RNSH

HANDBOOK OF PRACTICAL

ACUTE PAIN MANAGEMENT

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Trainees are to make use of these people for advice:

In hours

1. Consultant allocated to acute pain for the day – name available from daily allocations list
2. Duty director in theatres – ext. 32400
3. Pain fellow for ward consults – pager # 42016
4. Pain consultant on-call for the week – details available from dept. secretary (32488) or switch
5. Director Acute Pain Service, Dr Gavin Pattullo – contact through switch (9)

Out of hours

1. After hours anaesthesia provisional fellow – pager # 41438
2. Anaesthetic consultant on-call for the night (if still in theatre)
3. Pain consultant on-call for the week – contact through switch (9)
4. Director Acute Pain Service, Dr Gavin Pattullo – contact through switch

Other helpful numbers

Acute Pain Nurse – pager # 41219

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Introduction

There were a number of reasons for writing this handbook:
First, to bridge the gaping divide between what is written in the textbooks and what we do in real-life on the wards of RNSH.
Second, to address the disconcerting tendency of some to brush-off the acute pain round as a chore best undertaken with the least amount of effort. When examined simplistically there is an element of truth in this belief; but only when the aim of the clinician is simply acute pain *treatment* rather than the intended and more beneficial aim of pain *management*. Arguably any fool equipped with MIMS and a biro can wonder around the ward, satisfy many of the patient's needs and come back feeling good about themselves. However the results of such a pain treatment approach would testify to its failings. We would almost certainly encounter a backlog of patients unable to be discharged owing to unwanted drug side-effects, or GP's ringing for advice on treating chronic pain that should have been identified before patient discharge. These examples easily become reality and differentiate a clinician who just *treats* pain from one who practices actual *management*.
Finally this book was written to enable some form of continuity and uniformity amongst all members of the Acute Pain Team. The operating theatre is the place for one to display individual talents. However, from day-to-day all of us involved in acute pain management should generally be using the same routines and the same guidelines to arrive at a common goal. This handbook will help you to understand why continuity of care is important.

It is worth noting that RNSH has for many years been a world leader in pain management and is unique for having more pain specialists on staff than any other hospital in Australia. You are encouraged to make use of this extensive knowledge base. This is, after all, the reason why you are here as a trainee - to learn. If in doubt, ask. A list of 'who to call when' is provided on the inside front cover. There are several layers of senior cover and so trainees should never feel that advice is unobtainable.

To facilitate your learning the following texts are essential:

Acute Pain Management: Scientific Evidence (4th edition) 2015 ANZCA & FPM. Available at: http://www.anzca.edu.au/resources/college-publications

Therapeutic Guidelines: Analgesic. Version 6, 2012. Available at: www.tg.org.au

It is our hope this handbook will help to highlight just how much we can do to improve the acute pain management of our patients.

Ross MacPherson
Gavin Pattullo
February 2016

Why should we strive for optimal management of acute pain?

Numerous surveys have shown that when surgical patients are asked about their concerns relating to their upcoming surgery, the most common concern is post-operative pain.

It is self evident that pain is an unpleasant feeling and in the hospitalised patient, significant pain will result in disrupted sleep, impaired mobilisation and rehabilitation, and delayed discharge. What is often less well appreciated by doctors is that significant pain results in sympathetic nervous system stimulation which consequently affects almost every organ system in the body. The following are examples of the wide-ranging effects possible in each system largely as a result of this sympathetic stimulation:

	Immediate response to pain/sympathetic activity	Possible clinical endpoints
CVS	Coronary vasoconstriction, increase in HR, BP Catecholamine release	Ischaemia/infarction Arrhythmias
Resp	Muscle splinting → hypoxaemia Reduced mobility	Wound infection Pulmonary infection
Endocrine	Increased catecholamines, cortisol Salt and water retention → oedema Reduced insulin release	Accentuates stress response Wound breakdown Wound infection
GIT	Reduced intestinal motility → delay in restoring nutritional intake	Wound breakdown
Musc	Reduced mobility → muscle atrophy	Slow rehabilitation
Psych	Sleep deprivation	Anxiety, depression

Knowledge of these possible clinical endpoints (especially delayed wound healing) is useful when dealing with the stoic (usually older) patient who still views that putting up with pain is the best way to deal with it!

Over 20 years ago Ronald Melzak wrote a seminal paper in Scientific American (February 1990 Volume 262 Number 2) entitled "The Tragedy of Needless Pain" where he stated that contrary to popular belief, the author says, morphine taken solely to control pain is not addictive. Yet patients worldwide continue to be undertreated and to suffer unnecessary discomfort.
If we have known about this for so long why do we still manage pain so badly? In an early edition of the book Acute Pain Management- Scientific Evidence, the authors pointed out some key reasons for under treatment of pain. Some key reasons given included:

- The common idea that pain is merely a symptom and not harmful in itself
- The mistaken impression that analgesia makes diagnosis more difficult
- Fear of potential addiction to prescribed opioids
- Concerns about the development of opioid related adverse effects
- Lack of understanding of the pharmacokinetics of prescribed drugs
- The mistaken belief that opioids should not be given more than four hourly

If we can not only overcome these problems ourselves, but educate our colleagues to overcome them as well, we will be on the road to improved analgesia for our patients.

Lastly, we must consider the burden to the community and to the patient of chronic pain. When you think about it, all chronic pain has to start off as acute pain, and much as been written on preventing the transition from acute to chronic pain. At our level, each of us should be trying to optimise acute pain management not only to allow for a shorter hospital stay and more rapid return to function, but to reduce the risk of poorly treated acute pain transforming itself into a chronic pain state.

Conducting the round

The 'breaking in' of the RNSH trainee to the acute pain round usually occurs sometime in the first few weeks of their training. At this time there are usually thousands of thoughts whirling around the trainees mind as they go about the round – with few of these focused on how to actually conduct the round. These points should help:

Importance. Go into the round cognisant of the fact every consultation you have can have real effects on patient outcome. As a result, high importance must be placed on paying attention to what is said and done.

Responsibility. Many of our patients present as fairly straightforward pain management cases. However, many are also complex. Despite the fact that we get used to prescribing analgesic drugs from our days as an intern, we are often prescribing significant doses of opioids and other drugs. There is always the risk of adverse drug reactions and drug interactions. As anaesthetists, we need to take full responsibility for all the medications we prescribe and we will be held accountable for adverse events that may occur. Check and recheck your drugs, your dosage and the risk of interactions.
Lastly, your prescribing on the NIMC (National Inpatient Medication Chart) should be peerless. Every prescription should be legible, the dose and times clearly written, along with the indication and the number of doses if appropriate. There should be no risk that other staff could misunderstand your instructions. An example of recommended prescribing practices on the medication chart is provided in appendix A.

Professionalism. Out on the ward we are the visible face of the anaesthetic department and so our manner of conduct should fit with how we want our profession to be seen by others. Avoid a sloppy appearance as this projects an image of sloppy practice and one will then have to work harder to gain the respect of colleagues and patients. Always be polite. Seize any opportunity to educate other health professionals in a nurturing manner.

Rapport. For all trainees the principles of bedside interaction should be well practiced. A warm empathetic approach will go a long way.

Establish authority. Owing to the potent nature of the medications being dispensed it is imperative the rounding doctor clearly establishes themselves in the role of the one who has ultimate control of management. Trainees frequently run into trouble with difficult and demanding patients through a failure to establish the authorative role. This distinction is best made early on in the doctor-patient relationship and can be accomplished during the introduction, with for example: *"Good morning Mr. Smith, my name is Doctor Andrew Lee, I am a pain registrar doing my routine round to see how you are doing with our pain pump"*. This may sound a bit pretentious but the important points being made are: (1) the patient is seeing a doctor, (2) seeing a doctor with a specialty, and (3) a doctor who has a reason for being there. Please don't use the too often encountered alternative approach used by registrars of bumbling up to a patient without any introduction and mumbling some vague opening question alluding to how bad their pain might be.

Open-ended questions. Always start the consultation with open-ended questions and avoid narrowing in too early. If this open approach is not taken, the information gained is lacking in perspective and an incorrect treatment path can follow. For example, if asking for a pain score straight up, the reply of an 8/10 might lead to an increase in the PCA dose. This approach has missed the patient reporting how much better they feel today because they weren't drowsy on the PCA, were able to get out of bed and go for a walk, and so are actually better left on the lower dose.
So after the introduction the next question could be: *"how are you doing today?"* Most patients will then give a brief commentary on what is troubling them, in order of most to least. Having gained this valuable insight, hone in a little more. For example, the next question could be: *"and what about where you have just had surgery, is that troubling you?"*

Summarising. Patients with pain can present you with a bewildering mass of information. You need to be clear that you both agree with where you are at. It is often useful, after having listened carefully to the patient (as above) to give a summary back. *"So let me see if I've got this right. The pain in the foot was quite severe last night, but after you used the PCA device, the pain was relieved. It's very good now – and you've been able to have breakfast."*

Pain scores. Pain scores can be helpful. Scores are usually taken in the form of Numerical Rating Scores (NRS) of 0 to 10, for rest and activity. True Visual Analog Scores (VAS) require one to carry around a ruler-like device and so are mostly reserved for research situations. Patients who have trouble thinking in numbers can use the mild, moderate or severe quantitative scoring system.

When starting out it is reasonable to start by asking pain scores. Later with experience one will develop a feel for how to properly interpret pain scores. To be truly useful pain scores need to be taken in context with the rest of the patient's condition, signs and symptoms.

Pain scores are a made-up number resulting from combining a myriad of factors, many of which do not respond in a linear fashion to the pharmacological effect of our analgesic agents. The example most of us are familiar with is the heavily opioid narcotised patient in recovery who is still rating their pain as a 9/10 - but only when staff rouse them! Clearly, in this instance the pain score is not responding to what is obviously an effective and near maximal dose of opioid, since the patient is sedated. Management at this point would be to maximise the non-sedating analgesics of NSAID, paracetamol and LA. Document that even in the face of dangerous opioid levels the patient reported high pain scores. Henceforth other measures aside from pain scores should be relied upon to determine appropriateness of further opioid doses, for example respiratory rate and sedation score.

Be cautious not to rely on pain scores as a simplified and convenient indicator of overall pain management. An example from the US in recent years was the over-reliance on pain scores as a hospital performance indicator. This led to a spate of opioid overdoses as hospitals hastily implemented programs to reduce pain scores using the simplest means possible - opioids. This was a well intentioned attempt to improve pain management, but the implementers failed to comprehend that assessing the effectiveness of pain management is not as simple as asking patients for a number.

Psychology. The biopsychosocial model of pain aims to emphasise the importance of non-biological processes in the pain experience. So important is the psychology aspect that many now promote the term psychosocialbiological – to really emphasise psychology first and foremost. It is beyond the scope of this document to provide extensive coverage of psychology and also much of this is best learnt at the bedside. Nonetheless some simple first-step approaches can assist in managing the psychology side of things:

1. Expectation and conditioning: these processes largely underlie placebo (I shall please) and nocebo (I shall harm). This is mentioned in other parts of this book also. Placebo can be used to our advantage: "patients report these medications really help them to feel more comfortable". Nocebo can be avoided by, for example, not making mention of itch prior to use of IT morphine for a caesarean section.
2. Painkillers. The term 'painkiller' really belongs to advertising land! We do not really possess medications that are truly *pain killers* – though you could argue that local anaesthetic placed on a nerve can 'kill' the pain - all systemic medications mainly act to dampen down nociception or the pain experience. Giving patients a medication which has been extolled as a pain killer can set in train a host of unhelpful behaviours. If the medications do not kill the pain the patient can be left thinking: the dose must be too low, I need more medications, the doctor is incompetent, the doctor did not believe me and has given me the weaker drug, there must be something else wrong with me that the doctor has missed etc. Your interaction with the patient may be the only time they interact with anyone with knowledge and experience in pain management, so what you say can have a life-long impact on their behaviour. Rather than using the words 'pain killer', use terminology such as "these medications will make you more comfortable and allow you to do the things you need to do" or "these are pain relieving medications" or more simply refer to them as "pain medications".

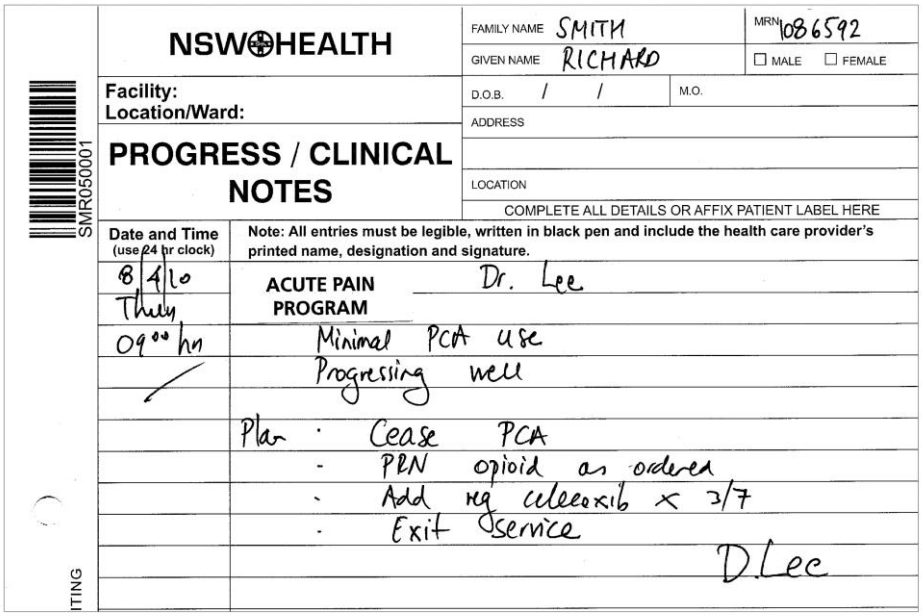
Differentiate between analgesia and no pain. We all like to pat ourselves on the back and take the credit when our patients do well. In the same way, there is a tendency to assume a patient is pain free because of our actions. Alternatively, what needs to be considered is that maybe the patient just doesn't have any pain! Needlessly medicating patients is not best practice. If it is just a simple regimen, well this runs little risk of harm and may be acceptable to continue, but if a more complicated regimen is being used then it really falls on us to ensure necessity. The ability to judge the difference between analgesia and no pain comes with clinical experience and common sense.

Feedback. A couple of minutes spent explaining to the patient the management plan can return large dividends down the track for everyone. As always, phrasing is important*: "we find that patients only need these medications for a few days whilst the worst of the body's response to surgery is occurring" (eg. NSAID)* or *"these strong medications (opioids) are only needed by patients for a couple of days after surgery and then you can be safely managed with medications like paracetamol".*

Interaction with treating team. We are engaged to assist the patient with pain management by consultation. Most teams respect this, and are grateful for our ongoing assessment and treatment of pain issues. Occasionally, you will find that the home team will interfere with your management. For example: while you have been carefully decreasing your patient's dose of OxyContin, a surgical registrar has come along, cancelled it all and written up codeine phosphate! On most occasions, it is easiest to just let this go, documenting in the notes that the surgical team is now responsible for pain management. If however, the new treatment is inappropriate or harmful – then speak up!

Patients in ICU. As in ICU's everywhere, the ICU consultants at RNSH prefer to make management decisions themselves. It is usually more polite to either write a suggested plan in the notes and leave the charting to the ICU team, or to speak to the ICU team first and then make any changes.

Documentation. Progress notes must be dated, timed, legible and signed.



NSW HEALTH

FAMILY NAME SMITH
GIVEN NAME RICHARD
MRN 1086592
☐ MALE ☐ FEMALE
D.O.B. / /
M.O.
ADDRESS
LOCATION
COMPLETE ALL DETAILS OR AFFIX PATIENT LABEL HERE

Facility:
Location/Ward:

PROGRESS / CLINICAL NOTES

SMR050001

Date and Time (use 24 hr clock)	Note: All entries must be legible, written in black pen and include the health care provider's printed name, designation and signature.
8/4/10 Thurs 0900 hrs	ACUTE PAIN PROGRAM Dr. Lee
	Minimal PCA use
	Progressing well
	Plan · Cease PCA
	· PRN opioid as ordered
	· Add reg celecoxib x 3/7
	· Exit service
	D.Lee

ITING

Oral opioids

IMPORTANT DEFINITIONS

Tolerance	state of adaptation in which exposure to a drug induces changes that result in diminution of one or more of the drugs effects over time.
Physical dependence	state of adaptation demonstrated by a drug specific withdrawal syndrome that can be produced by abrupt cessation, rapid dose reduction, decreasing blood level of the drug and/or administration of an antagonist.
Addiction / psychological dependence	primary, chronic, neurobiologic disease, with genetic, psychosocial, and environmental factors. Characterised by one or more of the following: impaired control over drug use, compulsive use, continued use despite harm, and craving.
Pseudo addiction	a reversible opioid-seeking behaviour that normalises once pain is under control

ORAL OPIOID SELECTION

There is a wide variety of controlled release and immediate release opioid preparations available. Sustained release morphine and oxycodone products are the most useful for baseline analgesia. These are named as the –contins; a not always so obvious abbreviation of *contin*uous.

At RNSH, we have a clear leaning towards oxycodone as the preferred oral analgesic either when oral opioid is needed or when transferring from PCA.

The usual prescribing format is Oxycontin b.d. (or t.d.s.- see below) coupled with breakthrough Endone (oxycodone immediate release) 5-15 mg Q3/24.

When prescribing Endone, it is imperative to specify a maximum daily dose. If there is concern that the patient has a tendency to overuse their PRN Endone it is useful to inform the patient what the maximum daily dose is. This will prevent confrontation between patient and nursing staff at 4 am that night.

It has been our experience at RNSH (although never published in the literature) that providing the Oxycontin dose on a t.d.s. basis, rather then b.d., often gives better "round the clock" analgesia. On examining the PRN chart we often find patients are asking for breakthrough Endone in the 3 or 4 hours prior to their next dose, suggesting that the previous dose is not quite holding them for the full 12 hours. When prescribing Oxycontin, don't forget to tick the "Long acting box" on the Medication Chart, to reduce the chance of drug error.

There seems to be an inherent pharmacological logic in having the same agent for both long term and prn analgesic, although there is no reason why MS Contin could not be used, and it is also available on the wards. Oxycodone is also available in oral liquid preparation for paediatrics and in parenteral form for injection. There are a few papers that suggest oxycodone in particular has some activity against neuropathic pain. The opioid receptor subtype responsible for oxycodone's activity is controversial. Some researchers have found that oxycodone has a significant effect on kappa receptors.

When assessing the adequacy of the Oxycontin dosage, it is obviously critical to check the degree of PRN Endone use. There are no hard and fast rules, but if the patient has used say 3-4 doses, and the use was related to specific activity (e.g. physiotherapy) then that's probably OK. Higher usage should prompt one to either increase the Oxycontin dose, or alter it to t.d.s.

After having increased Oxycontin dosage, it is important to check that this has been accompanied by a reduction in PRN Endone use. If this has not occurred, then it maybe time to sit down and have a talk with the patient about what are their triggers for requesting breakthrough pain relief.

A word or two on codeine: oral codeine when used alone is almost useless, worse than placebo and should never be prescribed despite the fact that generations of neurosurgeons have believed and will continue to believe that it is a fantastic drug!

CEASING OPIOIDS

Withdrawal reactions, albeit mild, will predictably occur in any situation where opioids have been taken for more than 7 days. For any dose greater than 40 mg daily where treatment has been for more than a week or so, opioid dose should be weaned off gradually, at the rate of about 10 mg/day, to reduce the risk of withdrawal reaction. Just warning the patient about possible symptoms (see below) is often enough. If symptom management is needed, low dose clonidine is the usual treatment, being mindful of the associated hypotensive effects of the drug.

Signs and symptoms of opioid withdrawal syndrome include yawning, sweating, lacrimation, rhinorrhea, anxiety, restlessness, insomnia, dilated pupils, piloerection, chills, tachycardia, hypertension, nausea/vomiting, crampy abdominal pains, diarrhea, and muscle aches and pains. These represent the complete spectrum of the syndrome. Many patients will just report mild dysphoria and anxiety.

Emergence of withdrawal symptoms varies with half-life of the particular opioid and can occur within 6-12 hours after the last dose of morphine or oxycodone, while longer (72-96 hours) following methadone . Duration and intensity of withdrawal symptoms can be variable and are related to clearance of the drug; withdrawal from morphine is short (5-10 days) but more protracted with methadone.

Whenever a patient is commenced on a long-acting opioid by the acute pain service (APS) the prescription becomes our responsibility. For most patients the APS will supervise the weaning process whilst they are still hospitalised. If discharging a patient from the service who is still on a long acting opioid then a clear plan for that prescription must be made. Most simply this is accomplished with a definitive cease date marked on the medication chart by two vertical parallel lines. If use is likely to run past discharge; then a letter should be provided to the LMO detailing your plan (proforma available from APS nurse), or referral made to the chronic pain service and the patient clearly briefed on the plan.

METHADONE

Methadone is an interesting drug and is sometimes useful if patients are having ongoing pain after days or weeks, or are having repeated procedures.

Anybody can prescribe methadone for pain. However, only specially accredited Drug and Alcohol Medical Officers can prescribe methadone for opioid dependant patients.

The following summary by Gazelle and Fine (reference below) is a great summary.

Methadone, a potent opioid agonist, has many characteristics that make it useful for the treatment of pain when continuous opioid analgesia is indicated. Although available for decades, its use has gained renewed interest due to its low cost and potential activity in neuropathic pain syndromes. Like morphine, methadone is a racemic mixture; one stereoisomer acts as a NMDA receptor antagonist, the other is a mu-agonist opioid. The NMDA mechanism plays an important role in the prevention of opioid tolerance, potentiation of opioid effects, and efficacy for neuropathic pain syndromes, although this latter impression is largely anecdotal.

Methadone is highly lipophilic with rapid GI absorption and onset of action. It has a large initial volume of distribution with slow tissue release. Oral bioavailability is high, ~ 80%. Unlike morphine there are no active metabolites; biotransformation to an active drug is not required. The major route of metabolism is hepatic with significant fecal excretion; renal excretion can be enhanced by urine acidification (pH <6.0). Unlike morphine, no dose adjustment is needed in patients with renal failure since there are no active metabolites. Methadone is available in tablet, liquid and injectable forms; oral preparations can be used rectally. Parenteral routes include IV bolus dosing or continuous infusion.

Unlike morphine, hydromorphone or oxycodone, methadone has an extended terminal half-life, up to 190 hours. This half-life does not match the observed duration of analgesia (6-12 hours) after steady state is reached. This long half-life can lead to increased risk for sedation and respiratory depression, especially in the elderly or with rapid dose adjustments. Rapid titration guidelines for other opioids do not apply to methadone.

Methadone is often used in very high dose in palliative care and chronic pain patients. Most of our patients however will commence dosage at 5-20 mg b.d..

The long half life of methadone also equates to a long time to onset. It is NOT indicated in acute poorly controlled pain, as it will take far too long to reach steady state. Rather, pain should be first controlled by conventional means and then converted to methadone.

Lastly, most people have heard about methadone and patient and family education is essential as they may misinterpret prescription of methadone to mean that their physician already believes they are an addict. If you are really worried about this, use the trade name "Physeptone" when referring to the drug, although obviously you need to be up front about what is going on.
Reference G Gazelle; PG Fine. http://www.mywhatever.com/cifwriter/library/eperc/fastfact/ff75.html

EQUIVALENCE TABLE

The following table is used as a guide when converting opioid doses. Bear in mind deriving such tables is notoriously difficult because of differences in drug action and the lack of experimental comparative studies. Trainees should not find themselves quibbling about minor differences between tables; rather they should concentrate on the order and direction of multiplication.

Equivalent dose to morphine 10 mg intravenous =

Morphine	20 – 30 mg oral
Morphine	3 mg epidural
Morphine	0.1 mg intrathecal
Oxycodone	15 mg oral
Oxycodone	10 mg intravenous
Codeine	200 mg oral
Fentanyl	100 – 200 mcg intravenous
Hydromorphone	2 mg intravenous
Hydromorphone	4 – 6 mg oral
Methadone	Chronic use: 1.0 mg oral Acute: 10 mg intravenous

Fentanyl patch

25 mcg/hr	=	Morphine 100 mg oral over 24 hours

Adjunct analgesics

The general strategy of modern acute pain management and post-operative care is for a rapid transition from parenteral feeding and medication to the enteral route. How well this can really be established is dependent on a multitude of factors, not the least of which include the capacity of the hospital to cope with so called "fast-track" recovery and early feeding and the wishes of the surgical team.

It may sound self-evident but transfer to oral medications can only occur when the patient is able to tolerate adequate oral intake. This usually means that they have eaten solid food and it has remained eaten! The fact that the patient has commenced oral fluids may mean that they can tolerate oral analgesia, but is by no means certain.

Most medications are better tolerated with food and some, e.g. NSAIDs, should only be given with food so they can't really be introduced until oral intake is established. Post-operative constipation is a major problem for the hospitalised patient and contributed to by opioids, dehydration, immobility and pain itself (through sympathetic nervous system stimulation).

PARACETAMOL

As soon as the patient is able to tolerate food, they should be swapped from parenteral to oral paracetamol. It is generally well tolerated. Only appropriate adult paracetamol preparations should be given to adults. That is either paracetamol tablets/capsules or soluble paracetamol should be given. Liquid preparations of paracetamol, designed for use in children, have a low concentration of paracetamol (usually 120mg/5ml) which means the adult patient is forced to consume vast quantities to get an appropriate dose. In order to improve palatability, sorbitol is usually used as a sweetening agent in these products, and consumption of high doses will result in abdominal pain and osmotic diarrhoea.

THE NSW Health Department has produced specific guidelines on paracetamol prescribing that should be adhered to. In order to really benefit from the opioid sparing effect of the drug it should be given regularly 1 gm q.i.d. in adults, reducing to 1 gm t.d.s. in the frail or elderly patient. When using this strategy, it is important to check that other paracetamol containing drugs have not been concomitantly prescribed on the p.r.n. chart.

Many patients refuse to take regular paracetamol on the basis that it's "just Panadol". Check their medication charts to see that if it has been prescribed, it's actually being taken. If not - ask why!

Many people are convinced that drugs containing paracetamol in combination with small amounts of codeine (such as Panadeine) or dextro-propoxyphene are superior in efficacy to regular paracetamol. The literature does not support this. Specifically, preparations containing d-propoxyphene such as Digesic and Doloxene Co, although widely used in the community for over 40 years, have not been shown to have any superiority over regular paracetamol and should be avoided. Recently the European Medicines Agency's Committee for Medicinal Products for Human Use (CHMP) has recommended that all d-propoxyphene containing products be withdrawn from use in Europe because of the unacceptably high risk of adverse events.

NSAIDS

The NSAIDs arguably represent the most useful and potent group of analgesic drugs in our armamentarium. While their adverse effect profile is well known, people often forget just how effective they are when stacked up against other analgesics, as demonstrated in the "league tables" by McQuay and his group. The table on the following page demonstrates how useful NSAIDs are when compared to other analgesics. The table is a little old, and so still contains rofecoxib (Vioxx) and pethidine. As an aside it also shows how useless codeine (alone) is.

The 2007 Oxford league table of analgesic efficacy (at least 3 trials or 200 patients)

Numbers needed to treat are calculated for the proportion of patients with at least 50% pain relief over 4-6 hours compared with placebo in randomised, double-blind, single-dose studies in patients with moderate to severe pain. Drugs were oral, unless specified, and doses are milligrams. Shaded rows are intramuscular administration

Analgesic and dose (mg)	Number of patients in comparison	Percent with at least 50% pain relief	NNT	Lower confidence interval	Higher confidence interval
Etoricoxib 180/240	248	77	**1.5**	1.3	1.7
Etoricoxib 120	500	70	**1.6**	1.5	1.8
Diclofenac 100	545	69	**1.8**	1.6	2.1
Celecoxib 400	298	52	**2.1**	1.8	2.5
Paracetamol 1000 + Codeine 60	197	57	**2.2**	1.7	2.9
Rofecoxib 50	675	54	**2.3**	2.0	2.6
Aspirin 1200	279	61	**2.4**	1.9	3.2
Ibuprofen 400	5456	55	**2.5**	2.4	2.7
Oxycodone IR 10 + Paracetamol 650	315	66	**2.6**	2.0	3.5
Diclofenac 25	502	53	**2.6**	2.2	3.3
Ketorolac 10	790	50	**2.6**	2.3	3.1
Naproxen 400/440	197	51	**2.7**	2.1	4.0
Piroxicam 20	280	63	**2.7**	2.1	3.8
Lumiracoxib 400	370	48	**2.7**	2.2	3.5
Naproxen 500/550	784	52	**2.7**	2.3	3.3
Diclofenac 50	1296	57	**2.7**	2.4	3.1
Ibuprofen 200	3248	48	**2.7**	2.5	2.9
Pethidine 100 (intramuscular)	364	54	**2.9**	2.3	3.9
Tramadol 150	561	48	**2.9**	2.4	3.6
Morphine 10 (intramuscular)	946	50	**2.9**	2.6	3.6
Naproxen 200/220	202	45	**3.4**	2.4	5.8
Ketorolac 30 (intramuscular)	359	53	**3.4**	2.5	4.9
Paracetamol 500	561	61	**3.5**	2.2	13.3
Celecoxib 200	805	40	**3.5**	2.9	4.4
Ibuprofen 100	495	36	**3.7**	2.9	4.9
Paracetamol 1000	2759	46	**3.8**	3.4	4.4
Paracetamol 600/650 + Codeine 60	1123	42	**4.2**	3.4	5.3
Paracetamol 650 + Dextropropoxyphene (65 mg hydrochloride or 100 mg napsylate)	963	38	**4.4**	3.5	5.6
Aspirin 600/650	5061	38	**4.4**	4.0	4.9
Paracetamol 600/650	1886	38	**4.6**	3.9	5.5
Ibuprofen 50	316	32	**4.7**	3.3	8.0
Tramadol 100	882	30	**4.8**	3.8	6.1
Tramadol 75	563	32	**5.3**	3.9	8.2
Aspirin 650 + Codeine 60	598	25	**5.3**	4.1	7.4
Paracetamol 300 + Codeine 30	379	26	**5.7**	4.0	9.8
Tramadol 50	770	19	**8.3**	6.0	13.0
Codeine 60	1305	15	**16.7**	11.0	48.0
Placebo	>10,000	18	**N/A**	N/A	N/A

When you see the huge array of NSAIDs available - how do we make a rational prescribing decision? As a drug group the NSAIDs show many similarities. They are well absorbed orally; they are all weak acids, they are all highly protein bound, and as such have small volumes of distribution, and all rely extensively on renal mechanisms of excretion. Furthermore, in terms of their clinical efficacy there are really no studies to suggest that any one agent has any superiority over another. However, where they do differ is in terms of their half-life, and hence speed of onset.

The earliest (and now abandoned) NSAID, phenylbutazone had a half life that could extend to 170 hours - and was associated with life threatening blood dyscrasias. Some modern agents such as piroxicam and ketoprofen have long half lives that make them suitable for one daily dosing - an

attribute that will improve compliance and convenience in the outpatient setting. However, the long half-life implies a longer time to onset of effects in most cases.

In the hospital setting, convenience of dosage must give way to safety, and since there is no difference in efficacy, the safest choice of NSAID is to choose an agent with a short half life. The options here are ibuprofen (one of the very early ones, but still the gold standard in many ways), naproxyn or diclofenac. These agents all have half-lives in the range of 2-4 hours and are usually given on a t.d.s or q.i.d. basis. They have a rapid onset of action and, critically, if you get into trouble with any side effects, they can be ceased, and you can be confident that the drug will be rapidly removed from the body.

When considering adverse effects an important, in fact the most important factor, is duration of therapy. The serious adverse effects of NSAIDs are well known, such as gastric ulceration, impairment of platelet function and bleeding. These are really confined to patients taking the medications on a long term basis. That is not to say these things can occur in short term therapy, especially in a patient with concomitant pathology, but it is unlikely. By simply restricting your prescribing of NSAIDs to 3-5 days the risk of developing a major complication is drastically reduced. Long term use of NSAIDs is fraught with danger. Tramer, in a recent review has calculated that for every patient taking continuous NSAIDs for more than six months, 1 patient in 1,200 will die from associated gastro-intestinal complications.

NSAIDs and asthma

The literature would suggest that any number of from 8-20% of patients with asthma will be at risk of NSAID induced bronchospasm, presumably as a result of increased leukotriene production. Clinical experience however suggests otherwise. Certainly there is a distinct sub-group of patients who have a historical triad of asthma, nasal polyps and aspirin sensitivity. In this group NSAIDs should be avoided. However in the general asthmatic population, NSAID induced bronchospasm seems to be rare. Because of the widespread availability of NSAIDs in the community, most people have taken NSAIDs at some stage of their life and simple questioning as to prior exposure will guide you as to whether NSAIDs can be used or not.

NSAIDs and orthopaedics

There is a widely held belief amongst some orthopaedic surgeons that NSAIDs should not be used in patients undergoing spinal fusion or joint replacement procedures on account that they increase the risk of non-union of bone and hence increase the risk of failure of fusion. These ideas sprang from studies done decades ago, mainly on rabbits that were administered very high doses of ketorolac and then underwent spinal fusion procedures. Those that received high dose ketorolac had a high rate of non-union. A retrospective study in the early 1990's also suggested that ketorolac (again in high and multiple doses) increased the rate of non-union in spinal surgery, although there are many confounders. The authorities at Bandolier are sceptical that NSAIDs really do have any adverse effect on bone. However we must concede that some papers exist suggesting the opposite. Our feeling is that NSAIDs are best avoided in spinal fusion patients but are OK when used in joint replacement patients, where as at RNSH, the surgeons have agreed to their use.

NSAIDs and renal impairment

Prostaglandins play a negligible role in the maintenance of renal blood flow in health. However, in the patient who is volume depleted for whatever cause the physiological response is the release of a range of vasoconstrictor agents such as angiotensin and aldosterone. To counter these, prostaglandin secretion to the kidney is also increased to maintain renal blood flow in the face of widespread vasoconstriction. These are the people, including any person in the peri-operative phase, who are at risk of renal impairment when given NSAIDs. The elderly, and those receiving mediations with renal activity are clearly at risk. Urine output should be adequate and serum creatinine normal before commencing NSAIDs. Lastly, the duration of treatment should not exceed three days. Following these simple rules will almost eliminate the risk of NSAID induced renal impairment.

COX-2 INHIBITORS

These drugs have had a big impact on prescribing in the community setting, as there is little doubt that their use is associated with reduced gastrointestinal adverse effects. They do not have any superior analgesic effects over NSAIDs and evidence that they have reduced effects on bleeding, renal effects or

effects on bone are less convincing. Because they provide the same level of analgesia as conventional NSAIDs, these agents have only a minor role to play in the hospital setting. Parecoxib (Dynastat) is approved for one intravenous dose for the relief of post-operative pain. Although it is usually given intra-operatively, it can be given on the wards and is a useful adjunct. The only NSAID available in parenteral form is ketorolac. Again, this agent has no superiority over other conventional NSAIDs and should only be used where the oral agent can't be.

TRAMADOL

When developed overseas in the 1970's the proposed role of tramadol was to replace full opioid agonists. Here at RNSH we have tended not to use it in that role, but rather as an adjunct in cases where opioid analgesia (either oral or parenteral) is failing to give adequate pain relief.
It does not have a traditional opioid side effect profile and can be used in combination with opioids. The usual dose is 100 mg q.i.d. It is doubtful whether smaller doses will produce much analgesia. Many patients coming from home, will be using the sustained release product (Tramal SR) which they can continue as an inpatient. Tramadol administered intravenously is associated with a high level of nausea and vomiting and should be given slowly via a burette. Tramadol is also available as oral drops.

Much has been made of the possible interaction of tramadol with anti-depressant medication and the development of the serotonin syndrome. While this certainly can occur, clinically it does not seem to be a common problem. Anti-depressant medications, especially SSRI's are so prevalent in the community it is always best to check. Generally we try and avoid the combination unless there is a compelling reason otherwise.
With regard to the development of the serotonin syndromes, this is really a dose related phenomena. In order to state with certainty that a patient has developed Serotonin syndrome, the patient must exhibit symptoms from each of the following categories (from Australian Prescriber 2003;26:62-3)

Clinical features of serotonin syndrome

Cognitive	Confusion, agitation, hypomania, hyperactivity, restlessness
Autonomic	Hyperthermia, sweating, tachycardia, hypertension, mydriasis, flushing, shivering
Neuromuscular	Clonus, hyper-reflexia, hypertonia, ataxia, tremor. Hypertonia and clonus are always symmetrical and often more dramatic in the lower limbs

Management of serotonin syndrome includes ceasing the causative agent(s), and then symptom treatment. Cyproheptadiene (Periactin), an agent with anti-histamine actions as well as being a serotonin receptor antagonist, is often advocated for the role of symptom management in serotonin syndrome.

PCA

The introduction of PCA approximately 20 years ago was a turning point in pain management because it allowed patients to rapidly titrate analgesia to their own needs. Importantly, this time-point also coincided with the start of awareness into the high levels of pain endured by many hospital patients. Prior to PCA, postoperative analgesia characteristically consisted of what are now considered 'barbaric' regimens; with sole reliance on IMI opioids administered at excruciatingly drawn-out intervals.

EVIDENCE

PCA is considered the gold standard for opioid delivery because it so effectively matches analgesic demand with supply. PCA's effectiveness is evidenced by the dramatic improvements seen in analgesia and patient satisfaction after the introduction of PCA. Although in truth, these improvements largely reflected the hopeless inadequacy of the analgesic techniques in use prior to PCA.
It is worth acknowledging that no significant benefit in morbidity and mortality has been demonstrated with the use of PCA (this is in actual fact not at all surprising when one considers the extraordinary requirements of a study designed to detect such a benefit). Why it is important to acknowledge this lack of any 'hard' morbidity or mortality benefit is that it means other well-considered opioid delivery techniques can be justified - provided they are made equally effective.

AGENT OF CHOICE

Despite much mythology, there is no one opioid with the least side effects for all patients. Rather, there seems to be individual variability in response to each opioid, such that for example morphine might work well for one patient but not for another.
Considerations when selecting an agent for use in PCA are:

Morphine

Advantages:	Morphine is a familiar agent with which most hospital staff are comfortable.
Disadvantages:	Metabolite accumulation, particularly in renal failure of M-3-G and the potent M-6-G, leading to prolongation of action.
Other:	Morphine can be quite sedating. This can be either beneficial or annoying to the patient. The other common side-effect seen with morphine is itch (pruritus). It is important to note that this itch is not a true allergy and is in fact just one of morphine's known pharmacodynamic actions (due to histamine release). The morphine itch commonly involves the face and upper chest. Often wheal and flare will be noted in the skin overlying the injected vein.
Usual dosing:	Adults: bolus 1.0 mg, five minutely Paediatrics: bolus 20 mcg/kg up to maximum 1.0 mg, five minutely Elderly: bolus 0.5 mg, five minutely

Standard strength: 60 mg in 60 mL

Fentanyl

Advantages:	Common agent for PCA in Australia mainly due to the lack of an alternative opioid for the morphine intolerant patient. Inactivity of the nor-fentanyl metabolite means fentanyl is an agent of choice in renal failure. Fentanyl is a cognitively 'cleaner' drug than morphine making its use preferable in the elderly.
Disadvantages:	Fentanyl PCA is a frequent culprit of inadequate analgesia. This arises most commonly from a failure to adequately load with fentanyl. Ideally loading should commence intraoperatively, otherwise loading can be achieved postoperatively with the use of a proper-sized PCA bolus dose. Correct fentanyl loading pushes the

pharmacokinetics beyond that dictated by the short t½α - with resulting poor analgesia - to one dictated by the much longer t½β – and thereby facilitating sustained analgesia.

Even when dosing is adequate there are at least two circumstances where fentanyl use has disappointing results. These are firstly, anxious often young patients who respond better to management with a morphine or hydromorphone PCA (presumably due to these agent's greater anxiolytic effects) and secondly, patients with a long period of use, usually at high doses - as seen with burns and ITU patients. The mechanism for this is not entirely clear, but possibilities include the development of opioid induced hyperalgesia or acute tolerance.

Caution: Particularly in the examples just given gargantuan doses of fentanyl can be consumed in a 24-hour period. In these circumstances care needs to be taken when converting fentanyl dose to an equivalent oral opioid. If the calculation works out to what seems to be a large and dangerous dose of oral opioid, then it probably is! Considerable apparent under-dosing in the conversion is usually needed in this instance to avoid over sedation upon introduction of the replacement opioid. As always, if in doubt ask a more senior person.

Usual dosing: Adults: bolus 20 to 25 mcg, five minutely
Elderly: bolus 10 mcg, five minutely
Paediatrics: bolus 0.3 mcg/kg, five minutely

Standard strength: 600 mcg in 60 mL

Hydromorphone

Advantages: Good clean drug. Well tolerated with favourable anxiolytic actions and neglible to no accumulation in renal failure. For these reasons, hydromorphone is the first choice for PCA use in some centres.

Disadvantages: Unfamiliarity with hydromorphone and its correct spelling can result in confusion with morphine and lead to dosing errors.

Dosing: Adults: bolus 0.2 mg, five minutely
(i.e. Hydromorphone is five times more potent than morphine).

Standard strength: 30 mg in 60 mL

Oxycodone

Advantages: Intravenous form of well tolerated opioid. Another preferred agent in renal failure owing to insignificant accumulation of parent drug and metabolites. Less itch than morphine. Easy dose calculation when stepping-down to oral analgesia.

Disadvantages: Higher cost compared with other opioids.

Dosing: Adults: bolus 1 mg, five minutely (= similar dosing to morphine)

The oral to IV conversion is ≅ 1:1, so 10 mg IV oxycodone is considered equipotent to 10 mg PO.

Standard strength: 60 mg in 60 mL

Tramadol

Advantages: Avoids opioid induced bowel dysfunction and so agent of choice in some centres following GIT surgery.

Disadvantages: Uncommonly used in PCA form at RNSH.

Dosing: Adults: bolus 10 mg, five minutely

Standard strength: 600 mg in 60 mL

Pethidine

No longer used at RNSH due to unacceptable risk of norpethidine induced seizures and an intolerably high addiction potential. In fact, the word "pethidine" should never be written by your hand - in view of the fact that so many alternatives are now available.

BACKGROUND INFUSIONS

The addition of a background infusion to the PCA infers no analgesic benefit, adding only a higher risk of respiratory depression and sedation - to a life-threatening degree for the opioid naïve.

A background should only be considered for the *replacement* of chronic high-dose opioid maintained patients when the normal dosing route can not be maintained. The most common reason for this is the fasting state in those on oral opioids. A reasonable cut-off for definition of high dose is upwards of 80 mg morphine oral equivalents per day.

Worked example: Replacing a dose of MS Contin 40 mg bd with IV:

Morphine daily oral equivalents = 80 mg
Morphine daily IV equivalents = 26 – 40 mg
Morphine hourly IV equivalents ≈ 1 mg

Therefore if PCA morphine is indicated, settings would be

Background infusion = 1 mg/hr (to replace oral opioid)
Bolus dose = 1.5 mg, 5 minutely (for breakthrough)

The addition of a background is unwarranted for patients maintained on doses less than 80 mg oral morphine equivalents because their requirements can be easily replaced with only 1 or 2 PCA demands every hour.

The addition of a background can also be avoided for high-dose oral opioid maintained patients when interruption to oral intake is not predicted, such as with limb orthopaedic surgery. In such a circumstance, the normal oral dosing is maintained and a PCA is added to provide breakthrough dosing - with appropriately up-sized boluses. Further discussion on this area is contained in the chapter on chronic pain.

PAEDIATRIC PCA

Only morphine and fentanyl are routinely used in PCA at RNSH for paediatrics. A 50 mL syringe is mixed to order for each patient producing a concentration calculated so that exactly 1.0 mL delivers the correct bolus based on weight.

Guidelines for patient controlled analgesia for paediatrics < 50 kg					
Drug	Dose in 50 mL syringe	Bolus dose	Drug concentration	Background infusion	Lockout
Morphine	1 mg/kg	20 mcg/kg = 1 ml	20 mcg/kg/ml	0 or 0.5 ml/hr = 0 or 10 mcg/kg/hr	5 min
Fentanyl	20 mcg/kg	0.4 mcg/kg = 1 ml	0.4 mcg/kg/ml	0 or 1 ml/hr = 0 or 0.4 mcg/kg/hr	5 min

Worked example for a 25 kg child needing a morphine PCA:

Take 25 mg morphine and make volume up to 50 mL in syringe,

∴ Concentration = 25 ÷ 50 = 0.5 mg/mL = 500 mcg/mL
(i.e. 1.0 mL = 500 mcg)
A correct morphine bolus is 20 mcg/kg
So a bolus for 25 kg = 25 x 20
= 500 mcg

Therefore, calculations confirm 1.0 mL contains the standard PCA bolus dose for morphine of 20 mcg per kg.

PCA ASSESSMENT AT THE BEDSIDE

When going about the pain round it is often necessary to undertake patient education. This is usually along the lines of reinforcing the aim of PCA: that of enabling daily activities to be undertaken

comfortably. Sometimes explanation is needed that elimination of all unpleasant sensations with the PCA is not possible. The majority of patients will already have a realistic expectation of the PCA along these lines and will find the PCA helpful and cease use at an appropriate time. For those patients where this is not the case, a number of points need considering:

- Excessive demand. When there is a large discrepancy between demands and successful doses try to find out why. Verify patient awareness of both the 5 minute lockout period and hand piece illumination when the next bolus is available.
- Inadequate bolus size. Irrespective of agent and size of bolus, PCA use should average 3 - 4 successful boluses per hour. If consumption is consistently greater than this over many hours then the bolus size may warrant increasing.
- Pain flare. For a transient pain flare initial management is to encourage 5 minutely dosing until pain is controlled. Remember on a standard morphine PCA, delivery of 12 mg morphine in 1 hour is possible – which is quite a potent dose. If this approach fails to control pain then increasing the bolus size may be an appropriate next step. The aim with this hesitant approach is to avoid accidental opioid overdose caused by premature, but well-intentioned, bolus upsizing when the problem was not with the bolus size but rather lay with technique.
- Incidental pain. Pain of onset with activity is notoriously difficult to cover completely with opioids. Remind patients to commence PCA use up to 30 minutes prior to activity so as to increase opioid levels in time for the planned activity.
- Alternatives. Is the PCA effective even when an appropriate dose has been delivered? If not, maybe there is an alternative. Some patients do even better back on their usual medications.
- PCA indications. What actually is the PCA being used for? Do not assume the PCA is being used for its original intention. Some examples of secondary uses encountered include chronic back pain rather than the new abdominal wound, and even for NG tube tolerance. “I’m pressing it because my daughter tells me to”, is sadly not an uncommon response. Patients using the PCA for sleep must have this behaviour discouraged and PCA withdrawal considered. The RNSH APS is not in the business of providing a pharmaceutical night-time sedation service. There are safer alternatives.
- Minimal PCA use. The PCA is interrogated and the patient has used only 5 doses over night. Why? With luck, the answer is that the patient has minimal pain. However, there may be a reluctance or inability to use the PCA because of fear of addiction, not being able to physically use the device, associated nausea and vomiting, or not understanding how the PCA works. Make sure none of these exist before ceasing the PCA.

WEANING FROM PCA

Modern patient management usually follows the regimen of as rapid a transition as practicable to oral intake of fluids and food with early mobilisation and discharge from hospital. The APS team can assist in this process by transitioning from PCA to oral medications as soon as practicable. PCAs are excellent devices for managing early post-operative pain, especially when GIT function has not returned. However they have significant limitations and patient’s pain management will often be improved when they switch to orals.

Working out how and when to step-down from PCA to alternative analgesia usually comes largely with experience and it would be beyond the scope of this guide to discuss every possibility. Nonetheless, a few principles need to be addressed here:

- Interrogate the pump. Bringing up the 24-hour pictogram of opioid consumption is very helpful in determining readiness for step-down analgesia. A pictogram with only the occasional blip usually signifies a straight forward step-down to PRN oxycodone IR (Endone). A pictogram showing an even spread of 1 - 2 boluses per hour points to an easy transfer to regular slow release oxycodone. In contrast, it would be somewhat adventurous to attempt transitioning a patient when the screen is all filled-in with the black of frequent dosing.
- GI function. Use of oral opioids should await the return of gastrointestinal function. Introduction too early will not only result in poor analgesia, but also runs the serious risk of overdose once bowel function returns and an overwhelming quantity of accumulated and unabsorbed opioid then undergoes absorption.

- Patient reluctance. Take care when communicating to the patient the decision to discontinue the PCA. For many patients, news the PCA is ceasing invokes fear, anxiety and a reluctance to relinquish the PCA. This response can be avoided by presenting PCA discontinuation in a positive manner and ensuring the patient understands the PCA is going to be replaced by an equally efficacious form of analgesia. For example: *"you are doing very well today Mrs. Jones so we can now change you to pain relieving tablets without the need for the PCA/button machine. These will actually work much better for you now and will mean you don't need to keep having these drips put in and will be freed from all this equipment and alarms".*
  Generally, understanding these issues and discussing them with the patient who wants to keep their PCA will result in a satisfactory outcome. However in some cases if the patient is particularly difficult to part from the PCA - despite an empathetic chat - it is permissible to leave it up for another day provided that a contract is made with the patient that the PCA will be coming down the next day.
- Overlapping opioids. When converting to oral long acting opioid from a PCA, the preferred approach at RNSH for many years had been to overlap the agents by 2 hours as a way to smooth the transition from one to the other. Due to a recent near fatal opioid overdose with this approach, the preferred approach now is to have no overlap time period. Therefore, cease the PCA and at much the same time administer the long acting opioid.
- Dosing of long acting opioid. The total PCA opioid consumption in the most recent 24 hr period can be used as a guide to dosing. The calculation is then undertaken in one of two ways:
  - Smart method: is to use the opioid equivalence table;

    e.g. morphine IV 60 mg = morphine PO 120 mg
    = oxycodone PO 80 mg
    = oxycodone SR 40 mg Q12H

  - Easy method: is to simply convert X mg Morphine IV to X mg oxycodone PO;

    e.g. morphine IV 60 mg = oxycodone PO 60 mg
    = oxycodone SR 30 mg Q12H

  The small difference in dosage for the two calculation methods is clinically insignificant. In practice the actual prescribed dose is adjusted down, predominantly to allow for the natural course of pain resolution. Patients for whom you suspect a significant proportion of PCA usage was for non-analgesic reasons should have this taken into consideration when determining the final oral opioid dose.
  As a guide; doses usually ordered at RNSH for stepping down from PCA to oral opioid are in the range of Oxycodone SR (OxyContin) 10 – 30 mg Q12H (or occasionally Q8H).
- Breakthrough dosing. Standard dosing is oxycodone (Endone) 5 – 10 mg Q3H. Dose may need to be upsized in opioid tolerant (see Chronic chapter). It is of great benefit to the patient and your colleagues if the step-down oxycodone is ordered on the medication chart at the same time as completing the PCA script. This order for oxycodone needs to be annotated with wording *"Once PCA ceased"* so as to prevent potentially dangerous piggy-backing of oral opioid on top of the PCA.

Epidural

RNSH has witnessed a drastic decline in epidural analgesia use over the past 10 years. This has arisen from a combination of greater fear of the potential for devastating complications with epidurals, and the publication of results, particularly from the MASTER trial, showing less than expected benefits with epidural analgesia. Reduced use results in lack of familiarity and loss of skills, leading to even further reductions in use and an ensuing state of negative reinforcement. Regrettably the rarity of epidural use has led to a reduction in exposure for the trainee such that any opportunity to be involved in the management of epidural analgesia should be seized upon.

EVIDENCE

Trainees should refer to the ANZCA Acute Pain Management: Scientific Evidence (Third Edition) for a more in-depth discussion of benefits. Recapping the salient benefits:

Cardiac	Thoracic epidural analgesia extended for more than 24 hours reduces incidence of postoperative myocardial infarction (Level 1 evidence)
Respiratory	Epidural local anaesthetics improve oxygenation and reduce pulmonary infections and other pulmonary complications compared with parenteral opioids (Level 1 evidence)
	Following open abdominal aortic surgery TEA reduces the duration of tracheal intubation and respiratory failure (Level 1 evidence)
GIT	Thoracic epidural improves bowel recovery after abdominal surgery (Level 1 evidence)
	The combination of thoracic epidural analgesia with local anaesthetics and nutritional support leads to preservation of total body protein after upper abdominal surgery (Level 2 evidence)
	Epidural analgesia is not associated with increased risk of anastomotic leakage after bowel surgery (Level 1 evidence)
Analgesia	For all types of surgery, epidural analgesia (except epidural analgesia using a hydrophilic opioid only) provides better postoperative pain relief compared with parenteral (including PCA) opioid administration (Level 1 evidence).

RISK VERSUS BENEFIT FOR MORTALITY

The mortality risk versus benefit of perioperative analgesic epidurals can be estimated based on current published data. The risk of permanent harm (including death) from an epidural is 1:10 000 (Cook 2009). While the benefit from an epidural has been calculated at one life saved for approximately every 500 epidurals placed (Wijeysundera 2008). This means for every 10 000 epidurals placed, 20 lives will be saved at the cost of one patient permanently injured inclusive of death. Certainly the numbers favouring epidurals are subtle, but are entirely consistent with modern practice and the rarity of preventable deaths.

INDICATIONS

For RNSH practice we emphasise the following points:

- Thoracic epidural analgesia (TEA) aids the recovery from upper GIT surgery, such as oesophagectomy or Whipple's. Patients undergoing these procedures have difficulty maintaining their nutritional balance due to the prolonged period of fasting. For these patients the use of thoracic epidural analgesia has been shown (by S Barratt) to limit whole-body protein loss, indicating a more favourable balance between the competing catabolic and anabolic states during the all important recovery phase.
- Lumbar epidural analgesia is of limited utility for our surgical patients outside of obstetrics. A key factor to consider is the shortening of bed stay times and the resultant need for rapid restoration of mobility - otherwise compromised by the presence of a lumbar epidural.

GETTING THE MOST OUT OF THE EPIDURAL

It is very easy for an epidural to fail. This is bad at any time, but even more so in the current climate where epidurals are only used when strongly indicated. Fortunately most failures can be prevented through fastidious attention to detail. This is where the edict: it's TEA or coffee, comes into play. One can choose to go for thoracic epidural analgesia (TEA) or more simply choose to forget about the epidural and go and get a coffee instead. Clearly, and it should go without saying, the latter is usually not in the best interests of the patient. The attention to detail demanded of an epidural includes:

Level of insertion. It is essential to insert the epidural at a vertebral level that enables coverage of the necessary dermatomes. This may seem obvious but it is so easy to misjudge the level if time is not taken to correctly identify the anatomy. Remember; bottom of the scapula with an unflexed spine is T7, otherwise count spinous processes up from the sacrum or down from C7. In the really difficult to palpate, ultrasound using the parasagital view can be very helpful to identify and count vertebral structures.
Choose a level that ensures coverage of the most cephalad extent of the wound, because (a) this end tends to be the most painful - as it is closest to the moving chest, and (b) this will provide maximal coverage of spinal afferents with the aim of limiting spinal-mediated stress responses and central sensitisation. If the surgical incision is limited to 6 dermatomes or less, analgesic cover will be assured by the simple action of siting the epidural at the mid-point of the affected dermatomes e.g. for an incision spanning T8 – T12, insert tuohy needle at T9/10 or T10/11.

Recommended level of insertion:

Thoracotomy	Level of surgical incision, usually T2 – T6
Upper GI (liver, Whipple)	T7/8
Nephrectomy - retroperitoneal	T9/10
Cystectomy - bladder	T10/11
Hemicolectomy	T11/12

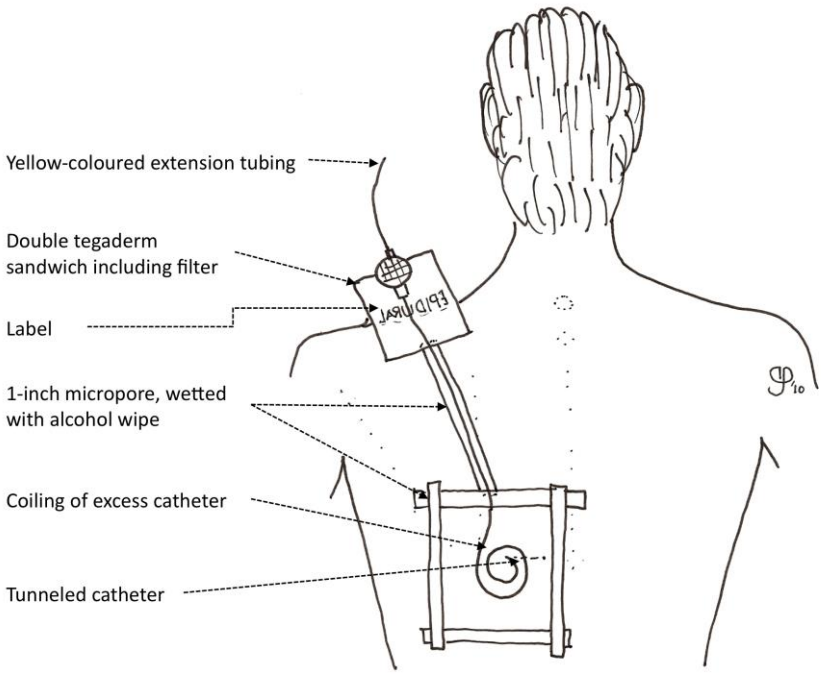
Check the block before proceeding with GA. Only rarely is it excusable to proceed with inducing GA without having first confirmed the presence of a block. Waking a patient up from a prolonged evisceration with an untested epidural and no residual opioid on-board belongs to a time we have long past. A clinically appropriate dose of lignocaine 1 – 2 % with adrenaline usually in a volume of 3 – 6 mL will produce detectable ice hypoaesthesia within a few minutes - well before taping has been completed. Failure to detect a block at this time point allows for epidural re-siting, or the decision made to abandon and a PCA ordered. Document in the anaesthesia chart the dermatomal extent of the test dose to make it simpler for colleagues to troubleshoot any problems with the epidural later on.

Insertion. The paramedian approach to the thoracic epidural space is recommended for insertion levels between T3 and T10. Skin entry point is made 10 – 15 mm lateral to the lateral border of the spinous process. Tuohy needle advancement is made at an angle of 90° to the skin in all planes until the lamina is purposefully contacted. Note is made of the depth of needle insertion at this point, since the epidural space should be encountered within 1.0 – 1.5 cm of this depth. Walk the tuohy needle off the lamina in a cephalo-medial direction and then commence advancement with guidance of the loss of resistance syringe. Aim to leave 4 – 6 cm of catheter in the epidural space. The sine qua non of the thoracic epidural space is an easy feed of the catheter 'like a hot knife through butter' - as they say.

This is in contrast to the lumbar epidural space where it is common to encounter some initial resistance to catheter advancement.

Securing. Frequently sighted on the ward are mobilising thoracic epidural patients dragging their IV poles behind them using their epidural catheter as a towrope – so better hope it is well secured! Tips for securing are:

- Tunnel the epidural catheter. This may well reduce the risk of dislodgement, and at the very least reduces the risk of bacterial colonisation – helpful when planning to keep the epidural for 2 days or more.
- Griplock the catheter. This should be placed so the notch of the device is firmly abutting the epidural catheter as it exits the skin.
- Prior to taping wipe off any dried betadine residue that can otherwise prevent adherence of the dressing to the skin.
- Ensure the entire length of the catheter and the filter is firmly secured to the skin. Tubing and filters flapping around in the breeze are just begging to be caught and become disconnected during patient transfers.
- The preferred technique to achieve securing of the filter is to take 2 large Tegaderms and then make a sandwich of these with the filter in the middle. Leave the proximal halves free to adhere to the patient in a mesentery-type fashion.
- The edges of the tegaderm as well as any uncovered catheter can be secured with 1 inch micropore wet with alcohol wipes which acts to greatly improve adhesiveness of the micropore.
- In a patient expected to be quite mobile postoperatively the pinch and pull phenomenon, contributing to outwards catheter migration can be pronounced, so choose a securing method to minimise this, such as: tunneling, and a Griplock.
- Finally, use the bright yellow *Epidural* label from the kit to clearly identify the catheter.



Recommended technique of securing the epidural catheter and filter.

Agents. The use of multimodal agents in the epidural space acts in just the same way as for systemic analgesia: to maximise benefits whilst minimising side-effects. The solution preferred for use at RNSH is subsequently a mix of 3 different agents, and owing to the contents is sometimes abbreviated to FAB:

Bupivacaine 1 mg/mL (0.1%)

Fentanyl 2 mcg/mL
Adrenaline 2 mcg/mL (1 in 500 000)

In reality, there is no one solution that works best for every patient in the fashion of one-size-fits-all. However the FAB solution seems to cover the patients we more frequently see, being elderly and often with multiple comorbidities. Bupivacaine is in a low concentration so sympathectomy induced hypotension is minimised, while still being potent enough to provide effective dynamic pain relief - the key benefit of local anaesthetic. Fentanyl concentration is also low so even at high delivery rates there is minimal 'spilling' of epidural fentanyl out into the systemic circulation.

The addition of adrenaline has a pharmacokinetic action to reduce washout of the other 2 agents from the epidural space. Adrenaline in the epidural space additionally provides a pharmacodynamic response in the spinal dorsal horn to produce analgesia. There is no evidence the use of epidural adrenaline significantly impairs spinal cord blood flow (this fact similarly applies to intrathecal adrenaline). Epidural adrenaline will also not affect the systemic blood pressure, either up or down, as the dose is miniscule.

A note on FAB solution availability: So as to ensure sterility and quality of contents, epidural solutions are provided by pharmacy (having been sourced from an external compounding pharmacy, usually Baxter). The inpatient pharmacy can be contacted on ext. 31101 to order up the solution if stock in the PACU safe is depleted. If there is a specific requirement one may need to call TPN pharmacy on ext. 31151. From time to time, epidural solution may be unavailable and so the need to prepare a small number of bags may arise. This is appropriate provided the solution is prepared with due regard to strict aseptic technique (which includes undertaking in a high air turn-over site such as the operating theatre), and that pharamacy sourced bags are reverted to as soon as they are available.

Commencing the infusion. It is advisable to commence the epidural analgesic infusate well before leaving the operating room. There are a number of benefits to this:

- Provides a smooth transition to postoperative analgesia.
- Allows early development of steady state drug concentration in the epidural space. Attaining steady state by the time of extubation enables analgesic adequacy of the infusate to be immediately apparent. To achieve these conditions start the infusion at the rate predicted to be necessary for postoperative analgesia (8 – 12 mL/hr) at least 2 hours prior to extubation.
- Minimises the risk of bacterial contamination by reducing number of line interruptions.
- Making the connection oneself avoids accidental misconnections being perpetrated by less experienced personnel.

A commonly encountered argument against commencing the infusate intraoperatively is the concern for haemodynamic instability. Counter points to this concern are: any haemodynamic instability induced by an infusion will be of slow onset - compared to the alternative and more risky bolus technique; will be of small magnitude, since the solution is very dilute; and readily overcome with standard doses of alpha and beta agonists. If there is still concern for haemodynamic stability simply reduce the rate, or switch off completely until satisfied that it is safe to recommence the infusion. Leave the epidural pump and lines all connected so the infusion can be readily recommenced.

CADD Solis Pumps. These were introduced in early 2014 to replace syringe drivers. When used for thoracic epidurals they are loaded with 500 mL bags of FAB, aka FAB500. The general access passcode is 123. More advanced functions, such as clinician boluses, require the entry of the code 456. Access keys are generally kept with the 'redkeys' in various locations including PACU.

One unique feature of the CADD Solis is software allowing the delivery of the infusate as intermittent boluses rather than as a continuous infusion. Experimental evidence and clinical experience demonstrates improved analgesia with bolusing of catheters due to flooding of the epidural space over a wide area. This ensures maximal cover of neural fibres and avoids the limited spread and selective channeling which is thought to occur with continuous infusions.

The Solis is pre-programmed with 3 modes of delivery:

1. Continuous infusion. Rate range is up to a maximum of 28 mL/hr

2. Programmed intermittent bolus (PIB). Default settings are 4 mL delivered every 20 minutes, first bolus is delivered 2 minutes after starting pump. Bolus volumes and intervals can be easily altered to match clinical need. Suitable maximum and minimum parameters for each of these settings have been pre-entered into the pump.

3. PIB + PCEA (patient controlled epidural analgesia). This is expected to be the most frequently utilised mode. Default settings are PIB of 4 ml every 30 minutes - this is the minimum the patient will receive. PCEA settings are 5 mL with a 10 minute lockout. The pump is programmed to avoid piggy-backing of a PIB delivery on top of a PCEA delivery, and vice versa. This means following a PCEA demand and successful delivery, the pump will only deliver a PIB if 30 minutes passes without a patient demand. Similarly, following delivery of a PIB, a PCEA demand will not be successful until the 10 minute lock-out period has passed.

IV access. On the ward no IV rapidly translates to no epidural. If there are tenuous veins looking like they won't hold out for the planned duration of the epidural then do everyone a favour and place a well-secured central line intraoperatively.

HOW TO REVIEW AN EPIDURAL PATIENT

There are a number of specific details to review for any patient with an epidural:

Pump and equipment. Equipment errors are rare but can be catastrophic, it is our job to act as key quality control officers. Make sure the bag is loaded with the 'as advertised' agents, check the integrity of the catheter, verify the presence of a filter and check the robustness of the securing technique.

Analgesia. The importance of determining whether the epidural is functioning as well as it should can not be understated. What we want to see with an epidural is dynamic pain relief. Dynamic pain relief is defined as analgesia with activity and is the key feature of correctly placed epidural local anaesthetic. Consider for a moment: being pain free at rest is relatively easy to achieve, and in fact is possible with just a plain old PCA without going to all the effort of an epidural. It is only when enquiring into dynamic pain relief that one will really determine epidural efficacy.
A good way to determine how well the epidural is providing dynamic pain relief is to firstly ask about pain at rest, then with the dynamic manoeuvres of a deep breath, a big cough and finally if able, ask patient to pull themselves forward (these criteria are an extension of the Prince Henry Hospital Pain Scoring system). Clearly, a thoracic epidural providing analgesic cover of a stimulus as intense as a big cough is functioning well. An epidural not even providing analgesia at rest is nonfunctional and necessitates immediate rectification.

Ice hypoaesthesia. Testing sensation to ice is important and should not be overlooked since this test provides valuable information on epidural function. The correct technique starts with demonstrating normal intensity of cold in an unblocked part of the body, usually the face, then go to a site where ice hypoaesthesia is expected and from there work cephalad and then caudally until the level of normal sensation is found. Assess both sides of the body to exclude unilateral block.

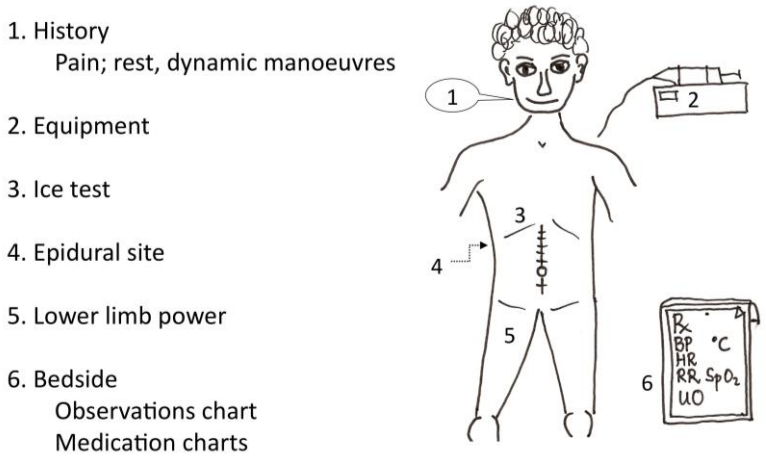
Site. Inspect site to: confirm correct markings of catheter length at the skin, absence of signs of infection, leaks or haematoma, and that the dressing is still well adherent and likely to remain so for the intended duration of the catheter.

Lower limb power. This must be documented. A correctly sited thoracic epidural in combination with a low dose solution should not lead to leg weakness. In fact, the majority of patients with a functional thoracic epidural are able to walk. Lower limb weakness may be present with a lumbar epidural, but should only be mild to moderate. Excessive weakness may be a sign of spinal cord ischaemia secondary to an epidural haematoma. A good neurological recovery from epidural haematoma requires prompt investigation and treatment, ideally within 8 hours. If in doubt, discuss with a senior colleague.

Anti-coagulant agents. Epidurals and thin blood don't mix. The introduction of LMWHs in the 1990s saw a spate of epidural haematomas. Analysis of causality pinpointed overdosing, notably in the elderly and those with renal impairment, and a failing to acknowledge the important relationship between timing of dose and catheter manipulations.
Review the medications chart for anti-coagulants, noting both the appropriateness of the dose and the timing. Also check for platelet altering drugs particularly NSAIDs.

The RNSH Neuraxial Interventions and Anticoagulant Guidelines (reprinted at the end of this chapter) clarify allowable combinations.

Vital signs. Review all vital signs for indications of common clinical states such as infection, hypovolaemia, haemodynamic instability and respiratory depression



Assessment of the epidural patient. Taking a Talley and O'Connnor-style approach from top to toe will ensure no points are overlooked.

HOW TO TROUBLESHOOT AN EPIDURAL

Owing to the quality of analgesia produced epidurals are often referred to as the Rolls Royce of analgesia. Unfortunately, this similarity can include the unreliability for which the Rolls was once notorious. Commonly encountered scenarios:

Pain with deep breath and cough, undetectable ice hypoaesthesia and an epidural infusion rate at maximum.
Diagnosis: Non-functioning epidural.
Management: Cease epidural, remove when appropriate and convert to alternative analgesia, likely to be PCA.

Pain-free including with dynamic manoeuvers, a low infusion rate and absence of ice hypoaesthesia.
Diagnosis: Most likely a patient with no pain, less likely to be a so-called 'hyper functioning' epidural.
Management: Will depend on situation. If day 1 after Whipple's, keep the epidural but be cautious with the duration of use and tentatively plan to remove early at 48 to 72 hours. If patient is post-operative day 4, then epidural removal and replacement with simple analgesia is indicated.

Block regression. Regression of block (narrowing of both field and intensity of analgesia) is commonly encountered after around 24 hours of infusion. Proposed mechanisms include channeling in the epidural space and even tachyphlaxis. Adrenaline containing solutions reduce the occurrence, and so if not already in the solution can be added. Otherwise, a 5 to 10 mL bolus of the analgesic solution (with volume titrated to cardiovascular stability of patient) and an increase in hourly infusate delivery will often restore analgesia.

Extensive block to ice, excellent dynamic pain relief, but with complaints of a deep dull pain.
Diagnosis: Sensing of unblocked visceral afferents.
Management: requires explanation and reassurance the epidural is working well. If allowable, increase the infusion rate to try and extend the block more cephalad above these afferents. If explanation does not improve tolerance, systemic analgesia may then be needed. Start with the simple agents of paracetamol, parecoxib single dose and then finally move to low-dose subcutaneous morphine (2.5 – 5.0 mg Q4H) only if necessary.

Unilaterality. Most unilateral epidurals can be rescued with a bolus and/or an increase in infusion rate. This is especially so if the epidural has previously been bilateral. It is uncommon for an epidural catheter to need pulling back to 3 cm in the space with the aim of 'centering' the catheter. Needlessly pulling back a catheter increases the risk of infection and dislodgement either immediately or later, through a slip-shodily reapplied dressing.

Patchy or incomplete block. The initial management steps of a patchy or incomplete block are to:

- Administer a bolus of a safe volume.
- Ensure solution is optimised through being a triple component solution of LA, opioid and adrenaline.
- Administer the highest safe infusion rate. In general the maximal delivery rate per hour should not consistently exceed 16 mL/hr. Short periods (of an hour or two) of up to 25 – 30 mL/hr may occasionally be necessary to restore a block.

If despite these maneuvers the block is still patchy or incomplete and the decision is to maintain the epidural, say because it is providing 90 % pain relief and the patient also wants to persist with the epidural, then the addition of systemic analgesia is warranted. Start with the simple agents of paracetamol and COX-2. Be wary of regular NSAID as the antiplatelet effect makes it relatively contraindicated in the presence of an epidural catheter and prophylactic LMW heparin (but unfractionated heparin is OK).

If pain severity warrants an opioid, management may be as simple as subcutaneous morphine e.g. 2.5 - 5.0 mg Q4H (i.e. be cautious with dosing) provided only 1 or 2 doses per day are expected. If consistent opioid dosing is likely, a low-dose PCA should be trialed (see below). Failing this, epidural abandonment is indicated.

Pain at another site. A site of pain remote to the area being covered by the epidural may warrant systemic analgesia. Examples include a fractured limb in combination with rib fractures. Start with simple analgesia and move up to a low dose PCA if necessary (see below).

Liver surgery patients can typically report referred pain to the shoulder tip, despite good epidural blockade. If severe this problem can often be successfully managed with a COX-2 inhibitor.

Hypotension. A number of points need consideration when managing epidural induced hypotension:

- Lumbar epidurals cause hypotension through vasodilatation. Management therefore is with IV fluids and α-agonists.
- Thoracic epidurals however predominately cause hypotension through cardiac effects; reduced ionotropy and chronotropy. Vasodilatation is only of the relatively small splanchnic bed and so plays less of a role. Management is with limited use of IV fluids and an emphasis on β-agonists. Clinically the simplest agents to use are ephedrine boluses or noradrenaline infusions. At times this may require ITU care to enable maximal effectiveness of the epidural.
- Definition of hypotension. The debate will continue for some time yet between what numbers, or the adequate perfusion of which organs, should be used to define hypotension. For the time being, a systolic more than 80 mmHg or within 20 % of preoperative baseline is generally considered adequate.

OTHER WARD MANAGEMENT ISSUES:

Adding PCA. Only rarely do we find it necessary to add IV PCA to an epidural - either because of poor coverage of the primary pain site or because of a secondary site of pain (e.g. a fractured foot in conjunction with a flail chest).

If a PCA is required, and because of the (very small) risk of opioid induced respiratory depression and sedation from the administration of an opioid by two routes – with one of them being the neuraxis - then for safety reasons, a low dose PCA should be instituted. Low-dose means a half to quarter normal dosing; for example, IV morphine 0.5 mg with lockout of 5 to 10 minutes.

In some centres when a PCA is added to an epidural, removal of the opioid from the epidural solution is advocated. This approach aims to avoid the risk inherent in delivering opioid by two differing routes. Unfortunately all this actually does is result in even worse analgesia, through loss of the

synergistic/additive affect between neuraxial opioid and local anaesthetic. Removing the epidural opioid potentially has the opposite effect to that intended; resulting in an even greater risk of opioid overdose due to the resultant poor analgesia and subsequent high PCA opioid consumption. So in a nut shell: all that is achieved by taking the opioid out of the epidural and adding it back in via a PCA is to turn a mildly dysfunctional epidural into a completely useless one. Far better to leave the opioid in the epidural and use a low-dose PCA.

NB. Due to the infrequency of adding a PCA to an epidural, the decision to do so should be discussed with more senior staff.

Epidural boluses on the ward. Ill-considered ward boluses can be fraught with danger. At the very least, breaking the line to inject a bolus increases the risk of bacterial contamination. However the main danger is presented if high concentration solutions are used. On the ward the lack of close blood pressure monitoring and resuscitative agents can see such a seemingly innocuous action as a bolus playing out into the sort of gut-churning ER episode no-one ever wants to be in. For these reasons, ward boluses are best administered utilising the analgesic infusate already connected to the epidural. The use of more concentrated solutions than this, and certainly no greater than ropivacaine 0.2%, on the ward is rarely necessary. Getting a result with dilute solutions may take a few minutes longer, but will have the benefit of lengthening your career through the catastrophes avoided!

Top-ups can be easily delivered using the 'Clinician bolus' function of the CADD Solis. This can be found in the drop down list of options after selecting 'Tasks' from the main screen. A higher level access code of 456 is entered instead of 123 to enable this option. The default clinician bolus volume is 10 mL, which is suitable in most circumstances.

Epidural cessation. Epidurals are generally ceased during daylight hours, so that staff are around to manage any problems. There is no benefit to 'weaning' the epidural, it is either on or it is off.

The choice of step down analgesia following cessation of an epidural depends on the clinical scenario. For example, a patient who has an epidural for 5 days following a Whipple can usually be stepped down to paracetamol +/- NSAID/COX-2 and only PRN SCI/PO opioid. At this point the wound discomfort, really just a hypersensitivity, is poorly responsive to opioid and adding a PCA at this late stage of recovery will only undo many of the beneficial effects from the epidural.

Prior to epidural cessation patients should be forewarned to expect a return of sensation. Doing so will prevent patients from confusing this new sensation with something they should be relying on opioids (often in excessive doses) to ameliorate.

Surgeries requiring only 2 to 3 days of epidural, such as hemicolectomy, can usually be stepped down to multimodal analgesia incorporating a judicious dose of oral slow release opioid for the first 24 – 36 hours.

Duration. The development of catheter related infection is the main reason to limit the duration of an epidural.

Epidural catheters maintained for less than 48 hours have a very low risk of infection. Beyond this magical 48 hour time period the risk of infection is known to increase rapidly, particularly when including patients at high risk for infection, such as the immuno compromised, diabetic, steroid dependant, or those with advanced malignancy.

There are times when it is appropriate to extend duration of catheterisation beyond 48 hours, including: for patients with no risk factors for infection, when insertion has been straightforward (since multiple attempts are associated with increased infection rate), when the proceduralist was meticulous with sterility, and when there stands to be benefit from extending the epidural period - such as for Whipples and oesophagectomy (usually 4 to 5 days). Ideally any plan to maintain the epidural beyond 48 hours should be discussed with the inserting anaesthetist and a high degree of vigilance maintained thereafter.

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Wijeysundera DN, Beattie WS, Austin PC, Hux JE, Laupacis A. Epidural anaesthesia and survival after intermediate-to-high risk non-cardiac surgery: a population-based cohort study. Lancet. 2008 Aug 16;372(9638):562-9.

	Timing of spinal/epidural insertion or removal	**Timing of next dose after any neuraxial procedure (insertion or removal)**	**Epidural catheter insitu**
Unfractionated heparin - prophylactic eg. heparin 5000 U bd	*Perform at least **6 hours** after last dose (See note 1)*	*Wait at least **1 hour** before giving next dose.*	*OK – up to 5000 tds*
Unfractionated heparin – IV therapeutic	*Wait **4 hours** and a normalised aPTT*	***1 hour***	*Contraindicated*
LMWH – prophylaxis dose eg. clexane 20 to 40 mg daily	*Perform at least **12 hours** after last dose*	*Catheter removal: Wait **4 hours** until giving next dose Following insertion of needle/catheter**: 6 hours postoperatively***	*Once daily dosing Ok, provided no additional haemostasis altering agents used (eg ASA) BD dose contraindicate*
LMWH – anticoagulant dose eg. clexane 1.5 mg/kg/day o clexane 1 mg/kg bd	*Perform at least **24 hours** after last dose*	*Catheter removal: Wait **4 hours** until giving next dose. Following insertion of needle/catheter: **6 hours** postop; unless dosing is twice daily, **24 hours** postop*	*Contraindicated – consult with specialist*
Warfarin	*Perform when: - INR < 1.4 -1.6 if warfarin being introduced - INR < 1.4 if warfarin has been ceased*	*INR dependent*	*Contraindicated – consult with specialist*
Clopidogrel	*Ideally wait 7 days*	*No specific recommendations*	*Avoid*
NSAID inc aspirin	*OK*	*OK*	*OK*
Dabigatran (Pradaxa)	*5 days*	*Min 24 hrs post op, and 6 hrs post catheter removal*	*NA*
Rivaroxaban (Xarelto), apixaban (Eliquis)	*3 days*	*6 hours*	*NA*

Notes

1. ASRA guidelines actually state no contraindication to neuraxial blockade when on ≤10 000 Units UFH per day. Time allowance of 6 hours based on ACCP and European guidelines.

Points:

- Heparin induced thrombocytopenia should be considered in all patients who have received heparin (particularly un-fractionated) and a FBC performed accordingly prior to any planned procedure.
- Warfarin and INR: the INR result is dependent mostly on the level of clotting factors with short half-lives, such that during introduction of warfarin the rise in the INR does not reflect the fall in all clotting factor levels - as clotting factors with a long half-life will still be present in high levels. As a result, there is a slightly greater margin of safety with the INR levels when warfarin is being introduced.

Adapted from:

Horlocker TT, Vandermeuelen E, Kopp SL, Gogarten W, Leffert LR, Benzon HT. Regional Anesthesia in the Patient Receiving Antithrombotic or Thrombolytic Therapy: American Society of Regional Anesthesia and Pain Medicine Evidence-Based Guidelines (Fourth Edition). Reg Anesth Pain Med. 2018;43(3):263-309.

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Neuraxial opioids

SINGLE DOSE INTRATHECAL MORPHINE

To provide meaningful postoperative analgesia with intrathecal (IT) opioids we rely mostly on morphine. The hydrophilic nature of morphine results in slow tissue take-up and a prolonged duration of action. The so-called 'selective spinal analgesia' produced with IT opioid can result in excellent analgesia with few side effects when compared to other parenteral routes. These benefits most noticeably apply when used for caudad incision sites. We have all convincingly witnessed this analgesic benefit with the use of intrathecal morphine for caesarean analgesia. The key is delivering opioid close to the anatomical site of action, the spinal cord. By not bathing the whole body in opioids, as happens with systemic administration, it is possible to lessen the multiple deleterious effects of opioid action on the brain of: sedation, respiratory depression, and nausea and vomiting.

The use of IT morphine brings a whole host of complexities and controversies and so its popularity waxes and wanes more than that seen for Ray Ban wayfarers.
Many of the concerns about IT morphine are lingerings from days past when fatal and near fatal respiratory depression and sedation resulted from IT morphine administration. This was due to a number of failings at the time, including: administration of large doses of morphine (1 - 2 mg!), failure to appreciate the need for appropriate monitoring of respiratory function and sedation level for an extended duration following administration, and finally, additional opioid doses within the first 24 hours were given at the full-dose rather than being appropriately down-sized.

EVIDENCE

The key summary from APMSE is that intrathecal morphine results in a reduction in overall morphine use and improved analgesia for the first 24 hours following surgery, particularly abdominal (level 1 evidence).
When abdominal surgery is being performed for cancer then IT morphine is well worth considering. This is because there is increasing concern for the tumour promoting effects of opioids and volatiles. Regional analgesia, including IT morphine, offers the ability to reduce both these factors and therefore potentially reduce tumour recurrence rates.

ISSUES TO CONSIDER WHEN USING INTRATHECAL MORPHINE INCLUDE:

Correct dose. The recommendations for dosing of IT morphine are largely dependant on the anatomical site of pain. The more cephalad the incision, the larger the required dose.

LSCS	100 - 150 mcg
TAH	200 - 300 mcg
Radical prostatectomy	200 - 250 mcg
Major abdominal	400 - 500 mcg

NB. Be attentive when measuring doses. Always use the micro dose (0.5 mg) ampoules and a 1 mL syringe.

Respiratory depression and sedation. Overall, the frequency of respiratory depression and sedation with single dose IT opioid is the same, or less than, for parenteral opioid. However, it is worth noting IT and parenteral routes share two similarities for the incidence of side effects: risk increases as dose is increased, and idiosyncratic hypersensitivity responses still occur. These idiosyncratic responses are problematic when they present in the form of extreme sensitivity since delivery of opioid into the IT space essentially functions as a long acting depot. A prolonged period of intensive management is then necessary.

The peak effect of IT morphine on sedation and respiratory depression is at 6 to 8 hours post administration. For most, this time falls safely within the immediate perioperative period of the operating theatre or recovery ward, when close monitoring is employed and so any excessive effect can be safely detected and rectified. The rise to peak effect of IT morphine is also favourable being slow and constant, resulting in a recognisable trend which can be safely enacted on in a considered and timely - instead of panicked - manner.
Following the peak depressant effect there is a steady decline to near-normal response by 24 hours.

Monitoring. The sedation resulting from opioids is a combination of direct sedative effect and CO_2 narcosis secondary to respiratory depression. It is sedation that leads to hypoxia and death. Although in general respiratory depression usually goes hand in hand with increasing sedation this is not always the case. And so a potential trap lies in being falsely reassured by seemingly normal respiratory rates. An example of this trap is to assume an opioid dosed patient is sleeping normally during the night solely on the basis of a normal respiratory rate. The way out of this trap is to awaken the patient at clinically relevant intervals verifying that the somnolence is not drug induced.
The most appropriate monitoring and its frequency following opioid dosing is a source of much debate. The current guidelines for monitoring at RNSH following IT morphine are:
Type: Sedation score, respiratory rate and oxygen saturation
Frequency: Hourly for first 8 hours, second hourly for next 8 hours and then fourth hourly up to 24 hours post injection.

Location: Ward versus ICU. IT morphine is not an independent indication for ICU care. We routinely follow this rule by not sending all our IT morphine caesarean patients to the ICU.
Ward-care eligibility for patients receiving IT morphine is based on patient and surgical factors. Healthy patients receiving IT morphine and undergoing surgery normally managed on the ward postoperatively can be safely nursed on a general ward.
ICU management for IT morphine patients is indicated when the presence of patient risk factors (cardiac and respiratory disease including OSA) or surgical procedure type (pancreatectomy, AAA) would normally mandate ICU care.

Supplemental analgesia. In general and whenever possible the risk of opioid overdose is minimised by avoiding the use of additional opioids within the first 24 hours following IT morphine. Attention to a number of details will greatly increase the chance of achieving this goal:

- Use regular non-sedating multimodal co-analgesics (eg paracetamol, NSAID). Of high importance is to ensure they are actually administered regularly.
- Education. Explain the use of IT morphine; remind the patient and staff the IT morphine is continually acting in the background to provide analgesia, and that if that's all they use, how beneficial it might be.
- Operation type. Some procedures lend themselves well to the sole administration of opioids by the IT route. Caesarean section is a good example owing to the low anatomical site of the pfannensteil incision and important patient motivational factors. Motivated mothers are often keen to avoid an approach that might slow their recovery, such as cumbersome PCA pumps. Also systemic opioids are usually of greater concern to lactating mothers.

Inadequate analgesia and need for supplemental opioid. We already know from opioid dosing by other routes that dose requirements are never a case of one-size-fits-all. This same principal holds true for the intrathecal route, albeit with a narrower range. This is the one reason why supplemental opioid may need to be offered to patients. Another reason for supplemental dosing is for when pain is more dynamic in nature, as experienced with upper abdominal incisions.
Supplemental opioid can be administered in two ways:

1. Low-dose PCA. This is the preferred delivery method should opioid supplementation be required. Low-dose defines a PCA of half to quarter of normal dosing. For an average person ordered a morphine PCA, this works out to 0.5 mg every 5 to 10 minutes. How drastically the PCA dose is reduced (either to half or quarter of normal) is dependent on the clinical circumstance. Take into account patient factors increasing opioid overdose risk (age, co-existing illness including respiratory disease) as well as the level of nursing care planned (ward versus HDU/ICU). If concerned for depressant effects then admit to ITU/HDU, or defer the addition of the PCA until need arises and then have an informed colleague assess the patient at that point in time. The low-

dose PCA is often normalised 24 hours after IT morphine dosing if there is still ongoing need for a PCA.

2. Oral route. When additional opioid consumption is expected to be low and GIT function has been maintained, then the addition of oral breakthrough can be safe and effective eg. oxycodone IR 5 mg Q3H PRN, but usually deferred until 12 hours has passed from the time of IT dosing.

Managing common side effects.

- Nausea and vomiting. Follow the multimodal RNSH PONV management protocol.
- Pruritus (itch). The exact mechanism of neuraxial opioid induced pruritus is unclear. A 5-HT receptor mediated process is suggested by the positive treatment response seen with ondansetron in a limited number of studies. In contrast to the mechanism seen with intravenous morphine, histamine release is not strongly implicated in the itch resulting from intrathecal administration.
    i. Ondansetron 4 mg IV Q12H. Sensible to use first-line as has no real downside, unlike the alternatives.
    ii. Promethazine 12.5 mg IMI Q6H. Although the sedative effect of anti-histamines is best avoided, use occasionally becomes necessary. Patients who become distressed by pruritus can require the sedative action of the anti-histamines so as to break the pruritus-scratch cycle. As with the pruritus from the intravenous route, sometimes the itch from neuraxial use just has to be tolerated for the analgesic benefit it provides.
    iii. Naloxone in a single dose of 80 mcg SCI Q1H or even an infusion of up to 2 mcg/kg/hr may rarely be needed. Larger doses can result in reversal of analgesia.

SINGLE DOSE EPIDURAL MORPHINE

Seldom utilised outside of obstetric practice.
Usual dose range is 3 to 5 mg morphine with dose varying depending on site of incision in a similar manner as for IT morphine.
Similar basic monitoring and precautions apply as for single dose IT morphine.

CONTINUOUS INTRATHECAL OPIOID

A specialised domain of the chronic and cancer pain service.
Similar basic principles of monitoring and precautions apply as for single dose IT morphine.

Perineural catheters

With the growing interest in prolonging the benefits of local anaesthetic we are seeing catheters being placed in a greater number of anatomical sites. This chapter will summarise only the sites likely to be encountered at RNSH.

Interscalene

There is strong evidence to support interscalene catheters over the single-shot approach. Catheters provide an extended period of reduction in both pain scores and opioid use. Some centres even achieve earlier discharge for their patients when sent home with a catheter in place for a further 2 to 3 days. Correct interscalene catheter placement requires a moderate level of technical skill.

Recommended for shoulder arthroplasty
Infusate: Ropivacaine 0.2 % at 6 – 10 mL/hr
Optimal catheter advancement in space: 1 - 2 cm
Duration: for elective surgery is usually 48 – 72 hours

Supraclavicular/Infraclavicular

Advantageous opioid sparing effect.

Recommended for finger reimplants, amputations and total elbow replacement
Infusate: Ropivacaine 0.2 % at 4 – 10 mL/hr
Optimal catheter advancement in space: 2 - 3 cm
Duration: 2 – 6 days depending on circumstance

Paravertebral

An alternative to thoracic epidural for thoracotomy. Usually best placed under direct vision by the surgeon.

Infusate: Ropivacaine 0.2 % at 6 – 10 mL/hr

Inguinal hernia repair/ilioinguinal

Frequently placed in the wrong plane and too far from the ilioinguinal nerve to be of any benefit. Other downsides include extra cost and lack of benefit over the optimal technique of single shot local anaesthetic with multimodal oral analgesia.

Not recommended

Femoral nerve

Femoral nerve catheters producing effective analgesia have the major drawback of the concomitant quadriceps weakness makes walking difficult and unsafe. This impaired mobility in the absence of compelling benefit over single-shot means a femoral nerve catheter is particularly not recommended for analgesia following knee replacement surgery – a surgery for which early mobilisation is thought to be paramount.

There is however some benefit from a femoral nerve catheter for patients after major knee ligament reconstruction, or those planned for prolonged treatment on a continuous passive movement (CPM) machine. Quadriceps weakness is not a major concern for these patients because they will either be mobilising with splint and crutches, or are going to be bed-bound anyway with the CPM.

For ideal femoral nerve catheter placement a dual-guidance technique is recommended. This will ensure the catheter is placed both safely and in close proximity to the femoral nerve. To maintain analgesia from an infusion it is essential to have close proximity to the femoral nerve. Whilst a remotely placed femoral catheter can achieve successful block of the femoral nerve when the large volumes of the initial bolus are given. This will not be the case when the small volumes of an infusion

are administered. The infusion has a large space under the fascia iliaca to spread beneath and might never reach the femoral nerve in sufficient quantities to produce analgesia.

Not recommended for knee arthroplasty.

Recommended for CPM, major ligament reconstruction.

Infusate: Ropivacaine 0.2 % at 6 – 10 mL/hr

Optimal catheter advancement in space: 5 – 8 cm

Sciatic nerve

Recomended for:

-below knee amputation. Often can achieve complete opioid avoidance in a patient population usually having multiple co-morbidities. Placement at the infragluteal level is favoured. (Note: surgically placed perineural sciatic 'sheath' catheters are too distal to be of benefit and are not recommended)

-

Infusate: Ropivacaine 0.2 % at 5 – 10 mL/hr

Optimal catheter advancement in space: 1 – 3 cm

Popliteal fossa

Target site is quite superficial and so catheters are relatively easy to place. Other benefits include a lack of significant impairment to mobility.

Recommended for fractures and major surgery of the foot and ankle, particularly when high doses of opioid would be required otherwise.

Infusate: Ropivacaine 0.2 % at 5 – 10 mL/hr

Optimal catheter advancement in space: 2 – 4 cm

TECHNIQUE OF CATHETER PLACEMENT

The technique to maximise successful catheter placement near peripheral nerves is still controversial. At this stage it would seem the technique of dual guidance with the use of a combination of nerve stimulator and ultrasound will likely emerge as the best overall approach. The use of dual guidance certainly seems prudent for femoral nerve catheter placement for the reasons already discussed. However owing to the confined anatomical space being sought at many of the other sites, ultrasound guidance alone will often suffice.

DELIVERY SYSTEMS

All catheters are to be run using CADD Solis electronic pumps, as they are reliable, cost effective in the long run and enhance safety.

The standard solution is ropivacaine 0.2% delivered from a 200 mL polybag. These are stored in the theatres pharmacy.

All catheters should be run in PIB mode (programmed intermittent bolus). The default setting is 5 mL q30 mins, which is suitable for the majority of catheters. Volume and frequency may need altering using the range mentioned under each catheter type.

DOSING

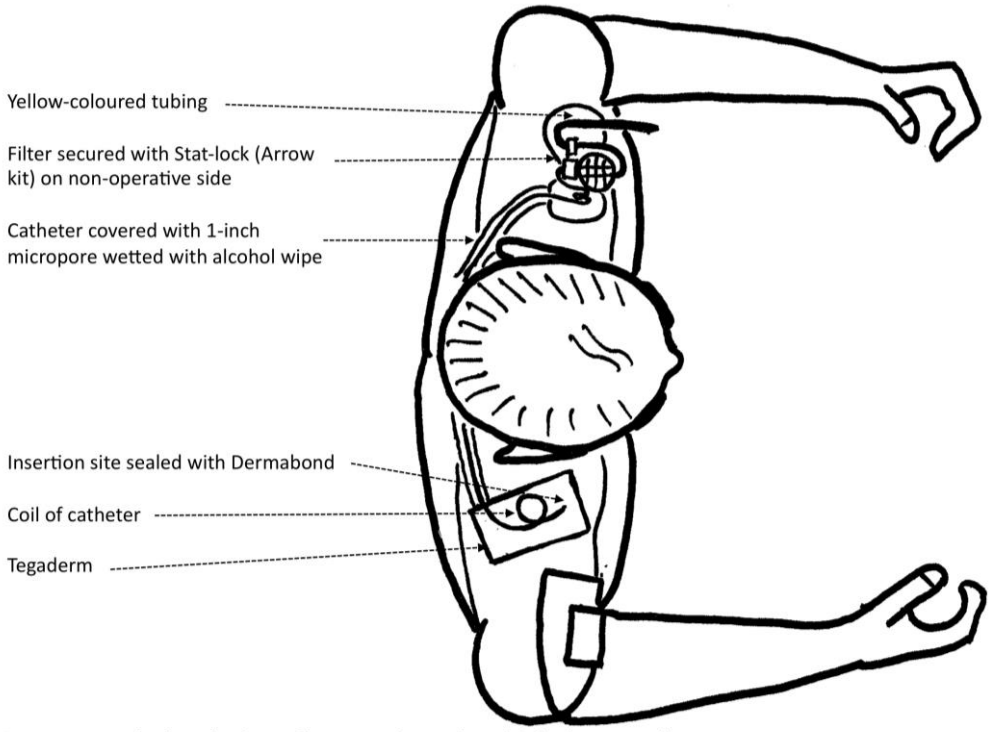
Aim to minimise LA toxicity by limiting dose of ropivacaine to a maximum of 20 mg/hr. Adjust dose down in the presence of low body mass and factors known to enhance LA toxicity - hypercarbia, hypoxia, debility, renal failure and heart conduction defects.

DURATION

Catheter duration should be determined for each case. For elective surgery 2 - 3 days is usually appropriate. For more extensive pain sites, such as an amputation, 3 - 4 days is justified.

SECURING

As for an epidural catheter, fastidious attention must be paid to the catheter securing technique to avoid premature removal. Dermabond applied to the catheter exit site on the skin can reduce leaking of infusate and hence dressing dislodgment. Additional securing techniques recommended (and not shown in the diagram below) now include a Griplock and tunnelling. Try to locate all catheter dressings away from surgical interference especially adhesive surgical drapes that can otherwise too easily pull out your catheter!



Recommended technique for securing a brachial plexus catheter

USE OF CO-ANALGESICS

A catheter system will be utilised when a high degree of postoperative pain is expected, meaning multimodal analgesia will be necessary also. This can include: paracetamol, COX-2/NSAID, gabapentinoid and an opioid.
The method chosen to add in the opioid component, oral or PCA, will depend on patient factors and the certainty of correct catheter placement. If choosing a PCA, avoid fentanyl. This is for the simple reason that need for the PCA usually starts several hours postoperatively at a time point when the all important fentanyl loading dose will be absent. Resultantly, there will be Buckley's chance of achieving therapeutic fentanyl levels in a timely manner. A better choice for this circumstance is morphine PCA.

REMARKS

As a final note: the decision to place a catheter should be based on benefit to the patient. Be wary to ensure catheter placement does not derive from a surreptitious desire to interest up your day.

Ketamine

Ketamine is invariably proffered up as an agent of great promise when managing 'difficult' pain patients. Unfortunately when used in this circumstance the result is often very disappointing. Usually because no amount of fancy pharmacology can overcome the large burden of unhelpful psychology often present in such patients – a case of wrong drug, wrong patient.

INDICATIONS

Overall the evidence to prove benefit with ketamine is marginal and the size of any clinical effect is modest. Despite this less than inspiring fact there are times when use is considered:

Intraoperative: Intraoperative use of ketamine can reduce 24-hour morphine consumption and PONV.
For greatest benefit, intraoperative ketamine use should be considered when high opioid requirements are predicted perioperatively, and particularly when the standard co-analgesics of NSAID, paracetamol, LA or gabapentinoids can not be effectively administered.

Procedural: In IV PCA delivery form co-administered with midazolam for use during burns dressings.

Ward: Episodes of acute on chronic pain - particularly 'neuropathic' pain. Ketamine in this circumstance is used as a third-line treatment.

To facilitate opioid dose reduction in opioid-tolerant patients.

Poor opioid responders. A diagnosis of poor-response should be reserved until an appropriate dose of an appropriate opioid has been administered. As an example, there is little sense in adding ketamine to an anxious patient receiving a fentanyl PCA. In this instance conversion to a hydromorphone or morphine, with their greater anxiolytic actions, should be trialed as the first step before introducing ketamine.

DOSING

Dosing limits in the conscious patient are largely determined by the potential for unpleasant dreams and hallucinations. These adverse psychomimetic effects of ketamine are a dose related phenomenon. The doses used in pain medicine are considerably less than those used in anaesthesia and so are usually associated with a commensurate reduction in adverse effects. Larger ketamine doses can be tolerated if a benzodiazepine is administered concomitantly, as with PCA on the burns ward, or when administered under general anaesthesia.

Intraoperative: Infusion 0.25 – 0.5 mg/kg/hr IV
Single bolus dose 0.25 – 0.5 mg/kg IV

PCA: For routine use on the burns ward only
Order is written as:
Syringe loaded with ketamine 200 mg, midazolam 10 mg and made up to a volume of 20 mL
Bolus dose: 1 mL, equals ketamine 10 mg and midazolam 0.5 mg
Lockout: 3 minutes
Route: intravenous

Ward: Infusion 0.1 – 0.2 mg/kg/hr. This usually works out to be 10 – 20 mg/hr, or 1 – 2 mL/hr of the standard RNSH 10 mg/mL ketamine solution.

ROUTE

The subcutaneous route is a standard for ward use at RNSH. This is advantageous as it frees up IV access for other infusions. The down-side is prolonged subcutaneous delivery can cause local skin irritation, so second-daily infusion site relocation is advised.
Ketamine for peri-operative use can be simply administered IV.

COMMENTS

Patients receiving ketamine can become quite obsessed with the infusion rate. So much so that when planning to switch off the infusion one needs to use a little bit of word magic to prevent patient panic. For example you could say: *"we find that this infusion takes about 3 days to dampen everything down and then after this time it is safe for us to manage you with tablets/a PCA/etc"*.

Caution: Always consider the safety of ketamine use on the ward before commencing therapy. Particularly when, as is commonly the case, administration will be in addition to other sedative agents including high doses of opioid and gabapentinoids. For this reason, senior consultation before commencing a ketamine infusion on the ward is recommended.

Analgesia for fast-track surgery

The development of fast-track surgery resulted from two simultaneous changes in the management of surgical patients: minimally invasive surgery, and a more aggressive approach to postoperative rehabilitation. Dr Henrik Kehlet a contemporary Danish surgeon is probably the most widely known proponent of fast-track. Fast-track is known to reduce hospital length of stay and complication rates. To accompany the marked change in practice by surgeons it is appropriate that we similarly modify our approach to postoperative analgesia. Our use of analgesics must be considered, so they compliment rather than harm, the benefits brought about by surgical fast tracking.

The recommended analgesia regimens place a strong emphasis on the use of multimodal analgesia by the following means:

- Coanalgesics including COX-2/NSAID, paracetamol and gabapentinoid administered at regular time intervals rather than PRN.
- Local anaesthetic. Provision of the highly sort after dynamic pain relief makes the liberal use of local anaesthetic very attractive. However it is possible to cause more harm than good with ill-considered use. Always deliver local anaesthetic in the correct anatomical site and only where benefit has been demonstrated.
- Opioid dose rationalisation. This is with the aim of lessening the deleterious opioid side effects of sedation, apathy, nausea, bowel dysfunction and urinary retention. It is these side-effects that can really slow a patients recovery. An opioid dose is aimed for that minimises these side-effects, but importantly still provides adequate pain relief. Opioid delivery by a PCA is often viewed as the nemesis of fast-track, and so is avoided wherever possible. This is because PCA with the comparatively unrestricted doses deliverable can amplify the unwanted opioid side-effects. PCA also necessitates the use of physically encumbering heavy IV pole with its attached pump. Being connected to drips and machines is also thought to perpetuate the 'medicalisation' of the patient and potentially slow their progress into the rehabilitation phase. For these reasons whenever possible opioid dosing is by the oral route using a combination of slow and immediate release agents.

ANALGESIA FOR FAST-TRACK SURGERY AT RNSH

A significant proportion of surgical cases at RNSH are emergency or complex so we do not see large volumes of fast-track applicable surgeries. Nonetheless, there are a small number of surgeries we can use as examples:

Hemicolectomy – classical or mini incision

Fast-track complimentary analgesia:

- Regular paracetamol and COX-2/NSAID
- Thoracic epidural. To avoid delaying discharge date the epidural is maintained for maximum of 48 – 72 hours. A correctly placed thoracic epidural delivering dilute strength LA has the benefit of allowing both mobilisation and normal bladder emptying, avoiding the need for mobility limiting urethral catherisation. Unfortunately, TEA is not utilised as frequently at RNSH as it could be - largely due to logistical reasons.
- As alternative to TEA: PCA for 24 – 48 hours, followed by oxycodone SR 10 – 15 mg Q12H X 2 days with breakthrough oxycodone IR.
- Note: a true laparoscopic hemicolectomy with a small incision (for the pathology specimen) can often be managed with oral analgesia alone.
- TIVA use encouraged for its advantage of reduced PONV and so enabling successful use of oral analgesia.

Total knee arthroplasty

Fast-track type analgesia: (see included guidelines)

- Regular paracetamol, COX-2 and gabapentin
- Local infiltration analgesia (LIA) preferred, else femoral nerve block
- Short-term PCA (24 hours), followed by oxycodone SR 10 – 30 mg Q12H for 4 days, with breakthrough oxycodone IR
- Spinal anaesthesia encouraged as allows for reduction in PONV, earlier return of cognitive function and reduction in wound infection rates.

LSCS

Fast-track type analgesia:

- Regular paracetamol and NSAID, ensuring the first dose is given in OT
- Neuraxial morphine: IT 100 – 150 mcg or EPD 3 mg
- Oxycodone IR 5 - 10 mg Q3H PRN commenced 24 hours post neuraxial opioid, earlier use permitted after phone consultation with anaesthesia or with specific instruction.

TKA ANALGESIA GUIDELINES

1. **Local anaesthesia** consisting of one, or both:
    1. Local infiltration analgesia (LIA) into periarticular tissues and wound, performed by surgeon. Dose: ropivacaine 0.2 % 100 mL with adrenaline 0.5 mg.
    2. Single shot femoral nerve block (FNB) – preprocedure.
        NB. Of questionable benefit when LIA is planned.
        Recommendations:
        Dose: ropivacaine 0.375% 30 mL (15 mL of 0.75 %) =112.5 mg
        Tips to reduce neural injury: ultrasound guidance, use of nerve stimulator to confirm loss of motor response on reduction of impulse to 0.5 mA, low injection pressure, use of modified fascia iliaca approach.

2. **GA** or **spinal** to complete anaesthesia.
    Note: Addition of IT morphine is not necessary for unilateral knees when local anaesthetic, either as LIA or FNB, is administered.

3. **Post operative opioid**
    Two options:
    1. Super-fast-track (Only recommended in presence of LIA)
        Oxycodone/naloxone SR (Targin) q12h or q8h to commence in immediate postoperative period and continue for 4 days only.
            Suggested dosing: Age < 80 yrs = 20 mg. Age > 80 yrs = 10 mg.
            Am dose to be given at 0600 hrs.
    2. Standard fast-track
        Postoperative PCA: using morphine, fentanyl or oxycodone.
        The order page of the PCA chart will be annotated by the anaesthetist with the wording: *To be ceased on postoperative day 1 just prior to first oxycodone/naloxone SR (Targin) dose.*
        Oxycodone/naloxone SR (Targin) 10 – 20 mg q12h to commence at 0600 am on POD 1 and continue for 4 days only.

4. **Oxycodone** (Endone) PRN 5-10 mg q3h po breakthrough (*when PCA ceased*).
5. **Multimodal analgesia**
    Paracetamol 1 g qid po for 5 days.
    Celecoxib 200 mg bd for 4 days
    Gabapentin 300 mg tds for 4 days. Reduce dose (or avoid) in elderly or frail patients.

Underlined = recommended

G. Pattullo Director APS August 2015

Neuropathic pain

DEFINITIONS

Nociceptive pain: that arising from stimulation of nociceptors
Neuropathic pain: that resulting from damage to the nervous system

INTRODUCTION TO NOCICEPTIVE VERSUS NEUROPATHIC PAIN

The days of dividing the source of acute pain as either purely nociceptive or neuropathic are well past us. Greater understanding of the biological processes, particularly of central sensitisation, and widespread use of the gabapentinoids has led to the realisation that neuropathic and nociceptive pain states share many similarities.
Furthermore, the clinical reality is most patients have pain resulting from injuries that can be thought of as stimulating both nociceptive and neuropathic systems. Few surgeons can perform surgery without dividing nerves and few injuries avoid damaging nerves. Our increasing use of the gabapentinoids is a response to these realities.

DIAGNOSING NEUROPATHIC PAIN

Making a diagnosis of neuropathic pain can be very tricky. This is because many of the classically taught signs and symptoms of neuropathic pain can also be present in nociceptive pain. Try holding your hand in a bucket of iced water, the words burning and tingling would almost certainly describe the sensation. But these are words usually equated with neuropathic pain, and yet clearly this is not neuropathic pain.
The following table highlights the similarities and differences in the signs and symptoms of neuropathic and nociceptive pain.

	Neuropathic	Nociceptive
Symptoms of burning, tingling, shooting	+++	++
Signs of allodynia, hyperalgesia	+++	++
Response to antineuropathic agents	+++	+
Response to opioids	+	+++
Natural rate of resolution	Slow	Fast

Diagnosing neuropathic pain as a major contributor to a patient's pain should only be made after integration of all the clinical information. It is acceptable to include in this having a suspicion of nerve injury based on the mechanism of the injury or type of surgery. For example, if treating a patient who has a traumatic amputation of the arm reporting burning pain it is reasonable to suspect brachial plexus neuropathic pain and promptly commence anti-neuropathic agents.

PHARMACOLOGICAL TREATMENT

There are many agents that can be used to treat neuropathic pain. Agents that should be considered when on the acute pain round because they are both efficacious and have the lowest side-effect profiles are:

1st line	nortriptyline, amitriptyline
	gabapentin, pregabalin
2nd line	tramadol, oxycodone, methadone
3rd line	carbamazepine

DOSING GUIDE FOR ORAL ANTINEUROPATHICS

Nortriptyline

Usual dose 10 – 50 mg nocte,
Better tolerated better than amitriptyline.

Amitriptylline

Usual dose 10 - 50 mg,
Routinely administered nocte to minimise impact of sedation.

Gabapentin

Usual dose 900 - 1200 mg per day
Max 3600 mg per day
Three times a day dosing
Tablet sizing: 100, 300, 400 mg
When used for acute pain (and in contrast to chronic pain setting) dosing is somewhat cavalier; starting doses are higher, dose escalation is rapid and there generally is no need to wean dose on completion of therapy.
Introduce low and escalate dose gradually in the elderly.
Dose-limiting side-effects most commonly encountered are sedation and dizziness.

Pregabalin

Usual dose 150 to 300 mg per day
Max 600 mg per day
Twice daily dosing
Tablet sizing: 25, 75, 150, 300 mg
Dose can generally be escalated more hastily than gabapentin in those likely to have problems, such as the elderly.

INDICATIONS FOR LIGNOCAINE INFUSION

Lignocaine infusion is a 3rd line treatment for neuropathic pain after use of the standard anti-neuropathics and opioids. For cases of severe neuropathic pain, lignocaine can be commenced simultaneously with the other agents in the expectation this will provide a 'bridge' while the other agents build up to their therapeutic peak.
Conditions for which use of lignocaine is appropriate, in order of decreasing likelihood of clinical response:

Peripheral nerve injury
Post-herpetic neuralgia
Diabetic neuropathy
Trigeminal neuralgia
Spinal cord injury pain

EVIDENCE

There is level 1 evidence supporting the efficacy of lignocaine for treating the pain of peripheral nerve injury. Despite this and for a number of reasons, the use of lignocaine infusions is limited to only a few specialised centres.
The mechanism of lignocaine analgesia is thought to be sodium channel blockade inhibiting the spontaneous firing of injured primary order neurons.

TECHNIQUE

Lignocaine is a potentially toxic drug so care must be taken with its use. At the dose ranges used the main concern is for worsening of any underlying cardiac conduction defect. Therefore an ECG needs to be reviewed prior to commencement of lignocaine.

Route:	Subcutaneous
Syringe contents:	Lignocaine 100 mg/mL (Xylocard 100) x 20 mL
Rate:	0.5 – 1.0 mL/hr. Range proportional to weight and presence of co-morbidities
Monitoring:	Blood lignocaine levels should ideally be checked at 24 hours to allow for an infusion rate reduction if level is too high
Duration:	Dependant on response, usually 1 to 5 days

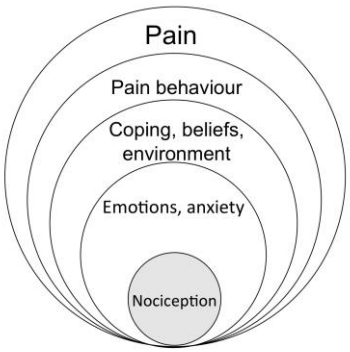
Management of chronic pain patients

Some anaesthetists really struggle with chronic pain patients as they are more comfortable with the predictability and totality of response that comes with general anaesthesia. They are easily unnerved when heading outside the operating theatre to deal with a chronic pain patient and their seemingly belligerent failure to respond to treatment in a predictable and effective manner. Rather than deal with this scenario these anaesthetists usually scurry back to theatre for the calming influence of the Datex monitor with all the pretty colours. But it does not need to be this way for all anaesthetists. Hopefully this chapter will help.

ASSESSMENT

Knowing what one is up against goes a long way toward simplifying management. There are a number of ways to aid rapid assessment of the chronic pain patient:

1. Old notes. Review old notes for details of any chronic pain specialist consults and the outcome. It may even be possible to contact the specialist to gather further information.
2. Onionskin. During assessment think about the onionskin diagram and all the layers adding up to result in the presentation of pain. Try to determine if any layers are over represented. If unable to remember all the onionskin layers then a simpler aid is recalling the first line of the IASP definition of pain: pain is an unpleasant *sensory* and *emotional* experience etc. Trainees should imprint this definition into their memory.



The onion-skin diagram

3. **Yellow flags.** Use the list of yellow flags to identify unhelpful psychosocial factors. Although more correctly yellow flags are applied to musculoskeletal pain, we do find broader clinical generalisations are possible. Yellow flags to look for:

   Cognitions: fear avoidance, catastrophising, sick role
   Emotions: depression, anxiety, stress
   Behaviours: rest, avoidance, pain behaviour
   Compensation (workers)
   Environment: workplace, solicitous partners
   Diagnosis: conflicting, reliance on passive treatments

4. **Red flags.** Always have in the back of the mind, the possibility of a new onset biopathological process such as: infection, fracture, tumour or neurologic injury. The classically taught red flags possibly indicating one of these processes include:

Infection:	Night sweats, redness, swelling
Fracture:	Pain with passive movement
Tumour:	Pain worse at night, pain worse when supine
Neurological:	Numbness, weakness, bladder or bowel disturbance

Identification of any dominant process will allow a better feel for how successful each of the management disciplines of bio-, psycho-, or social are going to be.

APPROACH TO MANAGEMENT

Management will be determined by the information gathered during assessment. Apply the following general principles:

Biological. During training it should have been drummed in that chronic pain is a biopsychosocial problem requiring multidisciplinary management. However when dealing with the chronic pain patient with acute pain, it is necessary to tailor this multidisciplinary approach to therapies readily implemented at the bedside, which are therefore largely going to be biological. So that none are missed, when implementing a biological plan go through a breakdown of the options:

Interventional:	Neuraxial: spinal, epidural
	Regional: single dose or catheter
Pharmacological:	Opioids
	Adjuvants: paracetamol, NSAID/COX-2
	Antineuropathics inc. gabapentinoids
	Others: ketamine, lignocaine, tramadol

Ensure a multimodal drug regimen is instituted since this is the protocol experts and textbooks all recommend. In some patients the overlay of negative psychosocial factors may be so overwhelming a multimodal approach seems futile. Even for these patients though, surprising responses to medications can be seen and so should at least be trialed. On the other hand, having realistic expectations from the start will avoid being disheartened if your drugs don't work.

Psychosocial approach. Owing to the processes underlying acute pain, a biological approach is clearly going to deliver the greatest bang for your buck. Nonetheless, paying attention to psychosocial factors can certainly improve response.
For example, if managing a catastrophiser, tailor the consultation style so as not to feed into this unhelpful behavior. One might say: *"You actually made a really good recovery from your surgery last time and all the signs are you are going to do just as well this time"* etc. One wouldn't want to say: *"well you've just had a massive operation, lost half your blood volume, your surgeon said it was the worst they had ever seen and boy are you lucky to be alive"!*

Power of placebo. This point is mentioned not with the intention of promoting placebo use, but rather to emphasise the treatment response benefits achievable through exploiting both expectation and conditioning. A management plan optimising the placebo response can only stand to aid both you and your patient. As an example: rather than just writing up gabapentin on the chart and walking away (leaving the patient to ponder what on earth these new pills are for), a new order for gabapentin could be accompanied with the wording: *"people find these tablets are very good for controlling the sort of pain you have"* or even, and depending on the patient, one could elaborate: *"it's only been quite recently that we have been able to justify the high cost of these tablets because people find they are so helpful"* etc.

DIFFICULT PATIENTS

Even within the chronic pain population there is a subset we can truly classify as difficult. Managing these difficult patients encompasses the above points with the addition of:

Don't dither, hesitate or be indecisive. Doing so to a seasoned chronic pain patient gives the impression of inexperience, or of not knowing what one is doing. Once such a bad impression is made it can be hard to gain the patient's respect.

Turn on the charm. This really just means being warm and professional, rather than patronising. Remember chronic pain patients have often suffered perceived mistreatment at the hands of health care professionals equipped with poor communication skills. If lack of social skills causes the patient to sense once again that they are about to be treated badly a protective response will be triggered. This response may come in the form of a pre-emptive verbal hit-out, or they may just clam-up. Either way establishing rapport will be a struggle.

Try to avoid arguments. Arguing just sees one going round and round in circles and ends in a lot of stress. This is especially so when dealing with borderline personalities. Borderlines relish the opportunity to prove others wrong and proving a doctor wrong is the grand prize. They are prepared to devote hours in achieving this goal - time we do not have.

Minimise negotiations with opioid misusers. Be firm with doses and clear in future plans. Failure to implement such an approach will see every dose, every day becoming a whole new battle. Document the plan so the next person who rounds is informed and there is no ambiguity.

Competency in opioid prescribing encompasses not only initiating opioid dosing, but also having the ability to wean when the time comes to do so. Unfortunately, too many doctors shy away from this responsibility. We in the hospital have a very important role to act as 'gate-keepers' to curtail inappropriate opioid use and so prevent the harm it can cause in the greater community.

OPIOID DOSING IN THE OPIOID-TOLERANT

The opioid tolerant patient presents in a number of ways; most commonly through use of opioids for pain, less commonly tolerance is encountered from prescription, usually of methadone, for maintenance of the opioid dependant. Irrespective of indication, there are two keys to management:

1. **Maintain baseline dose.** This will avoid precipitating either a pain flare or a drug withdrawal due to under dosing. How the baseline is maintained varies with the clinical scenario:

- Usual dosing route unavailable. Most frequently this arises with oral agents and the need for perioperative fasting. Oral opioids are therefore withheld and replaced with an intravenous equivalent.

  Worked example: Patient having surgery for which nil by mouth post operatively is expected and medications include oxycodone slow release (SR) 60 mg bd.

Morphine daily oral equivalents	= (60 x 2) x 1.5	= 180 mg
Morphine daily IV equivalent		= 180 ÷ 3 = 60 mg
Morphine hourly IV equivalent		= 60 ÷ 24 = 2.5 mg

  Therefore, PCA morphine settings would be:

Background infusion	= 2 mg/hr
Bolus dose	= 1.5 mg, 5 minutely

Setting the PCA this way, with a small under-dosing of the background, adds a margin of safety in case of any postoperative deterioration. Should this background dose be inadequate, the up-sized bolus will allow plenty of scope for the patient to boost dosing. It is reasonable to reduce the background infusion still further if greater concern for postoperative deterioration exists.

- Usual dosing route can be maintained. Alternatively, for the above example if resumption of diet is expected postoperatively and a PCA is still indicated, the maintenance oxycodone SR (60 mg bd) can be continued and PCA morphine ordered with bolus dose of 1.5 mg (Q5 min), but without a background.

  Dose calculations in the opioid tolerant can be tricky at first, so if in doubt check with a senior. Getting the dosing right hopefully will put an end to the baptism of RNSH nights of dealing with a screaming opioid-tolerant patient ordered a homeopathic PCA during the day by a less experienced colleague.

2. **Upsize the dose of breakthrough opioid in proportion to baseline dose:**

- PCA bolus dose. For the majority of patients on less than 300 mg/d morphine oral equivalents, a PCA bolus dose of morphine in the range of 1.5 to 3 mg is usually appropriate.
  For those patients maintained on doses greater than 300 mg/d it is possible to make mathematical PCA bolus dose calculations, but this gets terribly complex and dangerously fails to allow for the heavy burden of unhelpful psychosocial factors frequently present in such patients. For these reasons, consultation with a senior, or preferably the patient's own pain specialist, is advisable before completing the PCA order.
- Oral opioid breakthrough dose. A rule-of-thumb for calculating breakthrough oral opioid doses is to divide the usual daily dose by the number of times the duration of action (in hours) of the breakthrough agent goes into 24.
  For example, a maintenance dose of oxycodone SR 60 mg twice daily would require a breakthrough dose of oxycodone IR (expected duration of action 3 hours) of: 120 divided by 8 (which is 24 divided by 3) = 15 mg third hourly.

  A note of caution in relation to patients maintained on 'industrial' doses of opioid. There is potential for harm posed by the unique combination of staggeringly large calculated oral breakthrough doses along with the unhelpful psychosocial factors often present in such patients when added on top of any possible postoperative deterioration in medical condition. For such cases and to avoid harm, be sure to consult a senior, or again, preferably the pain specialist involved for advice on prescribing.

INTRATHECAL DRUG DELIVERY

When a patient has an IT catheter system, whether fully implanted or percutaneous, one needs to gather a fair bit of information. This will include: by whom, where and when the system was implanted, drug contents, reasons for use, and effectiveness.
Typically contents will be an opioid (hydromorphone or morphine) and clonidine. Less frequently encountered agents are local anaesthetics and baclofen (for muscle spasm of spinal cord injury).
On the whole, it is best not to interfere in any way with the pump:

- Turning the pump off will cause all sorts of headaches, not the least of which will be a calamitous drug withdrawal.
- A well-intentioned accessing of the side port to administer a drug bolus runs the risk of serious overdosing and then of drug withdrawal whilst the pump slowly reloads the catheter.
- Bacterial contamination of the pump with potential for disastrous consequences is also possible if the pump is accessed incorrectly.

Take home message: do not touch the pump! Consult the specialist if alteration to the pump might be necessary.
For patients presenting for surgery with IT pumps the need for supplemental analgesia will depend on the extent of surgery:

- Minor surgery. Can be managed by maintaining all usual medications and adding in a short course of multimodal analgesics in the usual fashion. If an opioid is necessary often only a modest increase in dose from normal is necessary. This surprising anomaly is important to note, since we would expect a major supersizing of breakthrough opioid dose given the equivalent systemic dose of the large IT doses usually being administered. Consultation with the pain specialist is once again recommended.
- Major surgery. Consultation with the pain specialist is now unavoidable.

BUPRENORPHINE

Use of buprenorphine, a mixed opioid agonist-antagonist, has undergone resurgence over the past few years. The low-dose patch form offers mild to moderate opioid analgesia while minimising the harmful side effects of sedation, confusion and constipation. This favourable side effect profile makes the patch attractive for use in the elderly. Much larger buprenorphine doses than are in the patch are given sublingually for maintenance in opioid abstinence therapy.
Two features of buprenorphine generate significant problems when treating acute pain in patients prescribed buprenorphine. The first is the low analgesic ceiling effect, making it ineffective for severe

acute pain. The second is opioid antagonism, partially inhibiting the action of pure opioids should their use be necessary. Clinical management of this varies with the delivery form and circumstance:

- **Buprenorphine patch.** Drug delivery rates with buprenorphine patches are 5, 10 or 20 mcg/hr. These doses are at the lower end of the range so the opioid antagonism is relatively simple to overcome with a standard dose of oxycodone IR 5 – 10 mg. However, if the need for opioid is going to be prolonged then management is simplified, and all doubt on the efficacy of the opioid removed, if the patch is just removed. Following removal, drug elimination is slow with a half-life of 12 hours and care must be taken to avoid accidental opioid overdose due to unopposed opioid activity.
- **Buprenorphine sublingual.** Clinical doses for opioid abstinence therapy are up to 32 mg/day , brand names encountered include Subutex, and Suboxone. Patients on these higher doses can be a little tricky to manage - often this is more to do with psychology than pharmacology. The clinical scenarios encountered include:
    - Emergency major surgery. In this circumstance, buprenorphine has usually been consumed in the preceeding 24 hours. Postoperative analgesia should focus on provision of neuraxial or perineural local anaesthetic with placement of a catheter to obtain maximal benefit. If a PCA is also needed, our experience is that a high-dose morphine PCA (eg 2 – 3 mg bolus) is usually satisfactory. Sublingual buprenorphine is continued. Note: this recommendation; of a morphine PCA and continuation of the buprenorphine, is contrary to the previously taught practice in this scenario of buprenorphine cessation and a high dose fentanyl PCA. Patients, seemingly paradoxically, consume less PCA opioid when maintained on their usual buprenorphine dose. Secondly, the use of fentanyl, perhaps owing to its pharmacodynamic properties, frequently failed to provide satisfactory analgesia to these patients.
    - Elective major surgery. Advise patient to continue on their buprenorphine preoperatively. Postoperative analgesia should focus on non-opioid based techniques. If a PCA is needed, again high-dose morphine is recommended. Continue usual dose of buprenorphine in post-operative period. Note: this recommendation is contrary to practice until very recently, which was preoperative buprenorphine cessation and rotation to an oral pure opioid agonist (eg MS Contin). In practice this was cumbersome to undertake as is was often resisted by the patient, who due to their personal experience of opioid dependence, were keen to avoid them altogether.
    - Elective or emergency minor surgery. Maintain buprenorphine dosing and maximise multimodal agents including local anaesthetic. The need for full opioid agonists can usually be avoided, however if needed may not always need to be dramatically supersized. Many clinicians recommend use of morphine oral tablets (eg Sevredol 10 – 30 mg) in this circumstance, rather than oxycodone, owing to reduced likelihood of misuse.

METHADONE

Whenever possible and appropriate, plan to continue a patient's methadone dose in combination with any additional analgesics. This is for two reasons: firstly, if the patient is being treated for opioid addiction this will maintain a stable drug concentration and hence a far more compliant patient, and secondly, trying to achieve equivalent opioid analgesia with other opioids can require an extraordinary number of tablets. Drug and Alcohol usually take responsibility for methadone prescriptions for addiction control whilst these patients are in hospital, therefore any management plan to alter the methadone dose is best discussed with them first.

OTHER MANAGEMENT ISSUES FOR THE OPIOID TOLERANT

- When formulating a management plan for the opioid tolerant patient a multimodal drug approach is expected. For reasons discussed earlier in this chapter the response can be varied and is patient specific.
- Always bear in mind some patients are maintained on long-term opioids because they have a number of unhelpful psychological factors preventing them from managing their pain without strong medications. Dealing with these patients can be particularly challenging.

- Despite what many chronic pain patients believe, it is not possible to safely extinguish all components of the pain experience with pharmacology.

Tip
Whenever possible and appropriate, involve the treating pain specialist in their patient's acute pain management. At a minimum, if the specialist is from RNSH, all this requires is a courtesy-type page to the Chronic Pain Fellow. Remember at RNSH there is consultant cover 24/7 for both Anaesthetics and Pain. Consultants are available (and expect) to be called for the difficult cases.

A day in the life of Dr Gaz Mann

Pain management in patients with severe burns

"You will never find the Professor of Greek in the Burns Unit" - Ross MacPherson 2003

Unlike many other types of pain, there have been very few high quality trials conducted on burns patients. Real evidence on best treatment and practice is lacking and most burns unit protocols reflect local protocols or long-held practices. In NSW there are two Severe Burns Units, one at RNSH and one at Concord Hospital. There is excellent communication between the two units and a high level of concordance with regard to patient management. Patients can (and do) move between the two units knowing that the drug protocols and management strategies will be essentially identical.
It is important to understand the likely course of events that befall the burns patient. On admission the most severe burns patients will most likely be admitted to ICU and remain intubated and ventilated for an initial period, only coming to SBU when they are stable, which may only be after a period of weeks.

ONGOING PAIN

Many patients have surprisingly little ongoing pain in between procedures, while others can have significant distress. Because many of these patients will be in hospital for weeks, we must be prepared for long-term use of opioids, and the development of tolerance. Even though many of these patients will commence early enteral feeding, and could therefore theoretically be commenced on oral analgesia, they will often be having regular painful procedures, necessitating parenteral analgesia. It is usually easier to keep patients on their PCA until the procedures have ceased, or at least are occurring only intermittently.

OPIOID ROTATION

The development of tolerance in these patients can be a significant issue. For this reason, chronic burns pain is one of the few instances encountered on the acute pain round where the need for opioid rotation is likely to be encountered. (The other circumstance where opioid rotation is most frequently undertaken is for chronic cancer pain and hence its practice is not often necessary on the acute round). Opioid rotation addresses the issue of within-patient variability in opioid responsiveness by identifing the opioid that provides maximal analgesia with the fewest side effects. For the chronic burns patient the most successful rotations are usually between oxycodone, morphine, hydromorphone and methadone. Rotation to methadone in particular often brings success – possibly because of its additional NMDA receptor activity.

PROCEDURAL PAIN

Initial burns treatment usually consists of debridement of the burns, changes of dressings, and where necessary, skin grafts. Skin grafts will always be carried out in OT, but debridement and dressings changes may be carried out either in OT or in the unit, depending on the extent of the procedure and the condition of the patient. These procedures may be carried out every couple of days during the patient's stay. It is important to recognise this fact since while the patient is undergoing repeated procedures, even though they may be intermittently eating, it's easiest to maintain them on parenteral analgesia until the conclusion of the procedures. Clearly, these procedures can be associated with considerable pain unless adequately managed.

There are three options namely, intermittent nitrous oxide, parenteral opioids, and ketamine/ midazolam PCA. Often, one has to wait until the patient undergoes their procedure to see which of these options will be most appropriate for them, although nursing staff are usually pretty good at predicting.

1. Entonox

Entonox is easy to use and has a high patient acceptance. In the past, nitrous oxide was used on a rather ad hoc basis, with, at the time, dire consequences. Consequently, it now has to be prescribed appropriately like any other drug. As well as giving clear instructions on its administration, folic acid and Vitamin B12 must also be prescribed concurrently while nitrous oxide is being administered. Checking homocysteine levels every week or so is also recommended.

2. Opioids

Administration of intermittent opioids is clearly the easiest option. This is best done by having an order for say IV fentanyl 15 mcg boluses every 2-3 minutes. Boluses should be commenced about 5 minutes prior to the commencement of the procedure. If the patient is already on a PCA, it can be used, but the higher levels of procedural pain coupled with the five minute lockout, makes this less than optimal.

3. Ketamine and midazolam PCA

Ketamine and midazolam administered via PCA or nurse administered, has proved a success in treating pain associated with large dressing changes. One key benefit is the avoidance of prolonged fasting periods prior to use, so allowing patients to maintain the high calorific dietary intake needed for wound healing.

The PCA order is recorded as:

Syringe Prescription:

Ketamine	200 mg
Midazolam	10 mg
NS 0.9%	to make volume
Total volume	20 mL (usually made up in a 60 mL syringe to fit the Alaris pump)

PCA Order:

Bolus	1 mL (= ketamine 10 mg and midazolam 0.5 mg)
PCA lockout	3 minutes

Despite all of these measures, some patients will still need to have a general anaesthetic for their dressings changes. It is important to recognise all of the above strategies have their limitations, and if the procedure is too painful, then there is no other option but to proceed to theatre. With the advent of the targeted Burns Theatre, these cases can usually be done quite promptly, but otherwise placing such patients on the general theatre list usually means prolonged fasting in a patient already in a highly catabolic state.

Many Burns units consider that there is a large component of neuropathic pain in burns injuries and routinely prescribe agents such as gabapentin or mexiletine. We don't do that as a routine at RNSH, but the possibility of a need for such agents needs to be kept in mind.

CO-MORBIDITIES

Many of the patients you will see in SBU are there because of misadventure, often involving drug abuse or ethanol. Many of these patients often have significant psychiatric and social issues to contend with. The importance of this is two fold. The first is that you must consider concomitant drug and alcohol use when prescribing in this group. Second there may be significant behavioural issues to be considered when interacting with these patients and lastly, multiple groups may be involved in the patients care such as liaison psychiatry and drug and alcohol. The importance of this is that all groups may be prescribing, and we need to ensure that out analgesic input is appropriate with medications being prescribed by other groups.

METHOXYFLURANE FOR OUTPATIENTS

The SBU has an extensive Outpatients service for patients who come for ongoing assessment and dressings changes. For some time now this clinic has been using Penthrox (methoxyflurane) inhaler for analgesia during dressings changes. Although the APS is not directly involved in this service or in the use of the Penthrox, you should at least be aware that it is being used in the SBU.

Pain and the spinal cord injured (SCI) patient

RNSH is one of just two hospitals in the state that deals with acute SCI. It is unlikely therefore that you will have come across such patients in your previous medical experience.

Obviously the recently arrived patient with a catastrophic injury will have a host of emotional and psychological issues that need to be considered and a caring and concerned approach to these patients is obviously paramount.

NEUROPATHIC PAIN

The outstanding issue with SCI is acute neuropathic pain, which can be severe. In the acute injury, patients with SCI usually present with the condition known as "spinal shock". Amongst other things hypotension and a complete (or near complete) cessation of gastro-intestinal tract function are common. This means that patients are usually on naso-gastric suction and are nil by mouth for some time after their initial injury until bowel function ahs recovered.

Because of this our management options are limited and subcutaneous lignocaine infusion is the most appropriate management. As oral intake is established, standard oral anti-neuropathic agents can be instituted. In addition to gabapentin, amitriptyline is often added in a night-time dose.

Patients usually remain in RNSH for some weeks to months following their injury before being transferred to Rehabilitation. During that time it is appropriate to slowly reduce anti-neuropathic agents to ascertain if they are still needed.

OTHER PAIN ISSUES

Almost all patients have received their SCI as a result of considerable trauma and we must not forget pain from other sites as a result of the accident. It is fairly standard practice for patients with acute SCI to be taken to theatre as soon as practicable after admission to stabilise the spine, if an unstable fracture is present.

Hence, as well as neuropathic pain, we must consider post-operative pain as well as other injury pain.

Almost all SCI patients will have what is called an "incomplete" lesion. That is to say, there is still some pain and sensory impulses being transmitted to higher centres even from below the level of the SCI. As well as this hyperalgesia may be present. Treatment of this type of pain can initially be via PCA or oral opioids as appropriate.

Depending on the level of the SCI, the patient may be able to use a standard PCA button. Where actuation by the hands is not possible, ward 7D can arrange the PCA to be breath actuated.

Some patients with SCI report severe muscle spasm. Treatment is almost always unsatisfactory and is managed either by APS or by the treating team. Both diazepam (2-5 mg t.d.s) or baclofen (5 mg t.d.s) have been employed.

THE "CHRONIC" ACUTE PAIN PATIENT

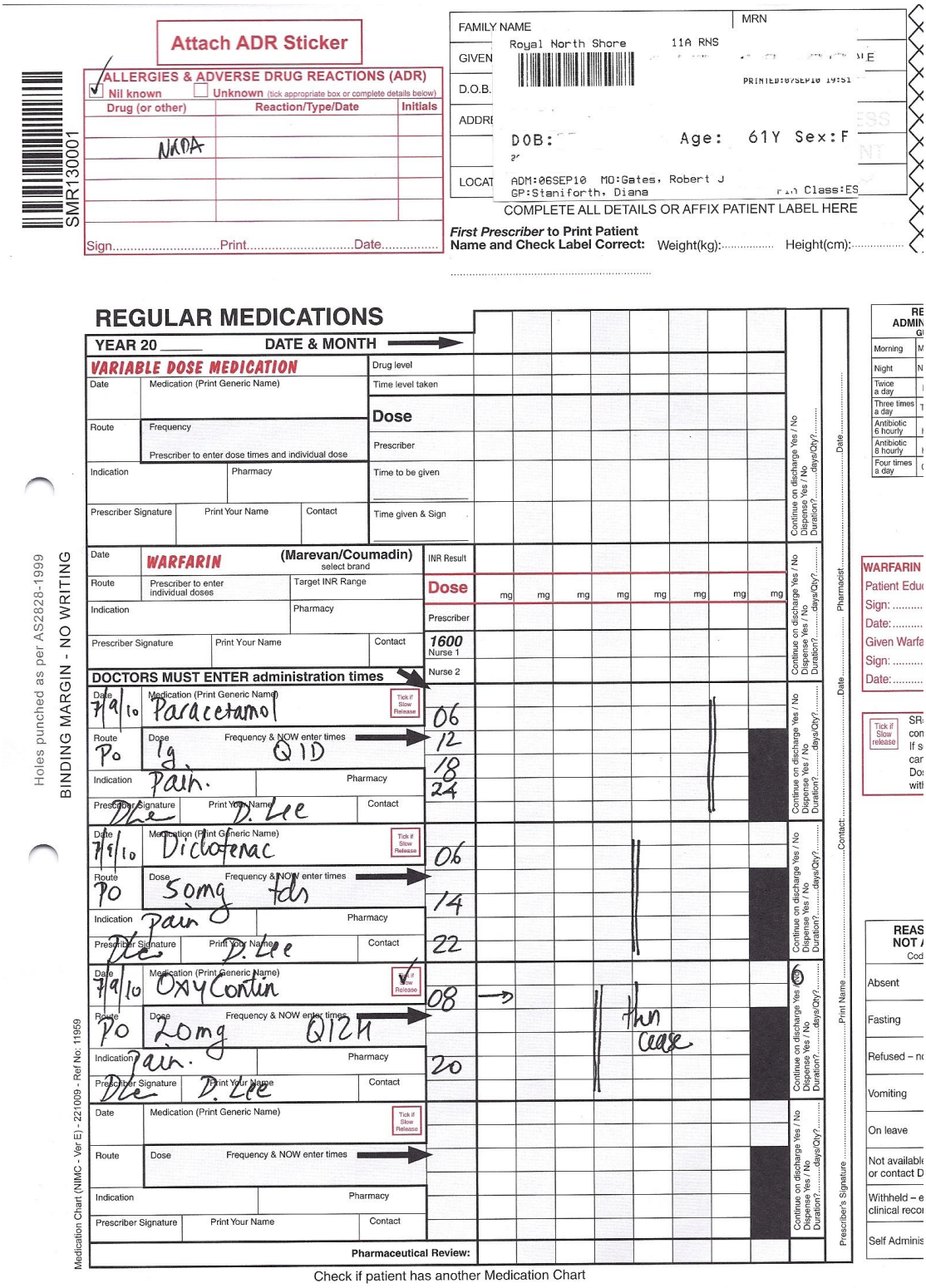
As with patients with severe burns, SCI patients usually remain under the care of the APS during their stay. Their pain issues are usually greatest in the early phase of their management but tend to resurface again once physiotherapy and mobilisation commences. In the early days, these people will be seen on a daily basis, but as their condition stabilises, we will usually see them once or twice a week, or as issues arise.

OVERVIEW

Most patients with SCI have an incomplete injury with some awareness of pain/sensation below the level of the lesion, and these sensations can be very troublesome indeed. Add to this that fact that many SCI patients will undergo some form of spinal surgery soon after admission, not as curative procedures, but to stabilise the spine, and you can see two pain types, neuropathic and nociceptive, intermingled creating quite a confusing picture.

Appendix A

Example of recommended prescribing practices on the National Inpatient Medication Chart



Attach ADR Sticker

See front page for details

AS REQUIRED "PRN" MEDICATIONS

Year 2010.

FAMILY NAME	MRN
GIVEN NAME	☐ MALE ☐ FEMALE
D.O.B. ____/____/____	M.O. NOT A VALID
ADDRESS	PRESCRIPTION UNLESS
	IDENTIFIERS PRESENT
LOCATION	

COMPLETE ALL DETAILS OR AFFIX PATIENT LABEL HERE

First Prescriber **to Print Patient Name and Check Label Correct:** ....

NOT A VALID ORDER UNLESS LEGIBLE

Field	Entry
Date	4/9/10
Medication (Print Generic Name)	Oxycodone
Route	PO
Dose & Hourly Frequency	5-10mg Q3H PRN
Max dose/24 hrs	60
Indication	pain
Pharmacy	
Prescriber Signature	DLee
Print Your Name	D. Lee
Contact	
Date / Time / Dose / Route / Sign	NOT WHILST ON PCA
Continue on discharge Yes / No; Dispense Yes / No; Duration?........days/Qty?........	

Each of the seven remaining blocks is blank and prints the following fields:

Date | Medication (Print Generic Name) | Route | Dose & Hourly Frequency PRN | Max dose/24 hrs | Indication | Pharmacy | Prescriber Signature | Print Your Name | Contact | Date | Time | Dose | Route | Sign | Continue on discharge Yes / No | Dispense Yes / No | Duration?........days/Qty?........

Date .... Pharmacist .... Date .... Print Name .... Prescriber's Signature ....

BINDING MARGIN - NO WRITING

Holes punched as per AS2828-1999

SMR130001

Check if patient has another Medication Chart

4