HAUORA TAIRAWHITI
PAIN MANAGEMENT HANDBOOK

March 2016

Acknowledgements to:
Dr Deon Stoltz, Gisborne Hospital Acute Pain Service Guidelines,
The Health Waikato Pain Management Workbook for Nurses and Midwives August (2007),
Hauora Tairawhiti Pain Management Committee 2015-16

Hauora Tairawhiti Pain Management Team
Date first created: July 2014
Reviewed & Updated March 2016

WARD/DEPARTMENT: _____

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See Paediatric Pain Management Manual in next section

INTRODUCTION

These guidelines have been developed by members of the HAUORA TAIRAWHITI Pain Management Committee as a combination of the Acute Pain Service Guidelines developed by Deon Stoltz, the Waikato based Pain Management Workbook and Hauora Tairawhiti IV policies and guidelines. We acknowledge the willingness of Waikato and particularly Deon in allowing us to use their work.

From Deon,
"Acute pain is defined as pain that is present in a surgical patient because of pre-existing disease, surgical intervention, or a combination of both disease and procedure related.

Pain assessment and treatment is a complex and difficult task. Acute pain comprises of sensory stimuli with an influence on the affect that can cause great suffering. It is important to listen to patients and believe them in the description of their pain, as with most things in life the experience of acute pain is quite unique to each individual. "Pain is whatever the person experiencing it says it is, existing wherever they say it does" (McCaffery, 1979)

This package is to provide guidelines that are based on current literature, and evidence based practise in the management of acute pain. Guidelines are not absolute rules, and need updating and reviewing on a regular basis. This workbook and guidelines is not an attempt to exclude the cognitive step in treating our patients but rather to give a departure point in treating acute pain. The rest of the journey with the patient may need further input or recommendations that falls outside the scope of these guidelines.

Not only as health care workers but also as compassionate fellow human beings it is our duty to deliver effective efficient pain relief to our patients. The results can be unpredictable, rewards diminutive. Patients may be discontent with their care, but this should only inspire us to be more individualistic in our approach to the patient with acute pain."

Deon Stoltz
DEPARTMENT OF ANAESTHESIA
Gisborne Hospital

It is important to note that any nurse (RN, EN & Obstetric Nurse) or midwife working with parenteral medicines is required to have **generic** IV certification. Specialty certification is required for administering IV narcotics, and for PCA and epidural management.

The information in this package is comprehensive, but not all inclusive. A list of approved resources is provided which supports the information given in this workbook. In addition, a reference list is provided for those who wish to follow up with further reading (on the back page). We hope this package can be used as an ongoing reference for nursing/midwifery and medical staff.

While we have done our utmost to consult and edit this workbook, we would appreciate any feedback. Please notify the Nursing Education Co-ordinator if you notice any glitches or have any constructive feedback.

Regards
THE HAUORA TAIRAWHITI PAIN MANAGEMENT COMMITTEE

SECTION 1

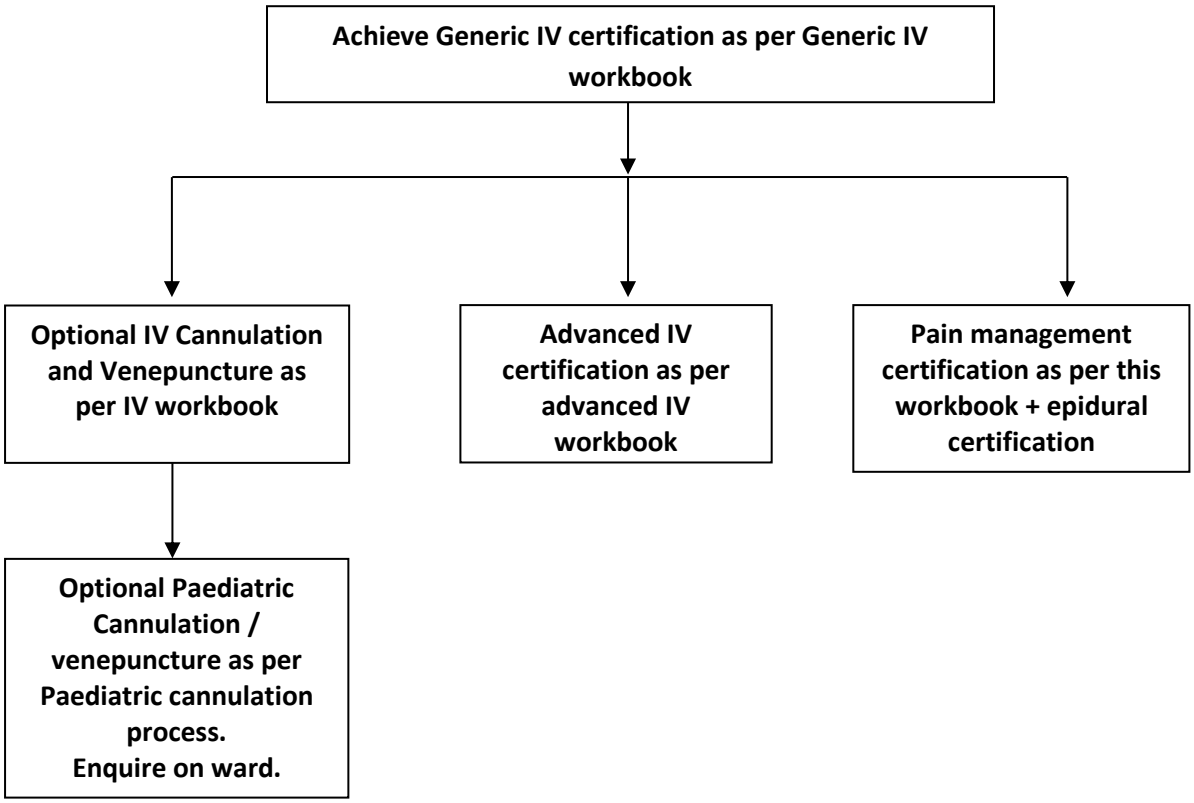
CERTIFICATION REQUIREMENTS AND PROCESS
PAIN MANAGEMENT FOR NURSES AND MIDWIVES

ACHIEVE GENERIC IV CERTIFICATION:

- On commencing employment, or returning after an absence of more than one year, the nurse or midwife is orientated to the IV competence assessment and validation process as per Generic IV workbook. Demonstration of equipment and procedures is given.
- If the nurse or midwife holds certification from another organisation, they need to show this to the Clinical Nurse Manager/Midwife Unit Manager. If this is comparable to HAUORA TAIRAWHITI standards, then these are accepted and placed on file.
- Please refer to HAUORA TAIRAWHITI organisational policy (2014) Intravenous Therapy Competence Assessment and Validation Process (Appendix 1)

ACHIEVE SPECIALTY COMPETENCIES: - *dependent on requirements of area worked*

- Cannulation and Venepuncture Certification (Optional)
- Advanced IV Certification
- Pain management
- Epidural management



IV NARCOTICS AND EPIDURAL MANAGEMENT PROCESS:

- Complete the basic IV certification process
- Read this pain management handbook.
- Familiarise yourself with relevant documents in the Tairawhiti DHB Intravenous Therapy and Related Procedures Manual-Best Practice Guidelines
- Complete certification written tests.
- Complete the pain management workshop; (if available)
- Pass three competency-based practical assessments. Preferably two assessments to be completed prior to study day. Assessed by:
  - Clinical Nurse Manager/Midwife Unit Manager
  - Nurse/Midwifery Educator
  - Other credentialed IV assessor
  - Duty Nurse Manager if credentialed assessors.

FINAL ASSESSMENT MADE AT THE PAIN MANAGEMENT WORKSHOP: (IF POSSIBLE)

- Send completed certification tools workbook and evidence of practical assessments to Nursing Quality Services for marking
- Gain 100% pass in the written assessments
- The evidence of completion is your marked workbook
- Midwives: It is recommended that midwives attend both the specific labour epidural workshop and the full pain management workshop with successful completion of the workbook and practical assessments as above.
- Epidural certification is only required in areas of use. However all staff to attend epidural part of pain management workshop and to read epidural part of this workbook so you have an understanding of the principles if you are deployed to an area where epidurals are used
- ***Revalidation is required annually and is part of the You-Time process.***
- The Clinical Nurse Manager/Midwife Unit Manager/or designated IV assessor will choose one core competency to be assessed and sign a certificate of competence when this has been demonstrated. (See Appendix 1)

THE CERTIFICATION WORKBOOKS AND ASSESSMENTS SHOULD BE SUCCESSFULLY COMPLETED BEFORE THE NURSE/MIDWIFE PERFORMS ANY PROCEDURES UNSUPERVISED

Once begun, the IV narcotics and epidural certification process should be completed as quickly as possible. It is expected that this take no longer than six months.

NB: To maintain consistency of practice, your practical assessment will not be valid unless this is carried out by a nominated IV assessor. Check within your clinical area to identify those IV assessors who are designated to carry out opioid and/or epidural certification assessments.

MIDWIVES:

It is compulsory that employed midwives complete the Hauora Tairawhiti epidurals in labour workshop within 12 months of commencement of employment and it is recommended that self-employed midwives undertake the workshop if caring for clients who have epidurals.

This provides evidence to demonstrate that you are maintaining clinical standards of practice in caring for women who become users of the Hauora Tairawhiti services for women and babies.
Section 88 specifies that Lead Maternity Carers may opt out of caring for women with epidurals in labour if they are not competent to do so, and would then hand over care to the core midwife.

ACUTE PAIN SERVICES: (APS)

Acute Pain Services have been around since the mid-1980s. Usually anaesthetist-led, acute pain teams offer a service that aims to provide expert support and advice for patients with pain, regardless of its aetiology.

The majority of patients have postoperative or trauma-related pain, but other patients may have sub-acute, chronic benign and malignant pain. Selected patients such as those with epidurals or peripheral nerve sheath catheters and PCA will be routinely seen by the pain team but patients still remain under the care of the primary care team.

It is hoped to set up an acute pain service at Hauora Tairawhiti in the near future, although at the time of writing this workbook this is not available. Currently, the prescribing anaesthetist manages cases on an individual basis apart from in neonates or paediatrics.

AREA SPECIFIC GUIDELINES:

Please note that in specialist areas such as Paediatrics, Midwifery, Palliative Care, there may be further guidelines specific to these areas.

LEARNING OUTCOMES:

Upon completion of the education process, you should be able to demonstrate knowledge of:

- Competence requirements for Hauora Tairawhiti
- Neurophysiologic mechanisms of pain, including the Gate Theory
- Appropriate assessment of chronic and acute pain
- Evaluation of effectiveness of analgesia given
- Monitoring requirements for opioid analgesics/local anaesthetics
- Mode of action of opioid analgesics and local anaesthetics;
- Benefits of opioid analgesics and mechanisms of action;
- Methods of administration of opioids, and local anaesthetics;
- Management considerations for minimising/treating complications;
- Specific nursing/midwifery responsibilities.

Should you have any questions regarding the content of this package, please contact:
Nurse Co-ordinator Education and Professional Development
Nursing and Quality Services (Ward 10, Second Floor)
HAUORA TAIRAWHITI

COMMON PAIN TERMS:

Pain

"*An unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage*" - International Association for the Study of Pain. Pain is always subjective for each individual, learning about the application of the word through experiences related to injury in early life.

Acute Pain

The normal, predicted physiological response to an adverse chemical, thermal, or mechanical stimulus associated with surgery, trauma, and acute illness. It is generally time-limited and is responsive to Opioid therapy, among other therapies.

Chronic Pain

A pain state which is persistent and in which the cause of the pain cannot always be removed or is difficult to treat. Chronic Pain may be associated with a long-term incurable or intractable medical condition or disease.

Nociceptors
Sensory neurons that are found in any area of the body that can sense pain either externally or internally

Hyperaesthesia (opposite = Hypo-aesthesia)
Where a non-painful stimulus is perceived to be stronger than normal (weaker than normal) but is not painful e.g. a light touch becomes a stronger (weaker) touch but is not painful.

Hyperalgesia (opposite = hypo-algesia)
Where a painful stimulus is perceived to be more painful than normal (less painful than normal) e.g. a pin prick is felt as a stronger (weaker) pinprick than normal.

Paraesthesia
An abnormal sensation which is not perceived as painful e.g. gently knocking your funny bone at the elbow causes non-painful tingling in your little finger.

Dysaesthesia
An abnormal sensation which is perceived as unpleasant or painful e.g. strongly knocking your funny bone at the elbow causing painful unpleasant tingling and pain in the little finger.

Allodynia (Greek - other pain)
Pain produced by a stimulus that would not normally cause pain e.g. lightly stroking the skin causing vibrational allodynia, putting your hands in luke warm water causing thermal allodynia.

Tolerance
A physiologic state resulting from regular use of a drug in which an increased dosage is needed over time to produce the same effect, or a reduced effect is observed with a constant dose.

Opioid Tolerance
The need to increase the dose of Opioid to achieve the same level of analgesia. Opioid Tolerance may or may not be evident during treatment and does not equate with addiction.

Physical Dependence
A physiologic state of neuro-addiction which is characterized by the emergence of a withdrawal syndrome if drug use is stopped or decreased abruptly, or if an antagonist is administered. Physical Dependence is an expected result of Opioid use. Physical Dependence, by itself, does not equate with addiction.

Addiction
A neurobehavioral syndrome with genetic and environmental influences that results in psychological dependence on the use of substances for their psychic effects and is characterized by compulsive use despite harm. Addiction may also be referred to by terms such as "drug dependence" and "psychological dependence". Physical dependence and tolerance are normal physiological consequences of extended Opioid therapy for Pain and should not be considered addiction.

Pseudo Addiction
A pattern of drug seeking behaviour of pain patients who are receiving inadequate pain management that can be mistaken for addiction.

Substance Abuse
The use of any substance(s) for non-therapeutic purposes or use of medication for purposes other than those for which it is prescribed.

SECTION 2

OVERVIEW OF ACUTE PAIN MANAGEMENT

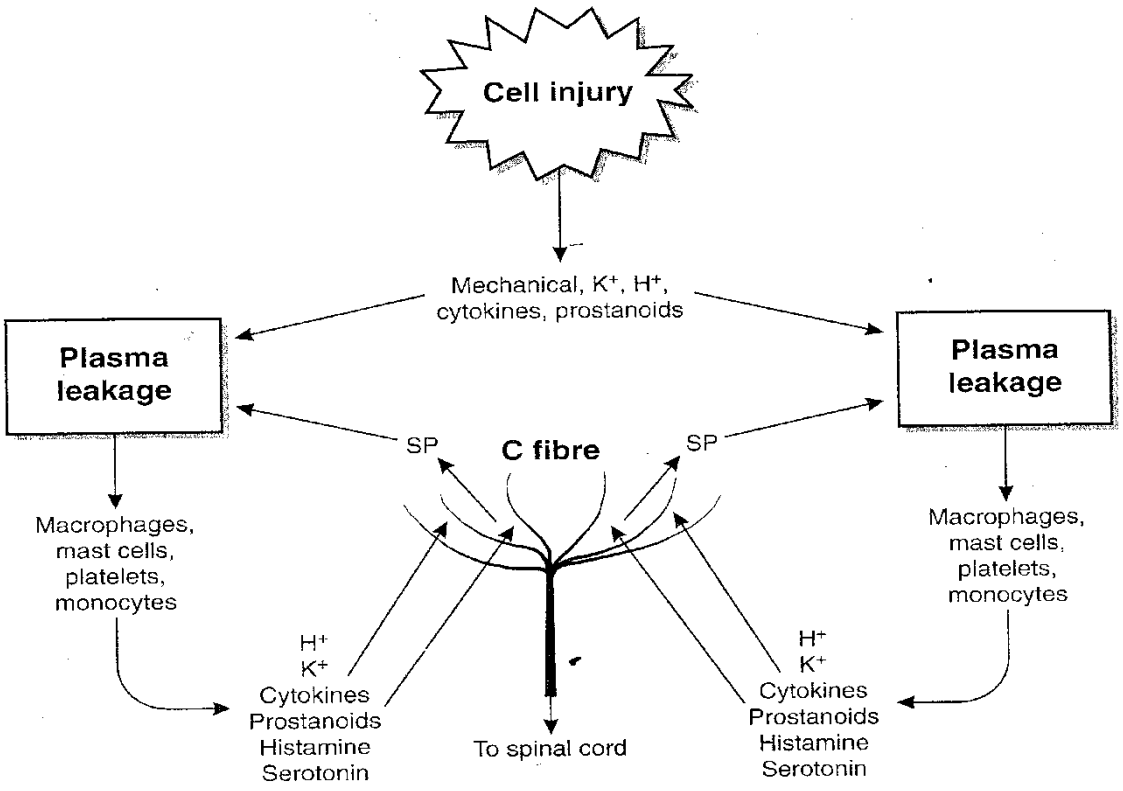
"Pain is a more terrible lord of mankind than death itself" - Albert Sweitzer

WHY DO WE SUFFER PAIN?

- Self-defense mechanism to protect us from noxious stimuli.
- To relay to the person that tissue damage has occurred, such as trauma and surgery.
- Severity of pain normally equates to severity of injury or tissue damage.
- Pain is usually self-limiting and decreases over time.

HOW DO WE FEEL PAIN?

Let's take an example like cutting your finger with a sharp knife.
Pain receptors in the skin are stimulated by the injury, due to the release of various chemicals by the damaged cells including histamine, substance P, serotonin (5HT), bradykinin and prostaglandins.



Pain signals are generated by these receptors which travel via the sensory nerves to the spinal cord.

The cell bodies of these sensory nerves are grouped together in a small swelling called the dorsal root ganglion.
In the spinal cord the pain signals are processed by a "computer" called the dorsal horn.

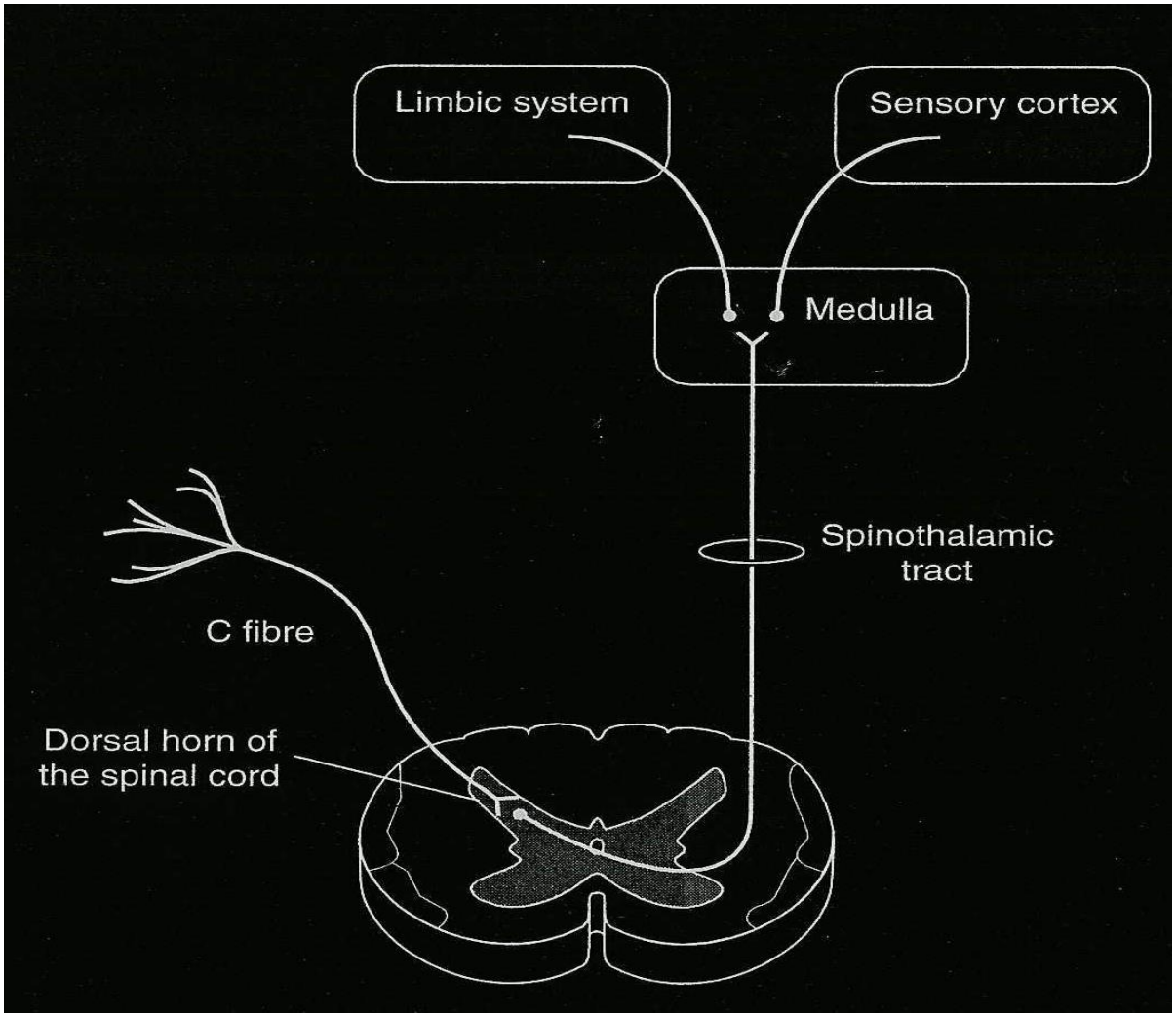
Signals come out of the spinal cord and travel via motor nerves to the arm muscles, causing the arm to withdraw quickly. This is an automatic reflex that does not involve the brain or conscious thought.

Depending on the settings in the dorsal horn computer (see Gate Theory and Dorsal Horn Sensitisation below), pain signals are also sent upwards in the spinal cord via the Spinothalamic tract (amongst others) to an area in the brain stem (base of the brain) called the thalamus.

Further processing occurs in the thalamus with signals being sent to areas controlling blood pressure, heart rate, breathing, and emotions. An acute pain event often causes a rise in heart rate, blood pressure, and breathing rate, as well as a change in emotions and behaviour e.g. shouting "ouch", contorted facial expressions, and behavioural displays such as waving the arm in the air.

Pain signals are also sent upwards from the thalamus to the primary sensory cortex (part of the outer surface of the brain dealing with sensory input). It is thought that some crude perception of pain and sensation occurs at the thalamic level, with much finer discrimination occurring in the primary sensory cortex.

There is initially a sharp fast onset of short- lived pain transmitted from the injured area to the spinal cord dorsal horn by large diameter high velocity sensory nerves (A-delta fibre nerves). This is followed by a dull, slower onset and longer lasting pain transmitted from the injured area to the spinal cord dorsal horn by smaller diameter low velocity sensory nerves (C fibre nerves).



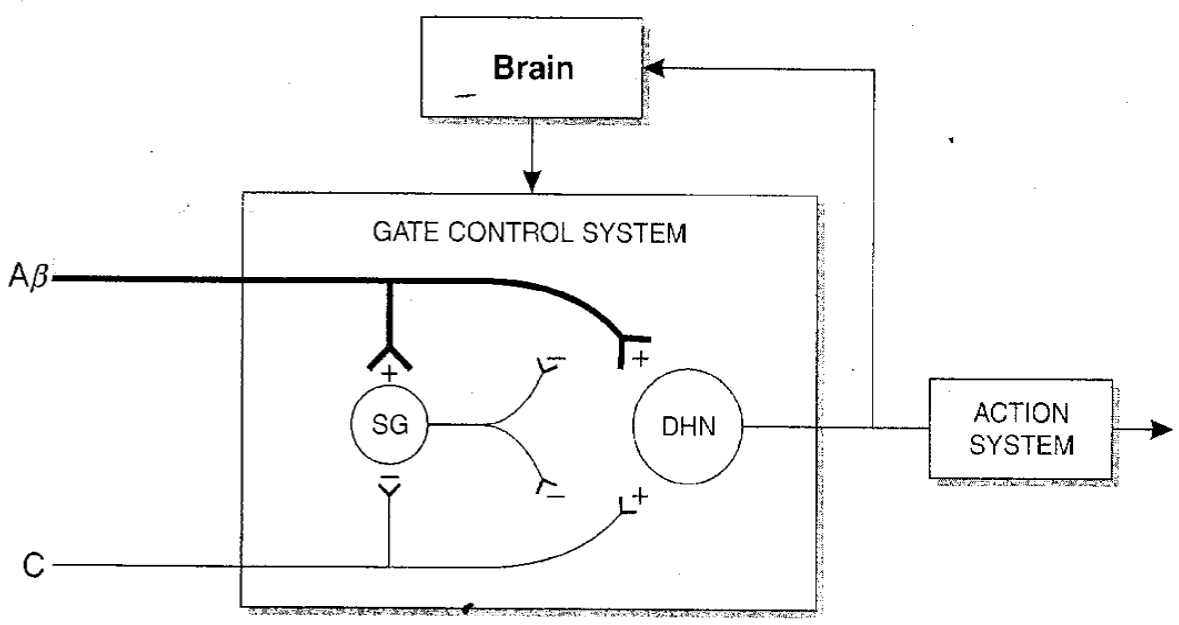
PAIN MODULATION: (Pain Gate Theory)

Rubbing an injured area often helps to ease the pain. Rubbing stimulates vibration receptors, sending signals to the dorsal horn via large diameter A-beta fibres.

These vibration signals enter the dorsal horn computer at the same time as the small diameter C fibre pain signals from the injured area

If the vibration signals are of the correct magnitude, they prevent further onward transmission i.e. closing the gate on pain.

Many other treatment modalities like TENS, acupuncture and heat produce pain relief by a similar mechanism. TENS stimulates the A-beta fibres, and acupuncture stimulates the A-delta fibres.



Gate control theory of pain. SG substantia gelatinosa. DHN dorsal horn neurone

CENTRAL SENSITISATION:

Modulation of afferent input activity in the dorsal horn increases or decreases depending on the activity of the other systems input on the synapse. Within hours of an injury, changes take place in the dorsal horn of the spinal cord that alters the way sensory impulses are processed. When these changes have occurred the dorsal horn is said to have become sensitised. This means that sensory and painful signals are more likely to be transmitted up the spinal cord to the brain, rather than being blocked at the dorsal horn level. Sensitisation is said to be dependent on N-methyl-D-aspartate (NMDA) receptor activation.

WIND UP:

NMDA (n-metyl d-aspartate) receptor activation increases dorsal horn cell output. The c-fibre stimulation at fixed frequency produces an output of progressive increases in frequency. NMDA receptor antagonists (blockers) like ketamine can help prevent sensitisation occurring.

Clinically dorsal horn sensitisation can be measured as changes in pain and sensory thresholds e.g. for temperature sensation the normal comfortable threshold will be lowered in the area of skin supplied by the sensitised dorsal horn.

Sensory thresholds can be altered for all the sensory modalities including vibration, heat, cold, light touch. Thresholds for pain can also be altered in two ways:-

- A stimulus that was not painful before is now perceived as painful.
- What would have produced a little pain now causes a great deal of pain.

Normally after an injury dorsal horn sensitisation reduces in line with tissue healing. However, in some people the sensitisation seems to go on for much longer, and may explain why some go on to develop chronic pain. In some of these people there is a continuing focus of pain in the periphery which continues to keep the dorsal horn sensitised, and in others the exact cause is unknown.

There is also a connection between emotions and dorsal horn sensitisation. In severe anxiety and depression states, lack of descending inhibition is enough to maintain the dorsal horn in its sensitised state.

EFFECT OF EMOTIONS ON MODIFICATION OF PAIN:

Emotions can also affect the gate in the dorsal horn computer. The normal state of affairs is that there are continuous descending signals from the brain to all the dorsal horn computers in the body.

These descending signals (descending inhibition) keep nearly all of the gates in a closed state, preventing unnecessary sensory information reaching the brain i.e. preventing sensory overload.

Emotions like anger and excitement tend to increase the degree of descending inhibition, making it harder for pain signals to gain access to the spinal cord and brain e.g. a footballer injures himself on the pitch but doesn't notice the injury until he stops playing. Distraction therapy also works by a similar mechanism.

Emotions like anxiety and depression tend to reduce descending inhibition, making it easier for pain signals to gain access to the brain and spinal cord e.g. patients with anxiety and depression have increased pain perceptions compared to normal people.

There aren't too many adults in this world who do not carry some form of emotional baggage around with them. Carrying this baggage around on a daily basis can seriously impair your ability to deal with many things in life, including relationships, work, and coping with pain.

Examples of baggage are:

- Guilt about things that they should or should not have done or said in relation to partners, spouses, children or parents.
- Emotional turmoil caused by bereavement, separation, divorce, a bad relationship, or even marrying the wrong person.
- Socioeconomic Distress - caused by the effects of sudden loss of income due to redundancy, battling with government departments about income support and disability living allowance etc.
- Childhood Abuse - emotional, physical and sexual abuse in childhood / early teens can have catastrophic effects on the ability to cope with life in general. Some adults have already come to terms with the past and no longer require any attention, whereas others are desperate for help and guidance, but are afraid to talk because of feelings of guilt or shame. Some abused children receive support afterwards from trusted family members, helping them to weather the storm. The most pernicious situation seems to be where the child was blamed by an adult (usually the mother) for leading the perpetrator astray. As adults this latter group seem to have the greatest disruption to their coping abilities, and require the most care and support.

Assessing and understanding people's emotional baggage is therefore very important when trying to understand their pain. There is a very close link between our emotions, beliefs and our behaviour. Read more about beliefs in the section below.

CHRONIC PAIN:
Commonly persists beyond the time of healing of and injury & frequently there may not be any clearly identifiable cause.

Referred to as persistent pain which occurs after an acute painful episode. Features of inflammatory, visceral, neuropathic or cancer pain may be components of pain of varying duration. Acute and chronic pain may represent a continuum rather than distinct entities. Chronic pain is very common after surgery if actively searched for. Attention to adequately managing acute pain post operatively may influence the progression to chronic pain.

CHRONIC PAIN AFTER SURGERY

Type of Operation	Incidence %
Amputation	30 - 85
Thoracotomy	5 – 67
Mastectomy	11 – 57
Cholecystectomy	3 - 56
Inguinal Hernia	0 – 63
Vasectomy	0 – 37
Dental Surgery	5 - 13

Pain management techniques can therefore be divided into three broad areas:
- Reducing the magnitude of pain signals coming from the periphery by either blocking the nerves that carry the pain or by doing something to the tissue that is generating the painful signal e.g. steroid injections reducing peripheral tissue inflammation.
- Reducing the degree of dorsal horn sensitisation by using analgesic drugs, TENS, Acupuncture, and spinal manipulation.
- Improving descending inhibition by examining patient beliefs, improving education, treating anxiety and depression, and by providing reassurance that there is nothing terrible going on

BELIEFS:
Most of our behaviour in life revolves around our own individual set of beliefs - for example:
- You believe that brushing your teeth is good for your gums and teeth, and therefore you do it twice a day
- I believe that sitting in front of this computer is going to make me rich one day, and therefore I sit here for hours on end typing away!
- We develop our beliefs from our own life experiences - for example:-
  - What we have witnessed with our own eyes (personal interactions and interpretations)
  - What we have been told by others (parents, media, education, health professionals)

Generally speaking if you believe something is good for you, you keep doing it, and if you believe something is bad for you, you stop doing it (avoid it).

Now let's take an example of two patients with acute low back pain seeing different doctors about their problem:
- Patient A consults Doctor A who says, "*the pain is due to an acute soft tissue sprain, the body has tremendous powers of healing, it's a self-limiting problem, no real harm has been done, keep as active as you can within the pain, and then you will have a 90% chance of it all settling down on its own without treatment in 2 weeks*".
- Patient B consults Doctor B who says, "*You've damaged your back whilst lifting at work. The x-ray you had yesterday shows early osteoarthritis. Rest if it hurts too much. Your pain is a warning that you've overdone things. It can only get worse with age. There is no cure for spinal arthritis*".

You can see that these two patients will come out of the surgery with completely differing ideas about their back pain. Their doctors have instilled different beliefs into their minds. From now on their behaviours in relation to their back pain are going to be completely different.

Patient A will have positive beliefs around his back pain, expecting that the pain will go away on its own, and that maintaining normal activities will be good for his back. It is quite likely that this patient will recover fully and go on to have a normal lifestyle.

Patient B will have a negative set of beliefs around his back pain, expecting that he is doomed for ever, that it can only get worse, that rest is the only cure, and that activity will cause more pain and therefore more damage. Because he's "been told by his doctor", he will now modify his behaviour, become less active, develop more back pain because of his inactivity, and slowly spiral downwards into disability, chronic pain and dependency.

If over a period of time Patient B is repeatedly told by his doctor and other health professionals (nurses, physiotherapists) that his back pain is due to spinal arthritis (spondylosis), the message becomes reinforced and more entrenched in the patient's mind.

Every time he modifies his behaviour (does less) in response to the pain, he becomes more unfit and more prone to having back pain, reinforcing his own beliefs. These beliefs can also be inadvertently reinforced by loved ones and colleagues at work by being over concerned about the pain and telling them to do less / take it easy.

When the pain comes on with every movement, and the pain in the patient's mind means that his back has been damaged further, several things then happen:

- The patient becomes frightened to do anything that may cause the pain - this is called Pain Avoidance Behaviour or fear of the pain.
- He anticipates the pain before he moves, causing him to hold his breath and guard his back, whilst tightening his back muscles - this is called Guarded Movements. Guarding only serves to increase the pain during movement, as most of the pain is muscular in the first place.
- Anxiety and depression develop over time with a tendency to overemphasize the pain experience, its cause, and its consequences (to make it seem worse than it actually is, to make the pain into a catastrophe). Anxiety and depression may also cause the patient to misinterpret the severity of the pain leading to a vicious spiral downwards.

When patient B eventually presents to a chronic pain clinic for help, he has firmly entrenched views about the pain, its cause, and how he should manage it. The clinic will examine his beliefs about the pain in order to try to help him, but the longer the abnormal beliefs have been held, the harder it is to change them and the stronger the emotional reaction during the process of trying to change.

The technical word for a belief is “cognition". Psychological treatment to try to re-educate the patient about his beliefs is called Cognitive Behavioural Therapy (CBT).

Many chronic pain clinics have multi-disciplinary teams (pain doctor, clinical psychologists and physiotherapists) who will try to use Cognitive Behavioural Therapy (CBT) to try to modify the patient's set of beliefs about the pain, in order for him to begin the long road towards physical and psychological rehabilitation. They often operate within a Pain Management Program. If his beliefs cannot be changed, then he will not modify his behaviour (not get fit), and not win the battle against chronic pain.

Some patients can manage their pain by combining CBT plus specialised physiotherapy, whereas others need some form of pain relieving procedure before embarking down this road.

Whatever technique is used, the messages are the same:

- They must learn as much about your pain as possible through education.
- They must stop thinking that pain equals more damage.
- They must learn how to control the fear of the pain, and stop anticipating it by guarding your muscles.
- They have to stop catastrophising about the pain, instead trying to minimise it in your mind (e.g. telling yourself it's only muscle spasm).
- They must be as active as you possibly can be, in order to prevent the negative consequences of inactivity.

A **VITAL MESSAGE** to all health professionals is therefore:

Careless talk can reinforce abnormal beliefs and impact on patient's quality of life.

Avoid words that focus on unrecoverable, disastrous and negative features of a painful condition (e.g., degenerative spine, crumbling disc, spinal arthritis).

PAIN CLASSIFICATION

Nociceptive Pain arises from the stimulation of specific pain receptors. These receptors can respond to heat, cold, vibration, stretch and chemical stimuli released from damaged cells.	**Non Nociceptive Pain** arises from within the peripheral and central nervous system. Specific receptors do not exist here, with pain being generated by nerve cell dysfunction

Somatic Pain	Visceral Pain	Nerve Pain	Sympathetic Pain
Source - tissues such as skin, muscle, joints, bones, and ligaments - often known as musculo-skeletal pain. **Receptors Activated** - specific receptors (nociceptors) for heat, cold, vibration, stretch (muscles), inflammation (e.g. cuts and sprains which cause tissue disruption), and oxygen starvation (ischaemic muscle cramps). **Characteristics** - often sharp and well localised, and can often be reproduced by touching or moving the area or tissue involved. **Useful Medications** - may respond to combinations of Paracetamol, Weak Opioid's OR Strong Opioid's	**Source** - internal organs of the main body cavities. There are three main cavities - thorax (heart and lungs), abdomen (liver, kidneys, spleen and bowels), and pelvis (bladder, womb, and ovaries). **Receptors Activated** - specific receptors (nociceptors) for stretch, inflammation, and oxygen starvation (ischaemia). **Characteristics** - often poorly localised, and may feel like a vague deep ache, sometimes being cramping or colicky in nature. It frequently produces referred pain to the back, with pelvic pain referring pain to the lower back, abdominal pain referring pain to the mid-back, and thoracic pain referring pain to the upper back. **Useful Medications** -	**Source** - from within the nervous system itself. The pain may originate from the peripheral nervous system (the nerves between the tissues and the spinal cord), or from the central nervous system (the nerves between the spinal cord and the brain) - see Nerve Pain. **Causes** - may be due to any one of the following processes: **Nerve Degeneration** - multiple sclerosis, stroke, brain haemorrhage, oxygen starvation **Nerve Pressure** - trapped nerve **Nerve Inflammation** - torn or prolapsed disc **Nerve Infection** - shingles and other viral infections **Receptors Activated** - the nervous system does not have specific receptors for pain (non nocicpetive). Instead, when a nerve becomes **injured** by one of the processes named above, it becomes electrically unstable, firing off signals in a completely inappropriate, random, and disordered fashion.	**Source** - due to possible over-activity sympathetic nervous system, and central / peripheral nervous system mechanisms. The sympathetic nervous system controls blood flow to tissues such as skin and muscle, sweating by the skin, and the speed and responsiveness of the peripheral nervous system. - see Sympathetic Pain. **Causes** - occurs more commonly after fractures and soft tissue injuries of the arms and legs, and these injuries may lead to Complex Regional Pain Syndrome (CRPS). CRPS was previously known as Reflex Sympathetic Dystrophy. **Receptors Activated** - like nerve pain there are no specific pain receptors (non nociceptive). The same nerve processes as mentioned above may operate in CRPS. **Characteristics** - presents as extreme

	usually very responsive to Weak Opioid's and Strong Opioid's.	**Characteristics -** These signals are then interpreted by the brain as pain, and can be associated with signs of nerve malfunction such as hypersensitivity (touch, vibration, hot and cold), tingling, numbness, and weakness. There is often referred pain to an area where that nerve would normally supply e.g. sciatica from a slipped disc irritating the L5 spinal nerve produces pain down the leg to the outside shin and big toe i.e. the normal territory in the leg supplied by the L5 spinal nerve. Nerve pain is often described as lancing, shooting, burning, and hypersensitive. **Useful Medications** - only partially sensitive to paracetamol, NSAID's, opioid's. More sensitive to Anti-depressants, Anti-convulsants, Anti-arrhythmics, and NMDA Antagonists. Topical Capsaicin, may be helpful.	hypersensitivity in the skin around the injury and also peripherally in the limb (allodynia), and is associated with abnormalities of sweating and temperature control in the area. The limb is usually so painful, that the sufferer refuses to use it, causing secondary problems after a period of time with muscle wasting, joint contractures, and osteoporosis of the bones. It is possible that the syndrome is initiated by trauma to small peripheral nerves close to the injury. **Useful Medications** - many of the features of sympathetic pain are similar to those of nerve pain, and therefore nerve pain medications may be useful (Anti-depressants, Anti-convulsants, and Anti-arrhythmics). Drugs which lower blood pressure by causing vasodilatation (nifedipine) may also be useful when used in combination. Treatment should include appropriate multi-modal medications, sympathetic nerve blocks, and intensive rehabilitation combining occupational and physiotherapy.

SECTION 3

PAIN ASSESSMENT AND MANAGEMENT

WHY TREAT PAIN?

Obviously for **humanitarian reasons,** but also to reduce the physiological effect pain has on the Cardiac, Respiratory, Gastrointestinal, Neuro-endocrine response, and it improves postoperative mobilisation.

PHYSIOLOGICAL:

Cardiovascular

Pain causes increased stress response with increased endogenous catecholamine resulting in increased Cardiac work load → Myocardial ischaemia

Respiratory

- Effects are increased in upper abdominal surgery, the elderly and the obese
- Cause a decrease in fractional residual capacity and tidal volume.
- Suppressed coughing effectiveness leading to Basal atelectasis → pulmonary shunts

Gastrointestinal

- Motility is impaired causing delayed gastric emptying and colon movement. Pain and opioids treatment are synergistic in causing constipation.

Neuroendocrine

- The stress response causes an increase in temperature, leucocytosis, protein catabolism, hyperglycaemia and increases in ACTH, ADH, cortisol, aldosterone, catecholamines**.**

Post-Operative Mobilisation

- Good pain relief aids early mobilisation resulting in decreased complications such as chest infections and deep venous thrombosis.
- Has been shown to decrease time in hospital.

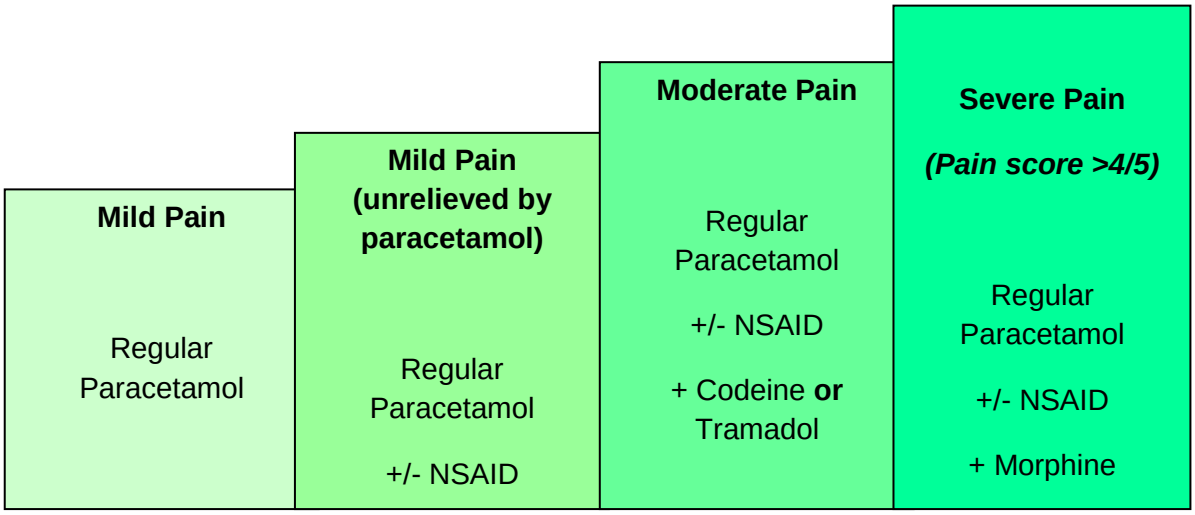
GENERAL APPROACH TO PAIN MANAGEMENT:

- Assess the pain
- Treat the underlying cause
- Non pharmacological
- Pharmacological
- Assess the efficacy and side effects of the analgesic intervention
- Modify the intervention as required
- Treat the analgesic side effects

Patient's pain control needs to be comprehensive and individualized and appropriate for the situation. The important questions that need to be taken into account are type and site of any surgery or injury, severity of pain, and degree of rehabilitation required.

Non-advanced analgesic techniques involve the use of combination of pharmacological agents that fall in categories of mild analgesics, non-steroidal analgesics, opioid and non-opioid analgesics and adjuvant agents. These are often used in combination to provide a multimodal/mechanism of analgesia. The cornerstone of acute pain management remains the use of opioids.

PAIN MANAGEMENT:
Pain should be managed according to the severity and nature of the pain and using the principles of the **WHO (World Health Organisation) Analgesic Ladder**.



The level of pain should be assessed regularly at least ***every 3 hours*** using a visual pain scale and the medication adjusted accordingly.

This emphasises the concurrent use of different techniques to maximise efficacy and moderate the adverse effects of the medications used.

Balanced Analgesia is:
The use of <u>regular</u> background paracetamol and/or NSAID with <u>additional</u> use of opioids as required.

Combinations must be used that are:
- Effective for type of pain
- Blocking pain at several sites
- Useful and advantageous
- Cause individual drug use reduction, and reduction in side effects
- Suit desired results e.g. Epidural and NSAIDS in bowel surgery, LA and paracetamol in kidney failure

What analgesics can be given together?
- Paracetamol
- NSAID or other Cyclo-oxygenase inhibitors (COX II , Parecoxib)
- Background opioid e.g. LA-morph/M-eslon, methadone, oxycodone SR, fentanyl patch
- 'Rescue' opioid, e.g. codeine, tramadol, morphine elixir, sevredol, oxycodone IR
- LA blocks
- Various adjuvants Ketamine

CHOICE OF AGENT:

<table>
<tr><td colspan="2">Nociceptive Pain arises from the stimulation of specific pain receptors. These pain receptors can respond to heat, cold, vibration, stretch and chemical stimuli.</td><td colspan="2">Non Nociceptive Pain arises from within the peripheral and central nervous system. Specific receptors are not involved and the pain is generated by nerve cell dysfunction.</td></tr>
<tr><td>Somatic Pain
Skin, muscle, joints, bones and ligaments. Often sharp and localised and sensitive to touch or movement.

Useful Meds
Paracetamol, NSAIDs, weak opioids, strong opioids.</td><td>Visceral Pain
Internal organs eg gut, liver. Often poorly localised, may feel like a dull ache cramping or colicky pain. Often referred to the back.

Useful Meds
Weak opioids and strong opioids.</td><td>Nerve Pain
May be caused by nerve damage or degeneration, infection inflammation, pressure.
The pain is random and often referred to the area the nerve would normally supply.

Useful Meds
Anti-depressants, anti-convulsants, anti-arrythmics, NMDA antagonists, capsaicin.</td><td>Sympathetic Pain
May be due to overstimulation of the sympathetic nervous system, often after an injury.
Often presents as allodynia with associated other symptoms.

Useful Meds
Anti-depressants, anti-convulsants, anti-arrythmics,drugs acting on the sympathetic nervous system.</td></tr>
</table>

Regular Analgesia

Paracetamol +/- a NSAID (and in cases of severe pain a strong opioid or codeine or tramadol) should be prescribed on the regular side of the medication chart while the person is in pain. PRN medications should be used when the pain is not controlled by these agents.

In acute pain the analgesia prescription should be reviewed daily.

Patient Categories

The patients' individual response to opioids differs or alters depending on their previous exposure to opioids, their condition and their stage of disease. Different sets of guidelines for different groups of patients apply therefore as follows:

a) **The Opioid Naive Patient -** Patients with **no recent history of opioid** use.
   Opioid naive patients in acute pain are far more susceptible to respiratory depression than patients who have been receiving regularly scheduled opioid therapy for 5 days or more. In addition, opioid naive patients taking benzodiazepines or other central nervous system (CNS) depressants are more susceptible to opioid-induced respiratory depression. Tolerance to opioid induced respiratory and other CNS effects generally occurs within 5 days of regularly scheduled therapy. They should therefore receive opioids as per the protocol or via PCA pump according to the following steps:
   1. Intravenous opioids according to intravenous opioid protocol.
   2. The Anaesthetist should be contacted by the primary team if the patient requires repeated administration of opioids, or if this administration is ineffective.
   3. PCA (with an appropriate programme and no background infusion) or alternative treatments will be started by the Anaesthetist.

b) **The opioid experienced patient**

Patients who have been for some time on a regular dose of opioids, for example oncology patients who are admitted on MST or methadone, are regarded as opioid experienced and of significantly reduced risk of respiratory depression. As they may require increased amounts of opioids to achieve pain control, the following steps should be considered:

- Early involvement of the Anaesthetist by the primary team with a view to commencing PCA.
- The use of a PCA will be preferably with a background infusion (only charted by anaesthetist) to substitute the previous opioid intake.
- Baseline analgesia to maximum levels.
- Consider other modalities, epidural, spinal opioids, nerve blocks, And local infiltration.

(For more details especially on methods of opioid administration, please see SECTION 4)

PAIN MANAGEMENT FLOW

All patients requiring pain management (including children) to have pain medications and anti-emetics **charted in the correct place on the medication chart.**

- All to be prescribed and offered baseline pain relief **(ie, <u>regular</u> (not prn) Paracetamol 6 hourly and/or NSAIDS for 48 hours)** unless contraindicated. *(Renal impairment, Platelet dysfunction or bleeding, ongoing risk for GIT bleeds, liver disease, asthma, etc)*.
- Explanation to be given to the patient regarding reason for this to be given (to prevent pain peaks and troughs).
- Add Codeine or other oral weak opioids if pain expected to be moderate.
- If severe pain is expected add the appropriate morphine protocol.

If pain **score >4/10** at any time consider further pain relief taking into account:

- Patient factors
- Medications given

<u>Morphine IV Rescue Protocol</u>

Intravenous (IV) titrated to pain score and observations as per **rescue IV morphine protocol.** (Page 46)
Paediatric morphine protocol (Appendix 4)
Pain relief regime to be reviewed at lease daily by the primary teams/Anaesthetist (post op patients).

- If pain score remains >4/10 after 30 minutes despite morphine IV titration.

  Contact Anaesthetist during normal working hours or after hours, contact on call H/S or on call Anaesthetist

- Increasing pain and pain relief demand that is not settling should be a red flag and the patient carefully evaluated for Nerve, ischaemic, or inflammatory pain.

ACUTE PAIN:

The aim of acute pain management is to provide subjective comfort inhibiting trauma-induced nocioceptive impulses, blunt autonomic and somatic reflex and enhance restoration of function allowing the patient to breathe, cough and move more easily, particularly postoperatively.

Acute pain is:

- Acute exacerbation of pain added to the basal pain physiology. It is usually self-limiting and there is progressive improvement over a relatively short period. It is a complex sensation which is difficult to define and difficult to measure.
- *The Affective* component leads to anxiety due to diagnosis of the underlying condition and fear and delay of analgesic treatment.
- A sensory appreciation of afferent nociceptor stimulation that elicits an affective (or autonomic) component. Both are subject to rational interpretation by the patient.
- Pain varies among individual patients.

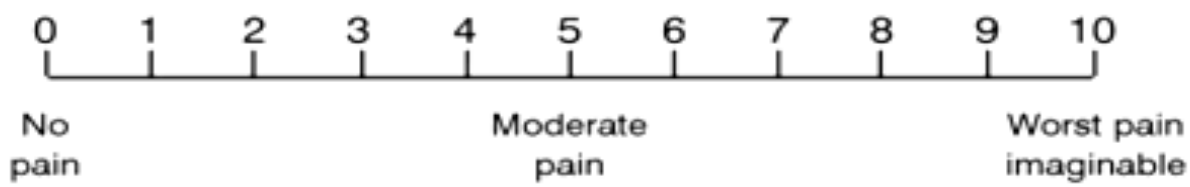
PAIN ASSESSMENT:

Visual Analogue Scale

No Pain I——————————————————————————I **Worst Possible Pain**

The individual is asked to place a mark on a 100mm line to indicate the level of pain. The score is the given by the measuring the number of millimetres between the no pain end and the patients mark on the line.

Numerical Rating Scales – Please rate the pain you have right now



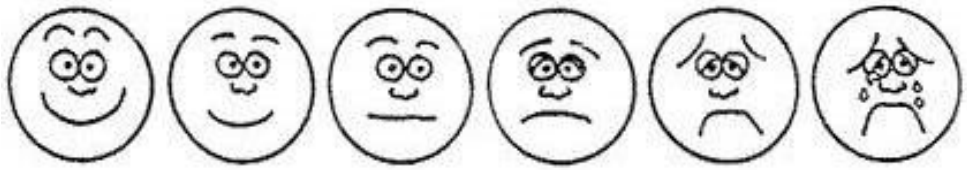
This is one of the most commonly used scales in clinical practice. This is a useful tool to measure changes in pain, and response to pain interventions.

Wong Baker Faces Pain Scale

Using a pain rating scale, like the one below, is helpful for young patients to communicate how much pain they are feeling.

Instructions:

Explain to the child that each face is for a person who feels happy because he has no pain (hurt) or sad because he has some or a lot of pain.



0	2	4	6	8	10
NO HURT	HURTS LITTLE BIT	HURTS LITTLE MORE	HURTS EVEN MORE	HURTS WHOLE LOT	HURTS WORST

Face 0 is very happy because he doesn't hurt at all.
Face 2 hurts just a little bit.
Face 4 hurts a little more (quite sore)
Face 6 hurts even more (very sore).
Face 8 hurts a whole lot more (really, really sore).
Face 10 hurts as much as you can imagine, although you do not have to be crying to feel this bad.

Ask the child to choose the face that best describes how he/she is feeling.

FLACC SCALE: (INFANTS)

Category	Scoring		
	1	**2**	**3**
Face	No particular expression or smile	Occasional grimace or frown, withdrawn, disinterested	Frequent to constant quivering chin, clenched jaw
Legs	Normal position or relaxed	Uneasy, restless, tense	Kicking, or legs drawn up
Activity	Lying quietly, normal position, moves easily	Squirming, shifting back and forth, tense	Arched, rigid or jerking
Cry	No cry (awake or asleep)	Moans or whimpers; occasional complaint	Crying steadily, screams or sobs, frequent complaints
Consolability	Content, relaxed	Reassured by occasional touching, hugging or being talked to, distractible	Difficult to console or comfort

Each of the five categories:
(F) Face; (L) Legs; (A) Activity; (C) Cry; (C) Consolability - is scored from 0-2, which results in a total score between zero and ten.

Others:
- Categorical scale: nil…mild…moderate…severe….excruciating
- Functional assessment: movement, cough
- Patient satisfaction

Special situations, e.g. developmentally delayed or unconscious patients

MONITORING
Regardless of the method of administration, monitoring should include the following baseline recordings:
- Sedation
  0 = Alert
  1 = Easily roused, occasionally sleepy
  2 = Easily roused, frequently sleepy
  3 = Difficult to rouse, deeply sedated
  S = Normal sleep, easy to rouse

Paediatric sedation score as per Children's Acute Pain Management Handbook **(Appendix 5)**
- Respiratory rate
- B/P
- Pulse
- Oxygen saturations
- Pain assessment – both at rest and following, or with activity.

The scale most commonly used is the 0 - 10 numerical rating scale, where 0 is **no pain** and 10 is the **worst pain imaginable**.

If the number scale is too difficult for the patient to understand, an alternative pain rating scale is the categorical rating scale which uses words to rate pain, such as nil, mild, moderate, severe, excruciating.

Monitoring to continue every 5 minutes for 15 minutes - then hourly.

SECTION 4

OVERVIEW OF COMMONLY USED MEDICATIONS

(FOR PAEDIATRIC DOSES - PLEASE SEE PAEDIATRIC BNF)

Measurement of efficiency of medications --→ Numbers to treat (NNT)

$$\text{NNT} = \frac{1}{\text{(Proportion of those with 50\% relief with the analgesic)} - \text{(Proportion of those with 50\% relief with the placebo)}}$$

Example

Ibuprofen – 27/50 patients had 50% relief for 6 hours
Placebo – 10/50 patients had 50% relief for 6 hours

NNT = 1 / (27/50) – (10/50) = 1 / 0.34 = 2.9

- ***NNT = 1 – 5 = effective treatment***
- ***NNT = 20 – 100 may be useful prophylaxis***

OXFORD LEAGUE TABLE FOR COMMON ANALGESICS

Drug Name	NNT	Drug Name	NNT
Ibuprofen 400mg	2.4	Oxycodone 10mg + Paracetamol 1000mg	2.7
Ibuprofen 800mg	1.6	Tramadol 100mg + Paracetamol 1000mg	2.7
Tramadol 100mg	4.8	Celecoxib 200mg	2.8
Tramadol 50mg	8.3	Morphine 10mg IM	2.9
Ketorolac 60mg IM	1.8	Naproxen 500mg	3.0
Diclofenac 100mg	1.9	Paracetamol 1000mg	3.8
Piroxicam 20mg	2.7	Codeine 60mg	16.7
Celecoxib 400mg	1.9	Aspirin 600mg	4.4
Paracetamol 1G + Codeine 60mg	2.2		

PARACETOMOL:

- First used 1893
- As effective as most NSAIDS for non- inflammatory pain
- Absorbed unchanged. Oral bio-availability 70-90%. Rectal bio-availability 60-80%
- Peak plasma 30-60 min, Plasma 1/2 life 2-3 hrs Hepatic metabolism with no active metabolites

Paracetamol	Simple analgesic and antipyretic, no anti-inflammatory action
Uses/Indications:	Analgesic, antipyretic. Mild to moderate pain and fever
Precautions:	Hepatic, renal impairment; prolonged use; children < 6 years
Drug Interactions:	Hepatotoxic drugs; anticoagulants; alcohol; hepatic enzyme inducers; drugs affecting gastric emptying, e.g. metoclopramide; cholestyramine
Pregnancy Category:	*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.
Side effects:	Skin rashes, headaches
Dose:	May be taken with or without food. Adults, children over 12 yrs - 500mg-1gm every 4-6 hours. Max. of 4gm in 24 hrs. Children - 15mg/kg every 4-6 hours. Max 60mg/kg in 24hrs without exceeding 2g or 3g/24hours depending on age (check NZFc for details)

All patients with pain that needs management should be offered regular paracetamol. The usual dose is 1g every SIX hours. **IV paracetamol should be reserved for use in Theatre and in patients who are nil by mouth and need regular pain relief.** *NB: paracetamol suppositories may also be considered in these patients.*

Paracetamol Costs

- 1g oral dose = 2c
- 1g IV dose = $1.10

If IV paracetamol is a restricted medication on the Hospital Medicines List (HML) and is only allowed for use in patients who are unable to take it by mouth or in whom oral absorption will be compromised. It must be reviewed every 24 hours.

NON-STEROIDAL ANTI INFLAMMATORY SALICYLATES: (NSAIDS)

Actions

- Peripheral action, Reduce nociception related to inflammation and mediators
- Modify the inflammatory reaction
- Antipyretic
- Reduce opioid use – up to 30%

Several specific receptors cox1, cox2 inhibitors

Drug Name	Tradename	COX2 :COX1 ratio	
Paracoxib	Dynastat	28000 :1	COX2
Celecoxib	Celebrex	9 : 1	
Diclofenac	Voltaren	4 : 1	
Ibuprofen	Nurofen	0.38 : 1	Mixed
Naproxen	Naprosyn	0.33 : 1	COX-1
Indomethacin	Indocid	0.23 : 1	

UNWANTED ACTIONS COX-1/MIXED:

Gastrointestinal Tract (GIT)

- Cause uninhibited acid secretion with reduced mucus and bicarbonate secretion, due to reduced mucosal blood flow.
- Combine with biochemical bridging of barrier mucosa to allow hydrogen ions to enter cells. Leads to dyspepsia, gastric erosion causing bleeding and perforations of upper gastrointestinal tract.
- Can also cause general GIT upset and present with nausea, vomiting, diarrhoea, and constipation.

Renal

- Renal blood flow reduction due to prostaglandin inhibition in renal cortex causing reduced glommerulo filtration pressure with reduced kidney function. Marked in renal disease, already poor renal perfusion states such as shock (septic or loss of volume) and dehydration.
- NSAID therapy in acute renal failure can cause salt & water retention causing congestive heart failure. It also suppresses renin and aldosterone secretion and can lead to high potassium
- Can also cause an allergic interstitial nephritis -nephrotic syndrome
- Clotting
- Non reversible block of cylo-oxygenase enzyme that affects platelet adhesion causing an increase in bleeding time and reduced clot formation.
- There is also prostacyclin and trombeboxane inhibition
- Bronchospasm,
- Anaphylaxis common
- Cyclo-oxygenase inhibition causes increased leucotrienes in the lungs which increases the inflammatory reaction causing bronchospasm

CNS

- N VII damage causing tinnitus, vertigo, metabolic alkalosis,
- Caution with Aspirin → Reyes encephalopathy syndrome seen in children less than12yrs of age with a concurrent viral infection

Reproductive

- Uterine contractions, prolonged labor
- Premature closure of ductus arteriosus,
- Premature newborn

PHARMACOKINETICS:

- Small intestine absorption
- Pka - 5, most insoluble in water, 99% Ionized at ph7
- Protein bound
- Oxidised, Hydrolyzed, excreted in urine

Ibuprofen	Nonsteroidal Anti-Inflammatory Agents
Uses/Indications:	Treatment of rheumatoid arthritis (incl juvenile RA), ankylosing spondylitis, osteoarthritis, other nonrheumatoid arthropathies. Soft tissue injuries, frozen shoulder, bursitis, tendonitis, tenosynovitis, low back pain. Mild to moderate pain such as dysmenorrhoea, dental and post-operative pain. Shouldn't be used as sole agent after major operations. Brufen syrup: for the relief of pain and fever in children.
Contraindications:	NSAID/aspirin sensitive asthma, rhinitis, urticaria; active GI bleeding, peptic ulcer; pregnancy, lactation (relative →febrile neutropaenia)
Precautions:	Prolonged use; history of GI bleeding, peptic ulcer; dehydration; haemostatic defects; cardiac, renal, hepatic impairment; history of heart failure, hypertension; asthma; monitor haematological, ophthalmological

	function; mask infection; withdrawal of concomitant steroids (reduce gradually); SLE; elderly;
Adverse Reactions:	GI disturbances including bleeding, ulceration; tinnitus; oedema, fluid retention; dizziness; headache; nervousness; rash; decreased appetite; visual disturbances; bronchospasm; hepatic reactions; blood dyscrasias; nephrotoxicity; Neutropaenia
Drug Interactions:	Warfarin; lithium; ACE inhibitors; beta-blockers; thiazide diuretics; frusemide; cardiac glycosides; methotrexate; corticosteroids; other NSAIDs, aspirin; cyclosporin; tacrolimus
Pregnancy Category:	**Category C** Drugs that, owing to their pharmacological effects, have caused or maybe suspected of causing harmful effects on the human fetus or neonate without causing malformations. These effects may be reversible.
Dose:	Should be taken with food. Do not halve tablets. Administer in divided doses. Adults: initially 1,200-1,800 mg/day; maintenance: 600-1,200 mg/day. Severe, acute conditions: max. 2,400 mg/day. Children: 20 mg/kg/day; max. 40 mg/kg/day (juvenile RA), 500 mg/24 hours (children < 30 kg).

Diclofenac Sodium	Nonsteroidal Anti-Inflammatory Agent
Uses/Indications:	Treatment of inflammatory and degenerative forms of rheumatism; acute attacks of gout; post-traumatic and postop-pain, inflammation and swelling; painful and/or inflammatory conditions in gynaecology; adjunct in severe painful inflammatory infections of the ear, nose and throat; migraine attack (suppositories only).
Contraindications:	Gastric, intestinal ulcer; aspirin/NSAID sensitive asthma, urticaria, rhinitis; proctitis (suppositories only).
Precautions:	History of GI disorders, ulceration; ulcerative colitis; Crohn's disease; hepatic porphyria; coagulation disorders; impaired cardiac, hepatic, renal function; extracellular volume depletion; mask infection; prolonged use (monitor LFTs, blood counts); elderly (esp low bodyweight); pregnancy, children.
Adverse Reactions:	Common: GI disturbances; raised LFTs; rash; headache; dizziness; vertigo; local irritation (suppositories only). Rare: GI bleeding, ulceration, perforation; hepatitis with/without jaundice; hypersensitivity incl anaphylaxis; others, see full PI.
Drug Interactions:	Lithium; digoxin; diuretics; other NSAIDs; anticoagulants; oral hypoglycaemics; methotrexate; cyclosporin; quinolones.
Pregnancy Category:	**Category C -** as for Ibuprofen
Dose:	Swallow whole with liquid before meals. Admin in 2-3 divided doses. Adults: initially 100-150 mg daily. Mild cases, long-term use: 75-100 mg daily Children >1 year: 0.5-2 mg/kg/day. Juvenile RA: max. 3 mg/kg/day.

COX 2 INHIBITORS:

COX-2 selective inhibitor is a form of Non-steroidal anti-inflammatory drug (NSAID) that directly targets COX-2, an enzyme responsible for inflammation and pain. Selectivity for COX-2 reduces the risk of peptic ulceration, and is the main feature of celecoxib, rofecoxib and other members of this drug class. COX-2 selectivity does not seem to affect other adverse effects of NSAIDs (most notably an increased risk of renal failure), and some results have aroused the suspicion that there might be an increase in the risk for heart attack, thrombosis and stroke by a relative increase in thromboxane.

PARACOXIB (DYNASTAT):

Parecoxib is a water soluble and injectable prodrug of valdecoxib. It is marketed as **Dynastat**. Parecoxib is a COX2 selective inhibitor in the same category as celecoxib (Celebrex) and rofecoxib

(Vioxx). As it is injectable, it can be used perioperatively when patients are unable to take oral medications. It is approved for short term perioperative pain control. Parecoxib has no effect on platelet function and therefore does not promote bleeding during or after surgery. In addition, has lower gastrointestinal toxicity profile compared to most other nonsteroidal antiinflammatory drugs (NSAIDs) including ibuprofen and naprosyn.
One study noted increased occurrences of heart attacks following cardiac bypass surgery compared to placebo when high doses of parecoxib were used to control pain after surgery. It is also important to remember that rare but severe allergic reactions (Stevens-Johnson Syndrome, Lyell Syndrome) have been described with valdecoxib, the molecule in which parecoxib is converted.
It is licensed for a single perioperative dose and should not be used in conjunction with any other NSAIDs.

FINALLY:
These are very useful agents especially if the pain in musculoskeletal in nature or has an inflammatory component. They commonly cause gastric upset and on rare occasions gastric ulcer and GI bleed. Care should be taken in patients who have other risk factors for GI pathology. Prophylaxis with a PPI (omeprazole) may be considered in patients who have risk factors but who would still benefit from treatment with a NSAID.

All NSAIDs are considered to be equi-analgesic in equivalent doses and the choice of agent is a matter of prescriber preference and presentation.

IV NSAIDs (Paracoxib and tenoxicam) should be reserved for use in Theatre and in Patients who are unable to take medication orally and who will benefit from a dose of a NSAID *NB: diclofenac suppositories may also be considered in these patients.*

OPOID ANALGESICS:

> *"Among the remedies which it has pleased Almighty God to give man to relieve his sufferings; none is so universal and so efficacious as opium"* ***Thomas Sydenham (1624-1689)***

The Analgesic Mechanism of Opioid Drugs
Opiates/opioids control pain by inhibiting the release of neurotransmitter substances, such as glutamate and Substance P. To do this they 'bind' with, or attach themselves to, specific opiate receptors within the central nervous system, blocking the transmission of the pain impulse.

Morphine was the first opioid. It is derived from opium and obtained from the plant *Papaver Somniferum*.

Morphine is the most commonly used strong opioid and should be considered for any patient who has severe pain. It is far better to give an appropriate dose of a strong opioid that to stick with weak opioids and fail to control the pain.

The normal IM/SC adult dose for acute pain is 7.5 – 10mg IM/SC when pain score is >4/10. Recheck pain score 45minutes after dose and give an additional dose of ½ the original dose if pain score still >4/10.

Morphine is a controlled drug and discharge prescriptions will need to be written on a CD Prescription Form. They can only be written for 1/12 and both the dose and quantity need to be in words and figures and written and signed by the prescriber.

Despite billions of dollars spent on attempts to develop 'better' opioids than morphine, these have largely failed. Morphine remains the "drug of choice" for the treatment of severe acute pain (eg, pain associated with trauma or following surgery) and pain associated with cancer.

It is a potent analgesic and sedative, with a rapid onset of action when given intravenously (about five minutes, with peak effect 10 - 15mins). Half-life in the plasma is about two to two and a half hours.

Opioids are said to have **agonist** properties because of the mechanism by which they act to relieve pain. Alternatively, any drug that reverses the effect of an opioid is called an **antagonist**.

Toxicology
Morphine and related opioids produce a wide spectrum of unwanted effects which are usually dose-related.

Central nervous System (CNS) Effects
- Respiratory depression – the primary mechanism involves a reduction in the responsiveness of the brainstem respiratory centres to carbon dioxide. This reduces the hypoxic drive.
- Drowsiness
- Mood changes – euphoria
- Apathy
- Loss of concentration
- Confusion
- Hallucination
- Muscle rigidity - chest wall rigidity can be severe with use of Fentanyl
- Pupil constriction (miosis) - due to excitatory action on the autonomic segment of the oculomotor nerve;
- Depression of cough reflex - in part a direct effect on the cough reflex centre in the medulla;
- Nausea/vomiting - direct stimulation of the chemotrigger receptor zone (CTZ) for emesis in the medulla (see **Section 6** on Postoperative Nausea and Vomiting).

Cardiovascular Effects
- Peripheral vasodilation and decreased peripheral resistance leading to hypotension. Caused by inhibition of baroreceptor reflexes and release of histamine
- Bradycardia.

Always rule out any other causes if your patient is hypotensive, and make sure that their hypotension is not being exacerbated by hypovolaemia.

Gastrointestinal Effects
- Small intestine - morphine decreases biliary, pancreatic and intestinal secretions causing
  - delayed digestion and increased water absorption
  - increased tone and reduction in propulsive contractions
- Large intestine - propulsive peristaltic waves are diminished causing:
  - constipation
  - increased bowel tone to the point of spasm
- Biliary Tract - constriction of Sphincter of Oddi causing:
  - increased pressure in common bile duct → spasms

Urinary Tract
- Ureteric spasm - amplitude and tone of ureteric contractions increased
- Urinary retention - tone of external sphincter and volume of bladder increased, as well as effects of morphine inhibiting urinary voiding reflex

Skin
- Flushing - dilatation of cutaneous blood vessels, particularly with injection of opioids;
- Sweating;
- Pruritis/urticaria) - histamine release with use of morphine and Pethidine.

Immune System

- Long-term abuse →depression of immune system.

Dependence and Tolerance

- Tachyphylaxis.
- Dependence, cravings, socially destructive, last for months or years.

Commonly used strong opioids are Morphine, Fentanyl, Pethidine, Oxycodone, and Methadone. These drugs are either naturally derived or synthetic opioids. Weak opioid drugs include codeine, tramadol, Dihydrocodeine (DHC) and nefopam.

Determinants of Opioid Requirements

- Magnitude of injury
- Age (eg. after laparotomy: morphine requirement is 100 – age mg/day)
- Other agents e.g. LA blocks, NSAIDS
- Tolerance to opioids
- Non-specific cross tolerance
- Organ dysfunction e.g. renal impairment prolongs morphine effect
- Psychological: these influences are common and legitimate
- Inter-individual variation
- Pregnancy – effects on fetus especially pre-term
- Lactation – effects on neonate/infant if breastfeeding

Race, gender and size have little influence on morphine requirement. Consider these factors when deciding first dose, e.g. of subcutaneous Morphine. Titrate to severity of pain (dependent upon pain score/s).

Signs of Opiate Overdose

- Drowsiness/unconsciousness
- Severe respiratory depression, usually presenting as decreased respirations (hypoxia is unlikely if on oxygen therapy)
- Small pinpoint pupils
- **BEWARE OF RESPIRATORY FAILURE** – even a small dose may of opioid may cause respiratory arrest
- Renal Failure – moderate or severe
  - Metabolites accumulate and morphine is long acting, with the risk of overdose
  - Pethidine metabolites can cause convulsions – observe closely for severe tremor and/or myclonus (brief, involuntary twitching of a muscle or a group of muscles)

Caution should be taken when a decision is made to give an IV dose of opioid within 60 minutes of an IM/SC dose, because of the time to peak and the unpredictability of the IM injection. Time of onset and time to peak is much quicker via IV route than via IM/SC.

eg,
- *Morphine SC/IM onset 15-20 mins. Peak 60-90 mins*
- *Morphine IV onset 3-5mins. Peak 10-15 mins*

Use of sedatives/other opioid analgesics with parenteral (IV) opioids is best avoided until discussed with anaesthetist.

MORPHINE SULPHATE:

Mechanism of Action

Central and peripheral inhibition of calcium entry into the cell causes prolonged depolarization of the neurons. Facilitation of potassium flux causes hyper-polarization→ increased threshold for depolarization. Inhibition of adenylcyclase alters ion channels and inhibits depolarization.

Opioid Receptors

μ **- Mu:** Analgesia, respiratory depression, Miosis, constipation, euphoria, somnolence, physical dependence, tolerance

δ **- Delta:** Analgesia respiratory depression, constipation, tolerance

κ **- Kappa:** Analgesia, constipation, dysphoria, hallucinations, somnolence, physical dependence, tolerance

Sites of Action

- Presynaptic terminal of nociceptor afferent→ Inhibition of Ca flux which prevents excitory neuro transmitter release.
- *Postsynaptic terminal* Potassium flux causes hyper polarization, less excitable. Ca influx reduces prolonged depolarization.

Activation in descending inhibition (PAG) reduces nociceptor input

- Bioavailability: Morphine, First pass effect, 30 % bio-availability
- Accumulation of active metabolites with repeated administration

Metabolism and Excretion

- Polar metabolites, kidney excretion
- Enterohepatic recirculation.
- Partly excreted in bile metabolized to parent opioid and re absorbed-→ secondary peak effect.

Morphine	Narcotic analgesics
Uses/Indications:	Analgesia effective in most pain. Less in neurogenic pain.
Contraindications:	Respiratory depression; premature infants; head injury; raised intracranial pressure; convulsive states; bronchial asthma; acute alcoholism; biliary tract post-op; MAOIs (+/- 10 days); heart failure secondary to chronic pulmonary disease.
Precautions:	Dependence, tolerance; adrenocortical insufficiency; hepatic, pulmonary, renal impairment; shock; hypothyroidism; prostatic hypertrophy; pregnancy, lactation; newborn, premature infants; acute asthma, COPD, reduced pulmonary reserve; debilitated; abrupt withdrawal; elderly.
Adverse Reactions:	Anorexia; nausea; confusion; constipation; sweating; vomiting; dependence; respiratory depression; hypotension; tolerance; circulatory failure; deepening of coma; allergic reactions; others, see full PI.
Drug Interactions:	CNS depressants; anaesthetics; hypnotics; sedatives; tranquillizers esp phenothiazines; skeletal muscle relaxants; MAOIs; dextroamphetamine.
Pregnancy Category:	Category C Drugs that, 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.

PETHIDINE:
This is a synthetic opioid that is commonly used for people who have adverse reactions to morphine or in whom the respiratory (e.g. bronchospasm) or muscle spasm side-effects (eg, biliary or renal colic) preclude its use.

Pethidine carries its own problem in the form of a metabolite that can cause muscle twitching, tremors and seizures. For this reason, it is contraindicated in patients with renal/liver impairment/failure and in people who have epilepsy.

Analgesic effects occur within 5 minutes of intravenous administration and peak concentration in plasma occurs at about 60 minutes. Half-life in the plasma is about 2-3 hours.

Atropine Effects
- Less effect on biliary sphincter than morphine
- Negative inotropic effect
- Passes across the placental barrier
- MAOI interaction causes muscle rigidity, hyperthermia, hypertension, seizure and death
- Active metabolite is norpethidine →CNS stimulant. Can cause mioclonus, mood disorders and seizures

Pethidine Hydrochloride	Narcotic Analgesics
Uses/Indications:	Opioid analgesic. Relief of moderate to severe pain unresponsive to non opioids; preoperative medication; analgesic adjunct in general anaesthesia; obstetrical analgesia.
Contraindications:	MAOIs (+/- 14 days); respiratory depression, poor respiratory reserve; convulsive states; pre-eclampsia, eclampsia; arrhythmias; cor pulmonale; diabetic acidosis; acute alcoholism, delirium tremens; severe hepatic disease, incipient hepatic encephalopathy; head injury, raised intracranial pressure.
Precautions:	Prolonged use; high doses; rapid IVI; abrupt withdrawal; pulmonary, hepatic, renal, cardiac impairment; MI; hypovolaemia; hypothyroidism; adrenocortical insufficiency; phaeochromocytoma; prostatic hypertrophy, urethral stricture; acute abdominal conditions; biliary colic, surgery; acute pancreatitis; glaucoma; diabetes; epilepsy; elderly, debilitated; pregnancy, labour, lactation, young children
Adverse Reactions:	Dependence; respiratory depression; CNS disturbances incl light headedness, dizziness, sedation, sweating, disorientation, bizarre feelings, hallucinations, psychosis, impaired alertness; nausea, vomiting, constipation; hypotension; others, see full PI.
Drug Interactions:	CNS depressants including alcohol; phenobarbitone, phenytoin; phenothiazines; MAOIs.
Pregnancy Category:	none

FENTANYL:
This is also a synthetic opioid, and is a potent analgesic and sedative estimated to be at least 80 times more potent than morphine as an analgesic, on a weight basis. Onset of action is almost immediate following intravenous administration and its half-life is dose-dependent. It is lipid soluble, can accumulate and has little CVS effect.

Fentanyl Citrate	Narcotic Analgesic
Uses/Indications:	Opioid analgesic. Short duration analgesia in anaesthesia and perioperatively. Supplement to general and regional anaesthesia. In combination with a neuroleptic for induction and maintenance of general and regional anaesthesia.
Contraindications:	Bronchial asthma; head injury, increased intracranial pressure; susceptibility to respiratory depression, e.g. comatose patients with possible head injury or brain tumour; MAOIs (+/- 14 days); myasthenia gravis; children ≤ 2 years.
Precautions:	Chronic opioid use; history of opioid abuse; severe pulmonary impairment (e.g. COPD, decreased respiratory reserve) or potentially compromised respiration; bradyarrhythmias; hypovolaemia; uncontrolled hypothyroidism; alcoholism; impaired hepatic, renal function; rapid IV injection; elderly, debilitated; pregnancy, labour, lactation.
Adverse Reactions:	Common: respiratory depression, apnoea; muscle rigidity; non epileptic myoclonic movements; bronchospasm, laryngospasm; bradycardia, other cholinergic effects. Less common: hyper/ hypotension; sphincter of Oddi spasm; dizziness; miosis; blurred vision; nausea; diaphoresis; itching; euphoria; seizures; anaphylaxis; dependence; arrhythmias; post-op depression; paradoxical CNS excitation; delirium.
Drug Interactions:	Azole antifungals; macrolides; protease inhibitors, e g. ritonavir; phenytoin; sibutramine; naltrexone; nitrous oxide; neuroleptics; CNS depressants, e.g. barbiturates, tranquillizers, benzodiazepines, opioids, general anaesthetics, alcohol; MAOIs; amiodarone; beta-blockers; Ca channel blockers.
Pregnancy Category:	**Category C -** Drugs that, 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.

ORAL OPIODS:

Codeine Phosphate

Codeine is a weak opioid drug. As such it has less analgesic efficacy than strong opioids like morphine but similar adverse effects especially constipation. Its main advantage is that it is not a controlled drug and therefore its use is simpler.

The analgesic effect of codeine is entirely due to its metabolism into morphine within the body and this does not happen in people who are **poor metabolisers**. This may be as many as 20% of the NZ population. If a patient has no response to a therapeutic trial of codeine ***3 doses*** or the patient cannot tolerate codeine phosphate - then the treatment should be changed to tramadol.

There are also some patients who are **ultra rapid metabolisers** of codeine (probably less than 1%) who may experience side effects from the morphine at otherwise normal dose of codeine. This is especially important for nursing mothers in whom the baby may experience morphine induced adverse effects. In nursing mothers who are given codeine both the mother and the infant should be monitored frequently for these effects.

Codeine phosphate	Narcotic analgesics low affinity for receptor
Uses/Indications:	Opioid. Relief of mild to moderate pain. Relief of symptoms of diarrhoea. As an antitussive in the control of non-productive cough. Pain associated with coughing.
Contraindications:	Respiratory depression; raised intracranial pressure; head injury; acute alcoholism; MAOIs (+/-14 days); diarrhoea associated with pseudomembranous colitis or caused by poisoning; during an attack of bronchial asthma; heart failure secondary to chronic lung disease
Precautions:	Hypothyroidism; adrenocortical insufficiency; prostatic hypertrophy; shock; renal, hepatic impairment; high doses, prolonged use; obstructive bowel disorders; myasthenia gravis; elderly, children; pregnancy
Adverse Reactions:	Constipation, nausea, vomiting; drowsiness; confusion; raised intracranial pressure, others, see full PI
Drug Interactions:	CNS depressants incl alcohol, anaesthetics, hypnotics, sedatives, tricyclics, phenothiazines; mexilitine; metoclopramide; MAOIs.
Pregnancy Category:	**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.
Dose:	Pain relief 15-60mg up to 6 times daily. Max.: 300mg/day; Non productive cough: 10-20mg 4-6 hourly. Max.: 120mg/day. Children: pain relief 0.5mg/kg every 4-6 hours as required; Non productive cough: 0.25mg/kg 4-6 hourly.

Dihydrocodeine Tartrate (DHC)	Narcotic analgesic
Uses/Indications:	Narcotic analgesic. Treatment of postoperative pain and pain associated with cancer. Treatment of opioid responsive, chronic, severe pain of nonmalignant origin after other conservative methods of analgesia have been tried (according to NZMA guidelines).
Contraindications:	Respiratory depression; acute asthma attack; MAOIs (+/- 14 days).
Precautions:	Head trauma; increased intracranial pressure; asthma; hypothyroidism; impaired respiratory, renal, hepatic function; severe corpulmonale; biliary tract disorder; pancreatitis; paralyticileus; prolonged use; history of opiate abuse; elderly; debilitated; pregnancy, lactation, children < 12 years.
Adverse Reactions:	Constipation; nausea; vomiting; biliary pain; headache; somnolence; confusion; dizziness; hallucinations; urinary retention; hypotension; respiratory depression; rash; urticaria; pruritus; physical dependence; tolerance; menstrual disturbances; decreased libido; infertility.
Drug Interactions:	CNS depressants incl alcohol; MAOIs.
Pregnancy Category:	**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.
Dose	Controlled release tablets. May be taken with or without food. Adults, children > 12 years: 60-120 mg twice daily; max. 240 mg/day. Swallow whole, do not crush or chew.

OXYCODONE:

Oxycodone should be considered a 2nd line agent and only used when morphine has been tried and failed or not tolerated for some reason.

This is a semi-synthetic opioid that has been more commonly used for the treatment of opioid-responsive cancer-related pain. However, it is an effective analgesic for postoperative pain management too, though not in the early postoperative period.

Unlike oral morphine which is subject to “first-pass” metabolism, oral oxycodone is well absorbed, and twice as potent as oral morphine. Oxycodone comes in an immediate release (oxycodone IR) preparation (capsule) and a slow release (oxycodone SR) tablet.

Clinical Use

In a 2008 review written by authors who "are members of advisory boards and speaker panels for Mundipharma," slow -release oxycodone was found to be superior to placebo in randomized controlled trials concerning diabetic neuropathy, post herpetic neuralgia, osteoarthritis, ambulatory laparoscopic tubal ligation surgery, unilateral total knee arthroplasty, and abdominal/ gynaecological surgery.

In 2001, the European Association for Palliative Care recommended that oral hydromorphone or oxycodone, "if available in both normal release and modified release formulations for oral administration," be second-line alternatives to oral morphine for cancer pain. There is no evidence that any other opioids are superior to morphine in relieving the pain of cancer and no controlled trials have shown oxycodone to be superior to morphine. However, switching to an alternative opioid can be useful if adverse effects are troublesome, although the switch can be in either direction, i.e. some patients have fewer adverse effects on switching from morphine to oxycodone and vice versa.

Pharmacology

Mechanism of Action

A group of Australian researchers has proposed (based on a 1997 study in rats) that oxycodone, unlike morphine (the effect of which is mediated by μ-opioid receptors), acts on κ-opioid receptors. Further research by this group indicates the drug appears to be a κ_{2b}-opioid agonist. However, this has been disputed, primarily on the basis that oxycodone produces effects typical of μ-opioid agonists.

Research by a Japanese group suggests that the effect of oxycodone is mediated by different receptors in different situations. Specifically, in diabetic mice the κ-opioid receptor appears to be involved in the antinociceptive effects of oxycodone, while in non-diabetic mice the μ_1-opioid receptor seems to be primarily responsible for these effects.

Absorption

After a dose of oral **oxycodone IR**, peak plasma levels of the drug are attained in approximately one hour; in contrast, after a dose of **oxycodone SR** , peak plasma levels of oxycodone occur in about three hours.

Distribution

Oxycodone (immediate release) in the blood is distributed to "skeletal muscle, liver, intestinal tract, lungs, spleen, and brain." Conventional oral preparations of oxycodone start to reduce pain within 10-15 minutes; in contrast, oxycodone slow release starts to reduce pain within 1 hour.

Metabolism

Oxycodone is metabolized to α and β oxycodol; oxymorphone, then α and β oxymorphol and noroxymorphone; and noroxycodone, then α and β noroxycodol and noroxymorphone. A study using conventional oral oxycodone concluded that oxycodone itself, and not its metabolites, is responsible for the drug's opioid effects on the brain.

Unlike morphine and hydromorphone, **oxycodone** is metabolized by the cytochrome P450 enzyme system in the liver, making it vulnerable to drug interactions. Some people are fast metabolizers resulting in reduced analgesic effect but increased adverse effects, while others are slow metabolisers resulting in increased toxicity without improved analgesia. The dose of **oxycodone SR** must be reduced in patients with reduced hepatic function.

Elimination

Oxycodone and its metabolites are mainly excreted in the urine; therefore, it accumulates in patients with renal impairment.

Dosage and Administration

Oxycodone can be administered orally, intranasally, via intravenous/intramuscular/subcutaneous injection or rectally. The bioavailability of oral administration averages 60–87%, with rectal administration yielding the same results.

Oxycodone is approximately 1.5–2 (i.e. a ratio of 1.5:1 to 2:1) times as potent as morphine when administered orally and equipotent (i.e. a ratio of 1:1) when administered parenterally.
There are no comparative trials showing that oxycodone is more effective than any other opioid. In palliative care, morphine remains the gold standard; however, oxycodone can be useful as an alternative opioid if a patient has troublesome adverse effects with morphine.

Side Effect

The most commonly reported effects include constipation, fatigue, dizziness, nausea, lightheadedness, headache, dry mouth, anxiety, pruritus, euphoria, and diaphoresis. It has also been claimed to cause dimness in vision due to miosis. Some patients have also experienced loss of appetite, nervousness, abdominal pain, diarrhea, dyspnea, and hiccups, although these symptoms appear in less than 5% of patients taking oxycodone. Rarely, the drug can cause impotence, enlarged prostate gland, and decreased testosterone secretion.

In high doses, overdoses, or in patients not tolerant to opiates, oxycodone can cause shallow breathing, bradycardia, cold, clammy skin, apnea, hypotension, pupil constriction, circulatory collapse, respiratory arrest, and death.

Withdrawal Related Side Effects

There is a high risk of experiencing severe withdrawal symptoms if a patient discontinues oxycodone abruptly. Therefore therapy should be gradually discontinued rather than abruptly discontinued. People who use oxycodone in a hazardous or harmful fashion are at even higher risk of severe withdrawal symptoms as they tend to use higher than prescribed doses. The symptoms of oxycodone withdrawal are the same as for other opiate based painkillers and may include "anxiety, nausea, insomnia, muscle pain, fevers, and other flu like symptoms."

Withdrawal symptoms have also been reported in a newborn whose mother had been injecting OxyContin during pregnancy.

METHADONE:

This drug works principally as an opioid but also has actions on the NMDA receptor (in similar fashion to ketamine). Although used primarily in the treatment of opioid addiction because of its long half-life, methadone is also prescribed for maintenance pain relief. It is rarely given intravenously, being more commonly given orally in tablet/elixir form. Because of its long half-life, the dosing of the drug needs careful monitoring and adjustment over a few days. With commencement of the drug or changing the dose, it is important to remember that it will take four to five days to reach steady state concentration. Usually prescribe a 12 hrly dose. It is well absorbed with a good bioavailability and reduced dependence.

TRAMADOL:

Analogue of codeine with good absorption and 70% bioavailability. It is metabolised in the liver, and excreted in the kidney. Active metabolite exit that have a greater affinity for receptors. Overdose as for opioids. Has little CVS and Respiratory effect in therapeutic doses. Side effects include dry Mouth, nausea, diaphoresis, vomiting and urinary retention. Serotonin syndrome possible

Tramadol is a weak opioid analgesic which also has some serotonergic activity. It should be used with caution in patients who are taking other serotonergic drugs eg, SSRIs. It is equianalgesic to codeine and an alternative in patients who are not suitable for codeine.

OTHER OPIODS – A SHORT NOTE:

The place of other opioids is beyond the scope of this formulary but the following points are worth noting:

- **Fentanyl** and **oxycodone** may have advantages in patients who are renal impaired.
- **Methadone** and **oxycodone** (along with tramadol) may have advantages in neuropathic pain.
- **Methadone** and **fentanyl patches** are often used in palliative care.

ALPHA 2 RCEPTORS:

Clonidine

Clonidine is a drug which is primarily used as an antihypertensive agent, however it now sees widespread use in a number of other fields e.g. as an additive to anaesthetics.

Clonidine is delivered as Clonidine hydrochloride and is an imidazoine derivative and a mesomeric compound. It's Chemical name is 2-(2,6-dichloro phenylamino)-2-imidazoline hydrochloride. It is a crystalline compound that is soluble in water, alcohol and lipids. When administered clonidine is transported by lipids by bonding to albumin and has a volume distribution of 2.1± 0.4L/kg. About 50% of the oral dose is metabolised by minor pathways in the liver. Clonidine is administered orally, by transdermal patch or by epidural injection. It works by stimulating the alpha-adrenergic receptors in the brain stem. This reduces the sympathetic outflows from the central nervous system leading to a decrease in peripheral resistance, renal vascular resistance and blood pressure (after 30 to 60mins). Common side effects include constipation, dizziness, drowsiness, dry eyes, dry mouth, nausea and hallucinations.

Clonidine Hydrochloride	Antihypertensive agents
Uses/Indications:	Treatment of hypertension either alone or in combination with other antihypertensives Injection: Treatment of acute hypertensive crisis.
Contraindications:	Severe bradyarrhythmia resulting from sick sinus syndrome or 2nd or 3rd degree A-V blocks
Precautions:	Taper withdrawal dosage; depression; renal impairment; mild to moderate bradyarrhythmia; cerebral, peripheral perfusion disorders; polyneuropathy; constipation; CHF; severe coronary disease; phaeochromocytoma; beta-blocker withdrawal; ophthalmological monitoring; pregnancy, lactation.

Adverse Reactions:	Sedation; dry mouth; sleep disturbances; headache; malaise; confusion; reduced lachrymal flow (caution contact lens wearers); elevated blood sugar (rare); decreased libido; gynaecomastia; Reynauds phenomenon; impotence; peripheral circulatory disturbances; GI upset; skin reactions; others, see full PI.
Drug Interactions	CNS depressants incl alcohol; other antihypertensives; TCAs; alpha-blockers; cardiac glycosides; NSAIDs; beta-blockers.
Pregnancy Category:	**Category B3** Drugs that 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[1] have shown evidence of an increased occurrence of fetal damage, the significance of which is considered uncertain in humans

NMDA RECEPTORS :

NMDA receptor antagonists induce a state called dissociative anesthesia, which is marked by catalepsy, amnesia, and analgesia. Ketamine and other NMDA receptor antagonists are most frequently used in conjunction with diazepam as anesthesia in cosmetic or reconstructive plastic surgery and in the treatment of burn victims Ketamine is a favored anesthetic for emergency patients with unknown medical history because it depresses breathing and circulation less than other anesthetics The NMDA receptor antagonist dextromethorphan is one of the most commonly used cough suppressants in the world

Ketamine Hydrochloride	NMDA receptor agonist
Uses/Indications:	General anaesthetic. Sole anaesthetic for diagnostic & surgical procedures not requiring skeletal muscle relaxation (best for short procedures but can be used with additional doses for longer procedures); supplement to low potency agents, e.g. nitrous oxide; induction of anaesthesia prior to administration of other general anaesthetics. Used in low doses as NMDA receptor blocker in opioid hyperalgesia, and acute pain management
Contraindications:	Conditions complicated by hypertension incl severe CV disease, heart failure, severe or poorly controlled hypertension, recent MI, history of stroke, cerebral trauma, intracerebral mass or haemorrhage
Precautions:	Monitor cardiac function; chronic, acute alcohol intoxication; elevated CSF pressure; significant renal, hepatic impairment; rapid IV admin; monotherapy in procedures involving the pharynx, larynx or bronchial tree; visceral pain; increased IOP; neurotic, psychiatric illnesses; porphyria; hyperthyroidism; pulmonary infection; intracranial mass lesions; head, globe injuries; hydrocephalus; hypovolaemia; dehydration; cardiac disease; seizures; mild/moderate hypertension; tachyarrhythmias; drug abuse; pregnancy, lactation; see full PI.
Adverse Reactions:	Elevated BP, pulse rate; hypotension; bradycardia; arrhythmia; respiratory depression; apnoea; laryngospasm; airway obstruction; diplopia; nystagmus; elevated IOP; psychological disturbances; tonic, clonic movements; anorexia; nausea; vomiting; hypersalivation; dependence, tolerance; injection site reactions; anaphylaxis.
Drug Interactions:	Halogenated hydrocarbon inhalational anaesthetics; barbiturates, narcotics; benzodiazepines; drugs with hypertensive effects, e.g. ergometrine; thyroxine; theophylline; antihypertensives; CNS depressants incl ethanol, anxiolytics,hypnotics, sedatives; antihistamines; thiopental; atracurium; tubocurarine.

Pregnancy Category:	**Category B3** - Drugs that 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[1] have shown evidence of an increased occurrence of fetal damage, the significance of which is considered uncertain in humans.
Dose	Low dose infusion for pain relief dose: 0.1mg/kg/hr or add to morphine solution PCA mg/ml.

OTHER DRUGS USED IN PAIN:

There are other drugs used in chronic pain and neuropathic pain and these include antidepressant drugs **amitriptyline** and **nortriptyline** and anticonvulsant drugs **carbamazepine**, **sodium valproate** and **gabapentin**.

Gabapentin is a special authority medicine and require approval for funding for neuropathic pain. A Special Authority Form (available from www.pharmac.govt.nz) needs to be completed before starting treatment with gabapentin for chronic pain. It is available in the Hospital medicines List (HML) for 8 days perioperatively for acute pain.

Hospital Medicines List (HML)
http://www.pharmac.health.nz/tools-resources/pharmaceutical-schedule/section-h/
(Please refer to the NZF or NZFc for doses)

NON-OPIOD ANALGESICS:

Paracetamol

Paracetamol Inj 10 mg per ml, 50 ml vial		1% DV Sep2014 to 2017
Perfalgan	2255707	$12.90 per 12
Paracetamol Inj 10 mg per ml, 100 ml vial		1% DV Sep 2014 to 2017
Perfalgan	2236621	$12.90 per 12

Intravenous paracetamol is only to be used where other routes are unavailable or impractical, or where there is reduced absorption. The need for IV paracetamol must be re-assessed every 24 hours.

Paracetamol Tab 500 mg
Any brand

Paracetamol Tab soluble 500 mg
Any brand

Paracetamol Oral liq 120 mg per 5 ml		20% DV Oct 2014 to 2017
Paracare	304905	$4.15 per 1000 ml
Paracetamol Oral liq 250 mg per 5 ml		20% DV Sep 2014 to 2017
Paracare Double Strength	306088	$4.35 per 1000 ml
Paracetamol Suppos 125 mg		
Panadol	734756	$7.49 per 20
Paracetamol Suppos 250 mg		
Panadol	734764	$14.40 per 20

Paracetamol Suppos 25 mg
Biomed 461172 $56.35 per 20
Paracetamol Suppos 50 mg
Biomed 398764 $56.35 per 20

Paracetamol Suppos 500 mg 1% DV Jan 2013 to 2015
Paracare 2172127 $20.70 per 50

Paracetamol with Codeine
Tab paracetamol 500 mg with codeine phosphate 8 mg
Paracetamol + Codeine (Relieve) 2383454 $2.11 per 100

Acute Migraine
Metoclopramide hydrochloride with paracetamol tab 5 mg with paracetamol 500 mg
Any brand
Aspirin
Aspirin Tab dispersible 300 mg
Any brand

Aspirin Tab EC 300 mg
Any brand

NON-STERIODAL ANTI-INFLAMMATORY DRUGS (NSAIDs):
Diclofenac sodium
Diclofenac sodium Tab 50 mg dispersible
Voltaren D 269905 $1.50 per 20

Diclofenac sodium Tab EC 25 mg 1% DV Mar 2013 to 2015
Apo-Diclo 793116 $4.00 per 100

Diclofenac sodium Tab EC 50 mg 1% DV Mar 2013 to 2015
Apo-Diclo 2025744 $16.00 per 500

Diclofenac sodium Tab long-acting 75 mg 1% DV Dec 2012 to 2015
Diclax SR 768278 $3.10 per 30
Diclax SR 2202174 $24.52 per 500

Diclofenac sodium Tab long-acting 100 mg 1% DV Dec 2012 to 2015
Diclax SR 2212846 $42.25 per 500

Diclofenac sodium Suppos 12.5 mg 1% DV Oct 2014 to 2017
Voltaren 645931 $2.04 per 10

Diclofenac sodium Suppos 25 mg 1% DV Oct 2014 to 2017
Voltaren 645958 $2.44 per 10

Diclofenac sodium Suppos 50 mg 1% DV Oct 2014 to 2017
Voltaren 747742 $4.22 per 10

Diclofenac sodium Suppos 100 mg 1% DV Oct 2014 to 2017
Voltaren 747750 $7.00 per 10

Diclofenac sodium Inj 25 mg per ml, 3 ml ampoule 1% DV Oct 2014 to 2017
Voltaren 263486 $13.20 per 5

Ibuprofen
IbuprofenTab 200 mg
Any brand

Ibuprofen Oral liq 20 mg per ml		1% DV Mar 2014 to 2016
Fenpaed	2090864	$1.89 per 200 ml
Ibuprofen Tab long-acting 800 mg		
Brufen SR	2255499	$8.12 per 30

Tenoxicam

Tenoxicam Tab 20 mg		1% DV Jan 2015 to 2016
Reutenox	2463121	$3.05 per 20
Tenoxicam Inj 20 mg vial		
AFT	2307022	$9.95 per 1

Parecoxib

Parecoxib Inj 40 mg vial		
Dynastat	2394804	$100.00 per 10

OPIOD ANALGESICS - Weak Opioids:
Codeine Phosphate

Codeine phosphate Tab 30 mg		1% DV Jul 2013 to 2016
PSM	206733	$5.80 per 100
Dihydrocodeine tartrate Tab long-acting 60 mg		1% DV Sep 2013 to 2016
DHC Continus	772887	$13.64 per 60

Codeine Phosphate is a Recorded Drug (RD) and must be written in the Controlled Drug Register and stored in the safe on the ward

Tramadol hydrochloride

Tramadol hydrochloride Cap 50 mg		1% DV Oct 2014 to 2017
Arrow-Tramadol	2351889	$2.50 per 100
Tramadol hydrochloride Tab sustained-release 100 mg		1% DV Oct 2014 to 2017
Tramal SR 100	2149508	$2.00 per 20
Tramadol hydrochloride Tab sustained-release 150 mg		1% DV Oct 2014 to 2017
Tramal SR 150	2149532	$3.00 per 20
Tramadol hydrochloride Tab sustained-release 200 mg		1% DV Oct 2014 to 2017
Tramal SR 200	2149524	$4.00 per 20
Tramadol hydrochloride Inj 50 mg per ml, 1 ml ampoule		1% DV Oct 2014 to 2017
Tramal 50	471208	$4.50 per 5
Tramadol hydrochloride Inj 50 mg per ml, 2 ml ampoule		1% DV Oct 2014 to 2017
Tramal 100	471216	$4.50 per 5

Strong Opioids

All strong opioids are Controlled Drugs (CD) and must be written in the Controlled Drug Register and stored in the safe on the ward.

Fentanyl

Fentanyl Inj 10 mcg per ml, 100 ml bag
Biomed 2392674 $210.00 per 10

Fentanyl Inj 50 mcg per ml, 10 ml ampoule 1% DV Sep 2012 to 2015
Boucher and Muir 2381168 $11.77 per 10

Fentanyl Inj 50 mcg per ml, 2 ml ampoule 1% DV Sep 2012 to 2015
Boucher and Muir 2381176 $4.50 per 10

Fentanyl Patch 75 mcg per hour 1% DV Aug 2015 to 2016
Mylan Fentanyl Patch 2351951 $13.60 per 5
Fentanyl Sandoz 2472902 $9.18 per 5
(Mylan Fentanyl Patch Patch 75 mcg per hour to be delisted 01 August 2015)

Fentanyl Patch 12.5 mcg per hour 1% DV Aug 2015 to 2016
Mylan Fentanyl Patch 2351927 $8.90 per 5
Fentanyl Sandoz 2472872 $2.92 per 5
(Mylan Fentanyl Patch Patch 12.5 mcg per hour to be delisted 01 August 2015)

Fentanyl Patch 25 mcg per hour 1% DV Aug 2015 to 2016
Mylan Fentanyl Patch 2351935 $9.15 per 5
Fentanyl Sandoz 2472880 $3.66 per 5
(Mylan Fentanyl Patch Patch 25 mcg per hour to be delisted 01 August 2015)

Fentanyl Patch 50 mcg per hour 1% DV Aug 2015 to 2016
Mylan Fentanyl Patch 2351943 $11.50 per 5
Fentanyl Sandoz 2472899 $6.64 per 5
(Mylan Fentanyl Patch Patch 50 mcg per hour to be delisted 01 August 2015)

Fentanyl Patch 100 mcg per hour 1% DV Aug 2015 to 2016
Mylan Fentanyl Patch 2351978 $14.50 per 5
Fentanyl Sandoz 2472910 $11.29 per 5
(Mylan Fentanyl Patch Patch 100 mcg per hour to be delisted 01 August 2015)

Methadone Hydrochloride

Methadone Inj 10 mg per ml, 1 ml vial
AFT 2265710 $61.00 per 10

Methadone Oral liq 10 mg per ml 1% DV Sep 2012 to 2015
Biodone Extra Forte 412325 $6.55 per 200 ml

Methadone Tab 5 mg
Methatabs 765503 $1.85 per 10

Morphine Sulphate

Morphine Inj 1 mg per ml, 10 ml syringe 1% DV Oct 2014 to 2017
Biomed 2308967 $45.00 per 10

Morphine Inj 200 mcg in 0.4 ml syringe
Any brand

Morpine Inj 10 mg per ml, 1 ml ampoule		1% DV Oct 2014 to 2017
DBL Morphine Sulphate	395013	$9.09 per 5
Morphine Inj 30 mg per ml, 1 ml ampoule		1% DV Oct 2014 to 2017
DBL Morphine Sulphate	394998	$12.43 per 5
Morphine Inj 1 mg per ml, 100 ml bag		1% DV Oct2014 to 2017
Biomed	2392682	$185.00 per 10
Morphine tartrate Inj 80 mg per ml, 1.5 ml ampoule		1% DV Sep 2013 to 2016
Hospira	615137	$35.60 per 5
Morphine Tab immediate-release 10 mg		1% DV Apr 2015 to 2017
Sevredol	242675	$2.80 per 10
Morphine Tab immediate-release 20 mg		1% DV Apr 2015 to 2017
Sevredol	242756	$5.52 per 10
Morphine Tab long-acting 10 mg		1% DV Sep 2013 to 2016
Arrow-Morphine LA	2383934	$1.95 per 10
Morphine Tab long-acting 30 mg		1% DV Sep 2013 to 2016
Arrow-Morphine LA	2383942	$2.98 per 10
Morphine Tab long-acting 60 mg		1% DV Sep 2013 to 2016
Arrow-Morphine LA	2383918	$5.75 per 10
Morphine Tab long-acting 100 mg		1% DV Sep 2013 to 2016
Arrow-Morphine LA	2383926	$6.45 per 10
Morphine hydrochloride Oral liq 2 mg per ml		1% DV Oct 2012 to 2015
RA-Morph	754293	$11.62 per 200 ml
Morphine hydrochloride Oral liq 10 mg per ml		1% DV Oct 2012 to 2015
RA-Morph	754315	$21.55 per 200 ml

Oxycodone hydrochloride

Oxycodone should be considered a 2nd line agent and only used when morphine has been tried and failed or not tolerated for some reason.

Currently we only hold oral forms of oxycodone at Gisborne Hospital

Oxycodone hydrochloride Cap immediate-release 5 mg		
OxyNorm	2179741	$2.83 per 20
Oxycodone hydrochloride Cap immediate-release 10 mg		
OxyNorm	2179768	$5.58 per 20
Oxycodone hydrochloride Tab controlled-release 10 mg		1% DV Oct 2013 to 2015
Oxycodone CR Tablets (BNM)	2452707	$6.75 per 20
Oxycodone hydrochloride Tab controlled-release 20 mg		1% DV Oct 2013 to 2015
Oxycodone CR Tablets (BNM)	2452715	$11.50 per 20

Oxycodone hydrochloride Tab controlled-release 5 mg

OxyContin	2194767	$7.51 per 20

Pethidine hydrochloride

Pethidine Hydrochloride Inj 50 mg per ml, 1 ml ampoule		1% DV Sep 2014 to 2017
DBL	500429	$5.51 per 5
Pethidine hydrochloride Inj 50 mg per ml, 2 ml ampoule		1% DV Sep 2014 to 2017
DBL	394971	$5.83 per 5
Pethidine hydrochloride Tab 100 mg		1% DV Mar 2013 to 2015
PSM	259950	$5.80 per 10
Pethidine hydrochloride Tab 50 mg		1% DV Mar 2013 to 2015
PSM	259942	$3.95 per 10

Local Anaesthetics

Bupivacaine hydrochloride Inj 1.25 mg with fentanyl 2 mcg per ml, 100 ml bag

Bupafen	2309009	$210.00 per 10

Bupivacaine hydrochloride Inj 1.25 mg with fentanyl 2 mcg per ml, 200 ml bag

Bupafen	2308991	$210.00 per 10

Neuropathic Pain

Amitriptyline

Amitriptyline Tab 10 mg		1% DV Sep 2014 to 2017
Arrow-Amitriptyline	2404451	$1.68 per 100
Amitriptyline Tab 25 mg		1% DV Jan 2015 to 2017
Arrow-Amitriptyline	2461250	$1.68 per 100

Gabapentin

Gabapentin Cap 100 mg		
Nupentin	2422751	$7.16 per 100
Arrow-Gabapentin	2443848	$7.16 per 100
Gabapentin Cap 300 mg		
Nupentin	2422778	$11.00 per 100
Arrow-Gabapentin	2443856	$11.00 per 100
Gabapentin Cap 400 mg		
Nupentin	2422786	$13.75 per 100
Arrow-Gabapentin	2443864	$13.75 per 100

Restrictions:

Acute Pain

1. For preoperative and/or postoperative use for up to a total of 8 days' use; or
2. For the pain management of burns patients with monthly review.

Neuropathic pain

The patient has been diagnosed with neuropathic pain (Re-assessment required after 3 months. Outpatient use requires the approval of a special authority number)

Intravenous Anaesthetics

Ketamine

Ketamine Inj 10 mg per ml, 10 ml syringe		1% DV Sep 2014 to 2017
Biomed	2461870	$14.00 per 1
Ketamine Inj 100 mg per ml, 2 ml vial		
Any brand		

Respiratory Depressant Antagonists

Naloxone hydrochloride Inj 400 mcg per ml, 1 ml ampoule		
Hospira	788457	$48.84 per 5

Antimigraine Drugs

Sumatriptan Tab 50 mg		1% DV Sep 2013 to 2016
Arrow-Sumatriptan	2347008	$29.80 per 100
Sumatriptan Tab 100 mg		1% DV Sep 2013 to 2016
Arrow-Sumatriptan	2347016	$54.80 per 100
Sumatriptan Inj 12 mg per ml, 0.5 ml cartridge		1% DV Sep 2013 to 2016
Arrow-Sumatriptan	2381656	$13.80 per 2

Prophylaxis of Migraine

Clonidine hydrochloride Inj 150 mcg per ml, 1 ml ampoule		1% DV Nov 2012 to 2015
Catapres	202266	$16.07 per 5
Clonidine hydrochloride Tab 150 mcg		1% DV Feb 2013 to 2015
Catapres	202274	$34.32 per 100
Clonidine hydrochloride Tab 25 mcg		1% DV Jul 2013 to 2015
Clonidine BNM	2432684	$15.09 per 112
Pizotifen Tab 500 mcg		1% DV Mar 2013 to 2015
Sandomigran	251666	$23.21 per 100

SECTION 5

OPIOID ANTAGONIST

NALOXONE HYDROCHLORIDE:

Naloxone is an essentially pure **opioid antagonist**, reversing the effects of opioids, most usually that of opioid overdose, though it can be used to reverse the distressing effects of histamine release (i.e. itch). It's mechanism of action is not fully understood but evidence suggests that antagonists compete for the same receptor sites as the opioids (agonists). Given intravenously, onset of action is apparent within 1-2 minutes, and it has a rapid distribution throughout the body. Plasma half-life in adults ranges from thirty minutes to 1 hour. In children bolus doses of 0.1mg/kg have a half-life of up to 70 min(neonates) Duration of action is dependent on dosage and route of administration, and requirement for repeat doses will also be dependent on the type, amount and route of administration of the opioid being reversed.

Administration *(see HAUORA TAIRAWHITI IV Therapy Manual page 244)*

Doses will vary between children and adults. The most rapid onset of action is achieved by IV administration, which is recommended in emergency situations, though naloxone can be administered by IM or subcutaneous routes too. It can be given either undiluted, diluted *or* as an infusion and titrated according to the patient's response. However, it must be remembered that since the duration of action of some opioids may exceed that of naloxone, the patient should be kept under close observation. Naloxone can be administered by an IV certificated RN/midwife or registered doctor.

NALOXONE TITRATION:	Make up 0.4mg Naloxone in 9ml Normal saline. Give 1 ml (0.04mg) IV every 2 minutes until patient is easy to rouse with respiratory rate >6/minute
RESCUE:	Give 0.2mg Naloxone IV stat and repeat after 2 minutes if indicated
PAEDIATRIC DOSE:	0.01mg/kg (10mcg/kg)

NB: It is important to remember that narcosis can recur and repeated doses of naloxone may be needed, especially if the patient has renal or liver impairment.

Adverse Effects

Abrupt reversal of opioid narcosis may result in:

- Significant reversal of analgesia
- Excitement - agitation, restlessness
- Tremulousness
- Nausea/vomiting
- Sweating
- Tachycardia
- Hypertension

NB: It should be used with caution in patients with pre-existing cardiac disease because cases of hypotension, hypertension, ventricular tachycardia and fibrillation as well as pulmonary oedema have been reported.

Please see specific maternity/neonatal unit guideline on reversal of narcotics in relation to administration to neonates, and paediatric IV morphine protocol for administration of naloxone to infants and children.

SECTION 6

POST-OPERATIVE NAUSEA AND VOMITING (PONV)
OPIOID INDUCED NAUSEA AND VOMITING AND CONSTIPATION

INTRODUCTION:

Nausea and vomiting are common and distressing sequelae of surgery. Indeed, adult patients will often rate nausea and/or vomiting as worse than any pain they experience in the postoperative period.

The incidence and severity of postoperative nausea and vomiting (PONV) have been decreasing as we learn more about precipitating factors, change operative procedures, and use better anaesthetic agents/perioperative medications. However, it is still far too common.

PHYSIOLOGY OF PONV:

Vomiting is essentially a protective mechanism designed to rid the body of poisoned/contaminated food. Feeling sick (nausea) is the trigger that will prevent further ingestion, while relaxation of the stomach to inhibit gastric emptying then leads to vomiting and the ejection of the offending material.
Nausea and vomiting can be divided into three distinct phases:

- nausea
- retching and vomiting
- post-vomiting

Nausea

This is a subjective sensation that may or may not lead on to vomiting, but is a separate entity.

It is worth noting that nausea can be more prolonged and less easily controlled than vomiting, and may be more unpleasant than actually vomiting.

Retching and Vomiting

Retching is the rhythmic movement that usually occurs in bursts immediately before vomiting, and is essentially **breathing in against a closed glottis**. The function of retching is unknown, but has been suggested that perhaps retching shifts duodenal and stomach contents to a more suitable position for vomiting to occur.

Retching can occur without actual vomiting and this makes it extremely unpleasant and uncomfortable for patients.

Vomiting is the forceful expulsion of upper gastro-intestinal contents through the mouth. The major driving force behind vomiting is the contraction of the rectus abdominis muscle.

Post-Vomiting Phase

Vomiting leaves the patient lethargic and weak; people find their legs tremble and can no longer support them if they are standing up. Although the nature of this process is little understood, vomiting itself is metabolically expensive and uses up considerable energy.

FACTORS CONTRIBUTING TO PONV:

Predisposition

- PONV is more likely in children than in adults.
- Incidence decreases with age.
- Women are more likely than men to suffer PONV, especially if they are pregnant or menstruating.
- Psychological stress and anxiety, and medical conditions such as uraemia and diabetes increase the risk.
- A history of motion sickness or previous PONV also increases risk.

Gastric Volume
High gastric volume (e.g. inadequate emptying time, bowel obstruction, swallowed air, or air insufflations after mask ventilation) may predispose to PONV. Prolonged fasting on the other hand, especially in females, increases the risk of PONV.

Premedication
Atropine, although useful as an antiemetic, may decrease gastric emptying and contribute to PONV. Likewise, opioids slow gastric emptying, increase the sensitivity of the Vomiting Centre (VC) to vestibular input and stimulate the Chemotrigger Receptor Zone (CTZ).

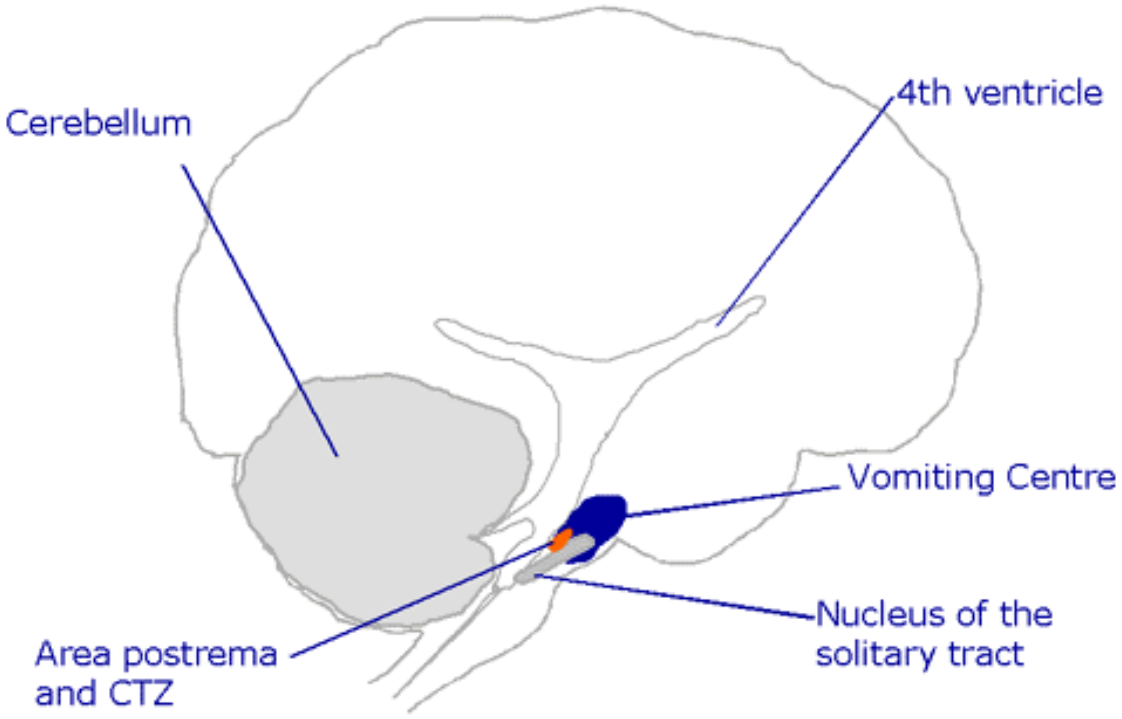
Anaesthetic Agents
Volatile agents increase the risk of PONV, as does Nitrous Oxide. Ketamine has also been implicated in the causing PONV. Propofol is associated with reduced emesis when compared to other induction/anaesthesia agents.

Surgical Procedure
Site of operation plays a part in incidence of PONV. Abdominal, gynaecological, eye procedures all lead to a higher risk of PONV, and laparoscopy because of manipulation of viscera and abdominal distension by gas.

Postoperative Factors
Hypotension, pain, swallowed blood, nasopharyngeal suctioning, all may increase risk of PONV.

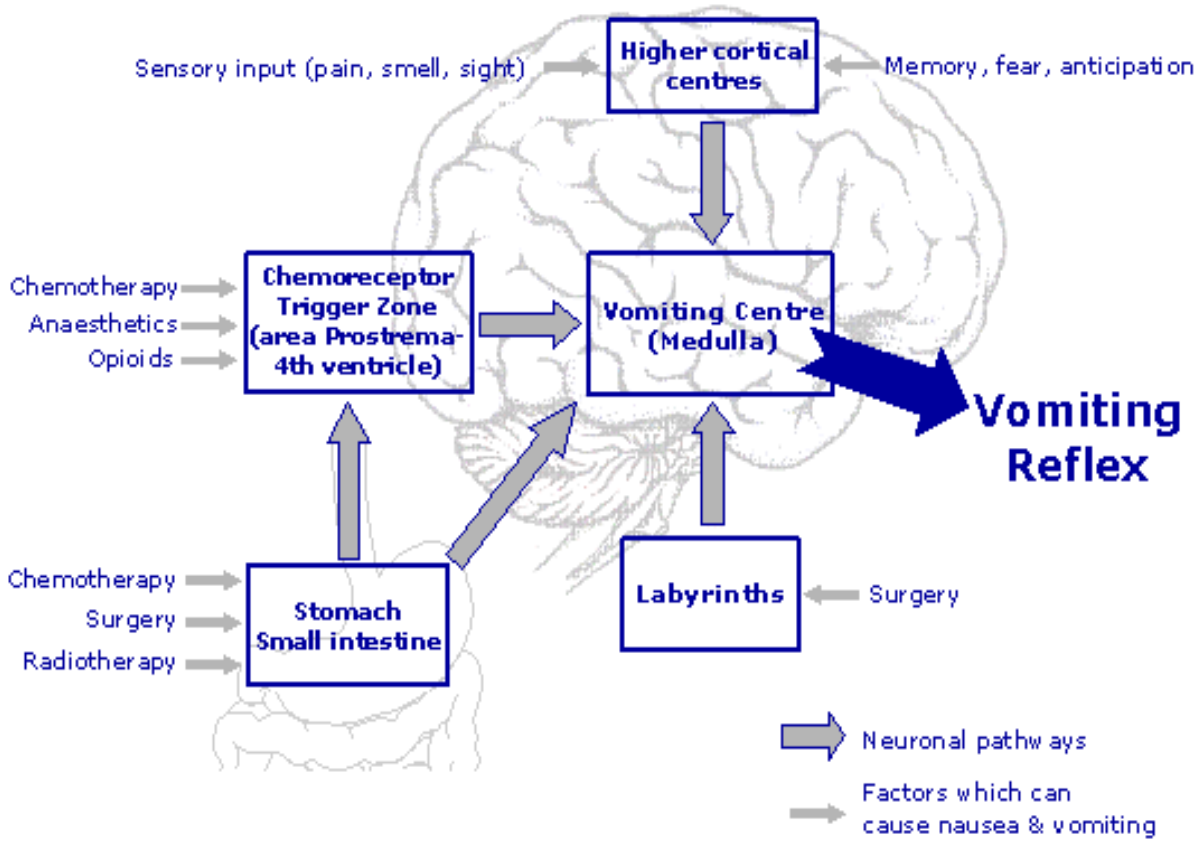
PROBLEMS ASSOCIATED WITH PONV:
PONV is debilitating and depending on its severity can lead to such consequences as fluid and electrolyte imbalances, Mallory-Weiss tears, depression and loss of morale. For the post-surgical patient, be they adult or child, it can also have a disastrous effect of postoperative recovery, with disruption of wound healing and immobility leading to longer hospital stays.

The Vomiting Reflex
The vomiting reflex involves:
1. ***DETECTORS*** - identify the need to vomit



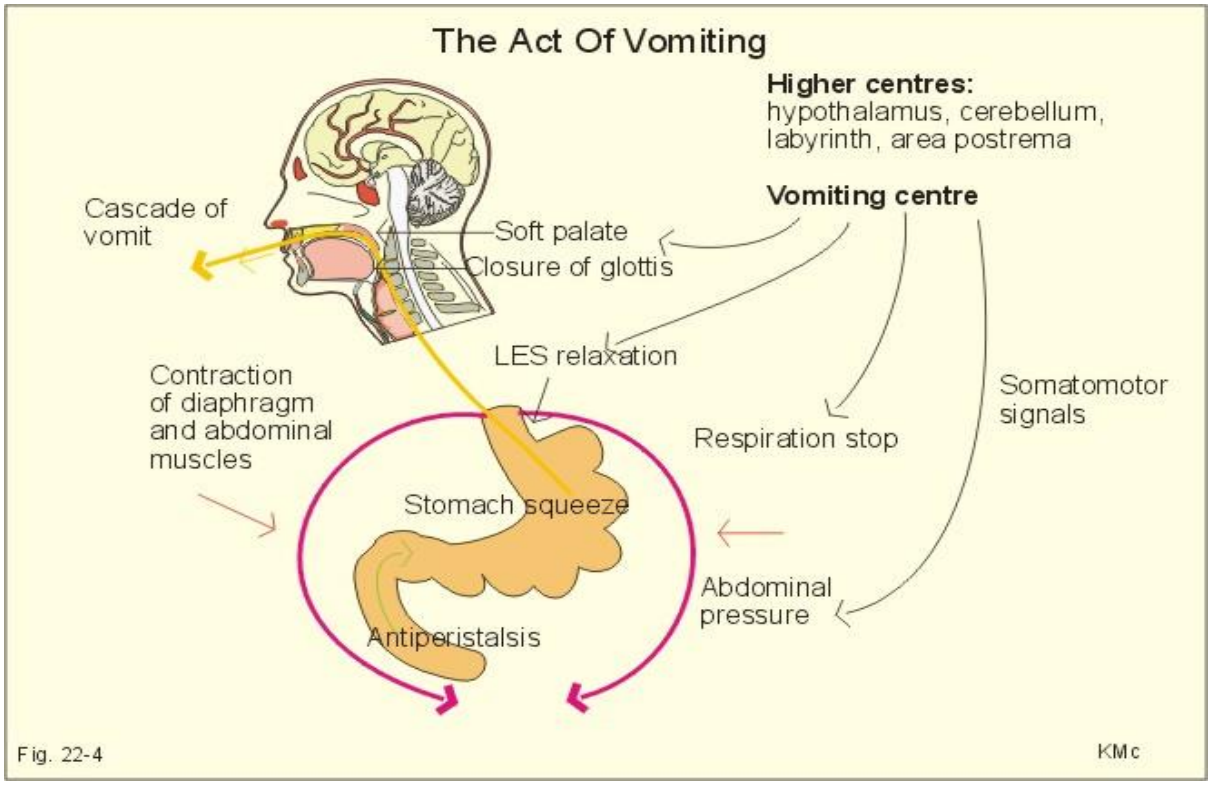
Main detectors are:

- **Gastrointestinal tract – chemoreceptors and mechanoreceptors**
  Stimulation of the gastrointestinal tract's chemo or mechanoreceptors leads to activation of the *vagal afferents*, which in turn stimulate the Vomiting Centre (VC) and lead to emesis.
- **CTZ** – specialised area in 4th ventricle known as *area postrema*
- **Other pathways** involved in vomiting. Although the CTZ and gastrointestinal system are most commonly implicated in the vomiting cycle, other pathways may be just as relevant in the post-surgical patient's situation. They are the following:
  - Pharyngeal stimulation
  - Higher brain centres - cortex and limbic centres - emotions, sights and smells, thoughts can all produce vomiting
  - Vestibulocerebellar pathway - motion is a potent cause of nausea and vomiting and results from stimulation of the labyrinthine apparatus of the inner ear.
  - Cardiac - vagus nerve has a cardiac branch, and may explain why heart attacks are often accompanied by vomiting.
  - Sympathetic activity from some other organs will also stimulate vomiting, for example urinary or gallbladder distension, or surgical procedures for gynaecological conditions, for example:



2. ***EFFECTORS*** - cause the vomiting

The "output" arm of the vomiting reflex consists of higher brain centres, the motor nerves co-ordinating respiratory muscle function, the vagal efferents which control gastric juice secretion and the sympathetic nerves which cause vasoconstriction, increased salivation, tachycardia



3. ***CO-ORDINATING CENTRE*** - controls the entire process

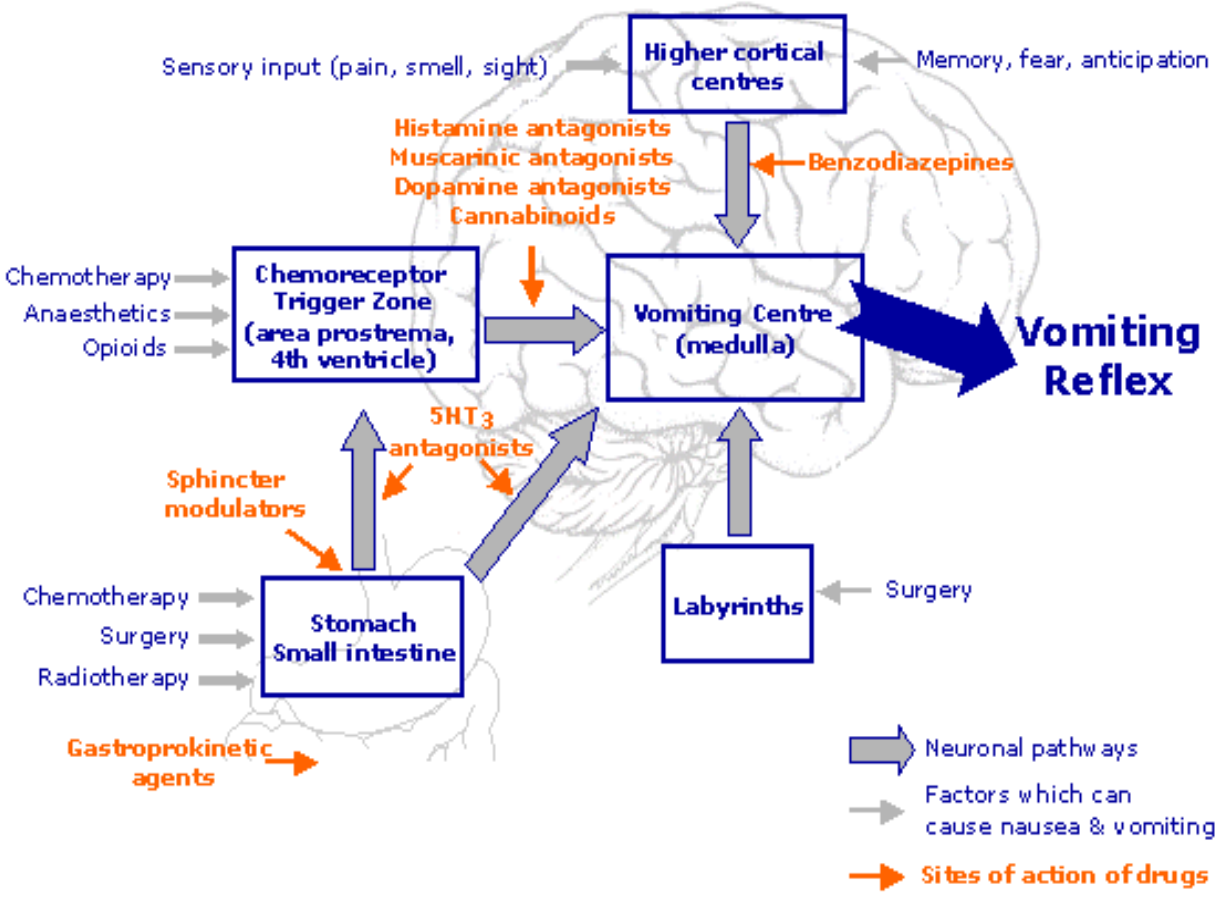
The vomiting centre thus receives input from gastrointestinal, ocular, pharyngeal, cardiac and tympanic afferents, and it co-ordinates the entire sequence of events. Located in the brainstem, it is a functional centre; its role is to detect and orchestrate the complex chain of events that cause nausea, retching, and may culminate in vomiting.

Transmitters involved in Vomiting Reflex

The main transmitters already identified as important are Dopamine D_2, Histamine, Acetylcholine, 5-HT (Serotonin). Within the body are *receptors*, which are **recognition sites on cells that will recognize messenger molecules that bind to and activate the receptor**.

If the binding that results is part of the biological function of that cell, the messenger molecule is an **AGONIST**, whereas inhibition of normal cell response is **ANTAGONISTIC**.

Anti-emetics have been developed to halt the normal cellular responses to messages being received from the gastro-intestinal tract, the vestibular apparatus, the CTZ, the cortex, and the vomiting centre, as the figure below indicates.



MANAGEMENT OF PONV:

Prophylactic Treatment

In high risk cases, prophylactic management is indicated. For example, those patients who will undergo surgery likely to result in postoperative nausea and vomiting and who have a history of motion sickness are candidates for premedication using an antiemetic, with the avoidance of opiates.

Class	Drug
Anti-cholinergic	scopolamine (L-hyoscine)
Anti-histamine	cyclizine promethazine
Dopamine antagonists	metoclopramide domperidone droperidol haloperidol
Cannabinoid	nabilone
Corticosteroid	dexamethasone
Histamine analogue	betahistine
$5HT_3$-receptor antagonist	granisetron ondansetron tropisetron

NB: Some anti-emetics are not safe for children, so check before using.

Intra-operatively, consideration is given to the anaesthetic agents most likely to minimise the effects of surgery, for instance use of propofol at induction and then for maintenance anaesthesia, and the avoidance of volatile agents.

KEY PRINCIPLES OF PONV MANAGEMENT:

The timing of antiemetic prophylaxis is still controversial. During shorter cases, anti-emetics can be given intravenously at induction of anaesthesia, whereas for longer cases, anti-emetics administered shortly before end of surgery is indicated.

Combination rather than single agent therapy is likely to be the most effective. Drugs that work at different receptors will give the best results, rather than using drugs from similar drug groups, e.g., too many antidopaminergics. This will only increase the likelihood of side-effects of the drugs, without benefitting the patient.

Regular versus PRN administration is also indicated, and this principle is similar to effecting analgesia. More constant serum levels are achieved, giving better antiemetic cover.

Minimising those other causes of nausea and vomiting. For instance, keep patients/children well hydrated, comfortable and quiet in the immediate post-operative period. Minimise movement wherever possible, and keep patients/children away from smells more likely to upset them.

Remember, the best intervention for postoperative nausea and vomiting is PREVENTION, so nurses and midwives need to regularly assess their patients for this distressing side-effect.

MANAGEMENT OF NAUSEA AND VOMITING:

Place in Therapy	Drug	Dose	Notes
1st Line One or more of these agents can be used either singly or in combination. Choose the most appropriate agent based on patient factors.	**Ondansetron** Inj 4mg. 8mg Tabs 4mg, 8mg Wafers 4mg, 8mg	For PONV: a single 4mg iv/im dose.	Ondansetron decreases gut motility.
	Metoclopromide Inj 10mg Tabs 10mg Syrup 5g/5ml	10mg q8h May be given iv/im/po	Metoclopromide increases gut motility. May cause dystonic reactions.
	Cyclizine Inj 50mg Tabs 50mg	50mg q8h prn May be given iv/im/po	Cyclizine decreases gut motility. It also causes drowsiness.
If the nausea and vomiting are still not controlled then a third line agent can be added to, or substituted for the second line agent.			
2nd Line One or more of these agents can be used either in combination or in place of the 1st Line agent/s.	**Methotrimeprazine** Inj 25mg/ml Tabs 25mg, 100mg	12.5 - 25mg q8h May be given iv/im/po	Methotrimeprazine increases gut motility. May cause dystonic reactions and anti-cholinergic effects.
	Droperidol Inj 10mg/2ml		Droperidol is a very useful agent when given pre-op.
	Promethazine Inj 25mg/ml Tabs 10mg. 25mg	25mg prn May be given im/po	Cause more sedation than cyclizine.
	Hyoscine Inj 400mcg	400mcg im/iv prn	Hyoscine decreases gut motility.
	Prochloperazine Inj 12.5mg/ml Tabs 3mg, 5mg	12.5mg q8h im 5mg q8h po	No effect on gut. 3mg buccal tablets available.

	Dexamethasone	4mg tds po, 4-8mg iv/im prn up to q8h
	Lorazepam	1-2 mg prn
PONV Most appropriate agent/s to be used in individual cases.	**Droperidol** **Dexamethasone** **Metoclopromide** **Cyclizine** **Ondansetron**	2.5-10mg at or prior to induction 4-8mg iv/im usually given in theatre 10mg q8h 50mg q8h prn 4mg IV pre-op or 16mg PO 1 hour pre-op followed by 4-8mg IV/PO q8 PRN (for 2 doses).
Spinal Anaesthesia	**Droperidol** **Dexamethasone** **Ondansetron**	Treatment aimed at nausea and vomiting and pruritis associated with spinal anaesthesia.

INDIVIDUAL AGENTS:

Ondansetron

Ondansetron is not an effective agent for preventing vomiting and should be in conjunction with other agents if the patient is vomiting. For the treatment of **PONV** the dose is 4mg IV pre-op or 16mg PO 1 hour pre-op followed by 4-8mg IV/PO q8 PRN (for 2 doses).

Ondansetron	Antiserotonergic Noncytotoxic and supportive therapy
Uses/Indications:	Antiemetic, $5HT_3$-receptor antagonist. Management of nausea and vomiting induced by cytotoxic chemotherapy and radiotherapy. Treatment (inj only) and prevention of postoperative nausea and vomiting.
Precautions:	$5HT_3$-antagonist cross sensitivity; subacute intestinal obstruction (monitor); hepatic impairment; phenylketonuria (wafers); pregnancy, lactation.
Adverse Reactions:	Headache; sensation of warmth, flushing; hiccups; LFT increases; constipation; injection site reactions. Rare: visual disturbances, dizziness (rapid IV only); hypersensitivity reactions including anaphylaxis; extrapyramidal reactions; seizures; chest pain; arrhythmias; hypotension; bradycardia
Drug Interactions:	Phenytoin; carbamazepine; rifampicin; tramadol.
Pregnancy Category:	**Category B1 -** Drugs that 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[1] have not shown evidence of an increased occurrence of fetal damage.
Dose:	Prevention of post-op nausea and vomiting - Adults: 4mg IV pre-op or 16mg PO 1 hour pre-op followed by 4-8mg IV/PO q8 PRN (for 2 doses). Hepatic impairment: max. 8 mg/day.

Metoclopramide

Metoclopramide hydrochloride	Antidopaminergic Antiemetics, antinauseants
Uses/Indications:	Restores normal co-ordination and tone to the upper digestive tract and relieves symptoms of GI dysfunction. Treatment of nausea and vomiting. Relieves symptoms of nausea and vomiting and gastric stasis associated with migraine. Promotes normal gastric emptying and restores motility in vagotomised patients and where postoperative symptoms suggest gastroduodenal dysfunction. Speeds up the passage of a barium meal and facilitates duodenal intubation procedures.

	Restrict use in patients < 20 years to: severe intractable vomiting of known cause, vomiting associated with radiotherapy and intolerance to cytotoxics, aid to GI intubation and surgical premedication. See full PI.
Contra-indications:	Phaeochromocytoma; use within 3-4 days of operations such as pyloroplasty or gut anastomosis; GI obstruction, perforation, haemorrhage
Precautions:	Establish diagnosis; reassess if vomiting persists; epilepsy; concomitant treatment with neuroleptics, centrally acting drugs; porphyria; prolonged use; high dose; hepatic, renal impairment; elderly; young adults; pregnancy, lactation, children.
Adverse Reactions:	Extrapyramidal reactions, usually dystonic type; raised serum prolactin levels; tardive dyskinesia; rashes, urticaria, pruritus; oedema; rare: drowsiness, restlessness, confusion, depression; diarrhoea; NMS; methaemoglobinaemia, sulphaemoglobinaemia
Drug Interactions:	Anticholinergics; opioid analgesics; phenothiazines; tetrabenazine; drugs acting at central dopamine receptors, e.g. levodopa, bromocriptine, pergolide; atovaquone; any concurrently administered oral medication incl aspirin, paracetamol
Pregnancy Category:	**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.
Dose:	Usual max. daily dose 0.5 mg/kg. Reduce in severe renal, hepatic impairment. Adults 20 years and over: 10 mg 3 times daily; 15-19 years, 60 kg and over: 10 mg 3 times daily; 15-19 years, 30-59 kg: 5 mg 3 times daily. Use syrup in children.

Prochlorperazine

Prochlorperazine	Antidopaminergics Antiemetics, antinauseants
Uses/Indications:	Anti-emetic dopamine and histamine antagonist. Treatment of vertigo, nausea and vomiting. Used for migraine, schizophrenia, acute mania and as an adjunct to the short-term management of anxiety.
Contraindications:	Renal, hepatic impairment; epilepsy; Parkinson's disease; hypothyroidism; phaeochromocytoma; myasthenia gravis; prostrate hypertrophy; history of narrow angle glaucoma; administration of discoloured (darkened) injection solution; rectal, IM administration in children; children weighing less than 10 kg.
Precautions:	Very hot, cold weather; pre-existing cardiac disease; hypocalcaemia; elderly; pregnancy, lactation; children.
Adverse Reactions:	Nasal stuffiness; dry mouth; insomnia; agitation; hypotension; cardiac arrhythmias; respiratory depression; mild leukopaenia; anticholinergic effects; CNS disturbances incl impaired alertness, drowsiness; akathisia; parkinsonism; tardive dyskinesia; acute dystonias, dyskinesias (children); hypo/hyperthermia; neuroleptic malignant syndrome; blurred vision; contact skin sensitisation; QT interval prolongation; hyperprolactinaemia; jaundice; photosensitivity; others, see full PI.

Drug Interactions:	CNS depressants incl alcohol, tricyclics; most antihypertensives; other anticholinergics; desferrioxamine; phenytoin; amphetamine; l-dopa; clonidine; adrenaline; antacids; anti Parkinson; lithium; oral anticoagulants; thiazide diuretics; guanethidine; hypoglycaemics
Pregnancy Category:	**Category C** Drugs that, owing to their pharmacological effects, have caused or maybe suspected of causing harmful effects on the human fetus or neonate without causing malformations. These effects may be reversible.
Dose:	Oral: May be taken with or without food. *Adults*: Prevention of nausea and vomiting: 5-10mg 2-3 times daily; Treatment of nausea and vomiting: 20mg immediately, then 10mg 2 hours later. Suppositories: Nausea and vomiting: 25 mg suppository immediately. Oral dose 6 hours later if required. IM: Nausea and vomiting: 12.5mg deep IM injection immediately. Oral dose 6 hours later if required.

Cyclizine

Cyclizine lactate	Anticholinergic Antiemetics, antinauseants
Uses/Indications:	Antiemetic, piperazine derivative. Prevention and treatment of nausea and vomiting associated with narcotic analgesics, general anaesthetics in post-op period, radiotherapy (esp in breast cancer) and motion sickness (when oral route is unsuitable). Pre-op in patients undergoing emergency surgery. Relief of vomiting and vertigo in Meniere's disease and other vestibular disturbances when oral route is unsuitable.
Precautions:	Glaucoma; obstructive disease of GIT; prostatic hypertrophy; severe heart failure; renal, hepatic impairment; abuse potential; pregnancy, lactation
Adverse Reactions:	Drowsiness; dry mouth, nose, throat; blurred vision; tachycardia; urinary retention; constipation; restlessness; nervousness; insomnia; auditory, visual hallucinations; hypotension; others, see full PI.
Drug Interactions:	CNS depressants incl alcohol; pethidine; anticholinergics.
Pregnancy Category:	**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.
Dose:	Adults: 50 mg IMI or slow IVI up to 3 times daily. Prevention of post-op vomiting: 50 mg by slow IVI 20 minutes before end of surgery. Pre-op in emergency surgery: 25 mg IVI before induction of general anaesthesia.

Hospital Medicines List (HML)
http://www.pharmac.health.nz/tools-resources/pharmaceutical-schedule/section-h/
(Please refer to the NZF or NZFc for doses)

Betahistine dihydrochloride Tab 16 mg		1% DV Jun 2014 to 2017
Vergo 16	2217090	$4.95 per 84
Cyclizine lactate Inj 50 mg per ml, 1 ml ampoule		
Nausicalm	2349655	$14.95 per 5
Cyclizine hydrochloride Tab 50 mg		1% DV Sep 2012 to 2015
Nausicalm	2231220	$0.59 per 10
Dexamethasone Oral liq 1 mg per ml		
Biomed	344842	$45.00 per 25 ml
Dexamethasone Tab 1 mg		1% DV Aug 2012 to 2015
Douglas	394947	$5.87 per 100
Dexamethasone Tab 4 mg		1% DV Aug 2012 to 2015
Douglas	277878	$8.16 per 100
Dexamethasone phosphate Inj 4 mg per ml, 1 ml ampoule		1% DV Apr 2014 to 2016
Dexamethasone-hameln	2451689	$25.80 per 10
Dexamethasone phosphate Inj 4 mg per ml, 2 ml ampoule		1% DV Apr 2014 to 2016
Dexamethasone-hameln	2451697	$17.98 per 5
Domperidone Tab 10 mg		1% DV Mar 2013 to 2015
Prokinex	2425874	$3.25 per 100
Droperidol Inj 2.5 mg per ml, 1 ml ampoule		
Any brand		
Hyoscine butylbromide Inj 20 mg, 1 ml ampoule		
Buscopan	770833	$9.57 per 5
Hyoscine butylbromide Tab 10 mg		
Gastrosoothe	2239787	$1.48 per 20
Hyoscine hydrobromide Patch 1.5 mg		1% DV Dec 2013 to 2016
Scopoderm TTS	311472	$11.95 per 2

Restrictions:

1. Control of intractable nausea, vomiting, or inability to swallow saliva in the treatment of malignancy or chronic disease where the patient cannot tolerate or does not adequately respond to oral anti-nausea agents; or
2. Control of clozapine-induced hypersalivation where trials of at least two other alternative treatments have proven ineffective; or
3. For treatment of post-operative nausea and vomiting where cyclizine, droperidol and a 5HT3 antagonist have proven ineffective, are not tolerated or are contraindicated.

Hyoscine hydrobromide Inj 400 mcg per ml, 1 ml ampoule		
Hospira	259144	$46.50 per 5
Levomepromazine Inj 25 mg per ml, 1 ml ampoule		
Any brand		
Levomepromazine Tab 25 mg		
Any brand		
Metoclopramide hydrochloride Inj 5 mg per ml, 2 ml ampoule		1% DV Sep 2014 to 2017
Pfizer	771759	$4.50 per 10
Metoclopramide hydrochloride Oral liq 5 mg per 5 ml		
Any brand		
Metoclopramide hydrochloride Tab 10 mg		1% DV Sep 2014 to 2017
Metamide	282081	$1.82 per 100
Ondansetron Inj 2 mg per ml, 2 ml ampoule		1% DV Sep 2013 to 2016
Ondanaccord	2425645	$1.82 per 5
Ondansetron Inj 2 mg per ml, 4 ml ampoule		1% DV Sep 2013 to 2016
Ondanaccord	2425653	$2.18 per 5
Ondansetron Tab 4 mg		1% DV Jan 2014 to 2016
Onrex	2441306	$5.51 per 50
Ondansetron Tab 8 mg		1% DV Jan 2014 to 2016
Onrex	2441314	$6.19 per 50
Ondansetron Tab dispersible 4 mg		1% DV Oct 2014 to 2017
Dr Reddy's Ondansetron	2369486	$1.00 per 10
Ondansetron Tab dispersible 8 mg		1% DV Oct 2014 to 2017
Ondansetron ODT-DRLA	2369494	$1.50 per 10
Prochlorperazine Inj 12.5 mg per ml, 1 ml ampoule		
Any brand		
Prochlorperazine Suppos 25 mg		
Any brand		
Prochlorperazine Tab buccal 3 mg		
Any brand		
Prochlorperazine Tab 5 mg		1% DV Jun 2014 to 2017
Antinaus	395838	$9.75 per 500

MANAGEMENT OF OPIOID INDUCED CONSTIPATION:

All patients on a regular opioid or on ongoing PRN opioids should be co-prescribed laxatives as per the table below.

All patients that are taking things orally should be given adequate fluids and appropriate bulk forming agents like kiwicrush or psyllium.

	Medication	Comments
1st Line	Docusate and Senna (Laxsol) 2 tabs bd	To be co-prescribed with a regularly or ongoing PRN opioids.
2nd Line To be added to therapy with the combination stimulant/stool softener above.	Lactulose 10-20mls bd Movicol Sachets 2 to 3 sachets daily	 Movicol is restricted to patients who do not tolerate lactulose and who need or have failed therapy with a rectal preparation.
Consider stopping other medications that may contribute to constipation eg ondansetron, cyclizine, anticholinergic agents.		
3rd Line These should be used acutely or if necessary added to regular therapy as above.	Bisacodyl Suppositories Glycerol Suppositories Micolette enemas Pico-salax or Fleet Danthron with Poloxamer	 *Danthron with Poloxamer is only for use in patients who are terminally ill. It is not currently available – check with pharmacy.*
Investigate for obstruction. If found discontinue stimulant laxatives and manage accordingly. Docusate may continue to be used on its own in these patients along with osmotic laxatives like lactulose.		

Hospital Medicines List (HML)

http://www.pharmac.health.nz/tools-resources/pharmaceutical-schedule/section-h/
(Please refer to the NZF or NZFc for doses)

Bisacodyl Suppos 10 mg
Dulcolax 270385 $3.00 per 6

Bisacodyl Suppos 5 mg
Dulcolax 270377 $3.00 per 6

Bisacodyl Tab 5 mg
Lax-Tabs 2199327 $4.99 per 200

Danthron Oral liq 25 mg with poloxamer 200 mg per 5 ml
Pinorax (S29) 2259435 $21.30 per 300 ml
(Pinorax Oral liq 25 mg with poloxamer 200 mg per 5 ml to be delisted 01 April 2015)

Restrictions:
Only for the prevention or treatment of constipation in the terminally ill.

Docusate sodium Tab 50 mg with sennosides 8 mg

Laxsol	2069482	$4.40 per 200

Docusate sodium Tab 50 mg 1% DV Jan 2015 to 2017

Coloxyl	252832	$2.31 per 100

Docusate sodium Tab 120 mg 1% DV Jan 2015 to 2017

Coloxyl	252859	$3.13 per 100

Glycerol Suppos 3.6 g 1% DV Jan 2013 to 2015

PSM	778338	$6.50 per 20

Lactulose Oral liq 10 g per 15 ml

Laevolac	2440210	$3.84 per 500 ml

Macrogol 3350 with potassium chloride, sodium bicarbonate, sodium chloride and sodium sulphate Powder for oral soln 59 g with potassium chloride 0.7425 g, sodium bicarbonate 1.685 g, sodium chloride 1.465 g and sodium sulphate 5.685 g per sachet

Klean Prep	790753	$14.31 per 4

Macrogol 3350 with potassium chloride, sodium bicarbonate and sodium chloride Powder for oral soln 6.563 g with potassium chloride 23.3 mg, sodium bicarbonate 89.3 mg and sodium chloride 175.4 mg
Movicol Half

Macrogol 3350 with potassium chloride, sodium bicarbonate and sodium chloride Powder for oral soln 13.125 g with potassium chloride 46.6 mg, sodium bicarbonate 178.5 mg and sodium chloride 350.7 mg 1% DV Oct-14 to 2017

Lax-Sachets	2396467	$7.65 per 30

Restrictions:
1. Both:
   - The patient has problematic constipation despite an adequate trial of other oral pharmacotherapies including lactulose where lactulose is not contraindicated; and
   - The patient would otherwise require a per rectal preparation; or
2. For short-term use for faecal disimpaction.

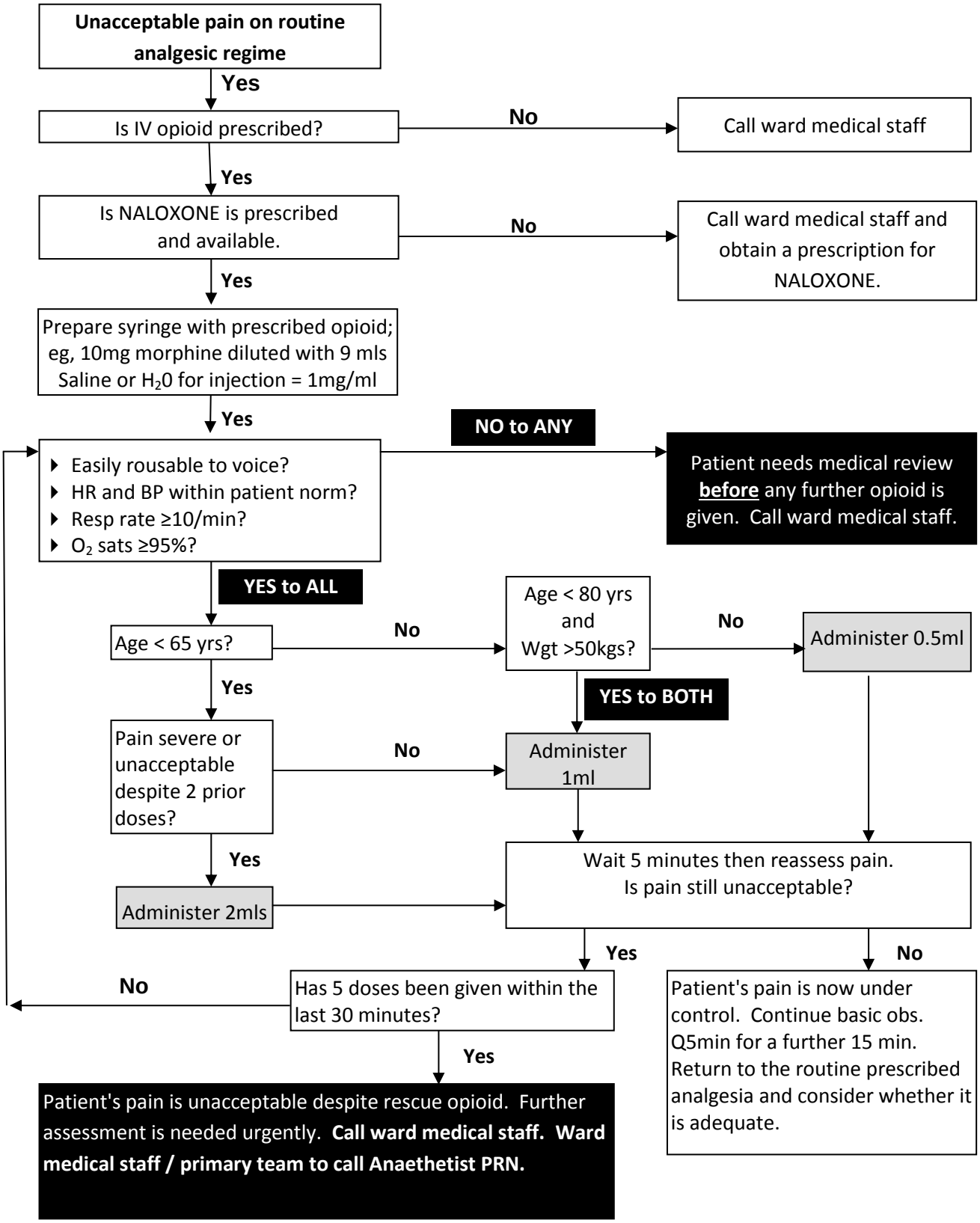
Sodium citrate Enema 90 mg with sodium lauryl sulphoacetate 9 mg per ml, 5 ml 1% DV Sep 2013 to 2016

Micolette	2262673	$19.95 per 50

Citric acid 12 g with magnesium oxide 3.5 g and sodium picosulfate 10 mg per sachet for oral solution
Pico-Salax

SECTION 7

HAUORA TAIRAWHITI IV RESCUE / BOLUS MORPHINE PROTOCOL



For subsequent doses consider current pain intensity, PLUS pain intensity prior to previous dose(s) and efficacy of IV morphine administered, duration of effect and side effects of previous doses.

Paediatrics - See Starship IV Morphine Protocol (Appendix 4).

SECTION 8

METHODS OF OPIOID ADMINISTRATION

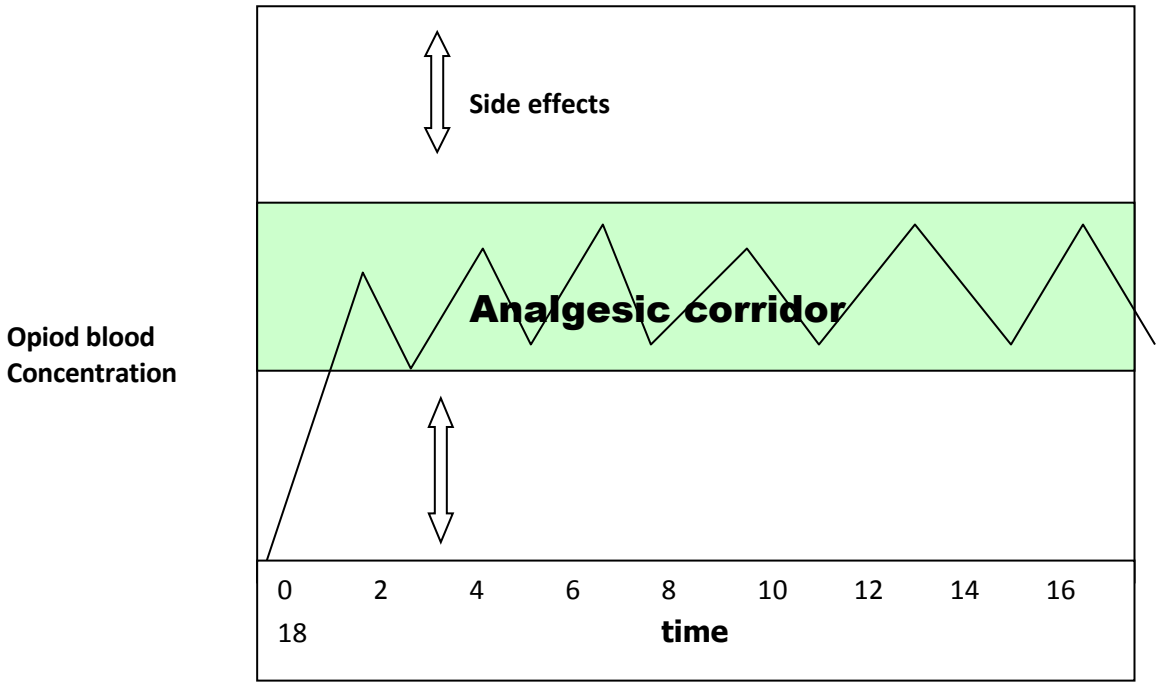
WHAT IS THE CORRECT DOSE OF OPIOID FOR AN ADULT?

Because of the variables involved the general answer to the questions is: *"the right dose is enough* ".

This ***right dose*** should be based initially on patient weight and age ~ and adjusted by titration. This titration should be based on the patient's analgesic response and side effects. Patients vary greatly in their dose requirements and responses to opioid analgesics.

Each patient has a fairly constant blood level range within which they will get pain relief. It can be thought of as an "Analgesic Corridor" (see diagram below).

Analgesic Drug Concentration



Below this level the analgesia effect is suboptimal, above it they have maximal analgesia but also have side effects.

This corridor may be at high or low blood levels but it stays relatively constant for each patient. Between patients there may be up to a fivefold variation in blood level of opioid needed for analgesia. Taking into account pharmacokinetic variables this means that there may be least an 8 to 10 fold variation in dose requirement between patients.

Whatever opioid and whatever route of administration the aim is to give enough to provide each patient with a **blood level which falls inside the analgesic corridor.** .

There is little correlation between weight of the patient and dose of opioid but there is good correlation between age of the patient and dose of opioid. So the initial dose is based on patient age but because of the 8 to 10 fold variation in total dose to analgesic corridor levels titration of subsequent doses will be needed.

Integral to achieving the analgesic corridor is the monitoring of effect. Regular pain assessments are required so the end point or goal is known to have been reached. This also facilitates opioids being given on a regular basis. The "when necessary", prn, administration has definite limitations in delivering consistent ongoing analgesia.

Commonly under the prn system pain is not reported until it is severe. The delay between time of reporting, administration of next dose, and absorption, therefore means a significant period of moderate to severe pain is experienced. This is far from ideal.

The prn system may have some merit late in the post operative course when the pain level is diminished. However, for the **first 48 hours** when the pain level is usually steadily high, opioids should be given on a regular, not a prn, basis. From early in the post operative period, following the initial titration and results obtained from regular pain assessments and the first few regular doses given, the correct dose and dosing interval should be decided upon. Under the 'supervision/monitoring' provided by the regular, at least three hourly, pain score assessments opioids should then be administered on a regular basis for at least the first 48 hours.

CONSIDERING THE PROS AND CONS OF THE VARIOUS ROUTES OF ADMINISTRATION OF OPIOD:

The preferred and most convenient route is the oral one. Numerous preparations in various strengths are available. However, most patients will require parenteral opioids because the oral route cannot be used, ie, nil by mouth, have nausea and vomiting, cannot swallow, have GIT obstruction, or require rapid onset of pain relief in an acute situation.

Intramuscular (IM) - For historical reasons most parenteral opioids in hospital are given intramuscularly (IM) to adults, despite the obvious disadvantages. IM injections have not only a delayed and unpredictable time of onset, they also result in variable blood concentrations depending on the site of injection, muscle perfusion and motor activity of the patient. Other disadvantages include the discomfort of the injection, increasing tissue damage with repeated injections and the potential for abscess formation.

The risk of IM injections is underestimated as the delayed onset of side effects, like respiratory depression might occur unnoticed. This can be minimised by the strictly regular observing and recording of vital parameters, such as respiratory rate, sedation and pain scores, BP and pulse rate.

This applies to whichever route of administration is used. It is inherent in the method of pain relief when using opioids. For the reasons already given this is the method of individualising pain relief by titration so that the appropriate dose and dose interval is determined as quickly and accurately as possible for each patient. This concept must be remembered to avoid approaching each patient with the same dose and dosing interval.

This applies particularly to IM, SC, PO and PR routes and less so to the IV route. Obviously there is a more or less standardised approach used in this method but it still needs to be individualised to each patient. Recognising and considering the somewhat complex nature of the above approach, the **ideal route of parenteral administration of opioids is the intravenous (IV) one.**

Intramuscular administration is not recommended for use in children.

IV bolus injections of opioids result in a rapid predictable onset of action, because obviously the agent is placed directly into the blood stream. It facilitates the stepwise titration of the patient's pain. However, it needs to be done with definite knowledge and care to avoid overdosage resulting in side effects including respiratory depression. A possible disadvantage of only using the IV titration of small doses of opioid is the nursing demands involved. However, to enable it to be used if desired {particularly in the acute setting) and to individualise but standardise the technique a protocol for IV opioid administration by nursing staff is attached **(Section 7)**.

The appropriate use of this protocol is the basis for the administration of opioids IV.in the acute setting. As inferred it is **not** recommended to use it more or less continuously.

PCA - The preferred device for longer term IV opioid use is the patient controlled analgesia pump (PCA). A correctly programmed PCA works similarly to the above mentioned protocol and is inherently safe, as long as continuous infusion and human error are avoided **(see Section 10)**.

Continuous IV infusions of opioids using burette systems and simple volumetric pumps [eg: syringe pumps] pose potential dangers such as manipulation/theft by unauthorised persons, dosing errors, erroneous pump settings, etc, and should not be used routinely in the opioid dependant patient.

Continuous IV or SC infusions of opioids, which are not controlled by the feedback loop of the IV protocol, PCA device, etc, have a five times higher incidence of respiratory depression, especially at night. There is a definite use in the opioid experienced patient (eg, cancer patient) however.

SUBCUTANEOUS:

For non-IV longer term opioid use, the options are IM, subcutaneous (SC), PO or PR. Each of these routes has its advantages and disadvantages for example: the placement of a loaded and non flushed SC butterfly needle or IV cannula avoids the regular needle sticks of IM injections, and produces good comparable absorption of morphine. Pethidine is not so good SC as it is an 'irritable' and higher volume solution.

Insertion and Use of Subcutaneous Needle

- SC morphine is injected into the fatty layer just beneath the skin and the subcutaneous tissue.
- Placement of needle or cannula is usually just below the clavicle or outer aspect of the arm.
- Morphine may be administered by either a subcutaneous bolus or syringe driver.
- Occasionally Fentanyl may be added to the syringe driver.

Equipment needed

- 23 or smaller gauge butterfly needle or IV cannula with bung. Alcohol swabs.
- Small Tegaderm dressing. Opsite spray.
- Morphine to prime the line.

Procedure

- Prime the infusion line with morphine.
- Choose insertion site and clean with alcohol swab. Pinch up a fold of skin and insert needle.
- Spray insertion site with Opsite, cover with Tegaderm dressing, writing on date of insertion.

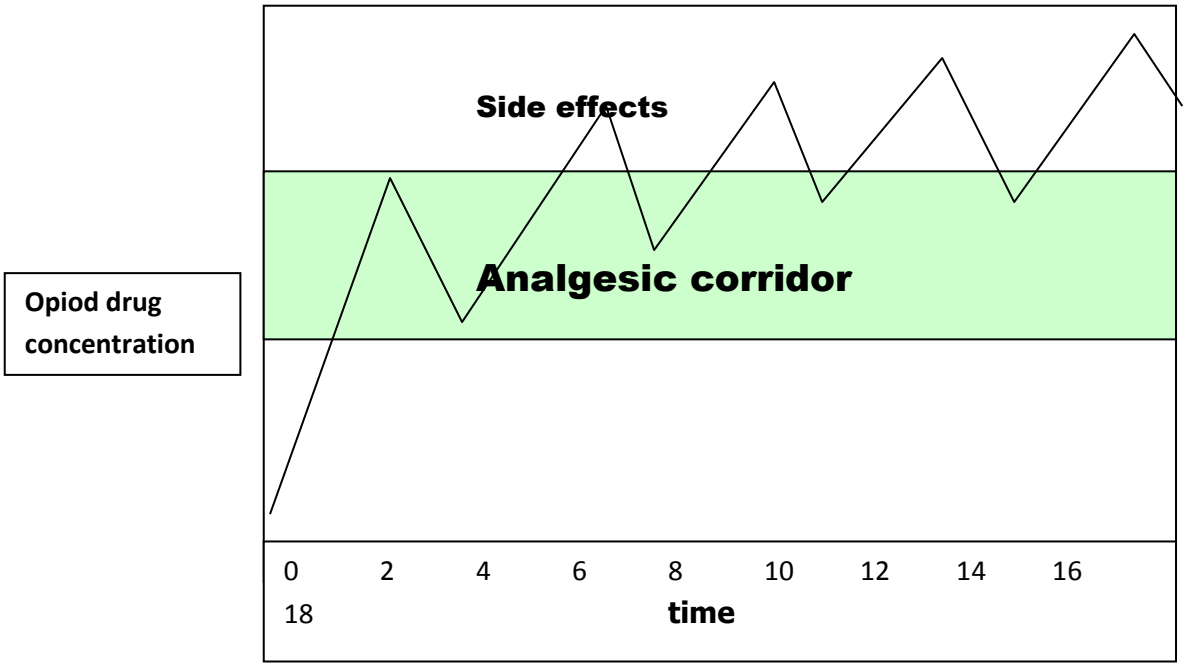
Injection Procedure

- Check order for dose of morphine.
- Draw up dose in 1 or 2 ml syringe and DO NOT DILUTE.
- Swab bung and inject morphine slowly over 1 to 2 minutes. Replace the bung daily.
- **DO NOT FLUSH EITHER BEFORE OR AFTER INJECTION.**
- The elderly need longer intervals between doses because of delayed absorption.

Care of Injection Site

- **The needle will not need to be changed for** 3 **or** 4 **days, unless site becomes red, painful or inflamed.**

Intramuscular (IM) Administration



The above diagram shows what can happen if Morphine or Pethidine are given at four hourly intervals. To last four hours (especially Pethidine) the dose may need to be of a size that will produce some side effects initially. This is commonly reported by patients who may say they are "allergic to morphine" when in fact, they become nauseated and vomited soon after the injection as their blood level peaked above their analgesic corridor. It is better to order the doses at lesser intervals (eg, 2 to 3 hourly).

Ideally for the first 48 hours, once dose and intervals are determined, the opioid should be ordered for, and effect monitored, at regular intervals, by the clock. Obviously this regular dose would be withheld if respiration rate and/or sedation score contraindicated it.

Later in the post op period when the need for opioid is diminished orders could revert to prn dosing.

Alternatively, if the ''as necessary", prn, approach was taken it could be ordered "2-3 hourly prn" with satisfactory observations/recordings. Ensure that prn means "offered frequently" and **NOT** waiting for patient to ask. A "little less more often" can result in blood levels staying within the analgesic corridor.

(Interestingly the concept of "four hourly dose" regimen seems to date back to a clinical study dose in the 1950's when the time between the injection of opioid and return of severe pain was found to be four hours!).

So integral to these schedules, the patients pain should be assessed at **regular** intervals to determine the efficacy of the opioid action, presence of side effects, or the need for dosage adjustment (up or down), or supplemental doses for breakthrough pain.

Finally a combination of these two approaches would be to order that initially the opioid dose be given at a regular interval (dose and interval to be determined/decided upon for/by each patient's response), unless the recordings contraindicated it or the patient declined the dose because of not being in pain or was asleep.

As an example:
Morphine 5 to 10 mg IM/SC Q3H unless recordings, patient, or sleep prevents.

By Spinal Injection
The spinal cord is rich in neurones with opioid receptors. By introducing opioid drugs directly into the fluid surrounding the spinal cord, it is possible to achieve very good pain relief- but with much smaller doses than when given by other routes. This is because they act immediately at the opioid receptors in the spinal cord, instead of having to be delivered there via the bloodstream. Because the dose requirements are lower, and there is less spread to other sites in the brain, some of the side effects associated with opioid drugs are also reduced.

Spinal opioids may be given as a 'single shot' technique. Here a very fine needle is introduced into the spinal sac in an area below where the spinal cord ends. A small dose of opioid is injected and the needle is removed. This technique has been used in the past for morphine because its pain relieving effects are long acting (6-12 hours) when given this way. Unfortunately, depression of breathing can potentially occur any time up to 24 hours later, which obligates extra observations. Another reason why morphine has fallen from popularity recently is that he itchiness caused by morphine can be severe and difficult to treat.

Shorter acting opioids like fentanyl can be used as an alternative to morphine however, they may not provide pain relief for very long. Giving repeated injections into the spinal sac increases the likelihood of causing a spinal headache from leaking cerebrospinal fluid.

In recent times, a technique, which combines a single injection of spinal opioid and an epidural, has gained popularity. This is called a 'combined spinal epidural' CSE. The epidural catheter is placed at the same time as the spinal injection and can be used later in labour if further pain relief is needed.

By Epidural Injection
Opioid drugs can also be given via epidural. Although the epidural space is separated from the spinal cord and fluid by a thick membrane (the dura), opioid drugs pass through this barrier fairly easily. The fine epidural catheter remains in the epidural space: this means that additional doses can be given whenever necessary - without requiring any more injections. For these reasons, most centres prefer to use the epidural, as opposed to the spinal, route when opioids are used in the management of postoperative and childbirth pain. When compared with the intramuscular and intravenous routes of injection, the opioid dose requirements are less.

Oral Administration (PO)
Oral opioids are not for severe acute pain, or pain experienced in the acute postoperative/postnatal period because of the unpredictability of pain in this period and patient/child opioid requirements. Also patients' GI tracts are often not functioning well, if at all, for a variety of reasons so use of oral opioids is contraindicated.

However Once the patient is able to drink properly, oral opioids can be very effective providing the difference between oral and parenteral doses is understood. Onset of action is a little slower and duration of action a little longer than IM/SC injections., Long-acting or sustained release preparations provide good background analgesia with the option for the quicker acting preparations available for 'break-through' /incident pain if required.

Oral opioids also allow for a 'step-down' regime, where those patients on parenteral opioids for longer than 7-10 days may require a weaning off opioid therapy.

There are various preparations of morphine and oxycodone, and the less strong tramadol, for use via oral route. These include short-acting and long-acting preparations

Because of the *first-pass* effect and difficulty in predicting absorption, conversions from parenteral to oral administration need to be carefully worked out. It is also important to remember that the majority of patients are *opioid naïve*. What this means is that they are not used to or tolerant to opioids. Prescribers should therefore err on the side of caution when choosing a dose and regularly review it for effect/side effect.

As a guide, an adult patient receiving PCA morphine of 50mg in 24 hours would probably need around 2.5 to 3 times this dose given orally. This converted dose may be given as a sustained preparation alone or as a combination of sustained and shorter-acting opioid. For children dosing and dosing intervals will need careful adjustment. The responsibility of nurses/midwives is to continue to monitor as prescribed the effects of the drug on the patient/child and report concerns or side effects.

The dosing intervals for oral preparation will depend on the form used. Morphine as elixir. tablets, granules, or MST tablets is suitable. While oral Pethidine tablets are available and efficacious it will produce higher levels of Norpethidine than parenteral Pethidine because of the "first pass effect" is not recommended for longer use. Sustained release morphine can be given as a daily, twice daily or three times daily drug, whereas the shorter acting elixir or tablets can be given up to 2 hourly.

As a general rule, for adult patients, LA-morph is the preferred long acting oral opioid, unless patients are admitted already taking and used to M-eslon.

Codeine phosphate is partly metabolised to Morphine and this most probably accounts for its effect.

Rectal Administration (PR)
It has the benefit that the drug does not pass through the liver before entry into the systemic circulation. Absorption PR is often irregular and incomplete but can be used if oral ingestion is unreliable.

Transdermal Fentanyl
For those adult patients who have been receiving Fentanyl via PCA but no longer require the intravenous administration of the drug, transdermal patches in 25, 50, 75, and 100 micrograms/hour are available. The patches stay on for 3 days, and the conversion from IV to transdermal is straightforward: one microgram IV = one microgram transdermal fentanyl. It is simply a matter of working out the total 24 hour IV dose and dividing it into an hourly dose for delivery via the patch.

The main contraindication to this delivery system is cost, so patient selection is important.

It takes about 12 - 16hours before the full effect of the drug is achieved via the transdermal route, and is recommended for the management of chronic rather than acute pain.

Using Methadone
Methadone, as many nurses and midwives will be aware, is the drug of choice for management of opioid addiction. On occasions, adult patients on the methadone programme will be admitted to hospital for surgical/obstetric care. Always make sure their dose of methadone is charted and given.

The dose of methadone they take daily is for their drug addiction, and **should not be considered as adequate analgesia for post-trauma/postoperative pain relief.** Issues such as dependence and tolerance are key issues to consider in the effective management of patients on methadone. These people therefore benefit from specialist advice regarding their pain management requirements so an anaesthetist should be contacted.

Patient
The patients' individual response to opioids differs or alters depending on their previous exposure to opioids, their condition and their stage of disease. Different sets of guidelines for different groups of patients apply therefore as follows:

More recently, methadone may be used instead of sustained release morphine for treatment of pain or for
weaning a patient off opioids. Again, the anaesthetist will determine the dose needed based on patient opioid
use and pain levels. The dose will be reduced over a period of time in keeping with the expectation that the pain is naturally subsiding as injuries/wounds heal and to avoid any symptoms of withdrawal for the patient.

As a rule of thumb, the following conversions are used:
5:1 IV morphine to oral methadone
10:1 Oral morphine to oral methadone
15:1 Oral morphine (high doses) to oral methadone

MANAGEMENT OF OPIOD COMPLICATIONS:

Constipation, nausea, vomiting, sedation and respiratory depression are potential side effects of opioid analgesics. Itching is a common side effect, particularly experienced by patients who receive opioids via intraspinal administration routes. General ways of managing side effects include changing the dosing schedule or route, or giving the drug in a way that produces relatively constant blood levels. This eliminates the high peak serum levels that often cause side effects. If decreasing the morphine dose is inadequate for pain relief, the time interval for giving the drug can be decreased, or the patient may benefit from a controlled release form of the drug. Adding an opioid-sparing agent, such as an NSAID or a co-analgesic drug, can decrease opioid induced side effects. Adding another drug that counteracts the adverse effect can also help decrease the undesirable side effect.

Constipation and Nausea
Opioid analgesics delay gastric emptying and bowel motility, and decrease peristalsis. As a result, constipation is one of the most common side effects experienced by patients who take opioids. Constipation is the only opioid side effect for which tolerance does not develop. Some patients have an increased risk of opioid induced constipation. Such patients include individuals, who are elderly, immobile, have gastrointestinal disease, or who are taking other medications that may also produce constipation. A high roughage diet, adequate fluid intake, and exercise are important strategies to prevent constipation, but are often not sufficient to prevent opioid related constipation. Patients who receive opioids around-the-clock are given stool softeners, suppositories, and laxatives to manage constipation as long as they continue to receive opioid therapy. Nausea is most common with the first dose of the opioid and usually decreases with subsequent doses. Slow and steady morphine titration is a strategy that can help reduce nausea, as is decreasing the dose of the opioid.

Respiratory Depression

Clinically significant respiratory depression in an adult is generally defined as less than 7 breaths per minute. This type of respiratory depression occurs most often when opioids are given by the neuroaxial route, or by intravenous patient controlled analgesia (PCA), than when given by the intramuscular route. Patients receiving neuroaxial narcotics, or IV PCA, should have frequent assessments of respiratory rate, as well as regular assessment of his/her level of consciousness, blood pressure, and pulse. Many institutions have a specific flowsheet at the patient's bedside, specifically for documentation of these vital signs.

Because tolerance to respiratory depression occurs over a period of days to weeks, the patients at highest risk are "opioid naive" patients – individuals who have not previously received opioid drugs and now, due often to trauma or surgery, receive regular daily doses of opioids.

Opioid naive patients in acute pain are far more susceptible to respiratory depression than patients who have been receiving regularly scheduled opioid therapy for 5 days or more. In addition, opioid naive patients taking benzodiazepines or other central nervous system (CNS) depressents, are more susceptible to opioid-induced respiratory depression. Tolerance to opioid induced respiratory and other CNS effects generally occurs within 5 days of regularly scheduled therapy. In the first few days of opioid therapy, patients should be monitored closely. To prevent respiratory depression, health care providers should carefully monitor all opioid-naive patients for both respiratory status and sedation level. **Healthcare providers should be aware that sedation precedes respiratory depression and that patients will not succumb to respiratory depression when they are awake.** A sedation scale should be used to identify increasing sedation levels. If sedation occurs, the dose of the opioid should be decreased to avoid opioid induced respiratory depression. The incidence of opioid induced respiratory depression can be high when patients are not carefully monitored for increasing sedation.

GUIDELINES FOR MANAGEMENT OF COMPLICATIONS:

Unrelieved Pain	If pain score >4/10 at rest or >6/10 on movement – call house surgeon. Anaesthetist will assist if pain relief cannot be achieved.
Sedation	Sedation precedes respiratory depression. Patients will not succumb to respiratory depression when they are awake or easily rousable. A sedation scale should be used to identify increasing sedation levels.(even at night) If sedation occurs, the dose of the opioid should be decreased to avoid opioid induced respiratory depression. The incidence of opioid induced respiratory depression can be high when patients are not carefully monitored for increasing sedation.
Respiratory depression	Follow chart below. Sedation precedes respiratory depression REMEMBER stimulation and encouragement to breathe may be all that is required for mild cases. For severe respiratory depression artificial ventilation may be required. Consider an arterial blood gas.
Hypotension	If hypotensive (e.g. below set parameters) allow no further opioid (stop PCA) until condition has resolved. Put patient 10° head down, give oxygen at 6L/min, record HR and BP q5-30min (according to condition), notify house surgeon.
Nausea / Vomiting	Follow prescribed antiemetic therapy. If continuing problems with nausea and vomiting, contact Anaesthetist for advice.
Distressing Itch	Ondansetron, Phenergan, Give 40mcg Naloxone IV q30 min prn to a maximum of 120mcg in 3 hours. See opioid spinal form for more details.

			LEVEL OF CONSCIOUSNESS					
			Alert / Easy to Rouse			Difficult to Rouse		Unrousable
Resp Rate			8-10	5-7	<5	>6	<6	ANY
Ensure Clear Airway (consider recovery position)			✓	✓	✓	✓	✓	✓
Stop PCA Basal			✓	✓	✓	✓	✓	✓
No further boluses until within prescribed parameters				✓	✓	✓	✓	✓
MONITOR	Sedation and Resp Rate		Q 1hr	Q 30min	Q 15min	Q 15min	Q 5min	Q 5min
	HR and BP		Q 4hr	Q 2 hr	Q 1hr	Q 15min	Q 5min	Q 5 min
	Pain Score		Q 4 hr	Q 2hr	Q 2hr	Q 1hr	Q 1hr	-
	Oximetry		Q 4hr	Continuous	Continuous	Continuous	Continuous	Continuous
TREATMENT	Oxygen Therapy		If SpO_2 <94%	SpO_2 <94%	Continuous	Continuous	Continuous	Continuous
	CALL	APS/Anaesthetist		✓	✓	✓	**Crash Call !!**	
	Naloxone (NARCA	Titration			✓	✓		
		RESCUE					✓	✓

- **NALOXONE TITRATION:** Make up 0.4mg Naloxone (Narcan) in 9ml Normal saline. Give 1 ml (0.04mg) IV every 2 minutes until patient is easy to rouse with respiratory rate >6/minute
- **RESCUE:** Give 0.2mg Naloxone IV stat and repeat after 2 minutes if indicated
- **PAEDIATRIC DOSE:** 0.01mg/kg (10mcg/kg)

Remember the short half-life of Naloxone. Treatment may need repeating. No opiod to be given within 1 hour of Naloxone except by the attending doctor. Turn off PCA.

Adult Restart Parameters
Heart rate > 50 min
Systolic B/P > 100mmHg
Respirations > 10/min
Breathing is of "normal" depth/rate/volume
Patient is easily roused.
When an advanced analgesic technique is temporarily interrupted due to side effects, monitoring must continue in accordance with the management of complications guidelines.

Paediatric Restart parameters as per Paediatric Morphine protocol.

Analgesic techniques should be restarted as soon as restart parameters are met. Pain relief should not be withheld until pain returns as it may then be difficult to regain control.

SECTION 9

NURSING/MIDWIFERY RESPONSIBILITIES FOR IV OPIOID ADMINISTRATION

1. Every nurse/midwife who accepts responsibility for the care of a patient receiving opioids, regardless of the route, is accountable for the patients' comfort and safety.

2. It is required that all nurses/midwives working in areas where opioids are administered via any parenteral route (IM, IV, S/C) must have generic IV medicine management certification.

3. Wherever possible a registered nurse/midwife who holds intravenous opioid certification should be assigned to care for patients receiving intravenous opioids including PCA. Where this is not possible, e.g. in wards/units where PCA is rarely used, nurses/midwives with generic IV certification may care for these patients. A specialty opioid certificated nurse/midwife should be identified for oversight of the infusion.

4. It is the responsibility of the nurse/midwife assigned to the patient to contact an intravenous opioids certificated nurse/midwife in a timely fashion to deal with the procedures outlined in 6 a, b, and c below.

5. It is the responsibility of the assigned nurse/midwife to ensure that they are aware of:
   - the monitoring requirements (see page 18 and page 54)
   - the management of adverse situations (see page 52-54)
   - the contact details of the certificated nurse/midwife and,
   - how to stop the pump in an emergency.

6. The specialty opioids certificated nurse/midwife overseeing the opioid infusion is responsible for the technical requirements related to the delivery system such as:
   a) changing the syringes and correctly positioning the syringe in the volumetric pump.
   b) use of specific tubing where appropriate.
   c) volumetric pump reprogramming (including giving a bolus dose, or altering the prescription).
   d) ensuring the rate/volume given matches what is left in the syringe/PCA.

7. The specialty opioids certificated nurse/midwife overseeing the opioid infusion needs to take responsibility for overall oversight of the patient eg, check the patient, not just the technical requirements.

8. Whenever responsibility for the patient moves from one assigned nurse/midwife to another, the receiving nurse/midwife must check:
   - the prescription is complete and legibly signed,
   - the settings on the PCA/syringe driver pump match the prescription.
   - medication in the fluid reservoir is labelled as the prescribed drug
   - ensuring the rate/volume given matches what is left in the syringe/PCA

If this is not understood, seek clarification.

SECTION 10

PATIENT CONTROLLED ANALGESIA (PCA)

INTRODUCTION:

PCA is a safe and effective pain relieving strategy for any patient who would require a minimum of 24 - 48 hours of regular subcutaneous/intramuscular opioid injections.

PCA allows small amounts of an opioid to be given intravenously, maintaining blood levels of that opioid within a range effective for that particular patient.

Several variables contribute to the difficulty in predicting the analgesic requirements of an individual patient, for example pharmacokinetic, pharmacodynamic, psychological, cultural and social factors. (Between patients there is a 8-10 fold variation in dose requirement). Individualisation is obviously required to achieve optimal analgesia.

This is achieved by first loading to and then maintaining at the opioid blood level/range for analgesia (analgesic corridor) for that particular patient.

The principle of PCA is that once 'loaded' the patients themselves can titrate their own pain relief by pressing the button of the PCA device. To deliver a preset amount of the chosen analgesic via the patients indwelling IV line which should be enough to be effective but small enough to have no side effects. A timer in the device precludes administration of a further bolus in a preset lockout interval. This interval should be long enough to give the drug time to exert is pharmacologic effect and so prevent inadvertent over dosage. (5 minutes)

BENEFITS:

The advantage of individualised IV pain relief results in less postoperative pain with reduced total drug dosages. While IM prn medication leaves the patient in pain from the moment of demand over the period of delay until the medication is injected to the final delay onset of action of IM medication (up to 35 minutes in some studies), the self-applied IV bolus gives immediate relief. On the other hand overdoses, possible in continuous IV infusion concepts, are more or less impossible, because the sedated patient will not press the demand button.

Further benefits described in multiple studies include greater spontaneous physical activity, fewer nocturnal sleep disturbances, improved early mobilisation, decrease in the duration of postoperative hospital stay and reduced nursing time requirements.

Finally, the patient acceptance is extremely high; patients control over their pain management certainly enhances the effect of the service. Reduction in anxiety and relief from incident pain may in turn enhance analgesic efficacy

GENERAL GUIDELINES:

1. The decision to use PCA should be made preoperatively so that the patient can receive instruction in its use. Obviously only patients able to comply with instructions are suitable candidates for PCA.
   The patient should be instructed in:
   - The rationale of PCA
   - Use of the pump
   - Explanation of safety features
   - Explanation of monitoring eg. pain, respiration, sedation scores
   - Likely duration of therapy

- A Ward Nurse/midwife assessed as competent in PCA procedures should give this instruction. The PCA patient information brochure includes the above and should be given to each patient due to have PCA.

2. Naloxone and an alternative analgesia must be charted:
   - Adult dose:0.4 mg should be readily available
   - Paediatric dose: 0.01mg/kg (10mcg/kg)
3. Oxygen is commonly ordered for the patient. Hypoxia due to respiratory depression, can cause problems more rapidly than hypercapnia.
4. A one way anti-reflex valve and anti-siphon device are part of the PCA extension set. The primary IV infusion line is connected to this so that opioid cannot pass back up the drip line if the IV cannula becomes blocked. This system is primed in the usual way (to purge air from the tubing) before connection to the cannula.
5. A quick guide to the use of the PCA machine is included on page 58 - 61 and should be readily available for reference in the ward.
6. Only registered nurses/midwives with a current IV/PCA Certificate should look after patients using a PCA. *(See Nursing/midwifery responsibilities for opioid administration)*.
7. Only an Anaesthetist may authorise use of a PCA. The anaesthetist must write and sign orders on the PCA Treatment Sheet and MR17 specifying the opioid used and programme prescription. *ie. mg per injection, lock out time,, basal rate ml/hr (usually zero), one hour limit (mgs)*
8. Anaesthetist or Recovery Staff will usually give loading dose of opioid in OT/Recovery Ward.
9. The Prescription is written on and according to Patient Controlled Analgesia prescription form by an anaesthetist.
10. Bags and tubing will need replacing every 72 hours, unless medications have been added to the bag; In this case the bags **must** be changed after 24 hours. This is due to additives change the stability of the solution which could crystallise.
11. Use only bags as outlined below

    MORPHINE 100 mg in 100 ml = 1 mg *I* ml (Available in pre-filled bags from pharmacy).

    If Pethidine is prescribed: it will have to be drawn up as instructed by anaesthetist – currently no prefilled bags,
    for example 100 ml normal saline bag – withdraw all the saline from bag, draw up pethidine 1000mg (approx. 20 mls) then add saline to make bag up to 100 ml (approx. 80 mls) – the concentration will be 10mg / ml.

    Fentanyl 1000mcg in 100mls = 10mcg / ml (available in pre filled bags from PACU and Pharmacy)

12. Only the patient may press the button (apart from when the patient is in PACU, or ICU, or when there are specific written instructions from the anaesthetist).
13. The PCA must be given via PCA set with a Y connector and a non-return valve. IV fluids must be infusing via this line for the duration that the patient is on the PCA.
14. The PCA must be connected directly to the IV cannula / CVL port – extension lines are not to be used between the Y connector and the patient.
15. In line with nurse's / midwife's role in maintaining patient safety, the rate of administration may be reduced by certificated nursing / midwifery staff in consultation with the Anaesthetist. Following verbal authorisation, the action taken must be documented in the clinical notes by the nurse / midwife and the prescription must be amended and signed for within 12 hours.
16. The PCA programme will be checked by two nurses each shift. It must also be checked after each bag change and if the recording sheet needs renewing (such as when completely used/full with recordings). For the latter instance the prescription will also need to be renewed -contact the anaesthetist for this.
17. **Inadequate analgesia (pain scores>4/10) requires review -contact the** anaesthetist**,** on-call House surgeon, or on-call Anaesthetist (after hours).

18. **Patients on PCA must NOT receive any other oral opioid analgesia unless discussed with the anaesthetist**.
19. Any patient on a continuous opioid infusion must be monitored in HDU/ICU and on continuous oximetry
20. ONLY the patient, not relatives, friends or nurses/midwives should activate the device unless otherwise ordered.
21. The use of the PCA should be discontinued ONLY after discussion with the anaesthetist.
22. All pumps will normally be kept in Recovery and must be returned there promptly when no longer required.

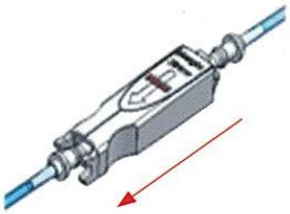
SAPPHIRE INFUSION PUMP QUICK TIPS

Sapphire

Getting Started

V Visual inspection of pack and set
A Arrow on cassette and filter in flow direction
C Close clamps
S Spike the bag

T Turn on the pump
O Open the door
I Insert the cassette and open clamps
P Prime the administration set and program pump
C Connect the patient and press start



DO NOT PRESS THE START BUTTON UNTIL LINE IS PRIMED

Verify Correct Delivery Mode – Top Right Corner

To change delivery mode

International Distributor of:

SAPPHIRE INFUSION PUMP
QUICK TIPS

Sapphire

CLINICIAN INITIATED BOLUS

Press to unlock patient

View/Edit

Clinician bolus >

Enter Password → OK

Set C. Bolus Dose → OK

OK

Confirm changes correct

"Bolus delivery of __ complete"

BAG CHANGE / REPEATING INFUSION

(Pump Message "Infusion Complete")

Mute → OK

Press to unlock patient

Repeat Last Infusion

Review and Confirm Program

If incorrect, press Back and select

New Infusion

OK

Start

REMOVE AIR IN LINE/ PRIME

(Pump Alarms "Air in Line")

Mute → OK

Press to unlock patient

Prime

"Disconnect set from patient before prime"

OK

Pause Prime

(when all air removed)

Quit Prime

Continue → OK

CHECK PATIENT HISTORY

Bolus history >

(from toolbar)

Review or Select History period required

OK → Back

CHANGING A SETTING

Press to unlock patient

Pause → OK

View/Edit

Make changes required and review program

OK → Continue → OK

REVIEW PROGRAM

Press to unlock patient

View/Edit

Review Screen Parameters

OK

Lock

Patient lockout

TURN PUMP OFF

(retain current infusion)

STOP

Press to unlock patient

ON/OFF

OFF

TURN PUMP OFF

(clear current infusion)

STOP

OK

Press to unlock patient

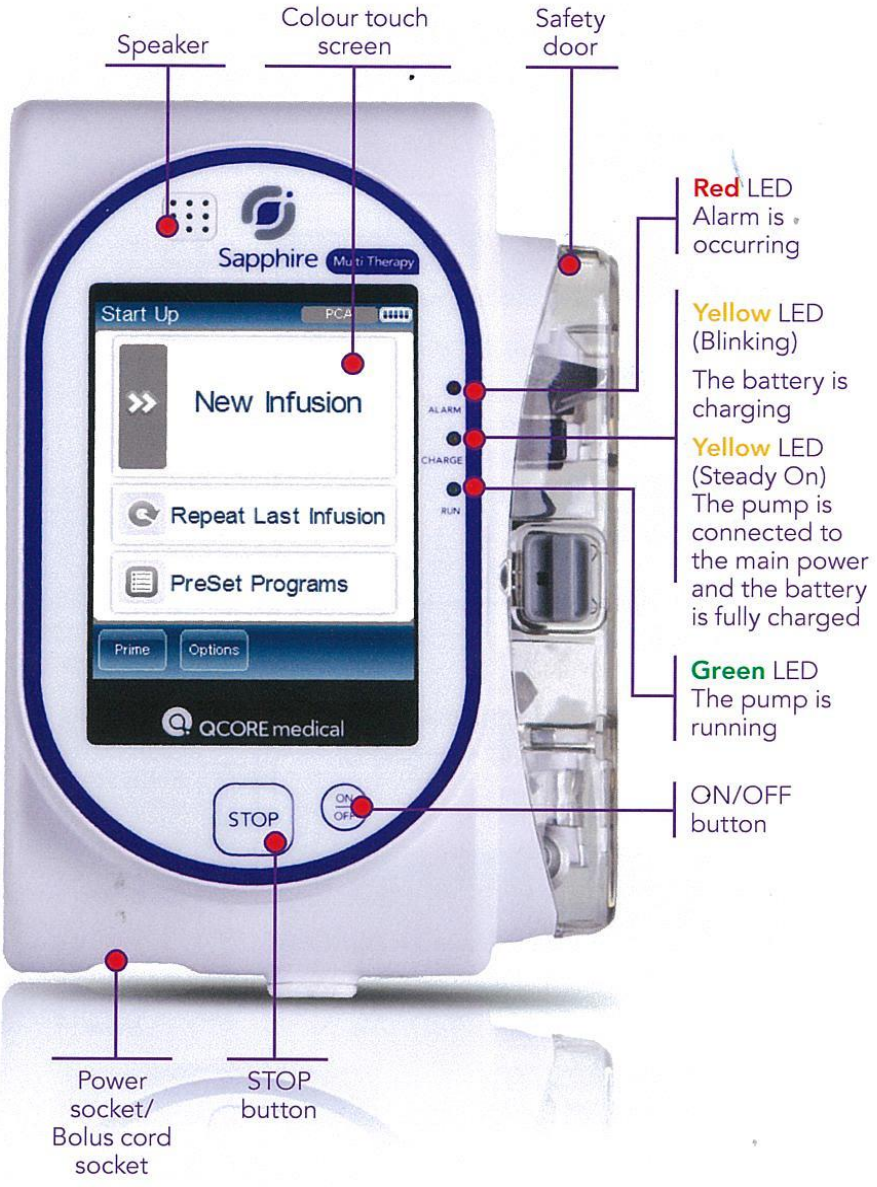
Quit → OK

ON/OFF

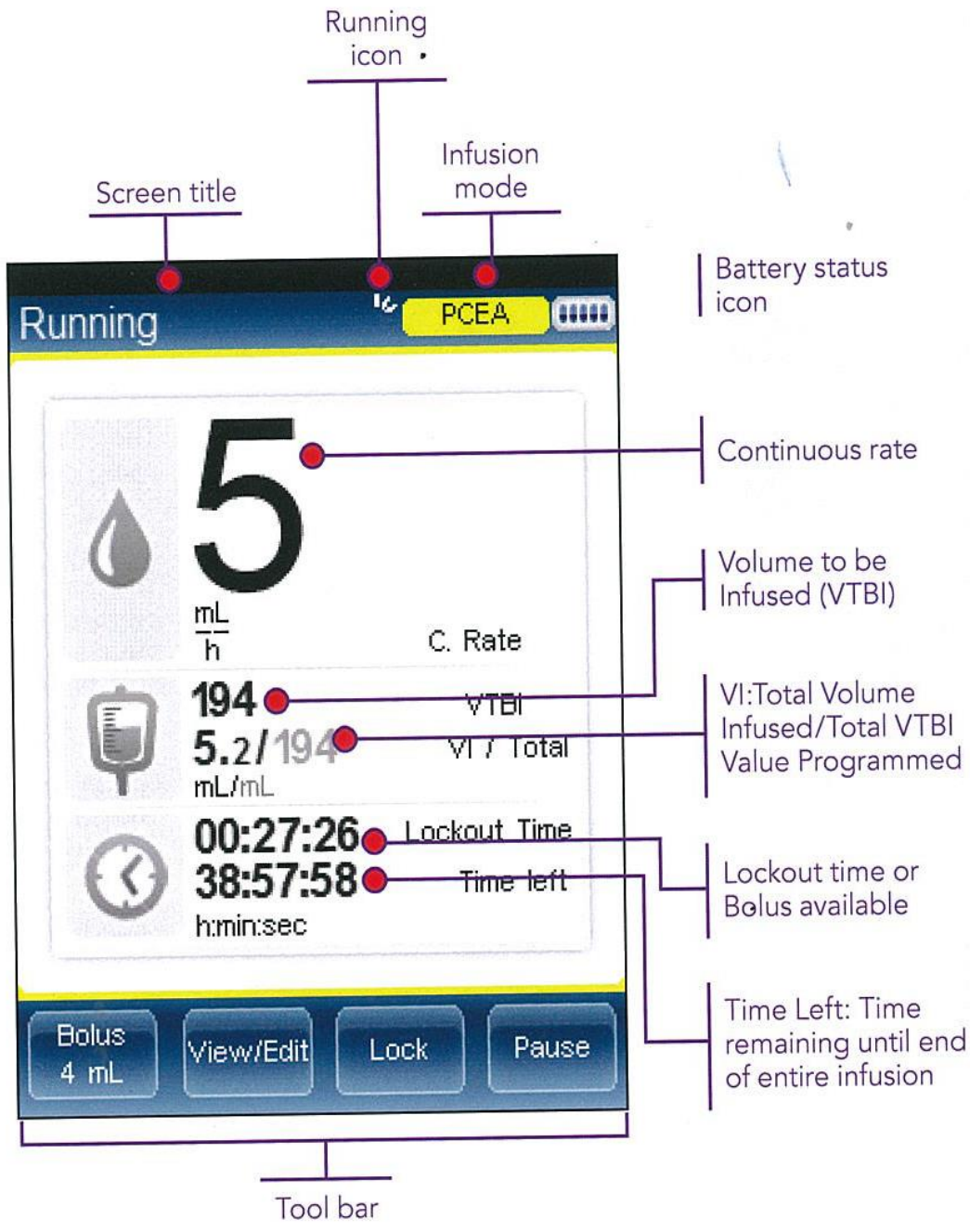
OFF

Sapphire™ Layout

Sapphire

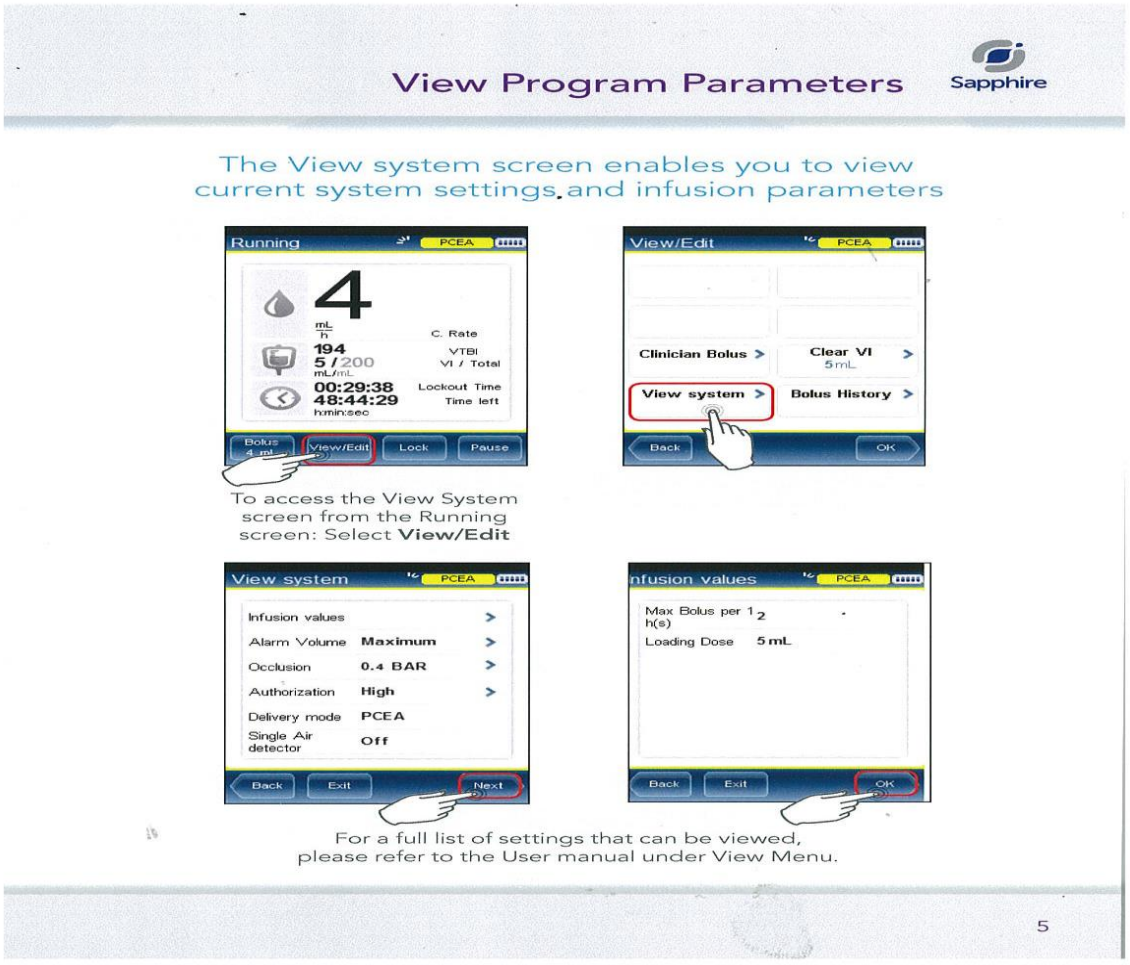


Sapphire™ Screen Layout
Pain Management



Reviewing a Programme (check latest version)

- **To view Program**
- **Press unlock patient**
- **Enter password and press ok**
- **Auth set to high /press ok**
- **Pause infusion /press ok**
- **Press view/edit (VTIB, Bolus, lockout viewed)**
- **Press view system then**
- **Press infusion values**
- **(concentration and max bolus per hour can be viewed)**
- **Press back button twice to return to main screen**
- **Press continue/ok**



PCA Settings

- A standard setting prescribed for the average adult patient will be 1 mg/ml Morphine with a lockout of 5 minutes, no background/basal infusion and an hourly limit of 10 mg
- Pethidine 10mg/ml with an hourly limit of 80 – 100mg
- Fentanyl – 10mcg/ml with a lockout 5 minutes, no basal or background infusion, Fentanyl bolus will be 10mcg with an hourly limit of 120mcg.
- These standard settings do not apply to all patients and have to be adapted to the individual patient by the Anaesthetist.
- **THE PATIENT WILL BE MONITORED REGULARLY ACCORDING TO THE PCA TREATMENT SHEET INSTRUCTIONS (Appendix 8)**.

PCA settings – Hard limits are the configuration settings within the Sapphire Pumps – if the anaesthetist charts a PCA outside of the hard limits then please contact PACU or the electronics department.
The pre-set PCA programmes are a guide, the anaesthetist can prescribe outside the pre-set program and will need to be programmed in as a new infusion.

HARD LIMITS	
CONTINUOUS RATE	0 – 5 mls
DEMAND BOLUS	0 – 5 mls
LOADING DOSE	OFF
BOLUS LOCKOUT	MINIMUM 3 MINUTES
PRE SET PCA PROTOCOL	MORPHINE 1mg / ml
CONTINUOUS RATE	0 mls
PCA BOLUS	1 mg
LOCKOUT	5 MINUTES
HOURLY MAXIMUM	10 mg
PRE SET PCA PROTOCOL	FENTANYL 10 mcg / ml
CONTIONUOUS RATE	0 mls
PCA BOLUS	10 mcg
LOCKOUT	5 MINUTES
HOURLY MAXIMUM	MAXIMUM 120 mcg

PATIENT MONITORING:

1. Baseline recordings: sedation level, respiratory rate, B/P, pulse, oxygen saturation, and pain assessment.
2. Post-administration (i.e. after first use by patient).
    - ¼ hourly for 1 hour
        - sedation level
        - respiratory rate
        - B/P
        - Pulse
        - SaO2
        - pain assessment
    - Repeat all of the above 1 hourly for 4 hours.
    - Thereafter 2 hourly
        - respiratory rate
        - sedation score

    - and 4 hourly

- pulse
- B/P
- pain assessment
- SaO_2
- total drug given recorded in mg or mcg (not in mls)

When a patient is on PCA monitoring, one set of observations must be transferred onto the Tairawhiti Early Warning System (TEWS) chart each shift.

3. The following should be recorded on the PCA observation sheet The necessary information can be obtained from the pumps main screen VI value (Total volume infused) memory via Inj/Att and
   - Total dose received (recorded in mg or mcg)

The anaesthetist needs to assess the patient's pain if it is not well controlled with the PCA and review the prescription every day. **This is not the responsibility of the House officer.**

Any nurse/midwife accepting responsibility for caring for the patient/child is ultimately accountable for practicing according to HAUORA TAIRAWHITI's protocols for IV Opioids/PCA and maintaining patient safety. There are NO exceptions.

DO NOT administer sedatives or oral opioids except by the order of an Anaesthetist.

Possible Side Effects
PCA is a safe technique because:
- The patient self titrates their own medication and will not do so if sedated by high blood levels
- Programmed safety features of the PCA Pump

Strictly regular monitoring and documentation of same by nursing staff.

When using PCA correctly on its own, few respiratory arrests have been reported. However, an additional continuous infusion can theoretically increase the risk of respiratory depression.

Generally additional IM opioids are absolutely contra-indicated.
The most common problem with a PCA is related to operator error in programming the pump, failure to clamp the tubing during bag change, wrong drug, or concentration of drug (eg, Pethidine), or pump use by others (eg, relatives). Due to the potency of the drug in the bag, extreme care has to be taken in changing the bag and programming the pump.

So apart from the patient and pump functions, the key to safety is the monitoring, which must strictly follow the instructions on the PCA Treatment Sheet.

In the rare event of excessive sedation(score ≤3), or respiratory depression (adult ≤ 8 breaths/mim), urgent intervention is required:
- Stop the pump.
- Give 6Lmin 02 via mask.
- Rouse patient, if necessary by painful stimuli.
  If patient rousable -maintain stimulation and encourage regular breathing. Inform anaesthetist.
  If patient not rousable - →**CRASH CALL**
- Assist respirations with airway and ventilation with bag and mask if necessary.
- Administer Naloxone (Adults) 0.1-0.2mg increments every 3 minutes until satisfactory respiratory rate. (Paediatric dose 0.01mg/kg (10mcg/kg)
- *Naloxone 0.4 mg should be readily available and a good idea to have prepared and ready for use*.
- Maintain rousability and breathing.
- Call anaesthetist STAT.

Other Side Effects
Nausea and vomiting should be able to be controlled by the prescribed antiemetic. If this is persistent despite treatment, call the on call anaesthetist for assessment. Antiemetics may be added to the PCA bag if prescribed by anaesthetist.

NURSE ADMINISTERED TITRATION VIA PCA (NCA):

Administration is by registered nurse / registered midwife holding PCA certification, or by a registered nurse / registered midwife with generic IV certification under the direct supervision of the PCA certificated registered nurse / registered midwife.

Only for use in PACU, ICU or with specific instructions from the prescribing anaesthetist.

Criteria for Use
1. Pain is severe and at operative site - if heavy use of PCA discuss with Anaesthetist.
2. Patient meets monitoring parameters.
3. Nurse / Midwife to remain in attendance during and for 10 minutes after completion of titration.
4. Naloxone to be in the room.

Monitoring
Every 5 minutes:
- sedation level
- respiratory rate

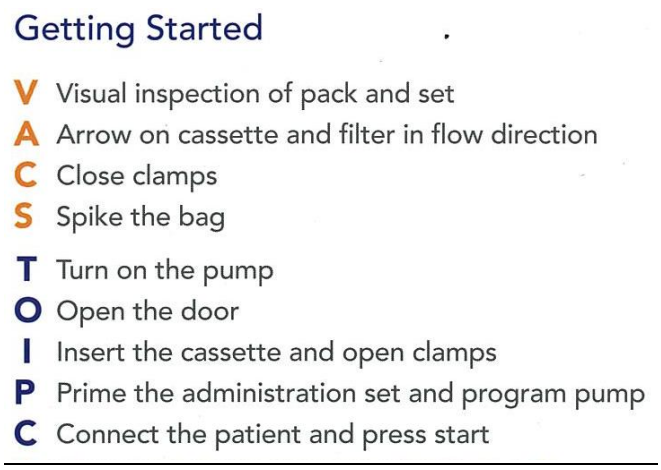
Every 10 minutes:
- pulse
- blood pressure
- pain score

This monitoring is to continue for 10 minutes after titration. If monitoring parameters are not met, no further boluses are to be given.

PCA PUMP MANAGEMENT:

The key required to unlock the PCA Pump is kept with the ward controlled drug keys.

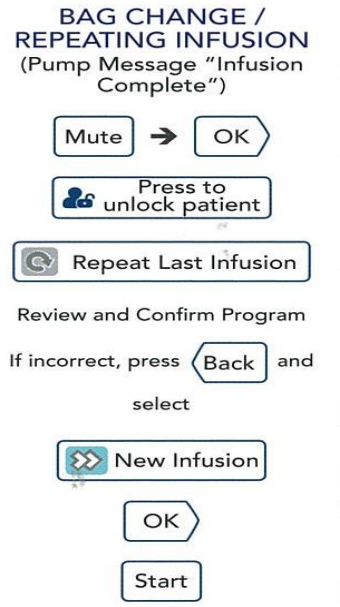
TO SET UP ALARIS IVAC *PCAM* PUMP:
Initial PCA Set-Up



Pump Protocols

There are two preset protocols programmed in these pumps. For safety reasons if at any time the protocol does not match to what is being infused, **check immediately**.. **If you have any doubt, check it out.**

To Change bag: *(When empty and at least every 24 hrs)*
Remember one of the personnel involved in this procedure must hold PCA Certification.



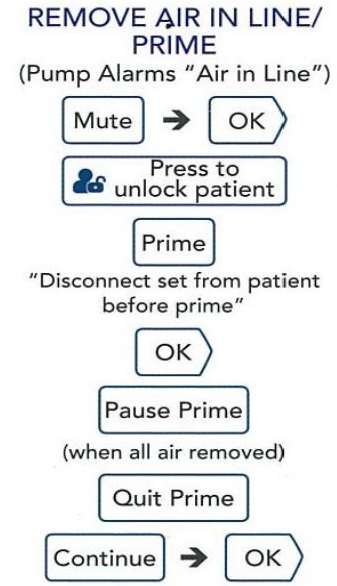
Pausing Infusion:
To pause the infusion and then re-start, in situations where the one hour maximum has been given or for nursing cares – mobilisation, showers or similar. If this is happening regularly, consider reviewing the need for PCA.

1. Assess patient's pain activity, planned and analgesia requirements

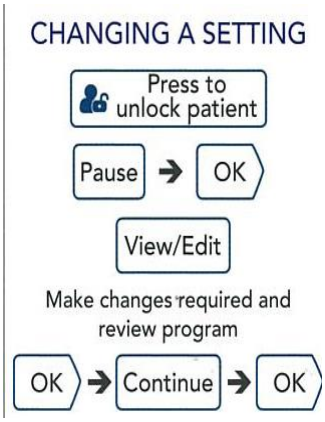
Restarting Infusion:

Air in Line:

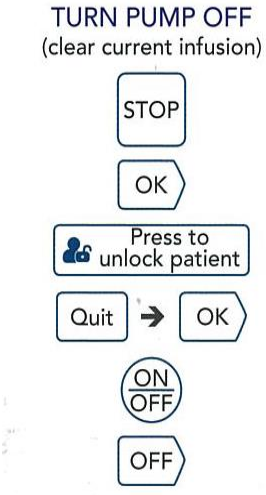
This occurs when the pump detects air in the line



Modifying the Protocol:



To Discontinue PCA:



The Alarms are Triggered When:

1. Air detected in line.
2. Low battery AC power source required – one is kept on each ward
3. There is an occlusion in the system.
4. The pump's internal mechanisms are not working properly.i .e mechanical error, pump fault, flow error etc
5. **A message will be indicated on the screen i.e door open – check administration cassette position and close the safety door.**

DOCUMENTATION: *(PCA Treatment Sheet) check name of sheet/chart*

This is individualised for each patient and contains:

- Patient details.
- Drug and concentration.
- Programme prescription.
- Management of respiratory depression.
- Observations/Infusion recordings section.
- Pain and Sedation Scores.
- Standing Orders especially including frequency of observations.

MOBILISATION:

Patients with a PCA can mobilise with the pump on the stand. Patients should be encouraged to use their PCA as necessary prior to any activity to help to control pain.

CLEANING AND DISINFECTING:

- The exterior surfaces of the pump may be cleaned with cleaning wipes with 70% isopropyl alcohol i.e. Azowipes
- The pump should not be immersed.
- Do not attempt to sterilise by ETO, steam or dry heat.

ROUTINE MAINTENANCE:

See Pump Operators Manual.

Any problems return pump to medical engineering with a note explaining what the problem was.

<u>If there are any queries or problems they should be discussed with the Anaesthetist</u>.

TERMINALLY ILL PATIENTS:

In cases where terminally ill patients require drugs for which the IV protocols stipulate patients be monitored, or that administration of these IV drugs requires specific nursing certification, it is inappropriate to transfer these patients to special units where nurses have the appropriate certification or for monitoring.

In the event of a terminally ill patient requiring therapy to relieve pain, the following process will enable a registered nurse / registered midwife, with generic IV certification to administer intravenous opioids.

The consultant in charge of the patient's care will:

1. Refer the patient to Hospice Tairawhiti services
2. Discuss with the Nurse Unit Manager and/or designate of the ward, the patient's needs and the clinical situation.
3. State in the clinical notes the clinical reasons for deviating from the established protocol.
4. Write in his/her own hand that the patient is terminally ill and requires this medication to relieve pain and distress.

5. Document the prescription on the HAUORA TAIRAWHITI Drug chart or the HAUORA TAIRAWHITI PCA chart
6. Use a Palliative Care standardised drug administration sticker

Drugs Used: Morphine, Pethidine, Fentanyl

Presentation: Morphine as stated
Pethidine as stated
Fentanyl as stated

Infusion Rate: Direct IV injection
Intermittent infusion
Continuous infusion - usually via a volumetric or syringe driver pump, but a PCA pump can also be used.

Prescription: Medical officer, following the above process, will write the prescription before administration occurs.
Prescription details must show:
a) maximum dose/hour
b) dilution
c) time to be given over (it is better to give a range e.g. Morphine 2-5mg IV into 20-100mL of crystalloid fluid given over 10-60 minutes prn)

OR

Morphine 20mg into 100mL of 0.9% Sodium Chloride given IV via an infusion pump at 10-30mL/hour.

Monitoring: Post administration - pain assessment as required to ensure desired comfort

SECTION 11

REGIONAL ANALGESIA

NEURAL BLOCKADE:

Introduction

A recent development in management of acute post-operative pain is to place a thin (+/- 0.5 mm) catheter next to the nerve or nerve plexus (a bunch of nerves grouped together) that supplies the particular part of the body where the pain originates from and to infuse local anaesthetic agents through the catheter.

Neural blockade has been performed for many years, and there is strong evidence to suggest that it has benefits for patient outcome, in terms of reducing surgical stress, morbidity and mortality (Schug, 2000). Individual nerves can be targeted for blockade, depending on the nature of the injury/pain problem. It blocks only a specific territory (that supplied by the nerve). The distal site of action of nerve block implies few "collateral damages" compared to an epidural or the use of parenteral pain killers (acting at the brain level). This is the case with a femoral nerve block. Alternatively, a number of nerves may be selected for blockade, as with an epidural.

A number of other nerve blocks can be performed:

- **Axillary** – lower arm/elbow/hand
- **Intercostal -** fractured ribs
- **Interscalene** – useful for upper arm, shoulder
- **Sciatic** – useful for lower limb pain, e.g. post TKJR, ankle fractures
- **Median -** ulnar nerve blockade for hands
- **Ilioinguinal –** useful for transverse incisions following such procedures as appendectomy and caesarean section.

Blocks can last from 2 – 12 hrs +, it depends on what local anaesthetic was used and the amount infused in.

The more frequently used sites of insertion are:

- femoral (for knee surgery)
- popliteal (for ankle and foot surgery)
- interscalene (for shoulder surgery)
- axillary (for arm or hand surgery)

Other insertion sites possible:

- intra-clavicular (for lower limb)
- paravertebral (indications depending on the level –neck/thoracic/lumbar- of the block)
- psoas compartment (for lower limb)
- median / radial … (hand surgery)

Other techniques used for postoperative pain control are:

- wound bed infiltration
- interpleural catheter – post open gallbladder surgery, post-nephrectomy, fractured ribs
- paravertebral catheter – post-thoracotomy
- stump infusions (sciatic nerve blockade) - following lower limb amputation

BRACHIAL PLEXUS ANALGESIA:

Anatomy

The brachial plexus supplies upper limb motor and sensory function. The roots of the brachial plexus form 3 trunks, each dividing into an anterior and posterior division. The six divisions then join to form cords. Each of the cords has two terminal branches and these are the nerves which supply most of the arm. As well, emerging from the plexus are small nerves – median, radial and ulnar.

Blocking the Brachial Plexus

- *Interscalene blocks* of the brachial plexus block nerves to shoulder and upper arm.
- *Axillary blocks* block nerve supply to the lower arm and hand.

The nerve stimulator is often used for this procedure to elicit parasthesiae. Once the nerve is identified by the nerve stimulator, a catheter is passed into the sheath. This is aspirated to make sure it is not in a blood vessel, then dressed with a transparent occlusive dressing.

Local anaesthetics used for axillary and interscalene blockade include bupivacaine and ropivacaine. If a single shot block is placed, it may last from 2 to 12 hours depending on the LA used.

Regardless of selected method of administration, the local anaesthetics block both sensory and motor nerve fibres, so the patient must be protected against injury whilst the arm/hand is numb.

FEMORAL NERVE BLOCKADE:

Anatomy

The femoral nerve (L2 – L4) runs round the postero-lateral wall of the pelvis, behind the **fascia iliac** lying on the psoas major and iliac muscles. The **femoral artery and vein** lie anterior to the fascia. When the vessels pass behind the inguinal ligament, the fascial sheath draws down around them. The femoral nerve lies behind this sheath and lateral to the vessels in the groin.

Blocking the Femoral Nerve

Femoral nerve block is used generally for surgery or injury to the leg involving the hip or knee.

A nerve stimulator or ultrasound may be used to locate the nerve for positioning of a femoral block/catheter. The femoral artery is palpated as it passes behind the midpoint of the inguinal ligament. A needle is inserted just below the ligament, and lateral to the artery. Typically, 2 'clicks' are felt as the needle passes through the fascia. Once in the nerve sheath, the anaesthetist will perform an aspiration test to ensure the needle is not in the artery or vein and then inject 10 – 20mls of local anaesthetic. An indwelling catheter can be sited next to the nerve and securely fixed, which allows subsequent administration of boluses of LA the duration of the nerve blockade can thus be as long as needed (usually 3 to 4 days, sometimes more – up to several weeks). The practice is to give a bolus of the full strength of the LA for the operation and then to use boluses of a more diluted solution of LA. This method is used at HAUORA TAIRAWHITI but only on rare occasions.

NURSING RESPONSIBILITIES:

Catheter Management

When a catheter is inserted, it is secured and dressed with a transparent occlusive dressing. A filter is attached. The insertion site MUST be checked at least once each shift if an infusion is used, or before any bolus (top-up) is given to look for signs of leakage, inflammation, discharge, swelling, pain. The dressing should be kept intact for the duration of the placement, and the placement site MUST be visible. DOCUMENT in clinical notes the condition of the site.

Giving a Top-up

Top-up's should only be given strictly under the direction of the prescribing Anaesthetist, The local Anaesthetics used are Bupivacaine 0.25% - 0.5% or Ropivacaine 0.2% - 0.75%. Always ASPIRATE the catheter before **slowly** injecting the medicine, to ascertain it has not migrated into the artery or vein.

The following observations must be documented:
Rousability, block level, pain and comfort scores with all vital sign observations documented on chart:

- ½ hourly for the first 4 hours
- Hourly for the following 12 hours
- 4 hourly once stable

If a top-up or bolus change to prescription occurs, change to every 5 minutes for 15 minutes then return to the above criteria.

If there are any concerns, then contact the duty anaesthetist.

EFFECTS, SIDE EFFECTS AND LIMITS:

Effects

CNB blocks the nerve supply to a specific territory for a chosen period of time. When performed with appropriate LA, it usually provides an intense blockade of the noxious stimuli and a moderate (or even none) blockade of the touch stimuli and motor pathway.

What can the Patient Expect?
The particular area of the body that is blocked should be free of pain and is usually totally numb just after the operation.

In the ward, during the continuous infusion of diluted LA, numbness is often observed, sometimes with a sensation of pins and needles (often when the initial dense block wears off)

In Clinical Practice
Neural blockade has proved to achieve very good pain relief after major orthopaedic surgery.
Neural blocks achieves better results than PCA (especially when an early rehabilitation is performed) and can compare with Epidural, as they induce less side effects and require less monitoring.

Side Effects

The incidence of side effects and complications (especially the serious and life threatening ones) is very low with Neural blockade but are the following:

Due to Catheter:

- Haematoma
- Infection (very rare if < 5 days)
- Nerve injury (very rare < 2/10 000)
- Leak around the catheter

Due to LA:

- Systemic toxicity (with neurologic and cardiac signs). Clinical signs of LA toxicity are tingling around the mouth, metallic taste, tinnitus, visual disturbance, numbness in the tongue, altered conscious state (up to coma) convulsions, cardiac arrhythmias.
- Motor blockade (risk of pressure areas and falls)
- Unwanted blockage of adjacent nerves (following interscalene block. A phrenic nerve block and Horner's syndrome can be noted, usually without requiring any specific treatment)

NB: CNB doesn't induce any changes in HR / BP / RR / sedation score.
If such changes are observed, another cause must be sought.

Limits
Neural blocks affect only one nerve territory. Sometimes, post-operative pain goes through several nerves (that why it can be less effective than EPA that blocks many nerve territories at the same time). For example, noxious stimuli after knee replacement go through femoral and sciatic nerve (that is not blocked by a continuous femoral blockade)

In Practice:
- CNB must be integrated in a "balance analgesia program" with systematic administration of paracetamol and NSAIDs (when possible).
- A "rescue" analgesia (IV morphine) must be prescribed and delivered when needed. When this is necessary, CNB allows an important decrease in morphine consumption and related side effects.
- In certain circumstances, a single shot nerve block with long lasting LA can be placed in addition to the CNB to block a painful territory during the first (and more painful) hours (eg. sciatic block in TKR)

Indications:
- Major orthopaedic surgery with intense pain expected to last more than 24 hours
- Orthopaedic surgery with early mobilization scheduled

Contraindications:
- Infection at the block site or septic condition
- Allergy to LA (extremely rare)
- Coagulation disorder

INTRATHECAL / SPINAL / SUBARACHNOID:
A spinal anaesthetic is the administration of local anaesthetic and/or a tiny dose of opioid into the CSF. This is done via a lumbar puncture. It blocks sensory and motor fibres of the bottom, legs and lower abdomen. With larger doses of local anaesthetic, higher blocks are achieved to upper abdomen and occasionally lower chest, with possible respiratory embarrassment.

If a patient has a spinal anaesthetic with the administration of any opioids, then the specific HAUORA TAIRAWHITI spinal record /intrathecal chart MUST be used

Monitoring of spinal anaesthetic includes:
Heart rate, blood pressure, respiration rate, rousability, pain and side effects (itching, nausea or vomiting and sedation) to be documented on the patients observation chart:
- ½ hourly for first 4 hours- then if stable
- 2 hourly for the next 24 hours
- Revert to ½ hourly for four hours if IV or SC Morphine is given

Common side effects and management of intrathecal opioids are:
- Pruritis / itching.
- Early Ondasetron and Dexamethasone intra-operatively seems to reduce pruritis.
  - If ongoing and severe, consider further doses of Ondansetron, Phenergan, Naloxone as per intrathecal spinal monitoring form.
- Delayed respiratory depression.
  - Respiratory depression can be late in onset and need to be treated with Naloxone as per intrathecal spinal monitoring form if indicated.

- Urinary retention.
  - Void bladder before coming to theatre if indwelling catheter is not part of the normal peri-operative procedure.
  - Reduce intra-operative fluids.
  - Consider draining bladder at the end of the operation.
  - Catheter if not able to pass urine and in discomfort
- Nausea and vomiting.
  - Treat with further anti-emetics as per Morphine spinal monitoring document
- Drowsiness.
  - Observe closely for respiratory depression and treat with Naloxone if necessary
- Constipation.
  - Consider laxatives (Coloxyl and Senna, or Kiwicrush) in patients receiving intrathecal Morphine.

Possible complications of the spinal procedure are generally rare but should be considered as serious and the Anaesthetist should be alerted at the earliest opportunity.

- Post Dural tap headache.
  - Significant ongoing headache, normally frontal and postural (Improved or absent in supine position), nausea and visual changes
- Epidural haematoma.
  - They may cause pressure on the spinal cord or cauda equina, which may present as pain, muscle weakness, or bladder and bowel dysfunction after spinal anaesthetic should have worn off. The diagnosis may be made on clinical appearance and time course of symptoms. It usually requires MRI scanning to confirm. The treatment is surgical decompression.
- Spinal infections.
  - Meningitis early, spinal abscess late.
- Meningism is the triad of nuchal rigidity (neck stiffness), photophobia (intolerance of bright light) and headache
- Sepsis signs.

DO NOT administer sedatives or oral opioids except by the order of an Anaesthetist.

SECTION 12

EPIDURAL BLOCKADE

Currently this modality is used most commonly for obstetric and postoperative analgesia. Less common applications are for trauma, pre-amputation and other selected types of chronic pain.

Epidural analgesia can provide intense, prolonged pain relief, particularly for patients undergoing major surgical intervention, and caesarean section. Likewise it has a place in the treatment of chronic pain, such as that experienced and suffered by cancer patients.

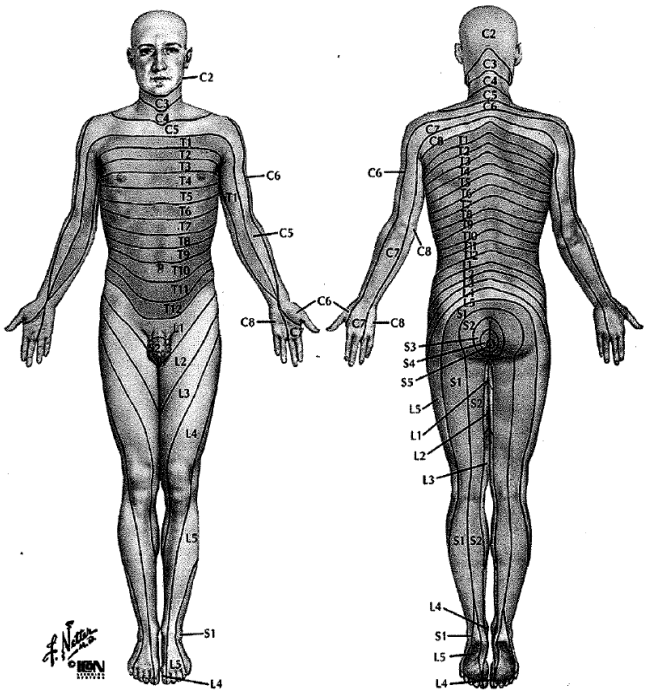
This is an infusion technique supplemented by top-up boluses if necessary. Patient controlled epidural analgesia (PCEA), where a basal rate of infusion is delivered and the patient is able to self-administer bolus doses following the same principles of PCA.

Integral to the success of epidural analgesia is the placement of the catheter in the epidural space at a level as close to the mid dermatone for the surgical incision as possible.

This can be performed at four levels:

- Cervical
- Thoracic
- Lumbar
- Caudal

and has varying physiological effects involving respiratory, cardiovascular and neuroendocrine function depending on the level used. Placement of high thoracic or cervical epidural pose a greater risk for respiratory embarrassment and complications and these must be managed in intensive care.



Administration of local anaesthetic agents in sufficient volume will interrupt the passage of sensory, motor and autonomic impulses in both dorsal and ventral spinal roots.

The early use of epidural analgesia was limited to these local anaesthetic drugs. However, increased understanding of the physiology of epidural neural blockade and the discovery that specific spinal receptors existed and that they modulated pain, lead to epidural (and intrathecal) administration of opioids for the relief of pain.

Benefits of this Technique

In contrast to other forms of pain relief, epidural analgesia is able to (or has the potential to) provide almost, if not complete relief after major surgery (Cousins & Bridenbaugh, 1989).

Given via the epidural route there is a lower opiate dose requirement and consequently some reduction of opioid related side effects. Decreased sedation and a more rapid return to pre-surgical levels of consciousness as compared to systemic opioids are acknowledged benefits (Macintyre & Ready, 2000).

Patients can mobilise earlier, are more able to co-operate in effective physiotherapy which all leads to decreased incidences of pulmonary and vascular complications. It is also suggested that it improves graft flow in vascular surgery patients through sympathetic blockade.

There is earlier return of bowel function and a decreased stress response in patients having epidural analgesia, facilitating earlier enteral feeding. With the benefits of better pain relief as well as earlier nutrition and earlier mobilisation, patients' convalescence is more rapid.

SPINAL ANATOMY

Relevant to our discussion of epidural analgesia are the following:

Epidural Space

This is located between the spinal dura mater and the vertebral column itself. It extends from the foramen magnum to the lower border of the second sacral vertebra. It is widest in the lumbar region, being approximately 5mm deep. This potential space shrinks with age.

The vertebrae themselves are held together posteriorly by short tough ligaments. [See Diagram 1]

Diagram 1

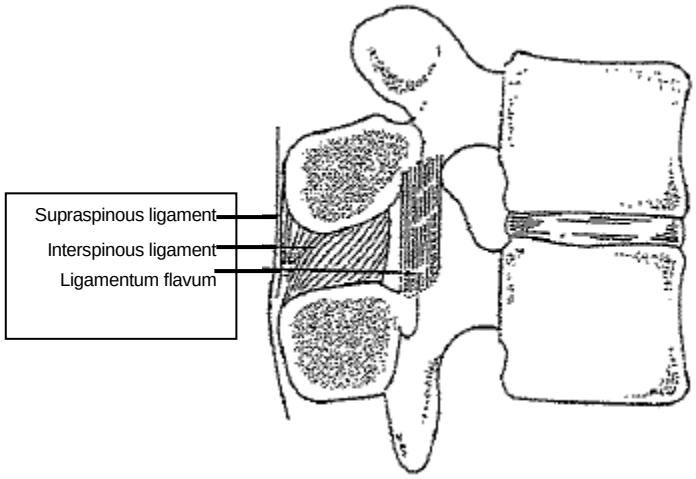


Figure 15.5 Diagram illustrating the ligaments

In reality, the epidural space is a potential space, occupied by the internal vertebral venous plexus, epidural fat, spinal nerve roots and lymphatics. The amount of epidural fat increases in pregnancy and in obese people.

The epidural fat acts as a "cushion" for the contents of the spinal canal. It is also highly vascular and constitutes an important pharmacological depot for injected local anaesthetics plus opiates such as fentanyl.

The average distance from the epidural space to the skin surface is variable, but generally around 5cms and when checking catheter placement, this needs to be considered.

Access to Epidural Space

Techniques for penetrating the epidural space are determined by the anatomical peculiarities of the spine at its various levels. The needle is directed in a median (midline on the spinal column) or paramedian (to either side of the spinal column) plane between the spinous processes.

As the needle is being advanced through the tissue and ligaments surrounding the epidural space, the anaesthetist feels distinct resistance to the attempted injection of air or saline. When the needle enters the epidural space, there is what is termed “a loss of resistance” (LOR).

Other Anatomical Considerations

The spinal cord is protected by both the bony vertebral column and three connective tissue coverings, called the meninges. We have referred to the **dura** already; the other meninges are the **arachnoid** and **pia mater** which directly covers the spinal cord.

For the purposes of our understanding of epidural analgesia, it is important to remember that puncture of the dura mater will probably mean puncture of the arachnoid mater too, since they are in close proximity to one another.

The subarachnoid space contains the cerebrospinal fluid (CSF) which means that puncture of the above can potentially lead to leakage of CSF, or contamination of this fluid.

Furthermore, when the epidural catheter is left insitu, there is a potential for the tip to migrate through the dura and arachnoid maters. The result can be infusion of epidural solution into the CSF. This would result in a high or dense block. Although extremely rare, it can happen, so vigilance in care of the patient is essential.

In adults, the spinal cord terminates at L1/L2 so although local anaesthetic (LA) at L3/4 will block nerve roots to the knee, for example, corresponding opioid receptors will be higher. This fact needs to be considered when placing an epidural catheter.

For epidural analgesia to be the most effective, the catheter needs to be sited at the level that corresponds with the mid-dermatomal level of the surgical wound/site of trauma/pain problem.

PHYSIOLOGICAL EFFECTS:

Changes to the sympathetic nervous system, and particularly the autonomic nervous system, result in specific effects on cardiovascular, temperature and, in women in labour, physiological response during second stage of labour. Many of these effects are related to the drugs used, and are detailed later in this section.

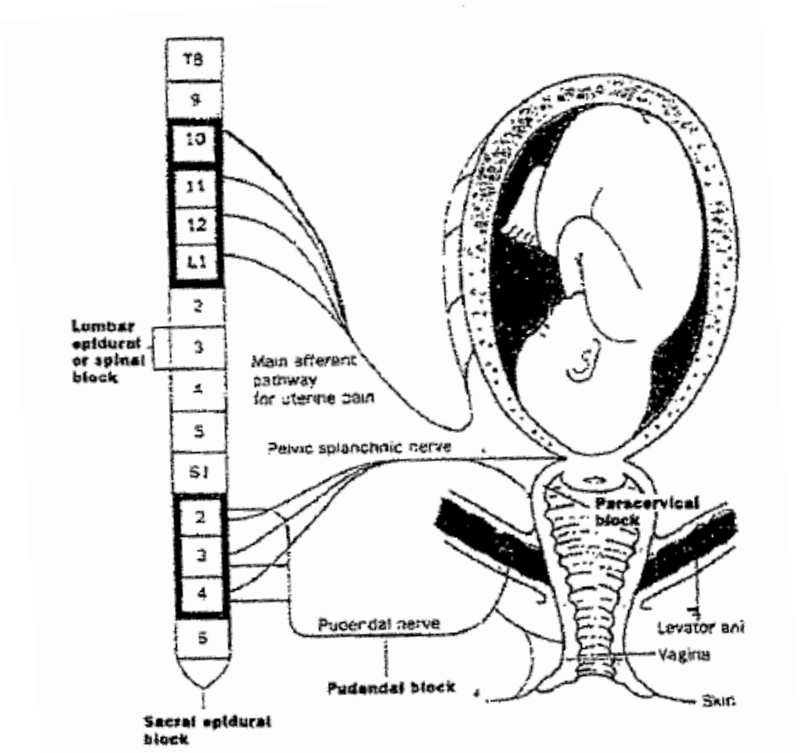
Effects on temperature

Shivering is a side effect of epidural analgesia (but also of anaesthesia without epidural), for reasons that are not entirely clear. It is not usually a problem. It may be that cutaneous vasodilation of lower limbs due to sympathetic blockade increases rate of heat loss when external environment is cold. However, if the external environment is hot, due to absence of a sweating response of lower limbs, a resulting rise in body temperature is likely. Another explanation is that it is due to stimulation of the thermoreceptor in the spinal cord when cold local anaesthetic is injected.

Second stage of labour response

Normally, parasympathetic nervous system increases oxytocin reflexles at full dilatation (Ferguson's reflex). With an epidural, this reflex is blocked; therefore there is less force with uterine contractions at full dilatation. As a result, the woman may need syntocinon augmentation to assist with contractions.

(See HAUORA TAIRAWHITI Epidurals in Labour Workbook 2008 for more information on this)



NURSING RESPONSIBILITIES IN MANAGING EPIDURALS

(Midwives: See HAUORA TAIRAWHITI Labour Epidurals workbook)

Pump Set Up and Administration

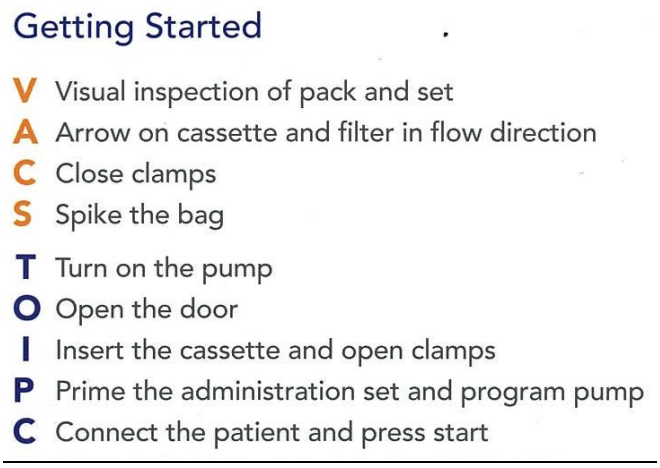
All Epidural analgesia infusions delivered at HAUORA TAIRAWHITI are administered via a Sapphire Infusion Pump, this pump is designed to help the patient to become mobile as soon as possible. The pump can be programmed to deliver an infusion and boluses can be delivered by way of PCEA or clinician administered. Clear step by step programming instructions, key and code entry, plus a lockable infusion bag cover make the pump secure and inaccessible.

Equipment Required

- Sapphire Infusion pump, pump keys, security code and instruction booklet
- epidural administration set
- Antibacterial filter should be attached to patient
- Epidural prescription and medication
- 2.5ml syringe
- Epidural stickers to label line

Pump Set Up

Depending upon prescription (infusion only or PCEA) programming will vary. The programming guide should always be available as a reference.



Monitoring

The 'acceptable' parameters for block level, blood pressure, pulse and respirations MUST be documented by the anaesthetist at the time of insertion of the epidural. HAUORA TAIRAWHITI epidural infusion record must be used for all epidurals. It must contain details of the infusion and prescription, and insertion of the catheter. The nurse must view the insertion site and check catheter position at skin at least **every shift.**

Rousability, block level, pain and comfort scores, with all vital observations – blood pressure, pulse, respiration rate and temperature, to be documented on chart:

- ½ hourly for first 4 hours
- Hourly for the following 12 hours, then if stable
- 4 hourly
- If a top-up/ bolus or change in prescription occurs then observations every 5 minutes for 20 minutes then return to above criteria,

When the infusion is commenced in maternity, or on a ward, initial observations are every 5 minutes for 30 minutes then return to the above criteria.

Monitor bromage and sedation scores, watch for side effects and follow the instructions and standing orders documented on the recording chart.

It is important that signs and symptoms of the complications are responded to in a timely fashion in order to avoid more serious complications and minimise distress to patients. Begin with base line observations

Reportable problems include:

- Numbness/tingling in the fingers or arms
- Bleeding or pain at epidural site
- Oxygen saturation <92% or difficulty breathing
- Urinary retention
- Inadequate pain relief

NB. For hypotension problems due to high epidural block, first line management includes:

- Head down tilt/ Elevate feet
- Increase IV fluids
- IV Ephedrine
- Oxygen
- **CALL the Anaesthetist**

Block Level/Changes in Sensation and Motor Function
Dilute concentrations of local anaesthetic solutions cause some loss of sensation to touch and temperature. These sensory losses are distributed over the dermatomes affected by the local anaesthetic. Sometimes if the epidural catheter is sited at the lumbar level, the patient may also experience some degree of motor blockade, though this is not usual.

A dermatome is an area of skin (cutaneous area) supplied by the sensory axons of a single spinal nerve. The entire body surface area is covered by sensory receptors and can be likened to a "map of dermatomes".

With the use of local anaesthetics an assessment of changes in skin sensation (level of block) is an integral part of patient monitoring. Should the level of the block become too high, patient safety is potentially compromised. In the first instance, it is likely that the patient's blood pressure and possibly heart rate would drop because of the sensory effects to the cardiac sympathetic chain.

Furthermore, skin and pressure area care becomes of paramount importance in these patients with sensory, and possibly some degree of motor loss. ***Strict attention to pressure areas such as heels and sacrum is mandatory for all patients receiving epidural analgesia.***

No hot water bottles are to be given to patients with epidurals.

Motor assessment should be done using the Bromage scale on the Epidural Infusion Record chart:

0 = able to move / lift legs freely
1 = unable to lift legs off bed / can bend knees
2 = unable to bend knees/can move ankles and toes
3 = unable to move legs

NB If a patient reports that he/she is unable to move their legs when previously they had been able to, please consider that the epidural catheter may have migrated in towards the intrathecal or subarachnoid space. This needs urgent management, so stop the epidural and contact the anaesthetist IMMEDIATELY.

Site Care/Dressings/Tubing
Infection at the site of an epidural catheter is relatively uncommon, however it can happen. Some studies report rates of between 4-20%.

It is therefore important to monitor the epidural catheter insertion site, once a shift, for any signs of localised inflammation, pain, or purulent discharge. If the catheter insertion site is not visible it must be redressed using a sterile technique.

Should you suspect a site infection, or the patient develops back pain or a lump at the catheter insertion site, stop the infusion, check the site and notify the Anaesthetist for advice.

Best practice guidelines indicate epidural tubing is changed at 72 hours – although as the epidural infusion is usually stopped before that time frame tubing changes are rarely done in this instance. (Waikato Pain Management)

Safety
As the RN/RM holding certification for epidural management, you are responsible for the patient's care and this includes their safety whilst receiving epidural analgesia.

All patients having epidural analgesia **MUST** have a functioning intravenous cannula in situ in case of emergency situations when IV access is imperative.

Current luer locks connect to both the epidural filter and to intravenous cannula with potential for catastrophic consequences should epidural drugs be given intravenously.

You **must** know how to verify catheter placement in the epidural space in the event of a catheter migration into the subarachnoid space in other words, in emergency situations only. In this situation, you MUST contact the Anaesthetist for management advice.

If advised to do so, attach a 2.5ml syringe on the bacterial filter and attempt to aspirate the catheter. Essentially no fluid should be aspirated. However if you do withdraw some fluid, say around 1ml chances are you are aspirating CSF.

Should this be the case, follow instructions from the anaesthetist.

NB: Women in labour with epidurals MUST NOT be left alone for twenty minutes following administration of a top-up

Infection Control
Patients with epidural catheters are at an increased risk of infection with potentially catastrophic results.

- The insertion site must be inspected every shift, leakage or drainage around the site must be brought to the attention of the Anaesthetist.
- Strict handwashing and aseptic technique before accessing the closed system in any way. This applies to making up the drug solution (when the need arises).
- Do not touch the immediate connections next to the bacterial filter.
- Minimise contact with ampoule necks (as these may be touched by aspirating needle).

Management of Disconnection (between filter and catheter)
If you find a disconnected catheter, unless you actually witness it happening, which is unlikely – i.e. the epidural catheter has become disconnected from the filter – wrap the tip of the catheter in a sterile paper guard or gauze, and seek immediate assistance from an anaesthetist.

Removal of Epidural Catheter
Do not remove epidural catheter without the Anaesthetist order to do so, the catheter can be safely left in place for up to 6 hours after stopping infusion. Catheter must not be removed within 12 hours after normal prophylactic anticoagulant dose has been administered, wait at least 2 hours after removal of epidural catheter prior to administering the next dose.

Consult with anaesthetist if other abnormal clotting profiles are possible or co-exist. See below.

Epidurals and Anticoagulants
Anticoagulants inhibit blood clotting so that if an epidural vein is damaged and bleeds, a haematoma may develop. This haematoma can put pressure on the spinal cord causing pain and paralysis (which can be permanent if not corrected quickly!).

The most likely time for haematoma formation is during or soon after insertion and/or removal of the epidural catheter, but can occur at other times also. Catheters can move or dislodge.

Agents that inhibit clotting include:

Aspirin/NSAIDs

- Affect platelets
- On their own are low risk with an epidural where an indication exists to continue them. i.e. risk vs benefit.

Low Molecular Weight Heparins (Clexane/fragmin)

- are not to be given at least 12 hours prior to insertion or removal.
- If therapeutic dose given wait 24 hours pre insertion and removal of epidural catheter.
- If blood present during insertion of epidural wait 24 hours post dose prior to removing catheter.
- Wait 2 hours post insertion or removal to recommence Clexane or warfarin dose.

Heparin

- s/c not before insertion or 6 hours prior to removal
- infusion not before insertion, and STOP 6 hours prior to removal – Check APTT (this includes dialysis patients) and notify Anaesthetist

Warfarin

- do not give to epidural patients at all
- need INR < 1.4 for 24 hours before insertion/removal

NB: If the epidural catheter falls out within the stated periods above, notify Anaesthetist.

NB: Giving anticoagulants after epidural catheter is removed – should wait a minimum of 2 hours to administer a dose of anticoagulant.

SECTION 13

EPIDURAL PHARMACOLOGY

The two main groups of drugs used for epidural analgesia are local anaesthetics [LA] and opioids. They can be used either alone or in combination depending on the individual pain situation. Furthermore, depending on catheter placement, certain combinations are potentially more advantageous than others. Together, the combination of LA and opioid offers more comprehensive analgesia than either drug alone.

Whatever drug is used, i.e. local anaesthetic or opioid, the medication infused **must** be **PRESERVATIVE FREE**, the reason being that preservatives may be neurotoxic with the potential to cause severe spinal cord injury.

OPIOIDS

Theoretically, the more lipid soluble (lipophilic) drug, the more rapidly diffusion occurs across the epidural space to the opioid receptors in the spinal cord. However, more is bound to epidural fat and is absorbed systemically. With rapid diffusion fewer drugs are available to migrate via the CSF to the brain causing unwanted side effects.

Most commonly used opioids are fentanyl, morphine and pethidine. *See table below for some guidelines re onset of action and duration of action of the opioids.*

Opioid	Onset of Action	Duration
Morphine	30 – 60 mins	6 – 24 hours
Fentanyl	10 – 15 mins	4 – 5 hours
Pethidine	10 - 20 mins	4 – 6 hours

Morphine is more water-soluble (hydrophilic) than fentanyl and prone to retention in the CSF. Furthermore, unlike fentanyl, which essentially "stays put" at the segmental level of administration and has a rapid vascular clearance because of its lipid solubility, morphine and pethidine will migrate to higher levels.

In the CSF, morphine particularly, will travel towards higher brain centres, e.g. the respiratory centre in the medulla, with side effects more apt to occur as a result.

However the ability of morphine to remain in the CSF does offer advantages such as the following:

- prolonged duration of analgesic action
- higher analgesic potency

Pethidine is used commonly and although an opioid, does have some LA effects such as an ability to cause symptomatic hypotension due to the sympathetic vasodilation.

LOCAL ANAESTHETICS

All local anaesthetics have a similar mode of action however they will differ to a greater or lesser extent in regard to:

- potency
- onset time
- duration of effect
- toxicity
- degree of motor block
- cost

The choice of drug will be mainly influenced by the individual patient's requirements as well as anaesthetist preference, but the three local anaesthetics you will see used are:

- Lignocaine
- Bupivacaine
- Ropivacaine

Mode of Action

Local anaesthetics cause a reversible block to the conduction of impulses along nerve fibres. In order for impulses to travel along nerve fibres, there is a wave of depolarisation followed by repolarisation.

Local anaesthetics act by preventing the changes in the cell membrane which lead to depolarisation and thus block impulse conduction. They achieve this by preventing the sodium ions from moving across the cell membrane into the cell, thus keeping the cell membrane in a polarised state.

Onset and Duration of Effect

Most local anaesthetics work very quickly, as a trip to the dentist demonstrates! Duration may vary between 30 minutes to 6 hours or longer, depending on the drug and dose given.

Obviously for purposes of epidural analgesia where infusions are continuous, the effects of the local anaesthetic may take even longer to "wear off".

Local Anaesthetics [LA] Toxicity

This should not occur using dilute LA agents. Staff responsible monitoring or administering the epidural must be aware of signs of LA toxicity that may result from mistaken IV due to migrated catheter. Signs and symptoms of central nervous toxicity are seen earlier than cardiorespiratory toxicity. Toxicity is most likely to occur at the time of top-ups, always draw back on the bacterial filter to check that **NO** fluid can be aspirated, if any blood or fluid is visible call the anaesthetist.

Local anaesthetic toxicity can be divided into 2 major categories: allergic reactions and systemic toxicity with effects on the CNS and cardiovascular systems:

1. **Allergic Reactions**
   Allergic reactions are extremely rare, but such reactions may be manifested by pruritus, urticaria, bronchospasm and culminating in anaphylaxis.
2. **Systemic Toxicity**
   In the context of epidural analgesia, systemic toxicity is most likely to occur with inadvertent intravenous injection, especially of boluses of LA, or tissue injection of excessive doses. Toxicity leads to CNS and cardiovascular system responses.

CENTRAL NERVOUS SYSTEM EFFECTS:

- light headedness
- tinnitus
- numbness of mouth and tongue
- visual disturbances
- irrational behaviour and speech
- muscle twitching
- generalised convulsions
- Eventually, generalised CNS depression occurs with respiratory depression followed by respiratory arrest.

CARDIOVASCULAR SYSTEM EFFECTS:

This is due to slowing of conduction in the myocardium, myocardial depression and peripheral vasodilation.

Signs/symptoms include:

- hypotension
- bradycardia
- cardiac arrest

MANAGEMENT OF EPIDURAL – CONSIDERATIONS:

Management of a patient with an epidural is based on an understanding of the physiological changes that occur as a result of the epidural and the medications used.

For midwives, please refer to HAUORA TAIRAWHITI epidurals in labour workbook, and attendance at the HAUORA TAIRAWHITI midwifery specific workshop is a requirement.

Contra-indications for use of epidural analgesia include:

- patient refusal
- localised or systemic infection
- hypovolaemia/hypotension
- coagulopathies (placental abruption, low platelets)
- structural deformities or invasive disease in spine
- neurological disease/deficit
- cardiac valve abnormalities, where imperative to avoid hypotension
- inadequate trained staff or facilities
- This technique is used cautiously for patients with a known head injury, unless managed in ICU.

COMPLICATIONS:

These fall into 2 categories:

- those that occur as a result of epidural catheterisation
- those that are caused by the drugs given via the epidural route

1. Complications of the Epidural Technique

Epidural Haematoma

When?

- after insertion
- after removal

Symptoms:

- back pain
- parathesiae ->paralysis

Treatment:

- call Anaesthetist IMMEDIATELY
- need MRI
- need urgent surgery within 6 hours

Epidural Abscess

When?

- 2-3 days after insertion and up to 2-3 weeks after removal

Symptoms:
- back pain
- parathesiae ->paralysis
- fever -Temp > 38 C

Treatment:
- call Anaesthetist IMMEDIATELY
- need MRI
- need surgery within 6 hours

Trauma to Spinal Cord or Nerve Roots
When?
- at time of insertion but persisting is uncommon and usually resolves
- Watch for abscess

Meningitis
When?
- can develop up to several days after insertion

Symptoms:
- fever
- headache
- neck/back stiffness

Treatment:
- notify Anaesthetist IMMEDIATELY

Dural Puncture
- Dural puncture headache
- Inadvertent subdural drug administration
- Often delayed onset (usually 24 hours after epidural insertion)

Symptoms:
- postural headache
- frontal or occipital stiffness
- bilateral
- photophobia/nausea

Treatment:
- bedrest supine
- encourage fluids – IV, oral
- caffeine
- notify Anaesthetist
- blood patch (or saline) if symptoms persist

Localised Site Infection
When?
- at any time after insertion, risk increases each day catheter left insitu

Symptoms:
- localised tenderness, inflammation, swelling, exudate
- fever

Treatment:

- notify anaesthetist urgently for advice –
- >stop infusion and remove catheter,
- take swab if exudate.

2. Complications Caused by Medications

These fall into two categories:

- local anaesthetics
- opiates

a) Local Anaesthetics

Because of their mode of action (see "Pharmacology") these agents can cause the following side effects (among others).

Hypotension - can be caused for two reasons:

- A reduction in the level of circulatory endogenous catecholamines that have been raised in response to the patient's pain. As the pain is relieved, the sympathetic response subsides and can lead to a fall in blood pressure, especially if there is an underlying fluid volume deficit.
- Direct action by LA's to block sympathetic nerve fibres in the same way in which it blocks pain – conducting sensory nerve fibres. The degree of sympathetic blockade is dose dependent, thus a higher concentration of local anaesthetic and accompanying high infusion rates can lead to hypotension - particularly postural hypotension.
- Pregnant women are particularly susceptible to supine hypotension.

NB: A sustained period of hypotension due to hypovolaemia can potentially put the patient at risk of Acute Renal Failure. Early diagnosis and action is imperative.

Management of Hypotension Suggestions:

Minimise effects by ensuring situations of hypovolaemia are responded to with appropriate management. This may include any or all of the following:

- Stop epidural infusion,
- obtain medical assistance,
- elevate bed-end,
- administer oxygen,
- fluids,
- vasoconstrictors

NB: Deaths have occurred where hypotension has inappropriately been attributed to epidural analgesia and internal haemorrhage overlooked.

- Close monitoring of blood pressure, pulse, urine output.
- Check for postural blood pressure changes, especially prior to first ambulation.

Leg Weakness

Caused by motor nerve blockade as well as sensory nerve block. The incidence is highest when the lumbar-sacral nerves are involved. Patients report "heaviness" of the lower limb(s) and some will even report numbness of the leg.

Also remember that these patients are more susceptible to pressure sores with motor block and lower limb numbness.

NB: A rapid onset of motor weakness is an indication that the epidural catheter may have migrated across the dura into the subarachnoid space and the medication is infusing into CSF (intrathecally).

Management of Motor Blockade Suggestions:

- Assess motor function along with monitoring of other vital signs by asking the patient to wriggle their toes and lift each of their legs
- Any sudden onset of motor weakness, stop infusion and notify Anaesthetist immediately
- Caution with mobilisation - take it slowly

Urinary Retention

Sensory and motor blockade occurs in the first 24 - 48 hours

Remember because of sensory block, a distended bladder does not cause discomfort or urge to void and it is important that the bladder is not left to become over distended.

Management of Urinary Retention Suggestions:

Most patients with epidurals have an indwelling catheter in situ. If they don't, assess ability to pass urine and take action to avoid urinary retention.

For women in labour, encourage to pass urine prior to epidural insertion/top-up. Continue to assist woman to void regularly thereafter and discuss option regarding the use of bed pain and/or catheterisation (see HAUORA TAIRAWHITI labour epidural workbook).

NB: If patients have not passed urine within 4 hours (6 hours MAXIMUM), they need a careful assessment of their bladder either by palpation or through the use of the bladder scanner.

Unpleasant Parathesia

Sometimes tingling or numbness can be experienced in relation to the use of LA. It will resolve with discontinuation of the infusion.

b) Opioid Analgesics

Respiratory Depression

Associated with morphine, occurs at two distinct intervals, although all opioids can cause respiratory depression.

An early depression occurring soon after administration i.e. within 1 hour reflects absorption into the systemic circulation and thence to the brain.

A later, more insidious "delayed onset" depression may occur after 6 - 12 hours, but may even occur up to 24 hours post administration (with morphine).

Management of Respiratory Depression

Naloxone in increment of 0.04 - 0.4mg may be all that is required.

NB: Duration of action of naloxone is short. Repeat administration may be needed.

Monitoring of respiratory rate, LOC and $Sp0_2$ are mandatory for a further 6 hours for pethidine and fentanyl and 24 hours once any administered epidural morphine is discontinued.

Urinary Retention
Its cause is unclear but may be related to opioid blockade of acetylcholine, which leads to a relaxed bladder detrusor muscle. This complication occurs most often in young men, though not exclusively.

Management of Urinary Retention
An indwelling catheter (IDC) is standard practice (except for women in labour with epidurals), usually because of other reasons, but this problem is thus minimised. Also, naloxone titration may work.

Nausea / Vomiting *(see section on postoperative nausea/vomiting for fuller explanation)*
Sometimes difficult to decide if it is the opioids but a clue is related to timing. Nausea due to morphine for instance will begin around 4 - 6 hours after administration because of the time required for the drug to reach the brain's Chemotrigger Receptor Zone (CTZ).

Management of PONV
There are a number of different anti emetic agents available. The clue is to consider combination rather than single agent therapy. eg. Maxolon, stemetil, scopaderm patch, droperidol, cyclizine, ondansetron.

Also consider minimising the opioid component in the solution being administered.

Pruritus
According to the literature, it is more common in obstetric patients, but can occur in any patient having opioids via epidural route.

Typically, patients report itchiness around the head, face, neck regions, though a rash is less common. Peak onset occurs 3 - 6 hours after administration.

Management of Pruritus
- lotions
- distraction therapy
- cool packs
- drugs such as phenergan

Even a small dose (0.1mg) naloxone may ease the distress of the pruritus.
Other drugs used include ondansetron and (rarely) propofol.

SECTION 14

ENDOSCOPY – MODERATE CONSCIOUS SEDATION

RESPONSIBILITIES OF RN IN ENDOSCOPY

Introduction

Moderate sedation or conscious sedation is a drug induced depression of consciousness – the patient is able to respond purposely to verbal commands, either spontaneously or with tactile stimulation. With moderate conscious sedation the patient is able to maintain their airway, maintain protective reflexes – clear secretions without aspiration, maintain spontaneous ventilation and cardiovascular function without interventions (Lippincott, 2013 – moderate sedation patient care)

Moderate sedation is commonly administered for procedures that are performed in endoscopy units with trained staff and the appropriate monitoring equipment must be available to ensure patient safety. Because the patient's response to sedation isn't always predictable health care providers must have an understanding of respiratory function and potential airway complications and differing levels of sedation. The medical officer within the endoscopy unit must have advanced life support skills (Level 7 CORE). The Registered Nurse administering the moderate sedation must have at least Level 5 advanced life support skills (CORE) and validated IV certification.

The medical officer undertaking procedures requiring conscious sedation takes responsibility for achieving conscious sedation – please refer to HAUORA TAIRAWHITI verbal order policy for endoscopy sedation. The RN administers increments of sedation and analgesia at the request of the medical officer and under the direct supervision of this officer. (Lippincott 2013 – moderate sedation; HAUORA TAIRAWHITI Policy conscious sedation, 2014)

To ensure the safe nursing management of adult patients undergoing intravenous conscious sedation for procedures in the Operating Theatre, conscious sedation may only be used in the Operating Theatre under the direct supervision of the prescribing medical officer (HAUORA TAIRAWHITI Policy conscious sedation 2014)

A patient receiving moderate conscious sedation requires sedation monitoring before the procedure, throughout the procedure and during the recovery period. The Registered Nurse who administers the sedation and who are responsible for the patient receiving conscious sedation should have an understanding of the pharmacology of the agents as well as the role of the reversal agent. The registered nurse who is responsible for the monitoring a patient receiving moderate sedation should not have any other responsibilities that would require them leaving the patient unattended or comprising the continuous monitoring during the procedure.

Some patients who require a procedure may not be appropriate for moderate sedation and required anaesthesia which is performed and monitored by an Anaesthetist. The surgeon will determine whether the procedure is performed under moderate conscious sedation or deep sedation or general anaesthesia.

LEVELS OF SEDATION:

The level of sedation is an important aspect to recognise as each patient will respond differently to the sedation - sometimes not always as intended. As indicated in Lippincott (2013 – moderate sedation). They have devised different levels of sedation.

Minimal sedation

Drug induced state during which patient respond normally to verbal commands. Cognitive function and physical coordination may be impaired - airway reflexes, respirations and cardiovascular functions are not affected.

Moderate sedation/conscious sedation
Drug induced depression of consciousness during which patient responds to verbal commands, either alone or accompanied by light tactile stimulation. Please note - that reflex withdrawal from painful stimulation is not considered a purposeful response. No interventions are required to maintain a patent airway and spontaneous respirations/ventilation is adequate. The cardiovascular functioned is maintained.

Deep sedation
Drug induced depression of consciousness which the patient is unable to be easily roused but responds purposefully following repeated or painful stimulation. Respiratory / ventilation function may be impaired and the patient may require assistance in maintaining a patent airway and spontaneously ventilation may be inadequate. Cardiovascular function is maintained.

General Anaesthesia
Drug induced loss of consciousness during the period when the patient is unrousable even with painful stimulation. The patient is unable to maintain ventilation therefore they require assistance with maintaining a patent airway; positive pressure ventilation may be required because of the depressed ventilation. Cardiovascular function may be impaired. (Lippincott, 2013 - Moderate sedation)

STANDARDS – HAUORA TAIRAWHITI POLICY 2014 – Conscious Sedation

	Action	**Rationale**
1	**Ensure that patient education and informed consent have been completed:** • Patient receives full explanation of procedure and type of sedation used. • Consent to procedure is documented	• To ensure patient understands and consents to procedure and sedation • Consent to be obtained prior to patient arrive to theatre
2	**Patient safety and check:** • Patient has been fasted prior to undergoing CS. The standard period of time the patient is required to be nil by mouth prior to CS is six hours • The area that CS is being administered in has oxygen, airway equipment, ventilation bag and tubing suction readily available. This equipment is to be checked and functioning prior to procedure	• To avoid risk of aspiration of gastric contents whilst patient level of consciousness is reduced
3	**RN assessment of patient prior to CS procedure includes:** • Baseline physical assessment - including pre procedure level of consciousness, respiratory rate, SpO2, heart rate and BP	• To assess patient baseline physiological parameters prior to procedure
4	**RN monitoring for patient during CS procedure:** • Patient is to be monitored using continuous SpO2 and / or cardiac rhythm monitor if available throughout procedure	• To monitor patient response to sedation and procedure
5	**Post Procedure care:** • Continued assessment of patient airway, breathing and circulation will continue until patient's level of consciousness returns to pre procedure level • Post procedure care includes patient education and assessment of readiness for discharge if applicable	• To maintain patient safety post procedure and conscious sedation

6	**Documentation:** • All care including all observations during procedure and post procedure is to be clearly documented on appropriate clinical notes	• Meeting Hauora Tairawhiti and Nursing Council standards

INTRA- PROCEDURAL MONITORING:

A monitor is only as good as the person supervising it. As per guidelines set by the endoscopy programme, the intra-procedural monitoring of the patient will be continuous with oxygen saturations and capnography.

Five to ten minute observations will be recorded and they will include blood pressure, pulse, respiration rate, oxygen saturations, and level of consciousness. It is imperative that the registered nurse monitors ventilation using capnography or assesses with observation and auscultation. Electrocardiogram will be used when required and the registered nurse should have a basic knowledge and understanding of cardiac monitoring and arrhythmia interpretation.

One of the main problems with sedation and analgesia are drug induced respiratory depression and airway obstruction. Monitoring the ventilation function can be assessed by observing depth, rate of the patients' respirations. (Odom – Forren & Watson, 2005)

Pulse oximetry - is a vital and valuable monitoring device to assess the oxygenation of the patient whilst receiving moderate sedation, pulse oximetry is used as an adjunct to clinical assessment for detecting hypoxemia. This tool provides non-invasive measures of the arterial haemoglobin oxygen saturation and pulse rate. Knowledge and the principles of oxygen transportation assist the nurse in interpreting data from the pulse oximetry. (Odom – Forren & Watson, 2005)

Capnography - end tidal carbon dioxide (ETCO2) monitoring determines the carbon dioxide (CO2) concentration in exhaled gas to provide information about the patients pulmonary, cardiac, and metabolic status - this information is important to aid the patient's management and can prevent compromise. Capnography is recommended for patients undergoing moderate sedation as it provides feedback from each breath and reflects apnoea immediately

In ETCO2 there is a photo detector which measures the amount of infrared light absorbed by airway gas during inspiration and expiration, the monitor converts these data to CO2 value and a corresponding waveform. The ETCO2 measurement can alert the nurse to hypoventilation from over sedation. The waveform reflects the course of CO2 eliminated during exhalation, and the registered nurse must have an understanding of end tidal CO2 during the moderate sedation phase.

(Lippincott - end tidal carbon dioxide monitoring, 2013)

PATIENT TEACHING:

Verbal and written instructions are provided to the patient and their family. This is provide in discharge summary and the patient information sheet provided by Day of Surgery Unit (DOSU)

Explanations will include:

- When immediate emergency care should be sought
- provide the patients escort with an emergency phone number.
- explain pain management strategies
- reinforce the importance of support at home to assist the patient.
- Outline relating follow up e.g. OPD appointment or GP

(Lippincott (2013) & HAUORA TAIRAWHITI Endoscopy Discharge Criteria Policy

COMPLICATIONS:

Responding to Complications of Sedation

COMPLICATION	NURSING INTERVENTION
Airway obstruction or respiratory depression	Reposition the head Suction the airway Insert an oral airway Administer Oxygen Encourage the patient to take a deep breath Stimulate the patient by rubbing their shoulder or legs Manually ventilate with a bag-valve mask
Over sedation	Maintain airway, breathing and circulation Have medication available for reversal of sedation immediately available and administer them as ordered, if patient is deeply sedated Naxolone for opiates Flumazenil for benzodiazepines Monitor respiratory status until stable
Cardiac Arrhythmias	Note baseline heart rate and rhythm Apical pulse for 1 minute Examine electrocardiogram pattern Ensure patent airway Monitor oxygen saturations Administer fluids and medications (antiarrhythmic) as prescribed
Hypotension	Investigate causes Support respiratory status Administer fluids and vasopressors as prescribed
Hypertension	Administer additional sedation or analgesia

Lippincott (2013) Moderate Sedation Patient Care.

PHARMACOLOGY:

Fentanyl

This is also a synthetic opioid, and is a potent analgesic and sedative estimated to be at least 80 times more potent than morphine as an analgesic, on a weight basis. Onset of action is almost immediate following intravenous administration and its half-life is dose-dependent. It is lipid soluble, can accumulate and has little CVS effect.

Fentanyl Citrate	Narcotic Analgesic
Uses/Indications:	Opioid analgesic. Short duration analgesia in anaesthesia and perioperatively. Supplement to general and regional anaesthesia. In combination with a neuroleptic for induction and maintenance of general and regional anaesthesia.
Contraindications:	Bronchial asthma; head injury, increased intracranial pressure; susceptibility to respiratory depression, e.g. comatose patients with possible head injury or brain tumour; MAOIs (+/- 14 days); myasthenia gravis; children ≤ 2 years.
Precautions:	Chronic opioid use; history of opioid abuse; severe pulmonary impairment (e.g. COPD, decreased respiratory reserve) or potentially compromised respiration; bradyarrhythmias; hypovolaemia; uncontrolled hypothyroidism; alcoholism; impaired hepatic, renal function; rapid IV injection; elderly, debilitated; pregnancy, labour, lactation.
Adverse Reactions:	Common: respiratory depression, apnoea; muscle rigidity; nonepileptic

	myoclonic movements; bronchospasm, laryngospasm; bradycardia, other cholinergic effects. Less common: hyper/ hypotension; sphincter of Oddi spasm; dizziness; miosis; blurred vision; nausea; diaphoresis; itching; euphoria; seizures; anaphylaxis; dependence; arrhythmias; post-op depression; paradoxical CNS excitation; delirium.
Drug Interactions:	Azole antifungals; macrolides; protease inhibitors, e g. ritonavir; phenytoin; sibutramine; naltrexone; nitrous oxide; neuroleptics; CNS depressants, e.g. barbiturates, tranquillizers, benzodiazepines, opioids, general anaesthetics, alcohol; MAOIs; amiodarone; beta-blockers; Ca channel blockers.
Pregnancy Category:	**Category C -** Drugs that, 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.

Midazolam

Midazolam	
Uses/Indications:	conscious sedation for procedures; sedation in intensive care; sedation in anaesthesia; premedication; induction of anaesthesia; status epilepticus [unapproved]
Contraindications:	severe respiratory depression; sleep apnoea syndrome; marked neuromuscular respiratory weakness including myasthenia gravis; acute pulmonary insufficiency
Precautions:	cardiac disease; patients with a low cardiac output; respiratory disease; neonates; children (particularly if cardiovascular impairment); risk of airways obstruction and hypoventilation in children under 6 months (monitor respiratory rate and oxygen saturation); history of drug or alcohol abuse; reduce dose in elderly and debilitated; risk of severe hypotension in hypovolaemia, vasoconstriction, hypothermia; avoid prolonged use (and abrupt withdrawal thereafter); concentration of midazolam in children under 15 kg not to exceed 1 mg/mL
Adverse Reactions:	gastro-intestinal disturbances, increased appetite, jaundice, hypotension, cardiac arrest, heart rate changes, anaphylaxis, thrombosis, laryngospasm, bronchospasm, respiratory depression, respiratory arrest (particularly with high doses or on rapid injection), drowsiness, depression of consciousness, confusion, ataxia, amnesia, headache, euphoria, hallucinations, convulsions (more common in neonates), dizziness, vertigo, involuntary movements, paradoxical excitement and aggression (especially in children and elderly), dysarthria, urinary retention, incontinence, changes in libido, blood disorders, muscle weakness, visual disturbances, salivation changes, skin reactions, injection-site reactions
Drug Interactions:	Antihistamines, antipsychotics, anxiolytics, azole antifungal agents, barbiturates Benzodiazepines, calcium channel blockers, certain types of antihypertensives cytochrome P450 enzyme inducers, cytochrome P450 enzyme inhibitors hypnotics, inhaled anaesthetics, macrolides, medicines which depress the CNS opiates, protease inhibitors, sedative antidepressants, sedatives
Pregnancy Category:	C: avoid regular use (risk of neonatal withdrawal symptoms); use only if clear indication such as seizure control (high doses during late pregnancy or labour may cause neonatal hypothermia, hypotonia, and respiratory depression)

Naloxone Hydrochloride (Narcan)

Naloxone is an essentially pure **opioid antagonist**, reversing the effects of opioids, most usually that of opioid overdose, though it can be used to reverse the distressing effects of histamine release (i.e. itch). It's mechanism of action is not fully understood but evidence suggests that antagonists compete for the same receptor sites as the opioids (agonists). Given intravenously, onset of action is apparent within 1-2 minutes, and it has a rapid distribution throughout the body. Plasma half-life in adults ranges from thirty minutes to 1 hour. In children bolus doses of 0.1mg/kg have a half-life of up to 70 min(neonates) Duration of action is dependent on dosage and route of administration, and requirement for repeat doses will also be dependent on the type, amount and route of administration of the opioid being reversed.

Administration

Doses will vary between children and adults. The most rapid onset of action is achieved by IV administration, which is recommended in emergency situations, though naloxone can be administered by IM or subcutaneous routes too. It can be given either undiluted, diluted *or* as an infusion and titrated according to the patient's response. However, it must be remembered that since the duration of action of some opioids may exceed that of naloxone, the patient should be kept under close observation. Naloxone can be administered by an IV certificated RN/midwife or registered doctor.

NALOXONE TITRATION:	Make up 0.4mg Naloxone (Narcan) in 9ml Normal saline. Give 1 ml (0.04mg) IV every 2 minutes until patient is easy to rouse with respiratory rate >6/minute
RESCUE:	Give 0.2mg Naloxone IV stat and repeat after 2 minutes if indicated
PAEDIATRIC DOSE:	0.01mg/kg (10mcg/kg)

NB: It is important to remember that narcosis can recur and repeated doses of naloxone may be needed, especially if the patient has renal or liver impairment.

Adverse Effects

Abrupt reversal of opioid narcosis may result in:

- Significant reversal of analgesia
- Excitement - agitation, restlessness
- Tremulousness
- Nausea/vomiting
- Sweating
- Tachycardia
- Hypertension

NB: It should be used with caution in patients with pre-existing cardiac disease because cases of hypotension, hypertension, ventricular tachycardia and fibrillation as well as pulmonary oedema have been reported.

Flumazenil - Anexate

Flumazenil - Anexate	
Uses/Indications:	reversal of sedative effects of benzodiazepines in anaesthetic, intensive care, and clinical procedures; overdosage with benzodiazepines
Contraindications:	life-threatening condition (e.g. raised intracranial pressure, status epilepticus) controlled by benzodiazepines
Precautions:	short-acting (repeat doses may be necessary—benzodiazepine effects may persist for at least 24 hours); benzodiazepine dependence (may precipitate withdrawal symptoms); prolonged benzodiazepine therapy for epilepsy (risk of convulsions); history of panic disorders (risk of recurrence); ensure neuromuscular blockade cleared before giving; avoid rapid injection in high-risk or anxious patients and following major surgery; head injury (rapid reversal of benzodiazepine sedation may cause convulsions); elderly; children
Adverse Reactions:	nausea, vomiting; less commonly palpitation, anxiety, fear; also reported transient hypertension, tachycardia, flushing, agitation, convulsions (particularly in those with epilepsy), dizziness, sensory disturbance, chills, sweating
Drug Interactions:	zopiclone, benzodiazepines, opiates, other medicines that work in a similar way to benzodiazepines, psychotropics, tricyclic antidepressants
Pregnancy Category:	B3: not known to be harmful

For more information and required readings please refer to Hauora Tairawhiti Endoscopy – conscious sedation self-learning package for nursing staff.

To become validated in performing conscious sedation in endoscopy the registered nurse must complete:

- Intravenous therapy and medicines management workbook.
- Pain management workbook and certification
- Complete conscious sedation self-learning package for nursing staff.
- Clinical competence demonstrated.

REFERENCES:

- Moderate sedation (2013). *In Lippincott's nursing procedures and skills.* Retrieved July 8th, 2014 from: http://procedures.lww.com/lnp/view.do?pId=729388&hits=sedation,moderate,sedated&a=false
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- Odom-Forren, J. & Watson, D. (2005) *Practical guide to moderate sedation / analgesia,* 2nd Edition. Elsevier Mosby, St Louis – Missouri.
- HAUORA TAIRAWHITI Policies
  - Conscious Sedation 2014
  - Verbal Orders for Endoscopy Sedation 2014
  - Discharge Criteria for Endoscopy 2014

SECTION 15

PAIN BUSTER:

Pain Buster – Closed Local Anaesthetic Infusion System

Purpose: This information sheet provides background material to ensure nursing staff are familiar with the Pain Buster infusion system and are aware of their responsibilities in maintaining and monitoring the system for post-operative analgesia.

Indication: Post-operative pain management – non narcotic pain relief system.

System Information: The Pain Buster (ON-Q) is a post-operative non-narcotic pain relief system which is designed to deliver a continuous regulated infusion of local anaesthetic to the intraoperative site/wound through specially designed catheters.

The system delivers a steady flow of local anaesthetic that bathes the surgical site for up to 5 days. The intention of the use of Pain Buster is to provide an even distribution of local anaesthetic for optimal post-operative pain relief.

Theatre Initiation:

- The Pain Buster device is loaded with local anaesthetic in the operating theatre and attached to the catheters which are put in place by the surgeon in the incision site, or near a nerve sheath for regional infiltration (the anaesthetist may position these catheters)
- A completed "Medication added" label is attached to the elastometeric bulb/ pouch of the Pain Buster in theatre once loaded with local anaesthetic.
- Infiltration catheters can be looped several times and firmly fixed to a flat surface such as the abdominal wall and secured with a V3000 clear dressing and reinforced if required with MEFIX.
- Wound catheters labels are applied to the catheters, and regional block catheters are to be labeled for nerve sheath infusion.
- Prescription for the Pain Buster is documented on the patient's medication chart – so the pharmacist is aware that one of the patient's regular medications is a local anaesthetic infusion.

Registered Nurse Responsibility:

- Every shift the RN must check and document the site for:

  -Redness, swelling, pain and any discharge at the catheter site

  -Blood in the catheter

  -Additionally check that all the clamps are open and there are no leaks in the tubing.

Any concerns please contact the surgeon

If the patient has <u>two or more of the following symptoms</u> this may suggest local anaesthetic toxicity. The infusion should be stopped by clamping the line and discussed with the surgeon as soon as possible.

-Dizziness / lightheadedness

-Blurred vision

-Drowsiness

-Ringing / buzzing in ears

-Confusion

Please refer to page 90/91 (Section 11 Regional analgesia – Effects, side effects and limits) section of the Hauora Tairawhiti Pain Management Handbook 2016.

Completion of infusion:

- The Pain Buster infusion is complete when the pump is no longer inflated; this depends on the size of the bulb/pouch. Small size may last 2 – 3 days in comparison to the larger bulb / pouch which may last up to 5 days.
- This system is a single use only and should not be refilled.

Removal of catheters:

- Sterile procedure
    1) Remove catheter site dressing
    2) Grasp the catheter close to the skin and gently pull on the catheter (this should be painless and the catheter should be easy to remove). Avoid tugging the catheter or quickly pulling the catheter.
    3) If the catheter becomes hard to remove or the line stretches – STOP as continued pulling may break the catheter and contact the surgeon.
    4) Once the catheter is removed check the tip for a black marking to ensure the entire catheter is removed.
    5) Swab site with chlorhexidine and cover with a sterile dressing for 24 hours.

References:

Hauora Tairawhiti Pain Management Handbook 2016

Canterbury District Health Board (2015) *Pain Buster (ON Q) Closed Local Anaesthetic Infusion System Policy.*

SECTION 16

LIST OF APPROVED RESOURCES:

1. Lippincott On Line procedural manual accessible: http://midlandlearning.elearning.ac.nz
2. Hauora Tairawhiti Medicine Management Policy
3. Children's Acute Pain Management Handbook
4. Notes on Injectable Drugs
5. Hauora Tairawhiti Formulary/MIMS on-line
6. Maternity/Neonatal Unit Guideline (on reversal of narcotics in relation to administration to neonates)
7. HAUORA Tairawhiti Labour Epidurals Workbook
8. HAUORA Tairawhiti IV Therapy Competence Assessment and Validation Process
9. ANZCA Guidelines on Acute Pain Management

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