Pain Management in Trauma

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- The stress response after multiple trauma is far greater than after elective surgery3 and includes cytokine and acute phase reactant release, impaired coagulation and immune response and accounts for a large portion of mortality in trauma patients. Provision of good pain relief will decrease the stress response and long term psychological sequelae of injury [1].
- Acute pain during trauma may progress to chronic pain. Effective early intervention may prevent or minimise the development of chronic pain, and appropriate psychological support and non-pharmacological means of treatment have a vital role to play [2].
- Non pharmacological measures such as splinting, reduction of fractures, irrigation of burns and covering burns and raw areas with saran wrap might help minimize pain.
- Analgesic agents can be administered via multiple routes- IV, IO, IM, Intranasal, Subcutaneous, Transdermal, Oral, oral transmucosal and inhalational.
- Ketamine is a useful adjunct to patient controlled analgesia regimens [3, 4]. There are now consensus guidelines available on the use of IV ketamine for acute pain management from the American Society of Regional Anesthesia and Pain Medicine, the American Academy of Pain Medicine, and the American Society of Anesthesiologists [5]
- There are multiple regional anesthestic techniques outlined in the chapter that can be utilized to provide analgesia to the trauma patient. Brachial plexus blocks for upper extremity analgesia, femoral and sciatic nerve blocks for lower extremity analgesia, fascial plane blocks (serratus anterior, erector spinae, paravertebral) and thoracic epidural for thoracic trauma.
- There is currently no evidence to suggest that regional anaesthesia prevents the diagnosis of compartment syndrome or delays its diagnosis if the patient is appropriately examined, though many surgeons still believe this to be the case [6–10]

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- An assessment of pre-existing nerve function should be performed and recorded before any block is attempted.
- For patients in severe pain and or unable to tolerate oral medication, an IV analgesic regimen in the form of patient controlled analgesia (PCA) can be initiated. Prior to initiating patient controlled analgesia consider age, ability to comprehend instructions, physical ability to use the infusion pump and comorbidities.

While the need to treat pain for humanitarian and physiological reasons is entirely obvious, the evidence is that pain is managed poorly and disjointedly at every stage of a trauma patient's journey [11]. Who is responsible for pain management now? Who is responsible for pain management in the next few hours? Who is responsible for ongoing pain management? Is there an overriding plan and will any interventions now limit analgesic options later (e.g. anticoagulation and regional anaesthesia)?

Assessing Pain

Pain, according to the International Association for the Study of Pain, is defined as an unpleasant sensory and emotional experience associated with actual or potential tissue damage or described in terms of such damage. There are multiple modalities of pain relief which can be utilised. However, the trauma patient poses some particular challenges that may limit how we can employ them.

The dogma that analgesia should not be given [12] lest it mask subtle surgical signs or cause gross haemodynamic instability has been debunked and is not supported by the current evidence base; however it is still a view held by some clinicians. The stress response after multiple trauma is far greater than that after elective surgery [13]. This response, includes cytokine and acute phase reactant release such as elevated levels of catecholamines, cortisol, growth hormone, and adrenocorticotropic hormone. It also causes activation of the rennin angiotensin system, impairment of coagulation and an altered immune response, and accounts for a large portion of the mortality in trauma patients. In several studies, inadequately treated acute pain has been shown to increase this response, resulting in higher morbidity [14]. The provision of not just adequate, but good pain relief is not only a humane aim but also will decrease the stress response and the long term psychological sequelae of injury [1].

The requirement for analgesia may also vary between injuries and between individuals; there may be up to a tenfold inter individual difference in the required doses of opioid analgesics such as morphine leading to a reluctance to give larger doses despite having a patient that is very much awake and still in pain. This is aside from concerns around long term issues such as addiction. The differences are because of physiological or pharmacological aspects (e.g. some patients will be ultra-rapid, rapid, slow or even non-metabolisers of some drugs such as codeine) and the psychological suppression or down-regulation of pain pathways depending on the context of the injury [15–18].

The anticipated duration of severe pain also plays a part in agent choice—e.g. for a short but painful procedure such as relocating a joint or fracture/dislocation, potent agents are needed to provide good analgesia for a short period. Once realignment has been achieved however, the amount of pain from the injury dramatically decreases. If a potent, long-acting analgesic and/or sedative is given (e.g. morphine and midazolam), then once the painful stimulus is removed there may be a rebound over-sedation or increased rate of side effects and complications such as respiratory depression.

The measurement of pain is essential in targeting the appropriate treatment, and as previously mentioned each individual's perception of pain will differ depending on circumstance. There may be a hyperacute suppression of what looks like a catastrophic injury—for example, the injured soldier missing an arm in the middle of a firefight but

who declines analgesia. At the other end of the spectrum is what appears to be a minor injury that ultimately requires multiple modalities of treatment, as is seen in chronic regional pain syndrome (previously known as Sudeck's atrophy, reflex sympathetic dystrophy or causalgia).

The time from injury and surrounding psychological issues also play an important role. Although the primary concern with pain in trauma is acute, this may progress to chronic pain. Consequently, the treatment goals may change from the complete alleviation of pain where possible in the acute phase, to strategies aimed at coping with pain on a day to day basis to allow a return of as much function as possible. Effective early intervention may prevent or minimise the development of chronic pain, and appropriate psychological support and non-pharmacological means of treatment have a vital role to play [2].

Firstly, an assessment must be made of the magnitude of pain as this will guide the level of intervention needed. A patient with a pain score of 1/10 will not require strong opiates, and conversely, a patient with a 10/10 rating will require more than paracetamol! Multiple systems can be used, but a 0-10 scale is the most commonly used in the UK with 0 representing no pain and 10 being the worst pain imaginable. Other indices are available (e.g. 0-3 scale, mild/moderate/severe rating, "smiley faces", FLACC scoring) and the clinician should ultimately use whichever one they are most familiar with and can be most easily understood by the patient; certain pain scores have been optimised for certain patient populations (e.g. FLACC for children and PAINAD for patients who have dementia). Potential pitfalls include patients who cannot speak either due to injury, age, language barriers or mental disturbance, or patients who are intubated and assumed to have an adequate degree of analgesia.

With the exception of ketamine [19], none of the standard anaesthetic induction agents or benzodiazepines have any analgesic action. Whilst anaesthesia may be considered as a highly invasive form of analgesia for humane reasons in some extreme circumstances, induction and maintenance of anaesthesia will not provide analgesia to obtund the physiological responses to pain. An intravenous opiate or opioid should be considered in these patients to treat pain even when anaesthetised. Opiates are naturally occurring compounds, i.e. morphine and diamorphine, whereas opioids refer to both the natural opiates and also the synthetically created compounds based on opiates, e.g. fentanyl, alfentanil, pethidine, etc.

A reassessment of pain after an intervention is always required as the clinician may be tempted to try and give repeated doses of drugs which are not indicated and run the risk of increased side effects without clinical benefit, or possibly not give more analgesia when it is needed. The likely clinical course of a patient should also be considered when selecting and implementing an analgesic strategy. An extreme example would be a patient who is trapped by their leg in a motor vehicle accident and currently complains only of 3/10 pain. If the scene becomes unsafe or a patient deteriorates drastically and there is a need for amputation for rescue purposes, ketamine analgesia/anaesthesia would be appropriate in this case. A less extreme example would be the same patient who is about to be extricated with a severe compound fracture of the leg. Although they may complain of 3/10 pain at the moment, moving the car and hence the fracture fragments which may be relatively splinted by the vehicle position will be painful. Either fentanyl, morphine, ketamine or some other analgesic reserved for severe pain may be indicated in anticipation of this.

The following segments of this chapter will be directed at specific analgesia modalities in certain kinds of trauma but it is far from comprehensive. Familiarity with drugs is paramount, as acute traumatic patients represent several management problems and it would not be appropriate to use a new drug or technique on these patients unsupervised for the first time. Existing experience with specific techniques (such as regional anaesthesia) or medications (such as ketamine) is essential before being tried in the trauma arena, as liability for the use of said techniques lies with the individual practitioner.

Pre-hospital practitioners may be faced with a large number of problems in the undifferentiated trauma patient, and good analgesia may make their subsequent management easier. Initial management with reassurance and simple splintage will provide a degree of relief which may be sufficient on its own; however several other options can be employed.

Non-Pharmacological Treatments

Splinting

Splinting not only provides analgesia when the patient is still but may also provide a degree of pain relief when the casualty is moved by preventing fracture ends from rubbing together. The reduction of fractures back into their normal anatomical alignment also decreases bleeding and the risk of fat embolus from long bone fractures. Specific devices may be used (e.g. Kendrick splints for lower limb fractures), or splints may be improvised (e.g. using a bed sheet or triangular bandage for an improvised pelvic splint) if a bespoke device is not available.

Reduction

The management of dislocations also relies on splinting techniques, and the dislocated limb should be splinted in the position it is found in if that is comfortable for the patient and there are no contraindications. An assessment of distal neuromuscular status should be made before and after moving the limb. Following a radiological assessment, limbs should be reduced into the neutral position and splinted in place with further orthopaedic input as needed for ongoing management.

Limbs that are ischaemic secondary to dislocation require urgent reduction; this may be in the pre-hospital environment without the benefit of access to x-rays pre-procedure. Reduction in these instances provides significant pain relief almost instantly. Be very cautious of using long-acting opioids before reduction, as over-analgesia post reduction can occur with a potential for complications such as respiratory failure, nausea and vomiting.

Irrigating

Burns should be cooled and irrigated with normal saline prior to dressing. In the case of chemical irritation, dry chemicals should be brushed away, followed by copious irrigation to dilute the irritated area. The inflamed area should be dried and be managed according to local burns protocols as needed.

Covering

Covering of raw, exposed tissue protects from further irritation from both friction and air moving across exposed nerve endings. Plain cling film (aka Saran wrap) is immensely useful in the pre-hospital and early emergency department management of such pain, particularly following burns.

Routes of Drug Administration

An ideal analgesic agent has high patient satisfaction with few adverse events and effects. Some drugs can be given by more than one route, e.g. diamorphine, fentanyl and ketamine can all be given intranasally as well as intravenously. The following is a very brief overview of the different routes available for administration.

Intravenous (IV)

The most frequent route of administration of drugs for rapid onset and most familiar to acute healthcare providers. It requires a cannula that is working and an intact vascular supply to the central circulation for onward distribution. Medications may be given in the form of a bolus, infusion or as patient controlled analgesia for narcotic pain medication.

Interosseous (IO)

The interosseous route was popular historically in paediatrics and is becoming more and more practised in the adult population with the development of devices like the EX-IO (Fig. 13.1), FAST-1, FAST-Responder and BIG (Bone Injection Gun). The IO route has all the advantages of IV access, is less likely to become dislodged and has few drawbacks on first assessment [20–23]. It is increasingly used in both military and civilian practice, with the caveat that the IO should be removed as soon as is reasonably practical (i.e. when reliable IV access has been secured). It should not be left in for more than 24 h, and depending on the device used it may render some military personnel non-deployable for up to 1 year regardless of other injuries.

The risks associated with IO access are misplacement, joint injury and osteomyelitis, though these complications are rare if the device is used correctly. There are very few contraindications to IO use, and these are all relatively self-evident—overlying skin infection, fracture proximal to the site of insertion (e.g. femoral or pelvic if tibial site used, humeral if humeral head used, sternal fracture or previous sternotomy if FAST-1 used) overlying burns, inability to locate anatomical landmarks, previous joint replacement at the insertion site or patient refusal.

The insertion of various devices has not been described as unduly painful, but the initial flushing and overpressure of the marrow cavity of the bones has been reported as transiently very painful and lasts a couple of seconds. There has been the suggestion of flushing the IO device with 2% lignocaine, and various protocols exist regarding insertion. However, in the severely obtunded patient, this is probably unnecessary.

Various sites of insertion have been described (medial malleolus, medial tibia, distal femur, pelvic crest, distal radius and humeral head), and whichever site is chosen must be cleaned and inspected for contraindications before insertion of the needle. All acute drugs that are given IV (with the exception of bretylium, a historical antiarrhythmic agent no longer commonly used) may be given through an IO needle. The speed of access has been reported to be faster than establishing IV access in some cases [23, 24], and the IO device has also been recommended in ALS guidelines for use in cardiac arrest due to ease of use and decrease in time to establish access in the arrested patient. The increased use of the IO route has led to several recent papers comparing the merits and drawbacks of each site. In an animal

Fig. 13.1 EZ-IO needles and driver. This is one of the most commonly used IO systems in use

model, sternal and humeral routes outperformed tibial in terms of producing higher mean maximal plasma concentrations of adrenaline in cardiac arrest, with the tibial route leading to a statistically significant lower plasma concentration when compared with the IV route. This difference was not seen with other IO routes, however the time taken to reach maximal concentrations was lower in the tibial group than other IO sites, and even lower than the IV route [25]. This may be significant, as a subgroup analysis of the Continuous Chest Compression trial showed that IO access was significantly associated with a lower rate of return of spontaneous circulation (ROSC) than IV access, despite marginally faster times to initial drug administration. There was no overall effect on mortality, or survival with favourable neurological outcome after adjusting for population variances [26].

Speed of establishing access is important when considering various IO routes in comparison to IV. A randomised controlled trial by Reades et al. [27] showed a higher first pass success rate with a lower time to initial success when using the tibial route compared to humeral IO or peripheral IV access, with tibial IO access is less likely to dislodge. In the FAST-1 sternal IO device, the time of spread to the central circulation has been shown with dye testing to be the same as drugs given via a central line [28].

Whilst IO access may be faster and perceived as easier in some cases, they are still not infallible. In a recent paediatric post-mortem study, up to 30% of IO devices were found to be improperly placed (i.e. not intramedullary) [29]. Whilst these findings may not be generalisable to adults (as the trial has not been done) they do serve as a warning that IO access may not be as easy as first thought.

Intramuscular (IM)

The IM route has been used historically, but due to unquantifiable differences in muscle perfusion in trauma has fallen out of favour. The speed of absorption of drugs from IM injections is highly variable and unreliable, and if the patient is hypovolaemic and not perfusing their muscles, then the drug may not reach the circulation to have an effect. This may be further compounded by multiple doses being given to achieve an effect, and when the patient is resuscitated and normal perfusion restored, a large amount of drug may suddenly be dumped centrally causing an overdose. There is a current trial in the design phase (The Trauma INtramuscular Tranexamic Acid Clinical Trial (TraumaINTACT)) [30] to use intramuscular tranexamic acid auto-injectors in trauma, where the skill of IV access is not available such as forward military units. Occasionally, tetanus toxoid immunisations are given IM in trauma, but this is the only routine use for the IM route in trauma patients.

Intranasal (IN)

Intranasal administration is a popular route in paediatrics and becoming more popular in adults. Ketamine, diamorphine, fentanyl and dexmedetomidine (Precedex) have been used with a mucosal atomiser for analgesia and anxiolysis. Drugs given by this route benefit from fast onset speeds and good bioavailability, with few if any contraindications and a bonus that naloxone can also be given intranasally if an opiate overdose is suspected. It is very good for children who are in pain and needle-phobic [31–34].

Subcutaneous (SC)

Subcutaneous injections are no longer used in acute trauma care (with the exception of giving prophylactic low molecular weight heparin for DVT prophylaxis), but subcutaneous drugs can be given in palliative care via syringe drivers.

Transdermal

The transdermal route can be used in chronic pain with fentanyl or buprenorphine patches, but these are inappropriate in the emergency setting as the onset time of medications given by this

route is measured in hours which precludes their use in acute trauma. However it is essential to look for patches that the patient may have been wearing at the time of their injury as this could cause either an overdose of opioid if left in position, or potentially an underdosing in acute pain if the patient is extremely opiate tolerant and a "normal" dose is ineffective in relieving acute distress.

Oral

This can be a useful route of drug administration in the ICU, and in minor trauma. However absorption from the GI tract may take a prolonged period in trauma due to hypoperfusion and shunting of blood away from the gut in hypovolaemia. Some drugs which are useful in managing subacute or chronic pain (e.g. amitriptyline, gabapentin, pregabalin) may have to be given orally as there is no IV alternative.

Oral Transmucosal

Transmucosal administration of fentanyl can be achieved via either "lollipop" or lozenges that are held between the lip and gum and is of use if immediately available as the patient is in control of their level of analgesia. American military practice [35] suggests taping the stick of the lollipop to the patient's finger so if they become obtunded then they will remove the lollipop when their hand drops out of their mouth. The relatively shorter half-life of fentanyl in these patients means that the duration of respiratory depression is less than with morphine. Once the drug source has been removed and the systemically absorbed fraction starts to redistribute, the patient will start to wake up. This practice has also crossed over into some civilian centres with good results [36]. However, there can be problems of dependence if used longer term which is why they are rarely used in UK.

The availability of fentanyl lollipops in the UK is less than on deployed operations, and unfortunately, they are not available in the JRCALC formulary at the time of writing, so their use is restricted to independent prescribers.

Inhalational

Entonox is a gas containing 50% nitrous oxide and 50% oxygen. It is a good analgesic, with a rapid onset and offset (approximately 6–8 breaths at either end). It requires a patient who can cooperate, but it reduces the inhaled fraction of oxygen from around 85% on a non-rebreathe mask to 50% on a mouthpiece, and so is not suitable for patients with shock or severe injury and a high FiO_2 requirement. It is, however, a useful agent for some patients and should not be forgotten, especially in pre-hospital care. It is contraindicated in chest trauma until a pneumothorax has been excluded and where the effect of nitrous oxide diffusing into air-filled spaces would be deleterious (for example, pneumocephalus). It is also contraindicated after SCUBA diving and in decompression illness. One practical point is that the pseudo-critical temperature of entonox (the temperature at which it separates into its individual components of O_2 and N_2O) is −6 °C. This means that around or below this temperature the cylinder should be repeatedly inverted to ensure an adequate mixture of the two chemicals. Failure to do this results initially in the oxygen rising to the top of the cylinder and no N_2O, and hence no analgesia, being given. This is followed by 100% N_2O being delivered when the O_2 has been preferentially inhaled first, and thus a hypoxic mixture is delivered.

Penthrox (methoxyflurane) is an inhalational agent which has been popularly used in Australasia for many years and has recently been marketed in the UK for adults. This may present a useful alternative to entonox as it does not suffer from the same logistical challenges and some contraindications, and has been found to be non-inferior to entonox in a recent review [37], though inferior to intranasal fentanyl and intravenous morphine [38]. Early experience has been largely positive, and the concentrations used provide

analgesia without causing renal failure—the reason penthrox was withdrawn as an anaesthetic gas in the first instance. Some studies and reviews in the paediatric population have shown safety and efficacy [39, 40], but it is still considered an off license medication for children.

Brief Pharmacological Comparison of Analgesics

Opiates (Oral, IV, IM, IO, Transmucosal, Intranasal)

Traditionally the gold standard analgesia in trauma but best used in conjunction with other techniques if possible. The standard opiate in UK practice is morphine and it is given at a dose of 0.1 mg/kg via the IV or IO route as an initial bolus in severe pain. However, a more practical approach may be to give a 3 mg bolus and repeat every 5 min until adequate analgesia is attained, as the wide variance in tolerance that has previously been described may lead to an inadvertent overdose in some instances. As a rule of thumb, it is always easier to give more opiate than it is to take opiate out of the patient that has overdosed! Of note is that the pharmacokinetics and pharmacodynamics of morphine means it does not reach its peak analgesic effect until 30 minutes after administration due to hepatic metabolism of the more potent morphine-6-glucuronide.

The antagonist to opioid overdose (naloxone) is a useful and rapid-acting drug. However, the half-life of naloxone is shorter than that of morphine. In practical terms this means an episode of secondary respiratory depression or unconsciousness may occur after the naloxone has worn off and before the morphine has been metabolised. Traditional teaching was to give both an IM and IV dose of naloxone as it was thought that the IM dose would be absorbed more slowly and thus have a more prolonged effect, but this has not been born out in clinical practice [41]. Naloxone can also be given intranasally [42] or subcutaneously with equal efficacy, or via an endotracheal tube in intubated patients.

Paracetamol/Acetaminophen (Oral, IV, Rectal)

N-acetyl-para-aminophenol, otherwise known as paracetamol or acetaminophen is a commonly used analgesic worldwide. It is available in a licensed IV preparation for management of acute pain. It should be given regularly instead of a PRN basis as it forms the foundation of the analgesic ladder and has been shown to decrease opiate requirement. This effect has been increased when IV paracetamol has been given in anticipation of a painful stimulus [43] (e.g. surgical incision) rather than as a reactionary medication.

The benefits of IV versus oral or rectal paracetamol are:

- a shorter time until maximum availability
- a higher dose bioavailability (by definition the IV dose is 100% bioavailable in comparison to 60% oral and 40% via rectal routes)
- less hepatic damage (the paracetamol is given systemically rather than being absorbed and metabolised by the liver via the portal circulation)
- less dependence on gut blood flow, which may be altered in trauma, or the patient may be strict nil-by-mouth in the case of some bowel injuries.

Despite the above, a recent study and systematic review found no clinical benefit of IV over oral forms of paracetamol in patients who were able to take oral doses [44, 45]. Maximal doses should be 4 g in 24 h for adults or one dose of 15 mg/kg every 6 h for paediatric patients or adults under 50 kg. Paracetamol administration should be documented in the patients drug chart, especially if given in theatre as there is the potential for inadvertent overdose if multiple administrations from different areas is not recorded in one chart. There is also the potiential for iatrogenic injury if preparations containing paracetamol and another drug are not identified as such (e.g. co-codamol, co-proxamol, co-dydramol), and paracetamol is prescribed in addition to these. Paracetamol overdose is treated with N-acetylcysteine (Parvolex) to

reduce toxicity of the toxic metabolite NAPQI and is given as a series of three IV infusions over 1, 4 and 16 h respectively. Oral dosing is possible, but IV is preferred.

NSAIDs (Oral, IV, IM)

NSAIDs inhibit prostaglandin synthesis in the arachidonic acid pathway producing clinical anti-inflammatory and analgesic effects. However, the inhibition of certain prostaglandins also causes a decrease in production of COX I and COX 2 which are gastroprotective, as well as decreased bicarbonate and mucus secretion in the stomach. Care is needed with renal function in hypovolaemia and in patients with pre-existing renal disease, but NSAIDs are very useful if the patient is normovolaemic with no ongoing bleeding. Caution is also advised in asthmatics, as up to 20% of patients with susceptible asthma may experience an acute asthma attack [46]. Typically, these patients will also suffer from allergic rhinitis and nasal polyps, and children with asthma also appear to be susceptible to this effect [47]. Depending on other injuries (renal, orthopaedic, GI bleeding) there may be relative contraindications to NSAID use, but where possible they should be used as they are well tolerated and opiate sparing. The "safest" NSAID in terms of GI side effect profile is ibuprofen (400 mg three times a day) as this has the least anti-inflammatory effect, but not the least analgesic effect. If this is insufficient, naproxen may be the next agent to consider [48]. Other NSAIDs such as diclofenac, meloxicam and ketorolac have the advantage that they are also available in an IV form, so may be used if the patient is nil by mouth for any reason. However, even when taking IV NSAIDS, patients at risk of GI bleeding should be given proton pump inhibitors [49] as the GI side effects are as a result of systemic absorption, not local toxicity.

Ketamine and S-Ketamine

Ketamine is a phencyclidine derivative and a racemic mixture of two optical enantiomers. The R- form is responsible for approximately 30% of the analgesic activity of the mixture and has been implicated in the side effect profile more than the S- form. In Europe, the S- form (often referred to as esketamine) is available as a purified preparation but is more expensive than the racemic form. However, the usage of s-ketamine has not crossed into mainstream UK or US practice in comparison to the relative resurgence of racemic ketamine in recent years, though it is gaining popularity as a rapid acting antidepressant [50]. Historical concerns about the deleterious effects of ketamine have made some clinicians wary of using it, but these fears have been proven ill-founded, and the evidence that these concerns were based on has proven to be of poor quality. Ketamine is enjoying a resurgence in both pre- and in-hospital trauma use for analgesia and induction of general anaesthesia [19]. It may be a useful adjunct to patient controlled analgesia regimens [3, 4]. There are now consensus guidelines available on the use of IV ketamine for acute pain management from the American Society of Regional Anesthesia and Pain Medicine, the American Academy of Pain Medicine, and the American Society of Anesthesiologists [5].

Neuropathic Pain Modulating Agents

Gabapentin/Pregabalin

Gabapentin is an anti-seizure medication that has been used in the treatment of chronic pain thought to be neuropathic in origin. It was thought to be a GABA analogue (hence its name), but further investigation has revealed that gabapentin does not have any effects on GABA receptors. The mechanism of action is not fully understood and thought to have some direct inhibition of calcium channel-medicated neurotransmitter release, but even this has been questioned as gabapentinoids do not consistently reduce calcium currents in laboratory studies [51]. Although its mechanism is not fully appreciated, it is an effective agent but it was initially thought to take several days if not weeks to exert an analgesic effect. However, more recent data suggests that it may also have a

role to play in acute pain and be opiate sparing. Some studies have concluded that gabapentin may not be any more effective than carbamazepine in neuropathic pain [52], is equally effective as pregabalin but cheaper [53] and may be effective in treating complex regional pain syndrome [54]. Its main side effects are dizziness, drowsiness and peripheral oedema, with an increase in depression, and suicidal ideation. Gabapentin should not be stopped abruptly as it may cause a withdrawal-like syndrome, potentially resulting in seizures. Pregabalin was released as a competitor to gabapentin, and the two drugs are structurally similar, however pregabalin is more potent, absorbed faster and has higher bioavailability [55]. It is also marketed for the treatment of neuropathic pain and post herpetic neuralgia, whereas this is an off-license indication for gabapentin. The dose of gabapentin is increased over the course of a week and if problems with dizziness, somnolence, insomnia or other side effects are experienced, the dose held at that particular level until tolerance is achieved.

Amitriptyline

Amitriptyline is a tricyclic antidepressant which has been used in the treatment of neuropathic pain for many years but also can be used in the treatment of post-traumatic stress disorder and insomnia related to this. A typical dose for neuropathic type pain is 25–50 mg at night as tolerated. The main side effects are anticholinergic symptoms such as dry mouth, blurred vision, urinary retention, nausea, increased sweating, constipation and prolongation of QTc. In relatively low doses it is well tolerated and has a synergistic effect with gabapentin.

Regional Anaesthesia

Systemic analgesia requires the administration of medications via one of the above routes and may cause unwanted side effects depending on the drugs used. One other potential method of analgesia would be a peripheral nerve block, if possible.

General principles of nerve blockade include ensuring that the standard of monitoring used when performing these blocks is the same as in patients undergoing general anaesthesia [56]. Patients should also be screened for coagulation defects which may present a relative or absolute contraindication for regional or neuraxial anaesthesia. Guidelines exist from the Association of Anaesthetists of Great Britain and Ireland (AAGBI) [57] and the American Society for Regional Anesthesia (ASRA) [58] which outline the accepted standards for testing, risks, limits and contraindications to performing peripheral and central nerve blocks in these patients. Occasionally there may be a patient who on risk:benefit analysis falls outside of these guidelines, they are largely the standard to which practitioners will be held and are essential reading.

There have been several case reports of regional anaesthesia used in the pre-hospital environment [59–61], and depending on the indication for use and the transit time to hospital, it may be an appropriate modality to use. Indeed, for secondary transfer or aeromedical evacuation, the supplementation or replacement of parenteral analgesia has many attractive advantages. It diminishes the risk of respiratory depression which may be deleterious at altitude, as well as the potential eu- or dysphoria of ketamine, it allows the patient to remain awake, promotes orientation and allows assessment of the casualty when they are awake. Regional anaesthesia should not be employed if it delays time to definitive care but is a useful tool in the analgesic armamentarium. The introduction of hand-held ultrasound machines has made regional anaesthesia pre-hospital more available than in previous years, and the Royal Flying Doctor Service of Australia has employed this technique on many occasions, and some regional anaesthesia is taught as part of their standard operating procedures. Landmark techniques are possible (and regional nerve blocks are also being increasingly used in emergency departments for certain patient groups (e.g. neck of femur fractures) with good results [62, 63]), but in some cases the use of a nerve stimulator or preferentially an ultrasound

machine is mandatory (e.g. supra- or infraclavicular blocks). It is worth explicitly stating that pre-hospital regional anaesthesia may preclude a repeat regional anaesthetic procedure for surgery (e.g. due to maximum dosing and risk of local anaesthetic toxicity) or make a proper surgical neurovascular exam difficult so should be chosen with care.

Compartment Syndrome

Orthopaedic surgeons are rightly concerned about the development of compartment syndrome. This is where increased pressure within a fascial compartment of a limb (classically following nailing of the tibia) increases due to muscle swelling. This swelling increases to a point where the venous drainage of the affected compartment is not possible, thus causing more swelling. The limb still has pulses as arterial pressure is much higher than venous pressure, but necrosis of the muscle begins, and the patient requires a fasciotomy (an operation to cut the fibrous band that separates compartments in the limb). The hallmark of compartment syndrome is pain out of proportion to the injury, with worsening pain on a passive muscular stretch.

There is currently no evidence to suggest that regional anaesthesia prevents the diagnosis of compartment syndrome or delays its diagnosis if the patient is appropriately examined, though many surgeons still believe this to be the case [6–10].

The diagnosis of compartment syndrome is mainly clinical and relies to a large degree on clinical suspicion and examination, as a normal compartment pressure measured by manometry does not exclude compartment syndrome completely. Recent work has looked at probes which measure localised tissue pH or infrared spectroscopy as an indicator of hypoperfusion in suspected compartment syndrome and may prove more reliable in future [64–67].

Specific Regional Anaesthetic Techniques

There are fundamental key blocks which theoretically may be employed in- or in some cases pre-hospitally for limb trauma. The exact details of how to perform these blocks are beyond the scope of this text, but there are many resources (such as the NYSORA website) for the interested practitioner to learn from with appropriate senior supervision and oversight.

It must again be reinforced that these blocks should be done in as aseptic a fashion as possible, and should not increase scene time or time to definitive care if performed pre-hospital. They may be appropriate in only an extremely small number of scenarios, usually when a prolonged transfer is anticipated, or other analgesic options are not practical. An assessment of pre-existing nerve function should be performed and recorded before any block is attempted, as well as any subsequent block performed, the time and dose of any agent given.

The peripheral nerve blocks outlined can also be utilised as a primary anaesthetic technique in some instances for surgery in appropriate patients, or more commonly are used to supplement general anaesthesia for postoperative pain relief. Either a single shot injection or a continuous nerve catheter [68] can be placed to allow for infusion of local anaesthetic for a prolonged analgesic effect [69]. These catheters if appropriately cared for can be left in situ for over four weeks [70], and have the added advantage they can be bolused for procedures such as bedside dressing changes, which may otherwise require further sedation or general anaesthesia.

The techniques outlined in this chapter can all be employed effectively, as can epidural analgesia/anaesthesia for lower limb injuries. The only difference in epidurals used in lower limb injuries compared to those in use for thoracic pain is that the catheter is inserted in the lumbar spine rather than at a thoracic level. The chances of nerve injury and profound hypotension are less, though the rate of post-dural puncture headache changes with type of needle used and procedure [71]. Primary anaesthesia for lower limb fractures can also be achieved with a spinal or sub-

arachnoid block, though this can cause profound cardiovascular changes (hypotension and vasodilatation after injection) and is limited to operative procedures less than 2 h in length. However, in the same way as adding opioids to an epidural potentiates its effects, intrathecal opiates can give up to 12 h postoperative relief. Although surgical anaesthesia is limited to 90–120 min with a single shot spinal, there is a degree of postoperative analgesia that may persist for up to 8 h or beyond in some patients. The use of intrathecal opiates in the elderly population is not without complications, and a more full discussion is available in the Silver Trauma chapter.

Spinal anaesthesia is not appropriate in the hypovolaemic, under-resuscitated patient, the coagulopathic or the patient requiring a prolonged procedure. However, in a small group of patients with longstanding and severe respiratory disease it may be considered an alternative to general anaesthesia for certain operations.

NB, hyperlinks in the following section will take the reader to videos demonstrating ultrasound-guided regional anaesthesia. The following section details the associated anatomy and describes the risks associated with each of the blocks. Readers are advised to be cognisant of the serious associated side effects. These nerve blocks are best performed under ultrasound guidance and by appropriately trained personnel.

Upper Limb Blocks

Brachial Plexus Anatomy

The brachial plexus is formed by union of anterior primary rami of C5-T1 nerves (Fig. 13.2). There may be occasional minor contributions from C4 and T2. As the nerve roots emerge from the intervertebral foramina, they converge to form trunks, divisions, cords, branches and terminal nerves. Three trunks (superior, middle and inferior) are formed between the anterior and middle scalene muscles. The trunks divide into anterior and posterior divisions. These fibres combine again under the clavicle to form three cords—lateral, medial and posterior (based on their relationship to the axillary artery). Each of the cords gives off branches before ending as a terminal nerve.

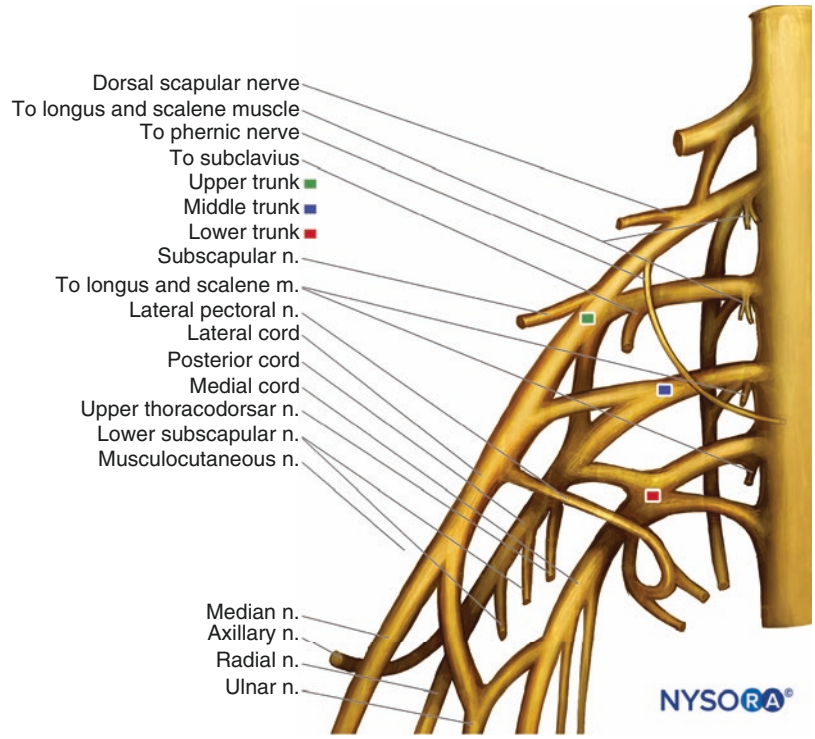

Fig. 13.2 Brachial plexus (Courtesy of NYSORA.com)

Interscalene Block

Video: Interscalene block [https://www.youtube.com/watch?v=OhFs-batCSY].

This block is indicated for procedures involving the shoulder and upper arm. The roots of C5-7 are usually more densely blocked and C8 and T1 are spared (ulnar sparing). Contraindications to the block include: Patient refusal, coagulopathy, local infection, allergy to local anaesthetics. The complications specifically associated with an interscalene block include ipsilateral phrenic nerve paralysis and pneumothorax, so caution must be used in patients with severe pulmonary disease or contralateral phrenic nerve palsy. Horners syndrome (due to proximal tracking of the local anaesthetic and blockade of the sympathetic fibres to the cervico-thoracic ganglion), vertebral artery injection, spinal or epidural injection and recurrent laryngeal nerve blockade have also been reported.

Supraclavicular Block

Video: Supraclavicular block [https://www.youtube.com/watch?v=9vW1uo7mKDc].

The supraclavicular block is at the level of the divisions of the brachial plexus. It is performed under ultrasound guidance and provides analgesia for upper limb, forearm and hand as well as the shoulder. There can be sparing of the ulnar nerve and it does not reliably anaesthetise the axillary and suprascapular nerves. Complications are similar to those for interscalene blocks and infraclavicular blocks. It is a suboptimal site for a continuous catheter because they can be easily displaced due to lack of muscle mass. Supraclavicular blocks are more commonly performed in the UK than infraclavicular blocks.

Infraclavicular Block

Video: Infraclavicular block [https://www.youtube.com/watch?v=Z9woYkyJl_U].

This block must not be performed without ultrasound. This block relies on depositing local anaesthetic under the clavicle and around the subclavian/axillary artery. This envelops the lateral, posterior and medial cords of the brachial plexus where they run in close continuity with the artery. Complications include inadvertent arterial puncture, bleeding, local anaesthetic toxicity and pneumothorax due to the proximity of the pleura. This block is suitable for anaesthesia or analgesia distal to the mid humerus for the distal upper limb.

Axillary Block

Video: Axillary block [https://www.youtube.com/watch?v=GaH-CO6OrV0].

Axillary blocks provide similar coverage to the infraclavicular block, however the point of injection is the medial humerus, thus avoiding the risk of pneumothorax. The traditional technique called for a trans-arterial puncture, but with the development of ultrasound-guided regional anaesthesia, it is no longer necessary to puncture the vessel as the nerves can be visualised. There is the potential to spare the musculocutaneous nerve as it is usually inferoposterior to the artery and occasionally difficult to visualise.

Lower Limb Blocks

Lumbar and Sacral Plexus Anatomy

The lumbosacral plexus provides innervation to the lower extremity (Figs. 13.3 and 13.4). The lumbar plexus is formed by ventral rami of L1-4. The femoral (L2-4), lateral femoral cutaneous nerve (L1-3) and obturator nerve (L2-4) arise from the lumbar plexus and provide motor and sensory innervation to the anterior thigh and sensory innervation to the medial leg.

The sacral plexus arises from L4-5 and S1-4. The posterior thigh and most of the leg and foot are innervated by the tibial and peroneal component of the sciatic nerve.

The possible complications associated with lower extremity peripheral nerve blocks

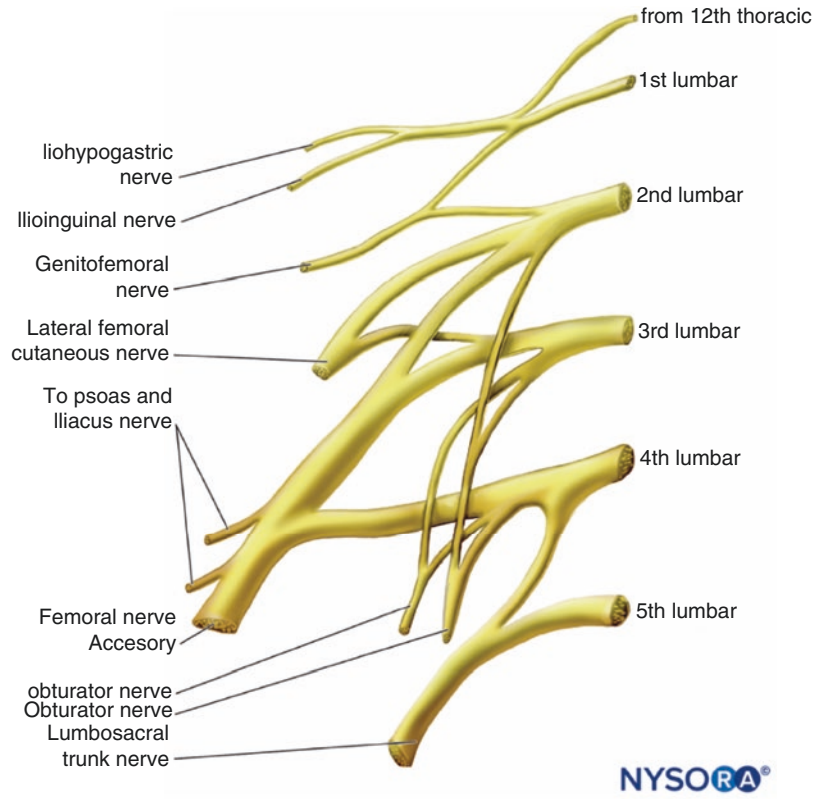

Fig. 13.3 Lumbar Plexus (Courtesy of NYSORA.com)

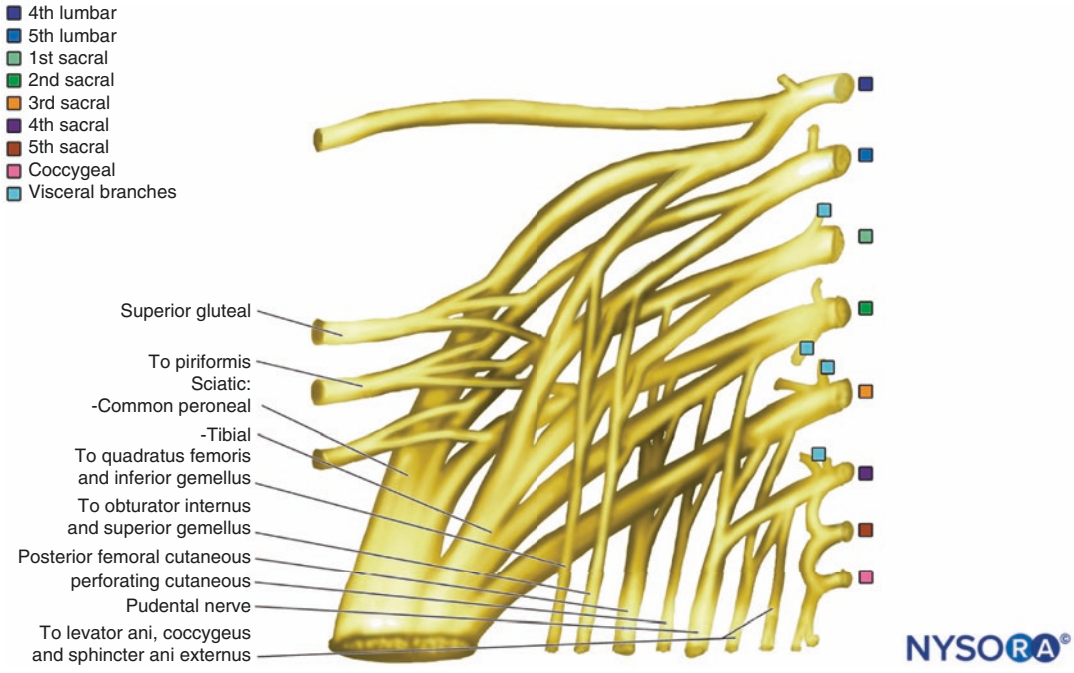

Fig. 13.4 Lumbarsacral plexus (Courtesy of NYSORA.com)

include infection, nerve damage, accidental intravascular injection, local anaesthetic systemic toxicity and allergic reactions to the local anaesthetic.

Femoral Block

Video: Femoral nerve block [https://www.youtube.com/watch?v=DwtvZ0tC9ng].

This block can be performed by a landmark technique as well as under ultrasound guidance and provides suitable analgesia for femoral fractures. The femoral artery is palpated as proximally as possible in the leg, and a needle is inserted 1–2 cm lateral to the pulse until two fascial pops are felt. The local anaesthetic is then slowly injected after negative aspiration unless resistance or pain on injection is felt. A variant on this, the fascia iliaca block, can be used in neck of femur fractures.

Video: Fascia Iliaca block [https://www.youtube.com/watch?v=p6X0IiYolIk].

Saphenous Block

Video: Saphenous nerve block [https://www.youtube.com/watch?v=C_Xmlqrn68Q].

The saphenous nerve is a branch of the femoral nerve which supplies some knee joint sensation and a small area of skin on the medial aspect of the leg below the knee. It is identified with ultrasound by tracing the femoral artery down the anteromedial thigh to the point where the artery starts to disappear (typically at the lower third). Look for the fascial "corner" just above the artery and infiltrate local anaesthetic to give excellent pain relief to the knee, but without quadriceps motor block.

Sciatic Block

Video: Anterior sciatic nerve block [https://www.youtube.com/watch?v=h14Ee2yAmUU].

Video: Popliteal sciatic nerve block [https://www.youtube.com/watch?v=qYM2sft8R2I].

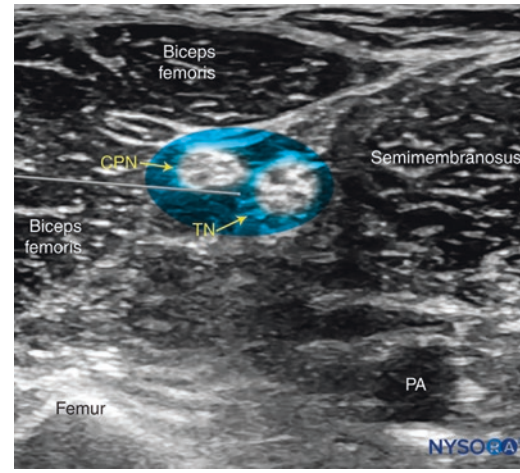

Fig. 13.5 Simulated needle path and local anesthetic distribution to block the sciatic nerve (at the level of bifurcation into the Tibial Nerve and Common Peroneal Nerve) in the popliteal fossa using the lateral approach. PA, popliteal artery (courtesy of NYSORA.com)

Video: Subgluteal sciatic nerve block [https://www.youtube.com/watch?v=rl8rZOEMveE].

The sciatic nerve supplies sensation to the knee joint and all the structures distal to the knee, (except the small strip of skin over the medial malleolus innervated by the saphenous nerve as described above). The sciatic can be blocked high at the buttock, sub-gluteally in the anterior thigh or in the popliteal fossa (Fig. 13.5).

Analgesia for Thoracic Trauma

Case Study

The patient is an 80-year-old male with a history of chronic atrial fibrillation on warfarin, hypertension, smoking and chronic obstructive pulmonary disease on home oxygen and spinal stenosis on opioids. He was involved in a motor vehicle accident and sustained left sided fractures of ribs 3-10, pulmonary contusions and a pneumothorax. He is admitted to the ICU and is complaining of severe pain and is unable to take a deep breath. What would be your analgesic plan?

As identified in the respiration and chest trauma chapter, the causes of perioperative morbidity and mortality in patients with thoracic trauma are airway obstruction, respiratory failure and haemorrhage. The anaesthetist is ideally suited and trained to deal with the problems of airway control, ventilatory and circulatory resuscitation, and adequate analgesia in all phases from pre-hospital care, through the operating room and ICU and eventually into the pain clinic in some cases. However, good analgesia is not the sole responsibility of anaesthetic staff—all clinicians who deal with trauma should have at least a basic understanding of common agents and techniques.

Pain from musculoskeletal trauma to the chest is a significant contributor to the failure of normal respiratory dynamics as previously highlighted. Inadequately controlled pain may cause hypoxic and/or hypercapnoeic respiratory failure. The inability to deep breathe and cough adequately leads to sputum retention, atelectasis and collapse/consolidation of lung tissue, which potentially may lead to superadded infection. This exacerbates hypoxia and leads to progressive respiratory failure which may result in a need for invasive ventilation if not addressed rapidly and effectively. The best way to avoid this predictable deterioration is to provide adequate analgesia from the first presentation of the patient.

The method and magnitude of analgesia required will depend more on the amount of pain suffered and baseline respiratory reserve than on the type and degree of injury sustained in many cases. A single lateral rib fracture in an elderly smoker with COPD may precipitate respiratory failure whereas multiple posterior rib fractures in a young fit person may be relatively well tolerated.

Key Points

Effective analgesia reduces stress, helps stabilise cardiovascular function, reduces oxygen requirements and allows early mobilisation. Analgesia is best achieved with a multi-modal approach combining several different drug types rather than relying on one technique alone and minimises the potential for side effects. Suitable analgesic components include:

Non-pharmacological Methods

Splinting, e.g. "Cough Lock" Support with Median Sternotomy

- Splinting by hand may offer some temporary relief, but binding or strapping may result in increased respiratory complications
- Surgical fixation of ribs may reduce pain, morbidity, mortality and length of stay [72–74]

Regional Anaesthesia

Single Shot or Infusion Catheter

- Intercostal Block
- Intrapleural/paravertebral blocks
- Thoracic epidurals (± epidural opiates/opioids)
- Serratus anterior plane block

Systemic Analgesia

- Simple analgesia with paracetamol or NSAIDs
- Opioids
- Low dose ketamine infusion
  - 5–10 mg IV loading dose followed by 0.5–2 mcg/kg/min infusion in a monitored High Dependency Unit (HDU) environment with 10–20 mg boluses as required 11. Confusion or agitation should be managed by reducing the dose of the infusion. Ketamine infusions are very useful in combination with any or all of the above techniques.

Regional Techniques for Thoracic Trauma

These can produce excellent analgesia with no sedation when performed well and allow almost normal chest movement with respiration. The technique chosen depends on experience and equipment available in addition to any contraindications and local agreements (e.g. site-specific requirement for HDU level care for all patients with epidurals). However, particularly high blocks may reduce the function of the intercostal

muscles and cause respiratory insufficiency on their own, so caution is advised with their implementation. With certain techniques (e.g. thoracic epidural) there may also be deleterious cardiovascular effects including hypotension secondary to sympathetic blockade causing vasodilatation and bradycardia if the cardio-acceleratory fibres are blocked and unopposed vagal tone predominates. If appropriately implemented and monitored then these techniques have a high success rate. In order to avoid complications then good nursing care and appropriate monitoring are as important as medical technical proficiency. Thus, placement of an epidural catheter may require a high dependency or ITU setting if ward staff are unfamiliar with epidural management or not exposed to epidural care on a regular basis.

Intercostal and intra-pleural blocks have the potential for local anaesthetic toxicity due to the relatively rapid uptake of drug from the pleural and intercostal spaces, as well as the risk of pneumothorax. The insertion of these blocks may be done before or after an operation while the patient is under general anaesthesia, or potentially awake in a sitting position if no operation is planned. However, the positioning required for these blocks in some patients may preclude them being done due to restrictions on movement (e.g. pelvic ex-fix in situ or unstable spinal fracture awaiting fixation) or severe pain. In the latter case, if the patient can be positioned or spontaneously move when they are awake for an intercostal or intrapleural block then they usually will not require one. Intrapleural catheters can be placed under direct vision at the time of operation if surgeons perform a thoracotomy.

Intercostal Block

Video: Intercostal block [https://www.youtube.com/watch?v=JVLZoxxthTY].

This block requires multiple injections, one for each rib fracture plus one segment above and below. The needle is placed just over the lower border of the rib at the angle of the rib with the injection of 5 ml of bupivacaine 0.25% at each site. The limited duration of action of each injection necessitates repeated injections. Good analgesia with bupivacaine or ropivacaine typically only lasts about 4 h. Single shot techniques therefore require repeated injections at multiple levels and is not practical for upper rib or posterior rib fractures. Therefore, this block is included for completion, but in practical terms is of limited value.

Intra/Inter-Pleural Block

Video: Interpleural block [https://www.youtube.com/watch?v=HBupyAtEkHw].

Insertion of an epidural catheter via a 16 g Touhy needle into the pleural space allows repeated or continuous injection of local anaesthetic. Various techniques have been reported for detecting the pleural cavity and rely on negative pressure within the space. 20 ml of 0.25% bupivacaine will produce several hours of good analgesia and may be repeated every few hours. It is effectively an intercostal block from inside the thoracic cavity, and the patient should be positioned during the bolus administration to allow the local anaesthetic to pool in the paravertebral gutter on the affected side. The intercostal nerve at this point is separated from the pleural space by the thin posterior intercostal membrane through which the local anaesthetic solution diffuses rapidly. This provides excellent analgesia for unilateral rib fractures or unilateral thoracotomy with bolus administration of local anaesthetic, and only requires one needle insertion into the chest wall. A catheter technique may be used for several days, but it does not work well in the presence of pleural fluids or pleural adhesions. In addition, the presence of a chest drain may result in local anaesthetic being lost from pleural space and an ineffective block.

Paravertebral Block

Video: Paravertebral block [https://www.youtube.com/watch?v=l97p0mbOv1E].

This block may be performed with either a continuous catheter technique to avoid repeated

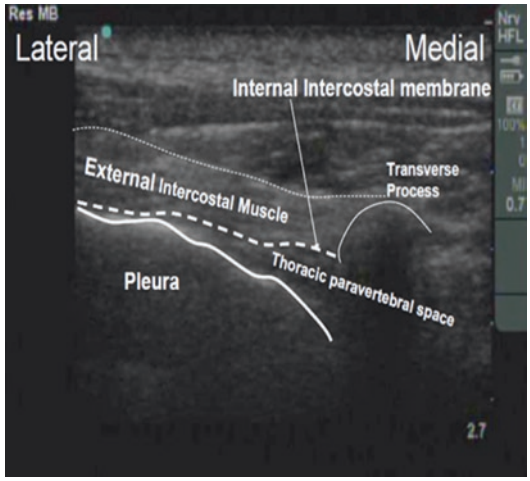

Fig. 13.6 Paravertebral space seen on ultrasound (Courtesy of NYSORA.com)

injections or as a single shot technique. Unfortunately, there is a less definite end point detected on insertion in comparison to epidural techniques (ultrasound image in Fig. 13.6). Drawbacks include the potential for LA toxicity, potential for sympathetic blockade, risk of pneumothorax and possible injection into a dural sleeve resulting in total or high spinal anaesthesia. Significant sympathetic blockade to the lung may predispose to bronchospasm and production of tenacious secretions.

Thoracic Epidural (+/− Opioid)

A midline or paramedian approach to the epidural space can be used and analgesia provided with a continuous infusion of local anaesthetic with or without opioid supplementation. Repeated bolus administration of local anaesthetic can be beneficial if a continuous infusion is not a practical proposition though this is rare. It is an excellent technique for unilateral and bilateral fractures of the middle and lower ribs. Epidural blockade may be used for several days but is not as effective for upper rib fractures, which would require very high blockade, increasing the likelihood of previously mentioned cardiovascular complications. This technique is contraindicated with thoracic vertebral injuries, other unstable spinal injuries, spinal cord injuries, overlying skin infection and in the presence of coagulopathy. A typical infusion regime is using 0.125% bupivacaine with 2 mcg/ml fentanyl at 6 ml/hour with a 6 ml bolus infusion with a 20-minute bolus lockout. If large numbers of boluses are demanded and the catheter is technically positioned correctly, the background infusion rate can be increased. Epidural solutions can be pre-made by pharmacy or reconstituted with strict aseptic technique on a volume by volume basis. The addition of opioids to an epidural has the effect of covering a less than perfect block, as epidural/spinal opioid receptors can be stimulated at lower doses than would be required systemically. This also has the effect of reducing (but not eliminating) the incidence of opioid side effects, namely nausea, vomiting, decreased level of consciousness and constipation.

One other potential complication of epidural analgesia is inadvertent dural puncture (estimated at 1% by the Royal College of Anaesthetists of all epidurals performed). This may cause a post dural puncture headache, or in severe cases an injury to the spinal cord in the thoracic region which may result in a permanent sensory or motor defect. Even in a perfectly placed epidural there is still the risk of infection and spinal abscess. Meticulous attention to asepsis and technique is essential. If there is any suggestion of spinal cord abscess or infection around the site then the infusion should be stopped, the epidural discontinued, and an urgent MRI requested regardless of the time of day or night. Unless caught and operated on early, spinal abscesses and haematomas have a very poor prognosis, so a high degree of clinical suspicion and a low threshold for intervention must be maintained. This is also why an epidural is contraindicated if there are overlying skin breaks, infections or systemic sepsis as the epidural and intrathecal spaces are sterile under normal conditions, and seeding of infection into them is a purely iatrogenic complication.

Serratus Anterior Plane Block

Blanco et al. [75] first described the regional anaesthetic technique of serratus anterior plane block in 2013. Local anaesthetic medication deposited superficial to the serratus anterior muscle and can provide analgesia to the hemithorax. It has been used in patients with rib fractures or following lateral thoracotomy as an excellent alternative to thoracic paravertebral blocks and thoracic epidurals in patients who have appropriate rib fractures and cannot be repositioned from a supine position. In certain patients who are intubated and have rib fractures, serratus anterior catheters can be placed prior to reducing sedation to provide effective analgesia and aid weaning from the ventilator [76–80]. If the rib fractures are lateral or anterior, then a serratus anterior block will likely be effective, but it will not provide analgesia for posterior rib fractures.

Local Anaesthetic Systemic Toxicity

Local anaesthetic systemic toxicity (LAST) results in CNS and cardiovascular symptoms [81–83]. The CNS symptoms include tinnitus, dizziness, blurred vision, paraesthesias, perioral numbness, seizures, agitation or restlessness. Eventually this can culminate in CNS depression, respiratory depression and cardiac arrhythmias. If toxicity is not treated at this point, further cardiac symptoms include ventricular dysrhythmias, ventricular fibrillation, myocardial depression and cardiac arrest (Fig. 13.7).

The Algorithm for Management of LAST Includes

1. Stop injecting local anaesthetic
2. Get Help:
   a. Consider Lipid emulsion therapy (intralipid) at the first sign of LAST
   b. Call for LAST rescue kit
   c. Alert nearest facility with Cardio-Pulmonary Bypass if appropriate
3. Airway management: Ventilate with 100% oxygen/avoid hyperventilating
4. Control seizures
5. Treat Hypotension and bradycardia

Cardiac arrest following LAST may be responsive to prolonged advanced life support with some adaptations to the universal ALS algorithm [84]. The ASRA recommendations are outlined in Fig. 13.8, below:

IV access and appropriate resuscitation facilities are mandated (including access to lipid rescue therapy) before commencing any form of regional anaesthesia under, the ASRA [85] and the AAGBI [86] guidelines for treatment of local toxicity. The same standard of monitoring should be applied to patients whether they are having a general or local/regional anaesthetic [56]. A more comprehensive discussion on local anaesthetic toxicity treatment is available on the www.lipidrescue.org educational website.

Analgesia for Neuro-Trauma

There are no specific treatment modalities for neurotrauma, the only caveat that must be born in mind is that specific treatments may cause neurological symptoms. For example, high doses of opioids can decrease the patient's level of consciousness and cause pinpoint pupils via stimulation of the Edinger-Westphal nucleus, both of which may alter findings in a neurological exam. Another example is ketamine, which has both pro- and anticonvulsant properties, so any focal neurological examination should ideally take place with the patient as free from impediment as possible. Frequently, the initial responder may have the most reliable examination findings as the patient may need high doses of analgesia, or even general anaesthesia before arriving at the hospital and the attention of a neurosurgeon or critical care team. It is important therefore to examine as thoroughly as possible and document clearly the highest GCS since injury and any lateralising neurological signs in the limbs or eyes before induction of general

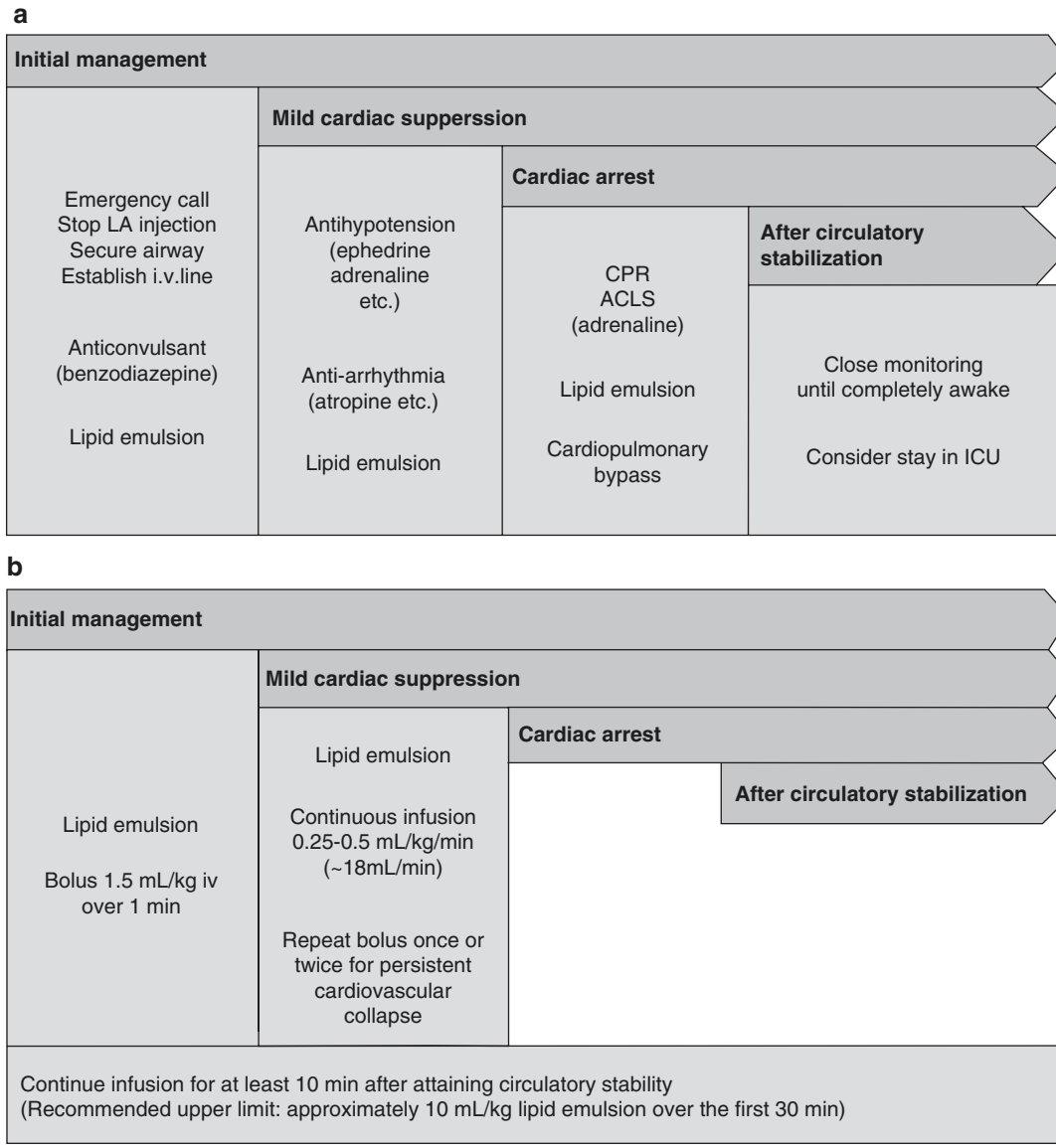

Fig. 13.7 Management of acute local anaesthetic toxicity. (**a**) Sequence of symptoms and required treatments. (**b**) Sequence of symptoms and program of lipid emulsion (20%) infusion. ACLS, advanced cardiac life support, CPR, cardiopulmonary resuscitation, ICU, Intensive Care Unit. From Sekimoto et al. [84]

anaesthesia or sedation. A focused neurological examination ("move your arms, your legs, close your eyes, where are you, what is your name, what day/week/month is it") will give a baseline to work from in hospital. It is also important to note the time of administration and dose of any neuromuscular blockers or other drugs which could change neurological examination findings in hospital.

It is essential to provide adequate analgesia to patients who are intubated and ventilated as pain may cause or substantially contribute to a raised intracranial pressure. Awareness under anaesthesia and coughing can also cause raised ICP, so assessment and maintenance of an adequate plane of anaesthesia even when not in the operating room (i.e. during CT scan, transfer inter- or intra-hospitally) are of vital importance.

Fig. 13.8 From Sekimoto et al. [84]

Table 3.Special considerations in cardiac life support for patients with local anesthetic-induced cardiac arrest†

1) If epinephrine is used, small initial doses (10 –100 μg boluses in adults) are preferable
2) Vasopressin is not recommended
3) Avoid calcium channel blockers and bete-blockers
4) If ventricular arrhythmias develop, amiodarone is preferable
5) In patients with cardiac toxicity avoiding the use of lidocaine and related class IB antidysrhythmic agents (e.g., mexiletine, tocainide) is crucial because they may worsen toxicity. Lidocaine has been used successfully bupivacaine-induced dysrhythmias, but its additive central nervous system toxicity is still a major concern.
6) In patients who do not respond to standard resuscitative measures, cardiac pacing and cardiopulmonary by pass may be introduced to improve the outcome. Cardiopulmonary by pass may serve as a bridging therapy until tissue levels of the local anesthetic have cleared.

†Adapted from ASRA guidelines.[4]

Clinical Pearls in the Management of Acute Pain in a Patient Following Trauma

It is important to perform a comprehensive assessment of the extent of trauma and injury, severity of pain, history of chronic pain, narcotic use and other comorbidities in the patient prior to initiating an analgesic plan in the acute period. Consider using regional anaesthetic techniques (peripheral nerve blockade or neuraxial techniques such as epidural analgesia) if the patient is an appropriate candidate. This assessment must take into account type, location and extent of injury, coagulation parameters, haemodynamics, allergies, infection, neurovascular injury and other comorbidities including cardiac and respiratory disease. A multimodal analgesic regimen should be initiated based on individual patient characteristics (severity of pain, allergies, contraindications to specific drugs). This regimen may incorporate several agents including paracetamol (acetaminophen), non- steroidal anti-inflammatory medications, neuropathic agents (gabapentinoids, tricyclic antidepressants), opioids analgesics, adjuvants like intravenous ketamine and lidocaine infusions. The WHO analgesic ladder (Fig. 13.9) provides a good outline for initiating therapy based on pain severity, though this approach was initially developed for treating pain due to cancer and is considered outdated by some.

Initiating Patient Controlled Analgesia in the Acute Setting for Pain Management

For patients in severe pain and or unable to tolerate oral medication, an IV analgesic regimen in the form of patient controlled analgesia (PCA) can be initiated. Prior to initiating patient controlled analgesia consider age, ability to comprehend instructions, physical ability to use the

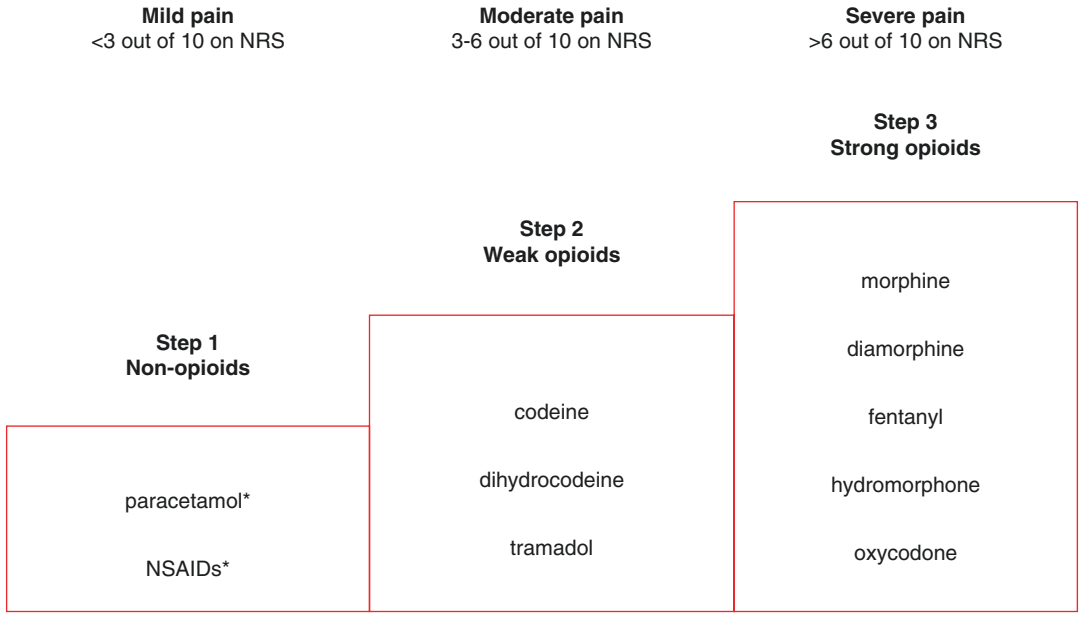

Fig. 13.9 WHO analgesic ladder

Table 13.1 Typical IV PCA setting for opioids

Analgesic	Typical Bolus Dose	Lockout period (minutes)
Morphine	1–2 mg	5–10 min
Fentanyl	10–50 mcg	5–10 min
Hydromorphone	0.2–0.5 mg	5–10 min
Remifentanil	0.5 mcg/kg	2 min

infusion pump and comorbidities. Comorbidities that increase the risk of respiratory depression include sleep apnoea, severe obesity, head injury, respiratory failure, renal failure (accumulation of potent metabolites like Morphine-6 glucuronide) and concurrent use of other synergistic medications such as benzodiazepines. Monitoring of patients using PCAs should include monitoring of sedation, respiratory rate and pulse oximetry. Medications used in PCA regimens include morphine, hydromorphone and fentanyl. Dosing strategy for PCA includes a bolus dose and a lockout period (Table 13.1). A bolus dose should be able to provide significant analgesia but should not cause respiratory depression. An appropriate lockout interval prevents repeat administration until a predetermined time period has elapsed. Even though the patient presses the demand button during this interval, medication is not administered. Lockout periods are tailored based on the drugs pharmacokinetics. Basal infusions should not be initiated in opioid naive patients and additionally have shown no improvement in analgesic effect, but instead are associated with higher risk of respiratory depression and side effects [87, 88].

Transitioning from PCA to an oral regimen is the next step in the acute pain management of a patient admitted for trauma. It is necessary to calculate the patients total daily opioid requirement. The first step is to assess the frequency of demands vs supply from the PCA machine to ensure the patient is not requesting analgesia greatly in excess of that delivered. Assuming that the doses are reasonably concordant with demands, add up the total dose of opioids given by the PCA in the last 24 h period and covert to oral equivalency of the new agent (typically oral morphine). This basal requirement is divided in two and given as a long acting analgesic twice a day. A bolus dose of 10–15% of the total requirement calculated is prescribed as a short acting opioid as needed for breakthrough pain. After the next 24 h the process is repeated again, looking at the total opioid dose given as basal dosing and adding the total dose of breakthrough boluses. If

there are only one or two breakthrough episodes, then the regime should remain unchanged. If there are multiple doses given for breakthrough pain, the basal oral dose is recalculated in the same way as previously for the IV requirement. Does should be tapered down as able over a few days as pain subsides in the acute period.

For example, a PCA shows that 70 mg of morphine has been used in the last 24 h and the pain specialist decides to transition to oral medications. The total dose is divided in half (70/2 = 35) and prescribed as a twice daily long acting opioid (oxycontin 35 mg BD) and 10 mg as a PRN dose of oxynorm for breakthrough pain. Analgesia should be given well in advance of anticipated painful procedures or physiotherapy sessions so patients can participate and achieve maximal benefit. This must be balanced against risk of over-sedation and inability to engage with the session, so feedback and a multi-disciplinary approach are needed.

When rotating between opioids, it is prudent to perform a dose reduction to account for cross tolerance. At the authors'institution some commonly used oral opioids in the acute setting include oxycodone, hydromorphone and morphine.

There are several apps, policies and methods which can be used for conversion between doses and drugs, and a paper by Nielson et al. [89] found general consensus amongst the recommended conversion factors internationally.

Chronic Pain After Trauma

Chronic post-surgical or post traumatic pain as defined by the International Association for Study of Pain is chronic pain that develops or increases in intensity after tissue trauma (surgical or accidental) and persists beyond three months [90]. The incidence of chronic pain after polytrauma ranges from 46 to 85% [91] and after burns is between 18 and 52% [92, 93]. Comparatively, chronic pain after injury to muscles, bones, joints (aka post traumatic arthritis) is about 18.7%. Chronic pain after trauma is often under recognised and poorly treated. Several studies [94, 95] have identified the following risk factors which are predictive of patients developing chronic pain;

- female sex
- injury mechanism
- injury regions (brain-brainstem- cerebellum, disc- vertebra, thorax/ skeletal, face, abdominal, spinal cord, thorax, upper extremity)
- previous history of alcoholism, anxiety or depression
- two or more rib fractures
- mild traumatic brain injury
- spinal cord injury
- back and spine problems.

It is important to recognise chronic pain after trauma and to attempt to identify the primary mechanism which may be nociceptive. This could be musculoskeletal or neuropathic (including sympathetically-maintained), and the physician should initiate appropriate multidisciplinary management. For nociceptive pain this will include opioids, while for neuropathic pain this also includes use of medications like neuropathic agents (gabapentinoids, tricyclic antidepressants), antidepressants (duloxetine, SSRIs and SNRIS), muscle relaxants (baclofen, tizanidine, cyclobenzaprine), non-steroidal anti-inflammatory medications and paracetamol. Consideration should be given to early initiation of physiotherapy and interventional management like sympathetic blocks for complex regional pain syndrome (CRPS) and peripheral nerve blocks for pain due peripheral nerve mononeuropathy. Cognitive behavioural therapy can be useful in appropriate patients, and a multidisciplinary approach should be taken to chronic pain management in trauma patients.

In patients with chronic pain which is neuropathic in origin and associated with a diagnosis of complex regional pain syndrome, failed back surgical syndrome with radicular pain and phantom limb pain secondary to amputation, implantable neuromodulatory devices like spinal cord stimulators may be considered in the treatment algorithm in the longer term. It is beyond the scope of this chapter to cover the mechanisms

and placement of neurostimulators for chronic pain, and review articles by Moisset et al. [96], Rokyta and Fricova [97] or Jeon [98] give good oversight.

Summary

Treatment of pain is one of the most important interventions that can be undertaken by any personnel involved in trauma. While there are many drugs with complex pharmacological actions and interactions that can be used, basic principles should not be forgotten in the acute phase. Treating pain can be an end in itself but can also have impact on patients physiology and outcomes, so it is important to get it right for a multitude of reasons. The development of complex pain syndromes following trauma can be as debilitating as the biomechanical or tissue effects of the injury itself, and chronic pain management is as much about assisting patients to cope with the impact of their pain as it is alleviating it. By giving appropriate treatment early enough, the development of chronic pain syndromes may be prevented.

Questions

1. Which of the following nerve blocks has the highest likelihood of ipsilateral phrenic nerve paralysis?
   a. Intercostal nerve block
   b. Interscalene nerve block
   c. Supraclavicular nerve block
   d. Suprascapular nerve block
2. Which of the following should be avoided in management of local anesthestic system toxicity?
   a. Use of intralipid
   b. Epinephrine
   c. Vasopressin
   d. Benzodiazepenes
3. The advantages of IV paracetamol over other routes of administration include all of the following except:
   a. shorter time until maximum availability
   b. higher dose bioavailability
   c. less hepatic damage
   d. Significant clinical benefit over other routes of administration.
4. Which of the following nerve blocks cannot provide analgesia to patient with thoracic trauma:
   a. Intercostal nerve block
   b. Interscalene nerve block
   c. Intrapleural nerve block
   d. Serratus anterior plane block
5. Which of the following regional anesthetic techniques has the highest systemic absorption of local anesthetic?
   a. Epidural
   b. Axillary
   c. Intercostal
   d. Femoral

Answers

1. b. Interscalene nerve block
2. c. Vasopressin is avoided in the management of LAST. Calcium channel blockers and Betablockers are to be avoided as well in treating arrthymias in the setting LAST
3. d. A recent study and systematic review found no clinical benefit of IV over oral forms of paracetamol in patients who were able to take oral doses [44, 45]
4. b. Interscalene Nerve block provides analgesia to the shoulder and upper arm
5. c. Intercostal nerve block has the highest systemic absorption of local anesthetic

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Further Reading

Regional Anaesthesia

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IO Access

http://www.jems.com/article/intraosseous/pain-management-use-io
http://bestbets.org/bets/bet.php?id=2515
http://reference.medscape.com/article/80431-overview

Intransal Drug Administration

http://www.lmana.com/pwpcontrol.php?pwpID=6359