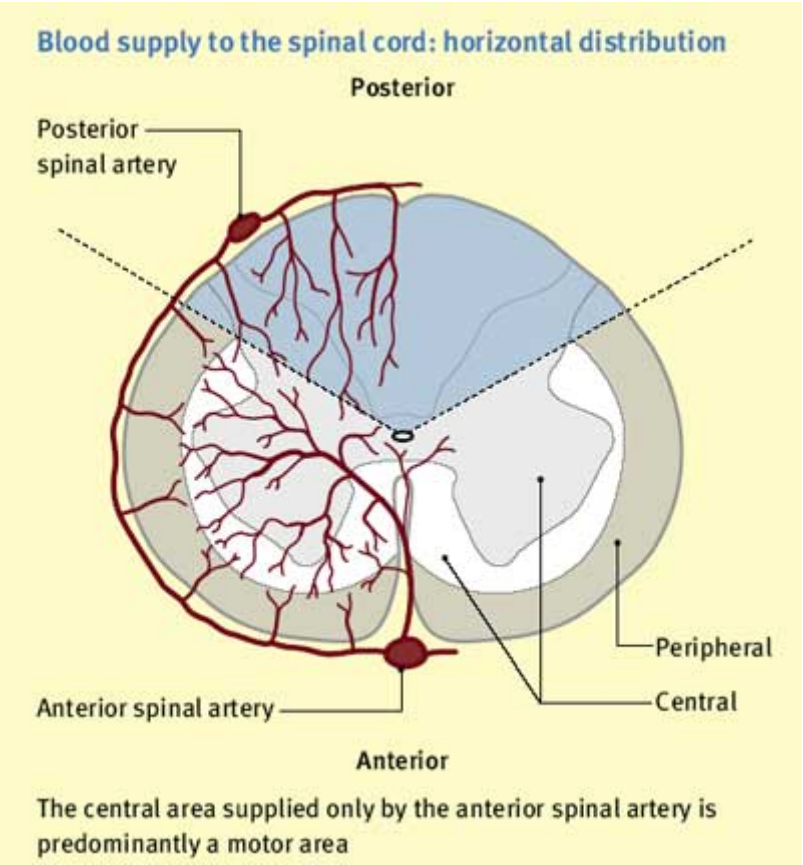
These are drained by the radicular veins and each in turn empties into the epidural venous plexus.

REFERENCES

1. Anatomy for Anaesthetists, Third edition, Blackwell Scientific Publications 1977.
2. Dommisse GF, The blood supply of the spinal cord. A critical vascular zone in spinal surgery. J Bone Joint Surg Br 1974 May; 56 (2) p225-235
3. Anaesthesia UK. http://www.frca.uk/article.aspx?articleid=100360

Suggested marking

Anatomy of arterial supply Description or diagram	3
Embryonic development	1
Area of supply	2
Vulnerability to injury	1
Variability of anatomy	1
Metabolic demands of cord	1
Venous drainage	1



QUESTION 10 **NON COMPUSORY**

Write notes on the gender influences in pain conditions and responses to treatment.

Gender: definition

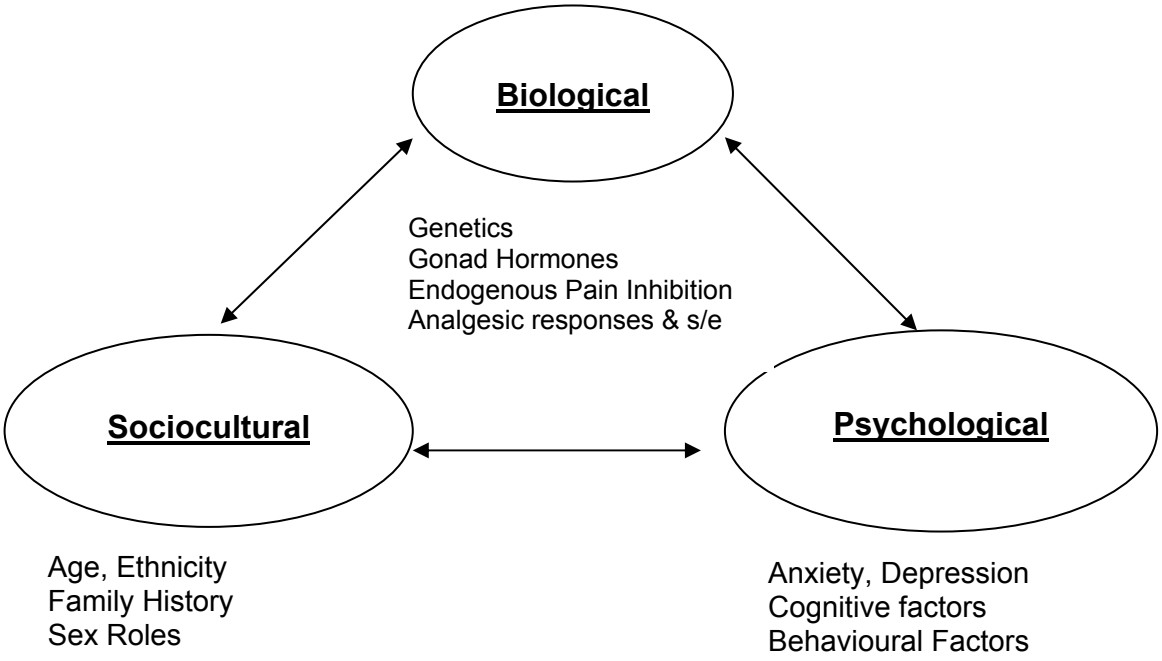
The term "**gender"** refers to modifiable, socioculturally shaped behavior and traits such as femininity and masculinity.

The term "'sex " refers to biologically determined aspects of femaleness and maleness.

The terms are commonly used interchangeably but are not the same.

This schematic view illustrates a framework of a biopsychosocial model as it relates to sex and gender influences in pain, with examples.

3 marks given for conceptual overview covering these three areas
7 marks given for examples and specific information about gender influences on pain (brief examples given)



Biological influences: differences in the physiological systems as a result of hormonal influences, between sexes, and as a result of hormonal variations

- Gender specific pains:
  - in females endometriosis, labour pain, dysmenorrhea, chronic pelvic pain in women, vulvodynia, gynaecological cancers, post mastectomy pain
  - in males prostatadynia, prostatitis, pelvic pain in men, prostatic cancer with secondaries

Epidemiological data indicate women are at greater risk of certain pain disorders and hormonal and genetic factors are implicated in some of these
Eg fibromyalgia, TMD dysfunction, migraine

Women have lower pain thresholds than men, with variation across the menstrual cycle and before puberty and after menopause associated with changing ovarian hormone function

Analgesic responses vary between man and women, and laboratory research may not correlate well with clinical research findings (for example Miaskowski's finding of reduced PCA morphine use by women post operatively was in contrast to laboratory research suggesting female rats required more morphine for analgesia)

Women and men in drug trials: Men have been overrepresented as candidates for research, and different responses bw men and women may not be evident because of this.
Sex or gender may be a risk factor for adverse drug reactions (more common in women)

Sex differences in pharmacokinetics; bioavailability, distribution (influenced by weight, differential passage across blood brain barrier) metabolism (cytochrome p450 and CYP3A vary, excretion.

Women have more adverse effects (nausea and vomiting) after opioid administartion

Sociocultural influences

MSK conditions: Men are more exposed to heavy lifting in labouring, women more exposed to high speed repetitious jobs in manaufacturing and office work.

Genital mutilation of females in less developed countries

High incidence of HIV AIDS in sub-saharan Africa, differentially more women afflicted experience pain

Implications of sexual abuse, association with chronic pain in later life
Women more likely to seek health care for symptoms

Psychological factors
Women are more likely to be given a psychogenic attribution for chronic pain than men.
Learned behaviours.
Influence and expression of depression in association with pain.

QUESTION 11 **NON COMPULSORY**

GPs are prescribing opioid patches for non malignant chronic pain. You are asked to give a lecture to GPs about this practice.

Give an outline of your lecture entitled "The use of transdermal opioids in non malignant pain".

Broad areas to consider:

- Opioids for Chronic Non-Malignant Pain (CNMP) in general
- Specifically transdermal opioids for CNMP

1. **Opioids for CNMP:**
   - Controversial, because of concerns about efficacy and safety, and addiction or abuse potential
   - Small subset only benefit
   - Long acting agents
   - Evidence stronger for short term (up to 6/12) rather than long term
   - Exhausted other reasonable measures
   - (some think that includes diagnostic blocks)
   - Comprehensive biopsychosocial assessment essential
   - Opioid risk screening
   - Goals and gains – should aim for and assess FUNCTION rather than analgesia alone
   - Care: obesity/OSA, other sedative/CNS depressant agents, prolonged QTc on ECG
   - Comprehensive risk : benefit information to patient / education
   - Written consent, agreement supply terms
   - Relative contraindication – past misuse of drugs or alcohol
   - TRIAL – with reasonable dose;
   - Failure to improve on trial leads to cessation
   - Implicit that continued dose escalation leads to wean or rotation
   - Careful patient selection
   - Therapeutic agreement [1]
   - Longitudinal care/monitoring with routine assessment of
     - degree of analgesia
     - functional daily activities
     - adverse events
     - aberrant behaviours

2. **Advantages of the transdermal route:**

   - constant drug level may help prevent recurrence of pain
   - and prevent abstinence symptoms
   - benefits of a parenteral route, avoiding first-pass metabolism
   - transdermal reapplication every 72 hours may enhance compliance

3. **Disadvantages of transdermal route:**

[1] Fishman SM, Kreis PG. The opioid contract. Clinical J. Pain. 2002; 18(4): S70-75.

- Cost usually high
- Application site pruritis, erythema – not major

4. **Transdermal Opioids available in Australia & NZ**:

    - Fentanyl
    - Buprenorphine

    They have different properties

    Fentanyl: strong mu, doses available should be mentioned:
    12.5, 25, 50, 75 mcg/hr
    Duration each patch 3 days

    Buprenorphine: mixed agonist/antagonist pharmacology
    Dose/sizes of patches should be mentioned: 5, 10, 20 mcg/hr
    May have antihyperalgesic effect in part via Kappa antagonist action
    Age and renal function little effect
    Probably best choice in elderly patients
    Each patch lasts one week

    General cautions: heat and fever may alter rate of delivery
    Constipation: natural and prescribed laxative measures from outset
    Disposal of reservoir used patches

5. **Evidence for patches specifically**:

    Fentanyl:

Collado F, Torres LM. Association of trqansdermal fentanyl and oral transmucosal fentanyl citrate in the treatment of opioid naïve pastients with severe chronic noncancer pain. J Opioid Manag. 2008 Mar-Apr; 4(2): 111-5

Level II-2 (ASIPP guidelines. Manchikanti L. Pain Physician 2008 Mar; 11(2 Suppl): S5 – S62)

    Buprenorphine:

Sittl R. Transdermal buprenorphine in the treatment of chronic pain. Expert review of Neurotherapeutics. 2005; 5(3): 315-323.

Pergolizzi J, Boger RH, Budd K, Dahan A, Erdine S, Hans G, Kress HG, Langford R, Likar R, Raffa RB, Sacerdote P. Opioids and the management of chronic severe pain in the elderly: consensus statement of an international expert panel with focus on the six clinically most often used World Health Organisation step III opioids, buprenorphine, fentanyl, hydromorphone, methadone, morphine and oxycodone. Pain Prac. 2008; 8(4): 287-313.

Marking Key points:

- Must mention Comprehensive **Assessment**, consideration of **risks**
- Opioid efficacy – limited group who benefit (~ 40% with 50% reduction)

- Should indicate there is **more than one drug to chose from**
- Know the pharmacology of the available transdermal agents
- Some advantages and disadvantages of transdermal vs oral route
- Set patient function gains/**goals**
- Describe **therapeutic trial**, **monitor & review**, assess aberrant behaviours
- Should achieve **stable dose**
- Control / Schedule status of the agents, prescribing limits (States vary?)

QUESTION 12 **NON COMPULSORY**

A 68 year old woman with metastatic breast cancer has uncontrolled bone pain in spite of regular paracetamol, NSAIDs and M.S. Contin 240 mg bd. She is requiring 50 mg mist morphine 2-4 hourly and continues to experience pain in spite of sedation.

Discuss the role of adjuvant pain therapy options for this patient.

Bone pain is common in the palliative setting and often results in both background and incident pain especially with weight-bearing. It is usually the result of metastatic disease with the most frequent cancers metastasizing to bone being breast, prostate and lung, with multiple myeloma often presenting as bone pain.
The analgesic options follow the WHO analgesic ladder commencing with simple analgesics such as paracetamol or the NSAIDs, graduating to weak opioids such as Tramadol and codeine, followed by the strong opioids e.g. morphine. There are however instances where these are inadequate, especially in the setting of incident pain, and the administration of bisphosphonates, radiopharmaceuticals, calcitonin or corticosteroids may assist the patient to achieve analgesia. Consultation with either the radiation oncologists regarding the role of localized radiotherapy for focal lesions, the orthopaedic surgeons regarding the role of internal fixation particularly of long bones, or the medical oncologists regarding the role of chemotherapy, may assist with pain control.
Patients with cancer present with complex pain problems that are often progressive in nature hence a comprehensive medical assessment is required to tailor an appropriate individualized plan of pain management. This will encompass an assessment of the pain, the status of the tumour including an expected prognosis, current and previous therapies, impact of the pain on sleep, function and mood and the presence of co-morbidities such as osteoarthritis which may be associated with significant pain. This assessment needs to recur frequently due to the 'fluid' state of cancer pain manaagement.
Multi-purpose co-analgesics

1. Corticosteroids
2. Ketamine

Drugs used for the management of bone pain

1. Bisphphonates
2. Radio-pharmaceuticals
3. Calcitonin

Other therapies

1. Radiotherapy
2. Surgery
3. Chemotherapy
4. Opioid rotation

Anaesthetic techniques

1. Local anaesthetic blockade
2. Spinal drug administration

Multi-purpose co-analgesics

1. Corticosteroids

Corticosteroids are analgesic in a variety of pain states in the cancer patient. Their mechanism of action is thought to be multifactorial including a reduction in oedema, tumour-reduction in steroid-sensitive tumours and a reduction in inflammatory mediators. Other situations in which corticosteroids are specifically indicated in the management of cancer pain include imminent spinal cord compression, radicular pain secondary to spinal metastatic disease and pain from visceral distension including hepatomegaly and bowel obstruction. Some side-effects may be beneficial including improved appetite, reduced nausea and malaise, however mood disturbance including steroid psychosis are not uncommon. The incidence of gastric ulceration is doubled with chronic steroid use and further compounded by combinations with NSAIDs. The COX-2 inhibitors may be safer. Long term use may be associated with hypoadrenalism.

2. Ketamine

The NMDA receptor antagonists have been used in the management of cancer pain including to improve the efficacy of opioids used in the management of bone pain. The general anaesthetic agent ketamine is the gold standard in this class. Multiple methods of delivery of ketamine have been described from intermittent injections to constant infusions to 'pulse' delivery to PCA. The major side-effects limiting further treatment are psychomimetic.

Drugs used for the management of bone pain

1. Bisphosphonates

The bisphosphnates were introduced into practice to treat hypercalcaemia, however their action as inhibitors of osteoclast function has seen them improve analgesia in patients with bone metastases or multiple myeloma. Pamidronate and zoledronate are intravenous preparations which have both been shown to produce opioid sparing analgesia in a variety of cancers including metastatic breast cancer and multiple myeloma. Prostate cancer metastases which tend to be osteoblastic, in contrast to the osteolytic metastases produced in breast cancer, respond less reliably to bisphosphonate therapy for analgesia. They are infused every 3-4 weeks. The major side-effects associated with the use of bisphosphonates are the potential for hypocalcaemia, a flu' like illness, renal impairment, gastrointestinal upset and rarely, ostenecrosis of the mandible which occurs more frequently in those with dental extractions and infections. There is a marked reduction in skeletal morbidity associated with the administration of these agents. Newer, more potent orally administered bisphophonates e.g. alendronate, which are available for osteoporotic crush fractures, may also be available to this patient.

2. Radiopharmaceuticals

Radio-isotopes are a means of providing radiation therapy to multiple sources of painful bony metastases. Concentration of radio-isotopes occurs as a result of selective uptake by re-mineralizing bones and the emission of beta particles selectively at the site of uptake. The most commonly administered radiopharmaceutical, strontium-89 is useful for metastatic bone pain. The major adverse effect associated with this is myelosupression which can be problematic in patients undergoing concurrent chemotherapy. Due to it's urinary excretion, urinary

incontinence and renal impairment are relative contra-indications to it's use. Costs can be prohibitive.

3. Calcitonin

Calcitonin can be used in the management of phantom pain associated with amputation for malignancy and for bony metastatic disease. Subcutaneous administration can be complicated by the development of an acute hypersensitivity reaction. Intranasal administration attenuates these effects. The analgesic effect is variably reported as useful. Nausea is a common side-effect, whilst hypocalcaemia may be induced by calcitonin.

Other therapeutic modalities

1. Radiotherapy

Radiotherapy may be useful as palliative management in patients with painful focal metastases. With low median survival times in this group of patients, a single treatment of 8Gy will produce a probability of pain relief in 80% of patients at 3, 6 and 12 months. Painful spinal metastatic disease and imminent spinal cord compression can be managed using high-dose corticosteroids and radiotherapy. Patients with good performance status will receive a high dose over several days, while those with poor performance status will receive a single dose of 8Gy for pain relief.
Recurrent irradiation may be associated with the development of myelitis.

2. Surgery

Surgical stabilisation of metastatic lesions in long bones is performed to prevent pathological fracture and for pain relief. Patients with good performance status' and who have extensive vertebral collapse secondary to metastatic disease may be candidates for anterior spinal surgery. This has a significant morbidity and mortality (up to 30% in this group of patients) associated with it, hence patient selection is important however, 71% of patients achieved pain relief. Surgery may also be considered in the situation of progression in spite of radiotherapy, and in relatively "radio-resistant" tumours.
Neurosurgical ablative techniques are uncommonly used in the management of pain in the cancer patient but may be considered. Percutaneous cordotomy is probably the most common technique used and is most applicable for unilateral pain in a patient who has a limited prognosis

3. Chemotherapy

Chemotherapy may have a role to play in the management of some tumours presenting with bone pain. Multiple myeloma and metastatic lesions from hormone sensitive carcinomas such as some breast and prostate cancers may respond to tumour burden reduction with chemotherapeutic regimens. Herceptin is a monoclonal antibody with proven efficacy in terms of treating metastatic bone pain in patients with breast cancer who display HER-2+. Other hormone therapies may be appropriate depending upon the hormone status of the tumour cells.

4. Opioid rotation

Patients who develop intolerable adverse effects whilst achieving inadequate analgesia with one opioid may sometimes benefit from switching to an alternative. The theory behind this relates to incomplete cross-tolerance between opioids and the knowledge that the relative potency of the of some of the opioids e.g. methadone and hydromorphone may alter with increasing dose. Differences in pharmacokinetics as the patient's state alters may contribute to worsening of adverse events especially cognitive effects which patients may find intolerable. Changing to a drug with a different pharmaocokinetic profile may improve this. Drug interactions may alter both the pharmacokinetic and pharmacodynamic profiles of a pre-existing opioid or deterioration in either hepatic or renal function may result in drug or metabolite accumulation. Opioid hyperalgesia may be another reason to alter or reduce the dose of opioid.
The major issues associated with opioid roation are the potential and consequences of, either under- or over-dosing on the new opioid (even with the use of drug equivalence tables).
Sometimes alteration of the route of administration may also be used to improve analgesic efficacy.

Anaesthetic techniques

1. Local anaesthetic neural blockade

Neural catheters can be placed to alleviate pain in patients with fractured long bones. Pain from a pathological fracture of the femur can be alleviated by femoral nerve block or insertion of a femoral nerve catheter to provide ongoing analgesia either whilst awaiting surgical stabilization or to allow for nursing care during terminal palliation. Similarly fractures of the humerus may be amenable to an interscalene block. Adverse effects of local anaesthetics are largely related to CNS excitability followed by cardiovascular collapse. Dose reductions for infusions should be considered in patients with impaired hepatic function and in those who are taking class 1 anti-arrythymics.

2. Intraspinal drug administration

There are a number of drugs that can be administered intraspinally for the management of intractable pain not relieved by other agents, or where the side-effects of other agents produce serious or unacceptable side-effects. Drugs that may be used include opioids usually morphine in a dose ratio of 1:10 for epidural opioids and 1:100 for intrathecal opioids. In addition other agents that can be added intraspinally are the local anaesthetics and the alpha-2 agonist, clonidine.
Phenol and alcohol can be administered intrathecally to patients who have lower limb or pelvic metastases from breast cancer, particularly if these have proven to be resistant to radiotherapy. The major problems associated with these agents are lower limb sensory loss, dysaesthesiae and loss of bladder and/or bowel function.
Intraspinal catheters can be complicated by infection, inflammation especially over time with the development of granulomas which may mimic neurological progression of disease, haematoma, CSF leak, opioid-induced hyperalgesia with paradoxical pain, and systemic opioid effects with high doses administered epidurally.

References;
Doyle D, Hanks G, Cherny N, Calman K. (Eds). Oxford Textbook of Palliative Medicine. 3rd Ed. 2004. Oxford University Press, Oxford.

Mercadante S. Opioid rotation for cancer pain: rationale and clinical aspects. Cancer, 1999: 86; 1856-1866.
Lussier D, Huskey AG, Portenoy RK. Adjuvant analgesics in cancer pain management. The Oncologist, 2004: 9; 571-591.
Cherny NI, Portenoy RK. Cancer pain: principles of assessment and syndromes. In: Wall PD, Melzack R. (eds) Textbook of Pain. 4th Ed. 1999. Churchill-Livingstone, Edinburgh. Ch. 45.

Marking schema:
Failure to address the question or listing all the therapies that are available for the treatment of bony metastases in breast cancer without providing a comprehensive discussion of any of these. Little or no comment about assessment of the patient – fail
A superficial assessment of the patient without reference to the interdisciplinary needs of the dying patient and failure to detail 5 approaches to pain management in this patient – pass
A good patient assessment with reference to the dynamic state of the patient's condition and an approach which is reasonably detailed describing 5 methods of analgesia that could be offered to this patient – good pass
A patient assessment making reference to the dynamic nature of this patient's disease and the interactions associated with therapies. Note the interdisciplinary nature of the situation. A detailed examination of 5 approaches to pain management for this patient, not necessarily inclusive of those mentioned above, but included in the discussion, the rationale, benefits and risks, where appropriate NNT and adverse effects associated with each – excellent pass

QUESTION 13 **NON COMPULSORY**

(a) State the diagnostic criteria of Major Depressive Disorder? (Name the classification used).

(b) What is the differential diagnosis for a person presenting with depressive phenomena?

(c) Why are antidepressant medications used for patients with chronic pain?

Information Resource

(a) ***State the diagnostic criteria of Major Depressive Disorder? (Name the classification used)***

DSM 4

Five or more of the following during a **two week period**, which represent a change from previous level of functioning.

At least one is either depressed mood or loss of interest or pleasure.

1. **Depressed mood**, most of the day, nearly every day, as indicated by either subjective report or observation made by others.
2. Markedly **diminished interest** or pleasure in almost all activities, most of the day, nearly every day. (as indicated by subjective account, or observation made by others)
3. Significant weight loss, unintentional **weight change** (more than 5% body weight in a month), or a change in appetite nearly every day.
4. **Sleep disturbance** (insomnia or hyper-somnia) nearly every day.
5. **Psychomotor agitation or retardation** nearly everyday (observable by others. Not only subjective feelings of restlessness or being slowed down.)
6. Fatigue, **loss of energy,** nearly every day.
7. Feelings of **worthlessness or guilt** nearly every day (not merely self reproach)
8. **Diminished ability to concentrate**, indecisiveness, nearly every day (either by subjective account or as observed by others).
9. Recurrent thoughts of death (not just fear of dying), recurrent **suicidal ideation** or a suicide attempt.

The symptoms cause **clinically significant distress or impairment** in social, occupational, or other important areas of functioning.

The symptoms are **not due to the direct physiological effects of a substance** (drug of abuse, medication) **or a general medical conditio**n (e.g. hypothyroidism).

The symptoms are **not better accounted for by bereavement** i.e. after the loss of a loved one, the symptoms persist for longer than 2 months or are characterised by marked functionally impairment, morbid preoccupation with worthlessness, suicidal ideation, psychotic symptoms, or psychomotor retardation.

(b). ***What are the Differential Diagnoses to consider for a person presenting with depressive phenomena?***

1. Mood change secondary to general medical condition
2. Mood change secondary to delirium, dementia
3. Mood change secondary to medication
4. Mood change secondary to substance use, or withdrawal
5. Disillusionment, demoralization
6 Other Psychiatric disorders – as listed above
7. Activity fatigue, sleep disorder

(c*). **Why are "Antidepressant" medications used for patients with chronic pain?***

Antidepressant medications are used in patients with pain in relation to

1. **Mood disorders**
2. **Sleep disorders**
3. **Analgesic adjuvant action**

1. Mood disorders
    - Major Depressive Disorders
    - Anxiety Disorders
        - Generalised Anxiety Disorder
        - Post Traumatic Stress Disorder
    - Adjustment Disorder especially irritability.

2. Sleep disorders
    - a strong association between sleep disorders and pain.
    - Pain is a potent cause of insomnia which can then have complications as a consequence. Long term sleep deprivation can lead to psychological, mood and physiological changes which interfere with recovery and rehabilitation.
    - Sleep disorders may also be a manifestation of the associated psychosocial disturbance.
    - association between insomnia and musculoskeletal disorders such as Fibromyalgia (Chronic Widespread Pain), demonstrated to improve with a small dose of trycyclic antidepressant medication.

3. Analgesic adjuvant action
    - excellent evidence base for many antidepressents
    - most research on Amitriptyline for neuropathic pain, particularly diabetic neuropathy
    - TCAs L1 evidence for diabetic neuropathy, post herpetic neuralgia and fibromyalgia
    - TCAs L2 evidence for central pain due to spinal cord injury and post stroke pain
    - L2 evidence for duloxetine, venlafaxine, mirtazapine in diabetic neuropathy, post herpetic neuralgia and fibromyalgia
    - NNT (Numbers Needed to Treat) of 2.9
    - Serotonin Noradrenalin Reuptake Inhibitors SNRI's have NNT's of between 5 and 15.

Pharmacology

- Antidepressant medications block reuptake of neurotransmitters, particularly Serotonin and Noradrenalin inhibiting nociception transmission.
- Amitriptyline also works by having an effect on sodium channels, acetylcholine, NMDA (N-Methyl D-Aspartate) receptors.

Adverse reactions

- NNH (Numbers Needed to Harm) of 3.7 for minor events, 22 for major adverse events.
- risk of death by cardiac dysrrythmia from overdose with trycyclic medication indicating caution, careful patient selection and supervision, particularly with suicidal patients.
- arrhythmia with tcas
- serotonergic syndrome in patients on multiple agents
- discontinuation syndrome(ssris),
- sedation
- postural hypotension (leading to falls)
- GIT disturbance (constipation, gastric slowing)
- urinary retention
- glaucoma (particularly for trycyclic antidepressants)
- risk of inducing mania in susceptible patients
- reduced saliva production, impairment of oral hygiene.

Prescribing

- commence a small dose of Amitriptyline (25mg)
- increase incrementally each 3 - 7 days depending on effect and tolerance
- at higher doses, particularly elderly, monitor with collateral information and ECG
- effective dose TCA may be 50 – 75 mgs
- usual therapeutic dose TCA for Depression is 150mgs
- maximum TCA 300mgs
- duration – 2 weeks to onset antidepressant effect, sooner for analgesic effect, continue at least 3-6 months if effective, possible for indefinite use.
- Research demonstrates a synergistic effect with non-pharmacological interventions

Non response

- Review diagnosis, treatment plan, compliance
- change to an alternative trycyclic antidepressant (Imipramine, Nortriptyline) is feasible .
- change to SNRI, or SSRI

QUESTION 14 **NON COMPULSORY**

The consumption of opioids in Australian and New Zealand has risen exponentially since 1990. Discuss the factors that have led to this trend.

Background:
Since 1990, there has been a substantial increase in the utilisation of opioids in Australia and New Zealand (and the United States of America and United Kingdom), based on a number of indicators such as community prescriptions. There have also been a decrease in the use of short-acting injectable opioids and an increase in the use of orally well-absorbed, long-acting opioids. While recognising that much of this supply may reflect increasing population size and aging of the population, better pain management and other improvements in treatment for a range of conditions, it nonetheless represents an increase in the total supply potentially available for diversion and non-medical use (RACP, 2008).

DRAFT ANSWER: Opioids are prescribed for acute pain, persistent cancer and non-cancer pain (NCP). The most dramatic increases in opioid consumption relates to persistent pain. It is difficult from available records to determine the proportions of this increase due to cancer and NCP. It is reasonable however to attribute a large proportion to NCP. This phenomenon is not isolated to Australia and New Zealand (Bell 1997).
The use of opioids in cancer pain is widely accepted. The role of opioids in NCP remains controversial (Fields 2007). Many factors have contributed to the increase in opioid consumption since 1990. This change in clinical practice appears to be primarily due to a ***change in attitude***, with more liberal use in both palliative care and chronic pain (Bell 1997).
Several factors have lead to these changes:

Changes in drug availability

A wide range of drug options (S8 & S4)
Since 1990 long-acting opioid preparations have come onto the market including:

- Slow release morphine (MS Contin, Kapanol & MS Mono)
- Slow release oxycodone (Oxycontin)
- Fentanyl transdermal preparations (Durogesic)
- Buprenorphine – transdermal preparations (Norspan)
- Tramadol (Tramal)

The introduction of these new drug preparations have been accompanied by extensive pharmaceutical marketing which has encouraged use in both cancer and NCP.

Effects of heroin drought and limited access of drug-dependent persons to opioid-substitution (OST) programs
There is an incentive for persons with opioid dependence to present as requiring treatment for pain rather than addiction. Access to OST programs in Australian and New Zealand is limited, with different regulatory frameworks. The degree of unsanctioned versus sanctioned use of prescription opioids is unknown.

Less restrictive government regulation

Drugs & Poisons Act (details vary from state to state)

In Australia there has been a tendency towards less restrictive requirements for authority prescribing e.g. in NSW recent changes have been made to the Act to limit authorisation to *dependent* patients only. Previously all patients were required to be notified if prescriptions continued beyond 8 week. Doctors are often reluctant to label patients as dependent and are unfamiliar as to how to best apply the term in practice (due to problems with definitions). As a result fewer requests for authority are made and less effective monitoring occurs.

Many patients receive opioids for prolonged period without authority. This removes the limit placed on dose escalation offered by peer review.

Pharmaceutical Benefits Scheme (PBS)

In Australia, since 1990 long-acting opioids have been approved for PBS subsidy for patients with persistent *NCP* that is non-responsive to non-opioid pharmacotherapy. Since 2006, the criteria for access to PBS-subsidised opioid have been eased: initiation in hospital or pain clinic is no longer required; and specialist involvement in the decision making process to continue opioid is no longer required (any two doctors can agree ongoing prescription is indicated).

No restriction is placed on the access to small opioid quantities (e.g. twenty tablets) .

As a result, monitoring of the *appropriateness of opioid prescribing* on the one hand and the degree of *unsanctioned use* (problematic use by those to whom opioids are prescribed and use by others to whom they are not prescribed) on the other is extremely difficult, the only source of information for clinicians at present being the Prescription Shopping Information Service.

There appears to be no relationship between holding a State Health Department Authority to Prescribe and being able to prescribe PBS-subsided opioids on a (Commonwealth DOHA) Authority prescription.

Evidence conflicting

The use of opioids in persistent NCP has been a subject of continuing and at times heated debate (Nicholas et al. 2006). The evidence supporting the long-term effectiveness of opioids is limited to a small number of RCTs (short duration) and case series (Ballantyne & Shin 2008). Despite this there is strong anecdotal support for long-term opioid prescribing in NCP.

Prescriber factors

Limited training

The training of medical practitioners in the assessment and management of pain remains limited. (Lin et al. 2007).

Limited time

Consultations are also extremely time-limited. Opioids are commonly prescribed to patients with social problems, high levels of emotional distress and unclear medical diagnoses (Bell 1997). Such patients are extremely time consuming to manage. Drug prescription remains a primary mode of treatment in medical practice: opioid prescription may be used instead of applying more time-consuming psychological treatments.

Limited consensus regarding indications and review

While many guidelines have been produced on the use of opioids in NCP, there is no overall consensus or "ownership" of those guidelines. There is no evidence that such guidelines are being followed. In the absence of a more comprehensive (non-pharmacological) approach to pain management opioid prescription is likely to be prolonged and ceiling dose more difficult to contain.

Suggested marking Q14:

Drug availability factors (2)
- Products and marketing
- Factors influincng illicit use

Regulatory factors (2)
- State authority to prescribe
- Commonwealth PBS subsidy
- Implications for monitoring

Prescriber factors (4)
- Lack of training
- Lack of resources including time
- Variety of guidelines but no consensus or review plan

Evidence for effectiveness (2)
- Limited evidence for long-term effectiveness
- Problems of escalating/ceiling dose

Total marks: 10

References:

Ballantyne JC. Shin NS. Efficacy of opioids for chronic pain: a review of the evidence. Clin J Pain 2008; 24(6):469-478.

Bell JR. Australian trends in opioid prescribing for chronic non-cancer pain, 1986-1996. Med J Aust 1997;167:26-29.

Fields HL. Should we be reluctant to prescibe opioids for chronic non-malignant pain? Pain 2007;129:233-234.

Lin JJ, Alfandre D, Moore C. Physician attitude towards opioid prescribing for patients with persistent noncance pain. Clin J Pain 2007;23(9):799-803.

Nicholas MK, Molloy AR, Brooker C. Using opioids with persisting noncancer pain: a biopsychosocial perspective. Using opioids with persisting noncancer pain: a biopsychosocial perspective Clin J Pain 2006;22(2):137-146.

The Royal Australasian College of Physicians, *Prescription Opioid Policy: Improving management of chronic non-malignant pain and prevention of problems associated with prescription opioid use,* Sydney 2008.

Pharmaceutical Benefits Scheme. *Highest Volume PBS Drugs by Generic Name, Year Ending June 2006, Expenditure and prescriptions twelve months to 30 June 2006.* (Available at) *http://www.health.gov.au/internet/wcms/Publishing.nsf/Content/A58720844CBFCB47CA257218000D91C7/$File/complete.pdf*

QUESTION 15 **NON COMPULSORY**

Discuss the differential diagnosis and predisposing factors in a patient presenting with chronic pelvic pain.

Background

Chronic pelvic pain (CPP) is a common problem and presents a major challenge because of its unclear etiology, complex natural history, and poor response to therapy.

CPP is poorly understood and, consequently, poorly managed. This condition is best managed using a multidisciplinary approach. Management requires good integration and knowledge of all pelvic organ systems and other disciplines including musculoskeletal, neurological, and psychiatric areas.

A significant number of patients may have various associated problems, including bladder or bowel dysfunction, sexual dysfunction, and other systemic or constitutional symptoms. Other associated problems, such as depression, anxiety, and drug addiction, also may coexist.

It affects approximately 1 in 7 women In the United States. The reported prevalence rate of significant pelvic pain was 3.9%.

Of all referrals to gynecologists, 10% are for pelvic pain Estimated direct medical costs for outpatient visits for CPP (women aged 18-50 y) is approximately $881.5 million per year (Mathias, 1996).

46.6 % of women at 80 pelvic pain clinic reported past sexual or physical abuse and had a positive screen for post traumatic stress disorder (PTSD)

Common causes of CPP in men include chronic (nonbacterial) prostatitis, chronic orchalgia, and prostatodynia.

Causes & differential diagnosis:

Various reproductive, gastrointestinal, urologic, and neuromuscular disorders may cause or contribute to CPP. Sometimes, multiple contributing factors may exist in a single patient.

Appropriate caution must be taken during treatment of patients with the following characteristics:

- Poor response to prior appropriate treatment
- Unusual unexpected response to prior specific treatment
- Avoidance of school, work, or other social responsibilities
- Severe depression
- Severe anxiety disorder
- Excessive pain behavior
- Frequent physician changes
- Noncompliance with past treatment
- Drug abuse or dependence
- Family, marital, or sexual problems
- History of physical or sexual abuse

Comprehensive systematic reviews

In women

- 122 studies (in 64,286 women) evaluated 54 risk factors for dysmenorrhoea,
- 19 studies (in 18,601 women) evaluated 14 risk factors for dyspareunia,
- 40 studies (in 12,040 women) evaluated 48 factors for non-cyclical pelvic pain.

- Age < 30 years, low body mass index, smoking, earlier menarche (< 12 years), longer cycles, heavy menstrual flow, nulliparity, premenstrual syndrome, sterilisation, clinically suspected pelvic inflammatory disease, sexual abuse, and psychological symptoms were associated with dysmenorrhoea.

- Younger age at first childbirth, exercise, and oral contraceptives were negatively associated with dysmenorrhoea.
- Menopause, pelvic inflammatory disease, sexual abuse, anxiety, and depression were associated with dyspareunia.

- Drug or alcohol abuse, miscarriage, heavy menstrual flow, pelvic inflammatory disease, previous caesarean section, pelvic pathology, abuse, and psychological comorbidity were associated with an increased risk of non-cyclical pelvic pain.

In men

Chronic pelvic pain syndrome in men years characterised by chronic perineal and penile pain with varying degrees of urinary and sexual dysfunction, is often labelled chronic prostatitis.

As the cause of the most prevalent, non-bacterial, forms of the condition remains unknown, and therefore no definitive diagnostic test exists, the diagnosis has relied on a combination of clinical features, exclusion of other diagnoses (such as bladder outlet obstruction), and the results of investigations, However, there is no generally agreed clinical definition that brings together the symptomatic features and investigative findings, so it is difficult to make reliable comparisons among the many descriptive and therapeutic studies in the medical literature—or to draw conclusions.

Conclusion Randomised controlled trials of interventions targeting these potentially modifiable factors are needed to assess their clinical relevance in chronic pelvic pain.

Treatment of chronic pelvic pain may consist of two approaches. One is to treat chronic pain itself as a diagnosis, and the other is to treat diseases or disorders that might be a cause of or a contributor to chronic pelvic pain. These two approaches are not mutually exclusive, and in many patients effective therapy is best achieved by using both approaches.

This is a fairly comprehensive list where there may well be more than one disorder contributing to the patient's pelvic pain.

Extrauterine reproductive disorders

- Endometriosis
- Adhesions
- Adnexal cysts
- Chronic ectopic pregnancy
- Chlamydial endometritis or salpingitis
- Endosalpingiosis
- Ovarian retention syndrome (residual ovary syndrome)
- Ovarian remnant syndrome
- Ovarian dystrophy or ovulatory pain

- Pelvic congestion syndrome
- Postoperative peritoneal cysts
- Residual accessory ovary
- Subacute salpingo-oophoritis
- Tuberculous salpingitis
- Vulvovaginitis

Uterine reproductive disorders

- Adenomyosis
- Chronic endometritis
- Atypical dysmenorrhoea or ovulatory pain
- Cervical stenosis
- Endometrial or cervical polyps
- Leiomyomata
- Uterine Cancer
- Symptomatic pelvic relaxation (genital prolapse)
- Intrauterine contraceptive device

Urologic disorders

- Bladder neoplasm
- Chronic urinary tract infection
- Interstitial cystitis
- Radiation cystitis
- Recurrent cystitis
- Recurrent urethritis
- Urolithiasis
- Uninhibited bladder contractions (detrusor-sphincter dyssynergia)
- Urethral diverticulum
- Chronic urethral syndrome
- Urethral caruncle

Musculoskeletal disorders

- Abdominal wall myofascial pain (trigger points)
- Compression fracture of lumbar vertebrae
- Postural anomalies

- Fibromyalgia
- Mechanical low back pain
- Chronic coccygeal pain
- Muscular strains and sprains
- Pelvic floor myalgia (levator ani spasm)
- Piriformis syndrome
- Rectus tendon strain
- Hernias (egg, obturator, sciatic, inguinal, femoral, spigelian, perineal, umbilical)

Gastrointestinal disorders

- Carcinoma of the colon
- Chronic intermittent bowel obstruction
- Colitis
- Chronic constipation
- Diverticular disease
- Inflammatory bowel disease
- Irritable bowel syndrome

Neurological disorders

- Neuralgia/cutaneous nerve entrapment (surgical scar in the lower abdomen; usually iliohypogastric, ilioinguinal, genitofemoral, & lateral femoral cutaneous nerves)
- herpes zoster infection
- Spinal tumors
- Degenerative joint disease
- Disk herniation
- Spondylosis
- Abdominal “migraine”
- Neoplasia of spinal cord or sacral nerve

Psychological disorders

- Personality disorders
- Somatoform Disorders
- Chronic visceral pain syndrome
- Substance abuse

- Chronic fatigue syndrome
- Depression
- Sleep disorders
- Substance abuse
- Sexual and/or physical abuse

Common causes of CPP in men

- Chronic (nonbacterial) prostatitis
- Chronic orchalgia
- Prostatodynia

Investigations generally depend on the focus and specialty of the consultant.

Generally consider:

- Routine haematology and chemistry
- Urine culture & cytology
- STD testing
- Cystoscopy
- Pelvic MRI
- Vaginal ultrasonography (identifies pelvic varicocities)
- Laparoscopy
- Laparoscopic pain mapping??
- Pregnancy testing
- Ca125 / PSA

References:

Mathias SD, Kuppermann M, Liberman RF, et al. Chronic pelvic pain: prevalence, health-related quality of life, and economic correlates. *Obstet Gynecol*. Mar 1996; 87(3):321-7

Bodden-Heidrich R, Küppers V, Beckmann MW, Rechenberger I, Bender HG. Chronic pelvic pain syndrome (CPPS) and chronic vulvar pain syndrome (CVPS): evaluation of psychosomatic aspects. *J Psychosom Obstet Gynaecol*. Sep 1999; 20(3):145-51

Howard F M. An evidence-based medicine approach to the treatment of endometriosis-associated chronic pelvic pain: placebo-controlled studies. *J Am Assoc Gynecol Laparosc*. Nov 2000; 7(4):477-88.

Howard F M. Chronic Pelvic Pain *Obstetrics & Gynecology* 2003; 101:594-611

Jamieson DJ, Steege JF. The prevalence of dysmenorrhea, dyspareunia, pelvic pain, and irritable bowel syndrome in primary care practices. *Obstet Gynecol*. Jan 1996; 87(1):55-8.

Sand PK. Chronic pain syndromes of gynecologic origin. *J Reprod Med*. Mar 2004; 49(3 Suppl):230-4.

McCrory P, Bell S. Nerve entrapment syndromes as a cause of pain in the hip, groin and buttock. *Sports Med*. Apr 1999; 27(4):261-74

Latthe, P, Mignini, L, Gray, R, Hills, R & Khan, K. Factors predisposing women to chronic pelvic pain: systematic review. *BMJ* 2006; 332; 749-755.

Meltzer-Brody,S, Leserman,J, Zolnoun,D, Steege,J,Green E, Teich, A. Trauma and Posttraumatic Stress Disorder in Women With Chronic Pelvic Pain. *Obstet Gynecol* 2007;109:902–8 *(***LEVEL OF EVIDENCE: II)**

Jarrell, J F, Vilos, GA,Consensus Guidelines for the Management of
Chronic Pelvic Pain. SOCG clinical practice guidelines; parts 1 & 2 August 2005