Opioid & Patient Controlled Analgesia

Theory

Name:	
Ward/Site:	Grade:
Date of teaching:	Date of Competency completion:

Theoretical Component:

The learner will have received PCA training and completed self assessment.

Completed competencies within 6 weeks of education.

Competencies can be assessed by Clinical Nurse Specialist, Education Co-ordinator or designated pain link nurse

Aim
The aim of this education package is to provide nurses with the knowledge and skills to effectively and safely care for a patient receiving opioids for effective pain relief.

Objectives

-This is a self-learning package. The nurse should be able to meet the following objectives by self-assessment.
-Show an understanding of the Scope of Professional Practice.
-Demonstrate an understanding of the physiology of pain.
-Demonstrate an understanding of the detrimental effects of uncontrolled postoperative pain.
-Demonstrate an understanding of the importance of regular pain assessment and formal written documentation.
-Demonstrate knowledge of the safe care and monitoring of the patient receiving opioid analgesics.
-State the actions and side effects of opioid and adjunct analgesics.
-Explain the principles behind opioid bolus titration.

Code of professional conduct
"As a registered nurse, midwife or health visitor, you are personally accountable for your practice. In caring for patients and clients, you must":

- Respect the patient or client as an individual.
- Obtain consent before you give any treatment or care.
- Protect confidential information.
- Co-operate with team members.
- Maintain your professional knowledge and competence.
- Be trustworthy.
- Act to identify and minimise risk to patients and clients.

These are the shared values of all the United Kingdom healthcare bodies.
-Nursing and Midwifery Council (June 2002)

Introduction
"Pain is a category of complex experiences, not a single sensation produced by a single stimulus". (SIGN 44, 2001)

Definitions of Pain

The most commonly used definition of acute pain is;

"An unpleasant and emotional experience associated with actual and potential tissue damage or described in terms of such damage". (International Association for the Study of Pain IASP 1979 p3).

Acute pain usually signals impending or actual tissue damage and thus permits the individual to avoid further injury (Melzack & Wall 2003). The complexity of acute pain requires an understanding of safe and effective principles to treat it.

Physiology of Acute Pain

Pain perception involves multiple interacting peripheral and central mechanisms. Pain is multi-factorial and involves physical, psychological and environmental aspects in every individual.

Acute pain is initiated by stimulation of nociceptors at the site of injury. There is associated release of a variety of inflammatory mediators, which both stimulate nociceptors directly and amplify their response to noxious stimuli. The pain signal is transmitted to the dorsal horn of the spinal cord and then onwards to a variety of sites in the central nervous system, including the cerebral cortex, resulting in conscious perception of the pain. At all points along the pain pathway the pain signal may be modulated to increase or decrease the duration and nature of the pain perceived.

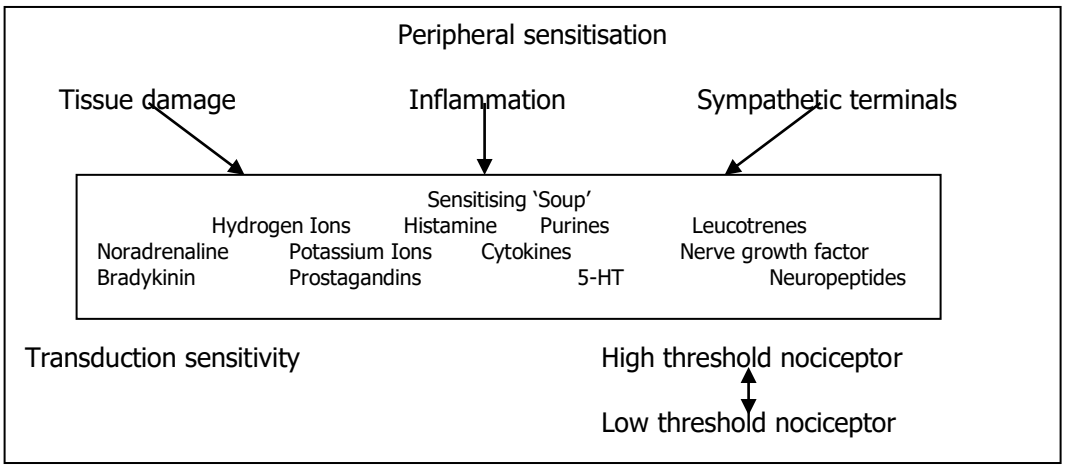


Figure 1 *The sensitivity of high threshold nociceptors can be modified in the periphery by a combination of chemicals that act as a 'sensitising soup'. These chemicals are produced by damaged tissue, as part of the inflammatory reaction, and by sympathetic terminals (from Woolf & Chong 1993).*

Transduction:
In the periphery, nociceptors which are free sensory nerve endings found throughout the body may be activated by pressure, chemical or heat stimuli. Multiple inflammatory mediators including substance P, serotonin, bradykinin and prostaglandins create an 'inflammatory soup' which reduces the threshold for stimulation of nociceptors making them more sensitive and which recruits additional nociceptors both at the site of injury and in adjacent uninjured tissue. This results in hyperalgesia (increased pain in response to a painful stimulus). This also explains the effectiveness of non steroidal anti-inflammatory drugs (NSAIDs) as analgesics since they inhibit prostaglandin production. The conversion of a painful stimulus to an action potential in the nerve ending is known as transduction. Abnormal processing by nociceptors in the absence of a recognised painful stimulus may also result in transduction. The resulting pain is neuropathic pain and has important features distinguishing it. These include the absence of ongoing tissue injury, the presence of hyperalgesia and allodynia (pain in response to stimuli not normally associated with pain eg. light touch) often stabbing and burning sensations are present and there is often a poor response to opioids.

Transmission:
This stage is responsible for the message being relayed from the receptors to the Central Nervous System(CNS). The two types of fibres involved in nociception are A delta (Aδ) and C fibres. These bipolar neurones relay impulses from nociceptors to the CNS and are termed nociceptor primary afferents (NPA). C fibres are small nonmyelinated neurones which conduct impulses relatively slowly and are responsible for aching delocalised burning types of pain (Puntillo 1988). The faster larger myelinated Aδ fibres are involved in the perception of well localised acute pain. Once the receptor has been stimulated, the information is passed along the afferent nerve to the spinal cord where the fibres enter the grey matter and terminate in laminae I, II or V of the dorsal horn (substantia gelatinosa). Here they synapse with second order neurones, which cross the spinal cord in the anterior commisure and ascend to many areas of the brain including regions of the thalamus and cortex.

Perception Of Pain:
Conscious experience of pain. It is important to remember that pain is a subjective phenomenon when discussing the physiology of pain. The perception and interpretation of an event as being painful depends on higher cerebral centres. Hawthorne and Redmond (1998) state that 'Pain' as such does not exist unless and until it is interpreted by the central nervous system (CNS).

Modulation:
In addition to peripheral sensitisation already discussed central mechanisms of sensitisation also exist.
Conversely, there are mechanisms for reducing or inhibiting pain transmission.
Pain signals themselves bring about changes in the excitability of neurones and the presence of transmitters and receptors at synapses in the pain pathway. The result is amplification of the pain signal and is termed 'wind up'. This is responsible for the phenomena of allodynia and secondary hyperalgesia (increased pain in response to painful stimuli in areas away from the injured area).

Inhibition of nociceptive impulses - neurons originating in the brain stem descend to the spinal cord and release substances such as endogenous opiods, serotonin (5HT), and norepinephrine (NE) that inhibit the transmission of nociceptive impulses.

Opioids exert their analgesic actions via opioid receptors distributed throughout the central nervous system activation of which partially inhibits transmission of nociceptive impulses. Opioid receptors have also been demonstrated at peripheral sites following tissue damage but the use of peripheral opioids at specific sites e.g. intra-articular has met with limited success.

Possible Harmful Effects of Untreated Acute Pain

- **RESPIRATORY_**- decreased lung volumes, atelectasis, decreased cough, sputum retention, infection and hypoxaemia.
- **CARDIOVASCULAR** - tachycardia, hypertension, increased peripheral vascular resistance, increased myocardial oxygen consumption, myocardial ischaemia, altered regional blood flow, deep vein thrombosis.
- **GASTROINTESTINAL** - decreased gastric and bowel motility.
- **GENITOURINARY** - urinary retention.
- **PSYCHOLOGICAL** - anxiety, fear and sleeplessness.
- **MUSCULOSKELETAL** - muscle spasm, immobility (increasing risk of deep vein thrombosis)

Advantages of Good Pain Management

- **RESPIRATORY** - improved lung expansion and ability to cough, reduced risk of atelectasis and chest infection.
- **CARDIOVASCULAR -** improved cardiovascular stability, decreased myocardial oxygen demand.
- **GASTROINTESTINAL -** earlier return of gut motility**.**
- **PSYCHOLOGICAL -** improved sleep, reduced anxiety and fear. Feels better
- **MUSCULOSKELETAL -** improved mobility (decreased risk of pressure sores, DVT). Potential earlier mobilisation.
- **ECONOMIC -** good pain control may shorten hospital stay**.**

Barriers to providing effective pain relief in the clinical area include;

Nurse/Doctor

- Lack of education and resources resulting in inadequate understanding of the multi dimensional nature of pain.
- Under utilization of pain assessment tools.
- The continuing belief of myths and misconceptions surrounding pain management.

Patient

Reluctance to report pain for several reasons including:-

- Desire to be a good patient.
- Fear of addiction.
- Fear that pain means the disease is progressing.
- Patients may not comply with treatment due to a lack of information and understanding of the concept of pain assessment and interventions.

Environment

- Lack of nursing staff to administer analgesia or medical staff to prescribe.
- Low priority of clinical staff to effectively manage pain.

These barriers to effective pain management may result in poor decision making skills by nursing staff thus perpetuating the carrying out of ritualistic practices including:-

-Underestimation of the severity of the patient's pain
-Overestimation of the effectiveness of interventions
-Establishing inappropriate treatment goals
-Administering a lower dose analgesic at longer time intervals than prescribed.
-Reluctance to administer parenteral analgesics.
-Withholding analgesics prescribed on a fixed time interval basis when the patient is not in pain.
-Administering the lowest dose of analgesic possible as opposed to the dose required to control the pain.

Pain Assessment

To be able to manage our patient's pain effectively it is imperative that we are able to assess and document their pain score by simple means. Pain score is also documented on the SEWS chart.
In this Trust we use both the Visual Analogue Scale and the Verbal Descriptor Scale (see below)

Visual Analogue Scale

No Pain **Worst Pain Imaginable**
0_____10

The patient is asked to mark a point on the line that they feel best represents their pain, on the reverse of the line it is marked out in centimetres and their score is measured from that line between 0 and 10 both at rest and at movement.

Verbal Descriptor Scale

Patient is asked on a score of 0 – 3 with 0 being no pain and 3 being the worst pain both at rest and at movement.

0 = No pain.
1 = Mild pain, it does not distress me.
2 = Moderate pain, it distresses me a bit.
3 = Severe pain, it distresses me a lot.

All patients should have their pain assessed at rest and on movement and documented at the same time as their vital signs and more frequent if pain is poorly controlled. Analgesia prescriptions should be tailored to individual patient requirements and efficacy reviewed regularly.

Key Points in Pain Assessment

- Pain is considered the 5th Vital sign
- Patients own verbal report of pain (due to pain being a subjective experience).
- Pain intensity (objective, using an appropriate tool at rest and on movement).
- Location of pain (ask patient to point to where the pain is, not always the wound!).
- Type of pain (intermittent or constant).
- Pain assessment following analgesia.
- Sedation level.
- Nausea and vomiting scoring.
- Vital signs respirations, pulse, blood pressure,
- Clinical signs: sweating, pallor.
- Behavioural signs facial expressions, crying, restlessness, guarding or rubbing of the affected area. (Absence of these does not indicate that patient's pain is controlled).
- Presence of pain that is different in nature to nociceptive pain e.g. burning, shooting, and stabbing.

Opioids
Opioid analgesics remain the cornerstone of acute pain management for moderate to severe pain. They produce analgesia by binding to opioid receptors both within and outside the central nervous system.

Morphine remains the gold standard opioid against which all new analgesics are compared. Although newer analgesics may possess useful qualities, they are not clinically superior in relieving pain. Morphine is metabolised mainly in the liver and the main metabolites are morphine 3-glucuronide, which has no analgesic action, and morphine 6-glucuronide, which is approximately twice as strong an analgesic as morphine. The glucuronides are primarily excreted via the kidney, and renal impairment will lead to a build-up of these metabolites. The most common side effects are sedation, respiratory depression, pruritus, nausea, vomiting, delayed gastric emptying, constipation, urinary retention and hallucinations.
PCA solution:

- 100mg in 50ml 0.9% sodium chloride
- drug concentration 2mg/ml
- standard bolus 1mg/0.5ml
- lockout 5 minutes

Tramadol is a synthetic analgesic, which produces analgesia by two mechanisms: an opioid effect and an enhancement of descending pain-modulating pathways. It is not as effective as morphine for the treatment of severe postoperative pain. The benefits of tramadol are that it produces minimal sedation and respiratory depression and it can therefore be a useful alternative to morphine for PCA use e.g. for patients who become excessively sedated with morphine.
PCA solution:

- 500mg in 50ml 0.9% sodium chloride
- drug concentration 10mg/ml
- standard bolus 10mg /ml
- lockout 5-10 mins **(maximum IV dose in 24hrs 600mg)**

Fentanyl is a highly lipid-soluble synthetic opioid that is metabolised in the liver to inactive metabolites and may therefore be a safer drug to use in renal failure. It has a more rapid onset of action than morphine.
PCA Solution :

- 1000mcg in 50ml 0.9% sodium chloride
- drug concentration 20mcg/ml
- standard bolus 10mcg
- lockout 5-10 mins

Side Effects of Opioid Analgesia

Respiratory Depression:
Respiratory depression may occur following opioid administration by any route. It may be slowly progressive or unpredictable in onset. Respiratory depression is often associated with an increase in sedation, which should be acted on promptly.

If respiratory rate is 10/min or less but patient is easily rouseable:

- Ensure patient has a clear airway and is in the lateral position if possible.
- Give oxygen 6 litres per minute via face mask.
- Continuously observe the patient.
- Stop administration of opioid.
- Ensure Naloxone is available.

Severe Respiratory Depression
If respiratory rate is 8/min or less and/or if the patient is very difficult to rouse:

- Ensure patient has a clear airway and is in the lateral or recovery position.
- Give 6 litres per minute of oxygen via face mask.
- Physically stimulate patient and consider hand-ventilation with Ambu bag and Oxygen.
- Stop opioid administration
- Call for help
- Administer Naloxone bolus intravenously:
  Dilute Naloxone 400ug in 10ml 0.9% sodium chloride (= 40ug/ml).
  Administer intravenously at 1ml/min slowly until respiratory rate is satisfactory and patient awake or easily rouseable.

If there is no response after a total of 400ug Naloxone, reconsider the diagnosis and contact emergency anaesthetist.

- Naloxone infusion:
  A Naloxone infusion may be indicated if there is an ongoing risk of respiratory depression:
  Dilute Naloxone 2mg (5 × 400μg ampoules) in 500 ml 0.9% Sodium Chloride or 5% glucose.
  Administer intravenously according to response.

Sedation:
Increasing sedation may be associated with compromise of the airway and should be recognised and managed promptly. Increasing sedation is also a predictor of impending respiratory depression. Increasing or excessive sedation should be managed by reducing the dose of opioid and/or considering alternative drugs. Sedation may be a particular problem in the elderly.

Nausea and Vomiting:
This is a very common side effect of opioid analgesia, which can be very distressing to the patient. It should be treated promptly and regularly. An antiemetic should always be prescribed when opioids are administered. Prophylactic treatment should be given to patients with a previous history of post-operative nausea and vomiting (PONV). See algorithm appendix 2 for management guidelines.

Pruritis:
Generalised itching may occur secondary to opioid analgesia and may be treated by antihistamine, or by small IV doses of naloxone in severe cases.
Chlorphenamine:
Dose: 10mgs IM or diluted by slow IV injection

Constipation:
Please refer to the Guidelines for the prevention and treatment of constipation
Drug and Therapeutics Committee bulletin No 32 September 1996 for further details.
An oral stimulant laxative and/or bulking agent are recommended for prevention and treatment.

Mild oral stimulant:	Senna 15mg (2 tablets or 10ml) at night.
Strong oral stimulant:	Bisacodyl 5 to 10mg at night.
Bulking agent:	One Fybogel sachet (3.5g) once or twice daily.

Hallucinations:
This can be a distressing side effect, which usually requires a reduction in dose or change of drug.
Hypoxia should be excluded as a cause of confusion or hallucinations in patients receiving opioids.
Psychotropic drugs should not generally be prescribed as they may cause excessive sedation in the presence of an opioid. If they are considered necessary they should be given in small doses titrated to effect e.g. Haloperidol 1mg iv.

Addiction:
The risk of addiction is considered negligible when opioids are used to treat acute pain. Provided that treatment has been titrated to the patients requirement and is discontinued promptly when no longer required.

Titration of opioid.

For an opioid to be effective it must reach a certain blood level, the effective range of blood concentration varies some fourfold to fivefold between patients. The amount of opioid that each patient requires will vary according to the severity of the pain stimulus. Therefore titration of opioids is needed in order to individualise treatment. To enable opioid analgesia to be titrated appropriate doses and dose intervals need to be ordered. See Protocol appendix 1.

"The key to effective titration of opioids is the use of incremental doses and careful observation of side effects and relief of pain".
Acute Pain Management: Scientific Evidence
National Health and Medical Research Council, Australia. (2005).

IV Opiate Administration Precautions:
The risk of respiratory depression and sedation with airway compromise is high with intravenous techniques and patients should be observed closely. This involves hourly respiratory rates and sedation scores.
Most patients should receive supplemental oxygen therapy for the duration of the infusion.

1. Dosage and concentration of drug must be accurately calculated, labelled and checked. The infusion device must be correctly programmed and must be checked hourly to ensure that the prescribed dose is given.
2. If an opioid is infused through the same cannula as a fluid infusion and the cannula becomes blocked the opioid pump will continue to work but will discharge its contents into the infusion line. When free

flow is established again, or the infusion connected to another cannula, a large bolus of opioid may be delivered. There are two ways of avoiding this:

- Use the desiginated PCA giving set with non-return valve and anti siphon valve.
- Use separate cannulae for fluids and opioids.

One of these methods must always be used

3. The pump must be mounted at or below the level of the patient's heart and an anti-siphon valve must be used to prevent rapid discharge of syringe contents due to gravitational effect.

All infusion techniques and equipment used for such techniques must comply with the Trust Guidelines and Medical Devices Agency (2003).

Patient Controlled Analgesia (PCA)

This technique involves the patient controlling the rate of administration of drug and thus dosing is better matched to individual requirements for analgesia. Bolus and Lockout time is prescribed and programmed accordingly to patient needs. Each time the patient presses the button the preset dose is administered unless this occurs during the "lockout" interval. The PCA machine can also provide a background infusion of opioid in addition to self-administration. However, this may increase the risk of side effects and should not be used routinely.

PCA should allow for a wide variation in opioid requirement because each patient titrates consumption to achieve a balance between analgesia and unwanted effects such as nausea. Under no circumstances should anyone else operate the button except in special circumstances e.g. patients with severe arthritis or in Intensive Care areas.

Analgesia must be established prior to commencing PCA. Loading doses maybe required prior to interventions i.e dressing changes, physiotherapy. Thereafter patients can resume self administration with PCA.

Patients should ideally be fully instructed in the use of PCA and be familiar with the hand trigger before surgery, instruction postoperatively is always reinforced. They must be able to operate the hand trigger (some patients with arthritis may have difficulty) PCA alternative handsets are available.

Adjunct Analgesics

Non-Steroidal Anti-Inflammatory Drugs

NSAIDS act by decreasing levels of inflammatory mediators generated at the site of injury, specifically prostaglandins. They have been found to have significant opioid sparing properties and can enhance the quality of opioid-based analgesia making them a useful adjunct in patients with PCA.

NSAIDS have **significant side effects.**

They are contraindicated in patients with **renal impairment, hypovolaemia, peptic ulcer disease, asprin sensitive asthma, coagulopathies or on anticoagulants and must be used with caution in the elderly.**

Most common NSAID's are Diclofenac Sodium, Ibuprofen and Piroxicam.

Paracetamol

Paracetamol is an important and often underestimated option in the treatment of mild to moderate pain and has been found to have opioid sparing properties when used as an adjunct with opioids. It has analgesic and antipyretic effects but is not considered to be anti-inflammatory. There is controversy about the mechanism of action but it appears to have a significant central action. Due to its opioid sparing properties most patients should be prescribed paracetamol 1gram 4 times a day. Paracetamol is available in oral, rectal & IV preparations.

(*Avoid rectal administration of any medication in patients with a low colorectal anastamosis). Paracetamol is contraindicated in patients with active liver disease and glucose 6 phosphate dehydrogenase deficiency where its use may precipitate liver failure.

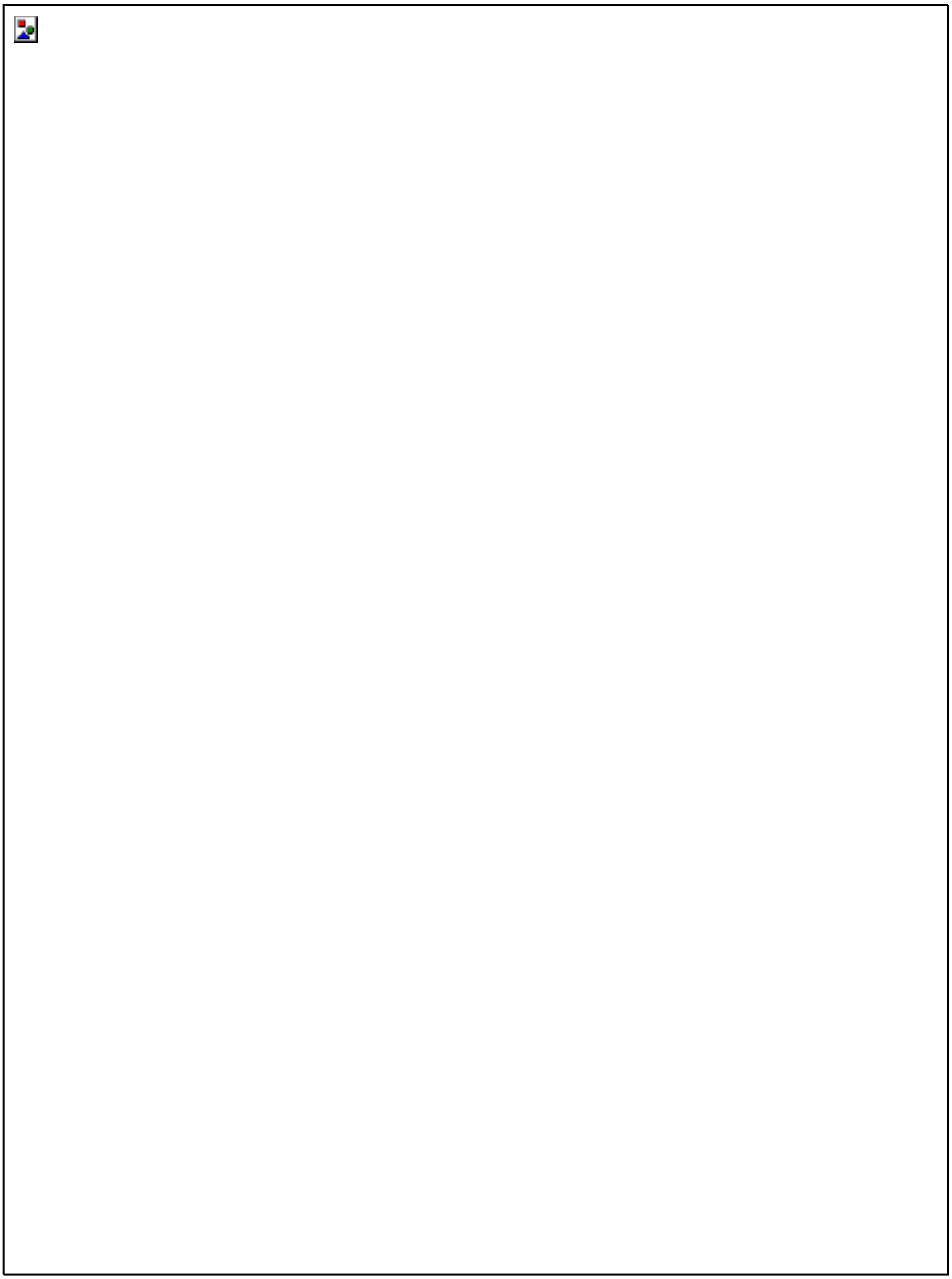
Discontinuation of PCA

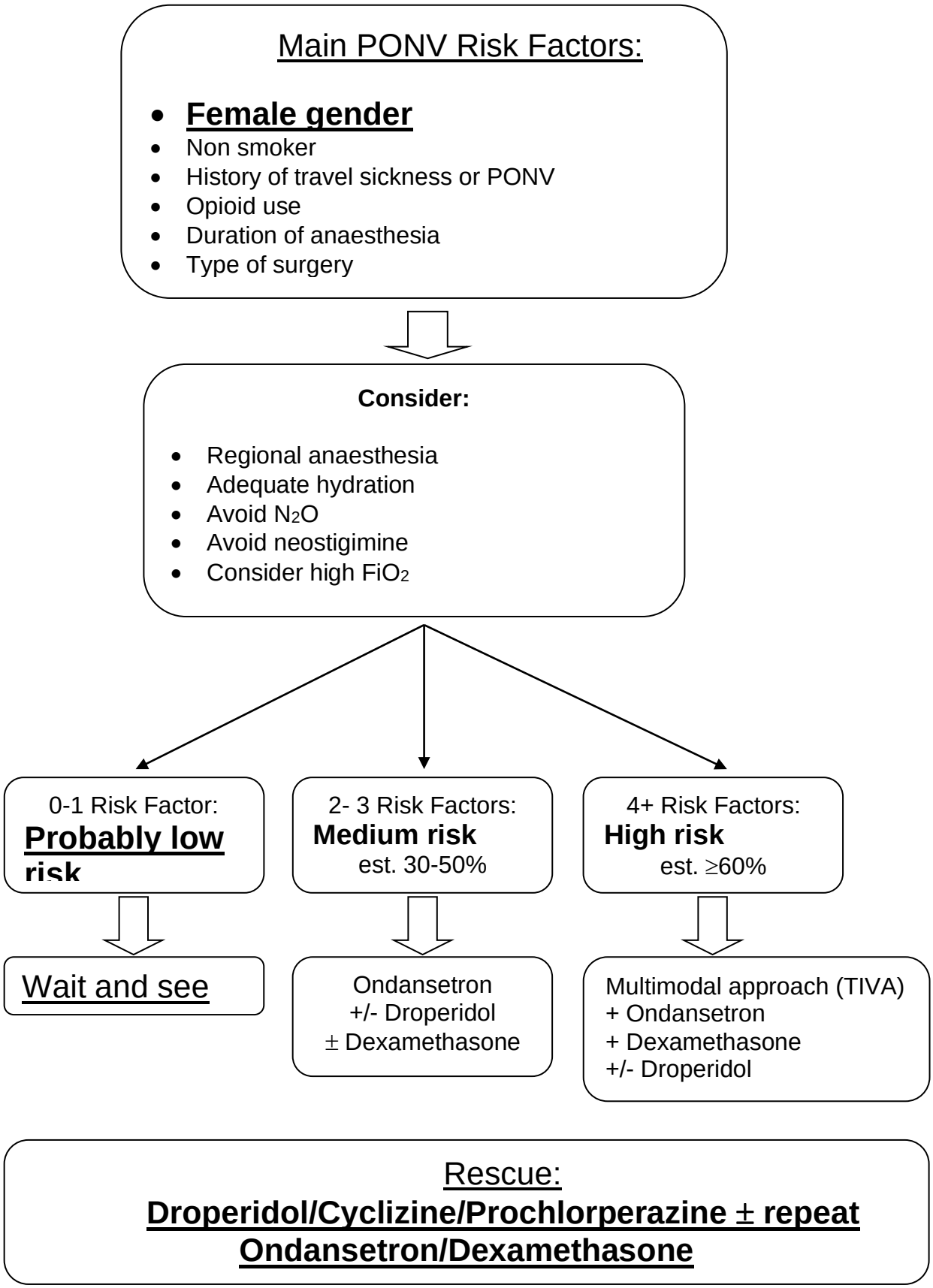
In general PCA should be maintained until oral medication can be used, step down analgesia should be prescribed and administered **at least one hour** prior to discontinuation of PCA to ensure continued effective analgesia. If PCA must be discontinued before oral fluids are established, other parentral opioids (subcutaneous or intravenous) will be needed.

We recommend that step down analgesia should consist of a milder opioid (e.g. codeine or tramadol) +/- NSAID + paracetamol. In addition to regular analgesia, rescue analgesia for breakthrough and incident pain should also be prescribed.

PCA Problem Solving

POSSIBLE PROBLEM	POSSIBLE SOLUTION
PAIN	Check the patients understanding of PCA and the correct use of the device. Check that the pump is working correctly. Check multimodal analgesia in progress, e.g. regular paracetamol and NSAIDS if not contraindicated. Ensure venflon is patent. Where is the patient experiencing pain? Has the pain changed in nature? Is there a significant change in the patients clinical condition? Ensure the patient has received adequate loading analgesia. Was the patient taking preoperative analgesics?
SEDATION	What is the sedation score and respiratory rate? Stop PCA Ensure that oxygen is administered. Monitor oxygen saturations. Alert medical staff (on call anaesthetist bleep WGH 8112 RIE 2140 and CNS Acute pain bleep WGH 8292 RIE 5247). Consider naloxone. Consider reducing bolus or stopping background infusion. Consider changing from morphine to tramadol Ensure only the patient is activating the demand button. **NB Patients will always become sedated before respiratory arrest. Acting on the sedation may prevent a respiratory arrest.**
NAUSEA	Is the patient receiving appropriate antiemetic therapy? Is the patient adequately hydrated? Rule out other possible causes. Consider increasing dose duration time. Consider change to alternative analgesia**.**
ITCH	Consider antihistamines - watch sedation. Consider small dose of naloxone. Consider change of opiates
REQUEST TO STOP PCA	Ascertain why the patient or medical team wants to stop the PCA. Consider the possible step-down analgesia options. Can the patient take oral analgesia? Will alternative analgesia be adequate?
Mechanical pumps alarm keeps being triggered.	Is there an occlusion? Is the pump plugged into the mains? Is it programmed correctly? Is the syringe fitted correctly? **If pump not functioning correctly for any other reason remove pump and return to medical physics.**




Main PONV Risk Factors:
Female gender
Non smoker
History of travel sickness or PONV
Opioid use
Duration of anaesthesia
Type of surgery
Consider:
Regional anaesthesia
Adequate hydration
Avoid N2O
Avoid neostigimine
Consider high FiO2
0-1 Risk Factor:
Probably low risk
2- 3 Risk Factors:
Medium risk
est. 30-50%
4+ Risk Factors:
High risk
est. ≥60%
Wait and see
Ondansetron
+/- Droperidol
± Dexamethasone
Multimodal approach (TIVA)
+ Ondansetron
+ Dexamethasone
+/- Droperidol
Rescue:
Droperidol/Cyclizine/Prochlorperazine ± repeat Ondansetron/Dexamethasone

References

Duthie, D J R (1994) The Physiology and Pharmacology of Pain. Anaesthesia, 2nd edition, vol 1, Nimmo, W S, Rowbotham, D J and Smith, G, p118-131, Blackwell Science Publications.

Hawthorne, J and Redmond, K (1998) Pain: causes and management, Blackwell Science, Malden MA.

International Association for the Study of Pain Sub Committee on Taxonomy (1979) Pain Terms: a list with definitions and notes on usage. Pain 6, p249-252.

McCaffery, M and Pasero, C (1999) Pain, Clinical Manual, Mosby, London.
Melzack R & Wall PD (2003) Handbook of Pain Management. Churchill Livingstone, Edinburgh.

Puntillo, K A (1988) The phenomenon of pain and critical care nursing. Heart and Lung, 17 (3), p262-271.

United Kingdom Central Council for Nursing, Midwifery and Health Visiting (1992) Scope of Professional Practice, London.

Woolf, C J and Chong, M S (1993) Pre-emptive analgesia – treating postoperative pain by preventing the establishment of central sensitisation. Anaesthesia and Analgesia 77, p362-379.

Bibliography

Power, I (1998) Acute pain management: scientific evidence. Ampersand editorial & design, Canberra.