REVIEW ARTICLE

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Complications of Regional Anaesthesia
Incidence and Prevention

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Abstract

The complications of failure, neural injury and local anaesthetic toxicity are common to all regional anaesthesia techniques, and individual techniques are associated with specific complications. All potential candidates for regional anaesthesia should be thoroughly evaluated and informed of potential complications. If there is significant risk of injury, then these techniques should be avoided.

Central neural blockade (CNB) still accounts for more than 70% of regional anaesthesia procedures. Permanent neurological injury is rare (0.02 to 0.07%); however, transient injuries do occur and are more common (0.01 to 0.8%). Pain on injection and paraesthesiae while performing regional anaesthesia are danger signals of potential injury and must not be ignored.

The incidence of systemic toxicity to local anaesthetics has significantly reduced in the past 30 years, from 0.2 to 0.01%. Peripheral nerve blocks are associated with the highest incidence of systemic toxicity (7.5 per 10 000) and the lowest incidence of serious neural injury (1.9 per 10 000).

Intravenous regional anaesthesia is one of the safest and most reliable forms of regional anaesthesia for short procedures on the upper extremity. Brachial plexus anaesthesia is one of the most challenging procedures. Axillary blocks are performed most frequently and are safer than supraclavicular approaches.

Ophthalmic surgery is particularly suited to regional anaesthesia. Serious risks include retrobulbar haemorrhage, brain stem anaesthesia and globe perforation, but are uncommon with skilled practitioners.

Postdural puncture headache remains a common complication of epidural and spinal anaesthesia; however, the incidence has decreased significantly in the past 2 to 3 decades from 37 to approximately 1%, largely because of advances in needle design.

Backache is frequently linked with CNB; however, other causes should also be considered. Duration of surgery, irrespective of the anaesthetic technique, seems to be the most important factor. The syndrome of transient neurological symptoms is a form of backache that is associated with patient position and use of lidocaine (lignocaine).

Disturbances of micturition are a common accompaniment of CNB, especially in elderly males. Hypotension is the most common cardiovascular disturbance

associated with CNB. Severe bradycardia and even cardiac arrest have been reported in healthy patients following neuraxial anaesthesia, with a reported incidence of cardiac arrest of 6.4 per 10 000 associated with spinal anaesthesia. Prompt diagnosis, immediate cardiopulmonary resuscitation and aggressive vasopressor therapy with epinephrine (adrenaline) are required.

New complications of regional anaesthesia emerge occasionally, e.g. cauda equina syndrome with chloroprocaine, microspinal catheters and 5% hyperbaric lidocaine, and epidural haematoma formation in association with low molecular weight heparin. Even so, after 100 years of experience, most discerning physicians appreciate the benefits of regional anaesthesia.

1. History and Definitions

The era of regional anaesthesia commenced with the discovery of local anaesthetics by Köller in 1884.[1] Within months of this discovery, cocaine was being used in ophthalmology, dentistry and general surgery in many medical centres all over the world. The initial enthusiasm with general anaesthesia was beginning to wane as the number of reported deaths associated with it increased. Intuitively, it seemed that the risk of complications would be less when procedures were performed under local anaesthesia. However, it quickly became evident that the use of local anaesthetics was not without risk either. Within 7 years of the introduction of cocaine, there were at least 200 reports of systemic toxicity and as many as 13 deaths.[2] Toxicity issues have tarnished the history of regional anaesthesia since its inception and continue to do so.

In 1898, Bier[3] introduced spinal anaesthesia to the world. This was indeed a dramatic discovery and one of the most important in the history of anaesthesia; the risk of systemic toxicity was no longer a concern with spinal anaesthesia. The injection of very small quantities of local anaesthetics into the subarachnoid space produced excellent surgical conditions for operative procedures below the waist. However, spinal anaesthesia came under attack in 1950 when a prominent neurologist in the US published a report describing 12 cases of adhesive arachnoiditis, which he firmly linked with spinal anaesthesia.[4] By some coincidence, there was a serious accident involving spinal anaesthesia reported in England around the same time. This was the famous Wooley and Roe case.[5] Although the true cause of this accident was never fully elucidated, the practice of spinal anaesthesia almost disappeared in the UK for 3 decades. Even today, patients express concern about the risk of back injury and paralysis with spinal anaesthesia.[6] The impact of spinal anaesthesia on the practice of anaesthesia has been enormous and is sustained. As we enter the 21st century we must acknowledge that spinal anaesthesia, which started in the latter part of the 19th century, will also be practiced in the 22nd century and probably long thereafter.

Epidural anaesthesia was discovered by Cathelin and Sicard[7] in 1901. This technique has also had a major impact on the practice of anaesthesia. The use of continuous epidural techniques was a very important advance in regional anaesthesia. Their subsequent use for analgesia and anaesthesia in obstetric practice has had a dramatic impact in reducing anaesthesia-related maternal mortality. In the most recent report from the UK on maternal morbidity, only 1 death was directly related to anaesthesia in the period 1994 to 1996 compared with 18 in the period 1982 to 1984.[8]

The discovery of the opioid receptors in the spinal cord by Snyder[9] added additional momentum to the importance of regional anaesthesia in the management of all types of pain. The management of acute postoperative pain, once passively in the hands of surgeons, has now rightfully found its place in the realm of anaesthesiology. Despite the dramatic advances in pain management, there is still much room for improvement in this area.

There is no doubt that central neural blockade (CNB) is still the mainstay of regional anaesthesia;

Table I. Classification of regional anaesthesia techniques

Central neural blockade
Spinal
single injection
continuous
Epidural (thoracic, lumbar or caudal)
single injection
continuous
Plexus blockade
Cervical plexus
Brachial plexus
Lumbosacral plexus
Peripheral nerve blockade
Single nerve blocks
Multiple nerve blocks
wrist
ankle
sciatic/femoral
intercostal
paravertebral
ophthalmic
other
Intravenous regional anaesthesia
Arm
Leg
Other
Sympathetic blocks
Stellate ganglion
Lumbar sympathetic
Coeliac plexus
Other
Miscellaneous
Interpleural
Other

however, the adverse effects associated with these techniques, for example hypotension, bradycardia, motor paresis, headache, bladder dysfunction, pruritis and respiratory depression, sometimes overshadow the benefits accrued.

There is increasing interest among anaesthesiologists in the use of peripheral nerve blocks (PNBs) in the management of postoperative pain, and recent generations of surgeons use local anaesthetics freely to infiltrate incision sites.[10] Regional anaesthesia zealots promote regional techniques because they intuitively believe that patients have a better outcome. There is no doubt that patients have better pain control, at least during the early phases of recovery. It is much more difficult to prove the other benefits of regional anaesthesia.

In order to discuss the complications of regional anaesthesia it may be useful to have a working definition. Regional anaesthesia involves the injection of local anaesthetic drugs towards neural targets either centrally or peripherally to produce anaesthesia in a region of the body supplied by those nerves. Conversely, local anaesthesia involves the injection of local anaesthetic drugs into the skin and subcutaneous tissues to produce anaesthesia of the skin in the area of the injection. Finally, topical anaesthesia involves the application of local anaesthetic drugs to mucous membranes and skin, resulting in diminished sensation in those areas. Table I provides a classification of regional anaesthesia.

It would be difficult to thoroughly address regional anaesthesia complications *in toto* in this review article. Therefore, we will confine our discussion to those areas that have most relevance to today's practice of regional anaesthesia in an operating room setting, in the adult population only. The following complications are common to all regional anaesthesia techniques:

- local anaesthetic toxicity;
- neural injury;
- failure.

These 3 broad categories will be discussed in detail. Then we will address specific complications associated with the more common regional techniques.

2. Local Anaesthetic Toxicity

The following simple mnemonic can be used to memorise the various components of local anaesthetic toxicity:

- **A**llergy
- **I**diosyncratic reactions
- **L**ocal
- **S**ystemic

2.1 Allergic Reactions to Local Anaesthetics

Fortunately, serious life-threatening allergic reactions to local anaesthetics are rare.[11] There are a number of reports in the literature, but it is difficult to give a true incidence.[12,13] Allergic reactions are more common following exposure to ester compounds than amide ones; there are numerous documented cases of contact dermatitis following exposure to procaine and related compounds. In contrast to this, contact dermatitis following exposure to lidocaine (lignocaine) is extremely rare.[14] A full range of allergic symptoms ranging from mild skin irritation to full-blown anaphylaxis has been described. Cross-reactivity between different ester compounds has also been described. *para*-Aminobenzoic acid (PABA) is a primary metabolite of ester local anaesthetics and is responsible for the majority of allergic reactions associated with local anaesthetics. Methylparaben is chemically related to PABA and is also a true allergen. It is used as a preservative to extend the shelf life of many compounds, including lidocaine.[15] An allergic reaction may be mistakenly attributed to lidocaine when in fact the cause is methylparaben.

Both the medical and dental professions and the public use the term allergy far too loosely. We need to be far more discriminating when it comes to labelling individuals as having a 'drug allergy', particularly in relation to local anaesthetics. Epinephrine (adrenaline) reactions, fainting spells and panic attacks (e.g. needle phobias) are often misdiagnosed as allergic reactions resulting in the unnecessary use of general anaesthesia when patients present for subsequent dental treatment. True allergic reactions to lidocaine are extremely rare and skin testing with preservative free lidocaine is a very rational approach.

2.2 Idiosyncratic Reactions to Local Anaesthetics

There are some reactions to local anaesthetics that defy a rational explanation. These we refer to as idiosyncratic reactions.

From time to time we may observe bizarre behaviour following injections of local anaesthetic drugs, which may more appropriately described as a hysterical reaction. The following is a true case scenario. A patient presented for oral surgery in the dental chair. A minute quantity of local anaesthetic was injected into the gingival margin, following which the patient became comatose and unrousable. After 3 hours the patient was still unconscious and underwent a computed tomography scan, which proved to be normal. The patient finally regained consciousness 5 hours after the injection of local anaesthetic. There were no permanent sequelae.

2.3 Local Effects of Local Anaesthetics and Additives

Local anaesthetics are innocuous substances when injected perineurally in appropriate quantities and concentrations. High concentrations of local anaesthetics are known to permanently damage neural tissue in some cases.[16] Substances added to local anaesthetic drugs may also damage nerves and other surrounding tissues. A change in the constitution of a preservative of chloroprocaine (sodium metabisulphite) resulted in several cases of cauda equina syndrome in the US in the 1970s.[17] The addition of ethylenediaminetetra-acetic acid (EDTA) to the same compound caused severe back pain in some patients following epidural anaesthesia.[18,19] More recently, 5% hyperbaric lidocaine has been linked with transient neurological symptoms (TNS) following spinal anaesthesia.[20] Finally, myelotoxicity is a recognised complication of intramuscular injections of local anaesthetic drugs.[21] It is thought that local anaesthetics cause a pathological efflux of Ca^{2+} from the sarcoplasmic reticulum, resulting in contracture, cell destruction and necrosis; regeneration of fibrils occurs within a few weeks. Of the local anaesthetics tested, bupivacaine resulted in the most damage, with procaine the least.[22] Damage was worse with repeated injection and when epinephrine is used.[23-25] All of these features are highlighted in a case in which a patient received an interscalene block and then developed intense neck pain and tenderness over the

sternocleidomastiod muscle, which persisted for 2 months.[26] In the clinical setting this myelotoxicity is largely unnoticed but it may account for diplopia after orbital nerve blocks, which may last up to 24 hours and rarely may be permanent.

2.4 Systemic Toxic Reactions

Systemic toxic reactions to local anaesthetic drugs occur more commonly as a result of accidental intravascular injection and much less frequently following the injection of an excessive quantity of local anaesthetic. The incidence of systemic toxicity has decreased remarkably within the past 30 years. Massey Dawkins,[27] in 1969, reported the incidence of seizures to be 0.2% following epidural anaesthesia. A recent study from France reported an incidence of 0.01%, which represents a 20-fold decline in 30 years.[28] A higher incidence of systemic reactions occur following PNBs, especially brachial plexus anaesthesia and caudal blocks in adults.[29]

The maximum plasma concentration (C_{max}) of local anaesthetic drug resulting from an accidental intravascular injection depends on a number of factors.[30] Clearly, it is directly influenced by the total dose of local anaesthetic injected, the speed and site of injection and whether the injection is administered intravenously or intra-arterially. The lungs are an important repository for local anaesthetic drugs; plasma concentrations of these drugs will be much higher if the lungs are bypassed (for example an accidental intra-arterial injection in the head, face or neck region).[31] The tension of CO_2 and pH also influence plasma concentrations of local anaesthetics. An elevated arterial CO_2 tension increases cerebral blood flow, and an acidotic state increases intracellular ion trapping and the amount of free drug available. This combination of factors has a synergistic effect on the seizure threshold (table II).[32]

Table II. Effect of acid-base status on seizure threshold. The table shows the dose of intravenous lidocaine (lignocaine), infused at 5 mg/kg/min, required to produce convulsions in cats as a function of arterial CO_2 tension ($PaCO_2$) and pH (from Englesson and Grevsten,[32] with permission)

$PaCO_2$	Dose (mg/kg) at pH			
	7.10	7.20	7.30	7.40
30			27.5	26.6
40		20.6	21.4	
60	13.1	15.4	17.5	
80	11.3	14.3		

Systemic toxic reactions occur much less frequently when local anaesthetics are administered to peripheral sites. A number of factors influence the degree of absorption that takes place from the periphery to the central circulation. The most important factor is the site of injection, absorption being more rapid in highly vascular tissues and less so in poorly perfused ones. Thus, absorption is fastest from the intercostal area, then in descending order from epidural, brachial plexus, lower extremity blocks and subcutaneous injection (fig. 1).[30] The addition of epinephrine to local anaesthetic drugs reduces the rate of absorption but the magnitude of this reduction is dependent on the local anaesthetic used. For example, the addition of epinephrine to lidocaine markedly retards uptake into the circulation but the effect is less marked with etidocaine (fig. 2).[30]

2.5 Symptoms and Signs of Systemic Toxicity

As the plasma concentration of local anaesthetic drug increases to the concentration that systemic toxicity occurs, there is a typical progression from effects on the CNS to effects on the cardiovascular system. However, this pattern of symptomatology may not be seen if there is a rapid intravascular injection.

Following common regional blocks, typical plasma concentrations of lidocaine vary between 3 and 5 μg/ml. Signs of toxicity may be observed when plasma concentrations reach 6 μg/ml, but convulsions do not usually occur until plasma concentrations exceed 10 μg/ml and cardiovascular collapse does not occur until plasma concentrations exceed 30 μg/ml (fig. 3).[33]

In the CNS, the amygdala is thought to be the site of action of the local anaesthetic drugs, since seizures are not observed in animals from which the amygdala has been experimentally removed.[34]

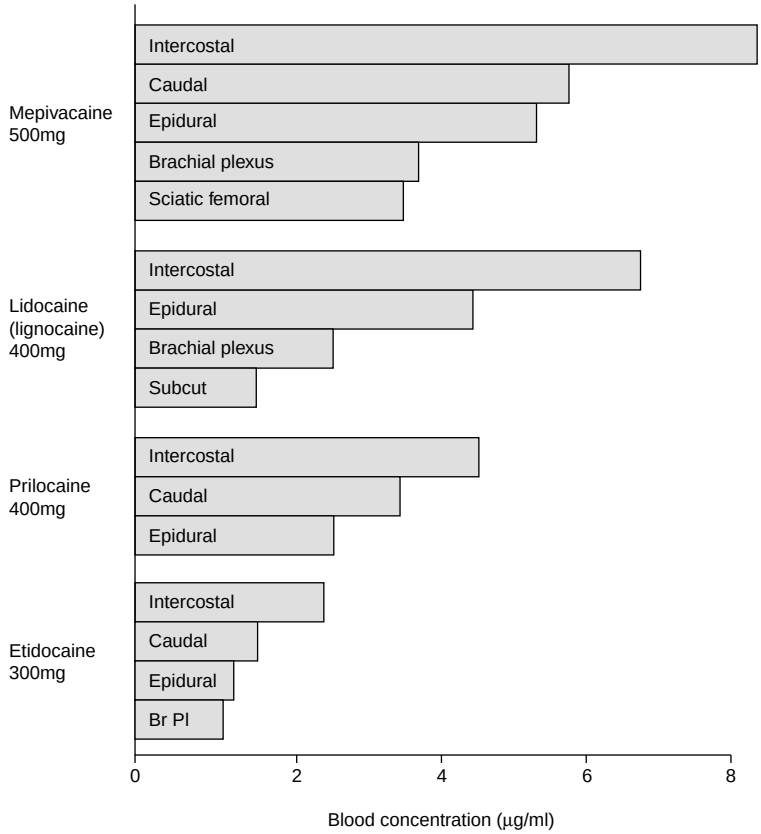

Fig. 1. Comparative peak blood concentrations of several local anaesthetic agents following administration into various anatomical sites (from Covino and Vassalo,[30] with permission). **Br Pl** = brachial plexus; **Subcut** = subcutaneous.

Benzodiazepines are known to accumulate in the amygdala, elevating the seizure threshold to local anaesthetics in animals.[35] This observation has not been reported in humans.

As can be seen in figure 3, lidocaine-induced cardiovascular collapse does not occur until plasma concentrations greatly exceed those that cause seizures. In contrast, bupivacaine causes cardiovascular collapse at plasma concentrations not too far removed from those that cause seizures.[36] All local anaesthetic drugs have an affinity for receptors in the sodium channel, including those in the heart. The problem with bupivacaine is that it enters the sodium channel rapidly, but leaves slowly ('fast in, slow out').[37] The end result of this electrophysiological anomaly is impairment of conduction and contractility, resulting in life-threatening re-entrant arrhythmias that are often refractory to treatment. Several deaths occurred before restrictions were placed on the use of bupivacaine in certain clinical scenarios.[38]

The single-isomer drugs, ropivacaine and levobupivacaine (the *S*-isomer of bupivacaine), have been developed in an effort to reduce the severity of cardiac toxicity, while preserving anaesthetic potency. Ropivacaine produces a sensory block similar to racemic bupivacaine, but with less motor block,[39-41] and is less cardiotoxic[42,43] However, a recent study suggests that ropivacaine may in fact be less potent.[44] Investigation of the pharmacoki-

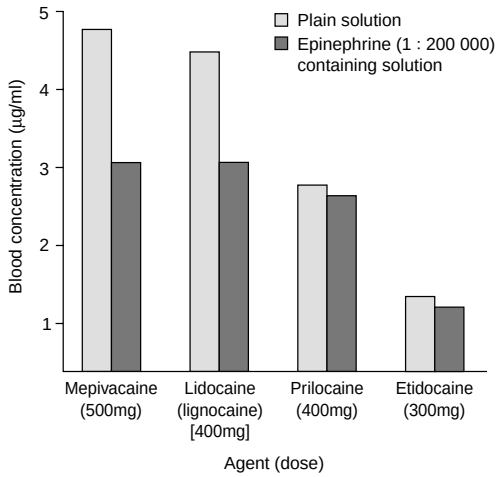

Fig. 2. Effect of epinephrine (adrenaline) on peak blood concentrations of various local anaesthetic agents administered epidurally (from Covino & Vassalo,[30] with permission).

netic properties of ropivacaine demonstrated a higher clearance, lower terminal half-life and lower plasma protein binding when compared with racemic bupivacaine.[45] In contrast, levobupivacaine results in a block that is clinically indistinguishable from that with the racemate,[46-48] and appears to have a reduced toxicity to both the heart and brain.[49-51] In a number of studies, the pharmacokinetic properties of levobupivacaine were shown to be very similar to those of racemic bupivacaine.[52-54]

2.6 Management of Local Anaesthetic Toxicity

The following mnemonic may be useful when dealing with both allergic and systemic toxic reactions to local anaesthetics:

- **S**top injection
- **A**irway
- **V**entilation
- **E**valuation of the circulation
- **D**rugs.

Control of the airway and ventilation is of paramount importance in the treatment of local anaesthetic toxicity, for reasons already outlined. Failure to do so results in profound respiratory and metabolic acidosis. To facilitate airway management, neuromuscular blocking drugs should be used if necessary.

The cardiovascular system should be evaluated without delay. Profound hypotension can occur in both allergic reactions and systemic toxicity, necessitating the use of vasopressors (for example epinephrine), while 'expansion' of the intravascular volume is necessary in the management of allergic reactions. Malignant dysrrhythmias, which occur particularly with systemic toxicity, should be controlled rapidly. Bretylium is recommended for recalcitrant arrhythmias,[55] and atrioventricular pacing and cardiopulmonary bypass are additional options in refractory cases. In addition, amrinone is recommended when conventional inotropes are ineffective.[56] Anticonvulsant medications such as thiopental and the benzodiazepines should be used judiciously as they may cause profound cardiovascular collapse. Bronchodilators, antihistamines and corticosteroids may be necessary to treat the bronchospasm and generalised oedema that can accompany allergic reactions.

In summary, there has been a significant decline in the incidence of systemic toxicity to local anaesthetics. There has been a similar decline in the incidence of serious cardiotoxicity to bupivacaine since it was first reported in 1979. However, these improvements should not lead to complacency. Patients can still succumb to the toxic effects of local anaesthetic drugs, and prevention of systemic toxicity should always be a priority. Test doses are strongly recommended and all local anaesthetic injections should be slow and deliberate with frequent pauses in between injections. Communication with the patient is strongly recommended at all times during and after local anaesthetic injections.

3. Neural Injury Associated with Regional Anaesthesia

3.1 Syndromes Associated with Spinal Damage

There are 3 well described syndromes associated with damage to the spinal cord, roots and coverings. Spinal and epidural anaesthesia are sometimes

linked with these syndromes. A brief description of these syndromes follows.

3.1.1 Cauda Equina Syndrome

This is a symptom complex involving the terminal portion of the spinal cord. The small autonomic fibres are chiefly affected. Patients present with autonomic disability, voiding and defecation problems, disturbed temperature control and sweating, and altered sensation to pin prick, temperature and proprioception in the distribution of the lumbar and sacral nerves. This syndrome may be related to vascular insufficiency or pressure ischaemia from injections of large volumes of solutions into the epidural space, particularly in patients with spinal stenosis.[57] It may also follow traumatic lumbar puncture,[58] injections of local anaesthetics containing preservatives and the use of microcatheters for continuous spinal anaesthesia.[59]

3.1.2 Adhesive Arachnoiditis

This is a sterile inflammatory response to intrathecal injection of chemical substances such as local anaesthetic drugs, radiographic materials, antibacterials and vaccines. When progressive, the subarachnoid space becomes obliterated by adhesions. Blood vessels become entrapped and may become endarthritic, leading to ischaemia of the cord. This syndrome is rarely seen as a complication of anaesthesia today, except when substances are accidentally injected.[60]

3.1.3 Anterior Spinal Artery Syndrome

Ischaemia or thrombosis of the anterior spinal artery gives rise to signs of lower motor neurone lesions at the level of the affected segments, with motor paralysis and preservation of sensation. Direct trauma, reduced perfusion pressure or venous congestion may cause anterior spinal artery syndrome.[61]

3.2 Trauma

Fortunately, serious permanent injury is rare following regional anaesthesia. Most neural injuries are associated with either paraesthesiae or pain on injection. Needle damage or pressure generated during injection of local anaesthetics account for most neural injuries.[28,62]

There is ongoing debate among anaesthesiologists about the safety of deliberately seeking paraesthesiae in regional anaesthesia.[63] There is also concern about performing regional anaesthesia in comatose/anaesthetised patients.[64] We do not have enough experimental data to answer either of these questions definitively.

Neuropraxia is one of the most common injuries following regional anaesthesia. The usual manifestation is a complaint of an area of persistent numbness that is not connected with patient positioning or the surgical procedure. This numbness gradually regresses over a period of weeks and is rarely observed beyond 3 months.

3.3 Infection

Infection is a rare complication of regional anaesthesia, although both epidural abscess[65,66] and meningitis[67,68] have been reported following CNB. A recent study from Denmark[69] reported an incidence of epidural abscess of 1 per 1930, which is higher than the 1 per 7500 reported in an earlier study.[70] The risks of infection are increased in immunocompromised patients, those taking cortico-

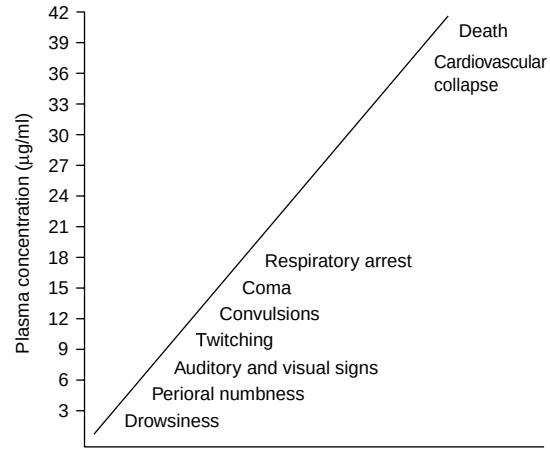

Fig. 3. Concentration-toxicity profile of lidocaine (lignocaine) [from Mather and Cousins,[33] with permission].

steroids and when epidural catheters are used. Meticulous adherence to sterile techniques is strongly recommended when performing CNB. It is also worth remembering that epidural abscesses can occur spontaneously (a reported incidence of 0.2 to 2 per 10 000 hospital admissions per year),[71] and that lumbar puncture has been safely performed in potentially bacteraemic patients.[72]

3.4 Toxicity

Neural toxicity occurs rarely following injection of local anaesthetics and additives. Several cases of cauda equina syndrome were reported in the 1970s following a formulation change in the local anaesthetic chloroprocaine.[17] More recently, 5% hyperbaric lidocaine has been linked with several cases of TNS.[20] Occasionally, toxic substances are accidentally injected perineurally, resulting in devastating destruction.

3.5 Ischaemia

Fortunately, ischaemic injuries are among the rarest reported following regional anaesthesia. When they do occur, several factors play a role, including hypotension, abnormal positioning, vascular disease, diabetes mellitus and clamping of major vessels.[62,73] The addition of epinephrine to local anaesthetic solutions may be controversial. No reduction in spinal cord blood flow was demonstrated in animals given both epinephrine and phenylephrine, but there was a significant reduction in dural blood flow.[74]

It would seem prudent, however, to avoid the use of epinephrine in patients at greater risk.

3.6 Compression

Compression from abscess formation is rare. However, the incidence of compression from bleeding has increased with the introduction of low molecular weight heparins (LMWHs) in North America.[75] Early diagnosis is of crucial importance when dealing with compression injuries (6 to 12 hours), otherwise permanent injury is likely.

3.7 Idiopathic

Finally there are some unexplained neurological injuries associated with regional anaesthesia. Anaesthesiologists must not always assume blame for unexplained neural damage; instead, they should be an integral part of the investigating team.

4. Failure of Anaesthesia

Failure of regional anaesthesia, in a sense, is not a complication. However, if in the course of administering general anaesthesia we failed to produce unconsciousness in 10 to 20% of cases, it would be considered a very serious issue. Furthermore, failure of regional anaesthesia, if not handled appropriately, may lead to serious complications; therefore, some discussion of failure of regional anaesthesia is necessary.

The failure rate of regional anaesthesia varies depending upon the block performed. One can expect a success rate of greater than 95% with ophthalmic anaesthesia. This can be attributed to the anatomy of the orbit, where the local anaesthetic is injected towards the apex of a cone-shaped bony structure occupied by the optic nerve. On the other hand, failure rates as high as 30% have been reported with brachial plexus anaesthesia.[76] The nerves of the brachial plexus in the axillary region are multiple, several millimetres apart and separated by fibrous bands. Therefore, a single injection of local anaesthetic into the axillary sheath will take a longer time to reach all the major cords and branches of the brachial plexus.

Regional anaesthesia is not well taught in many programmes in North America, contributing to higher failure rates.[77,78] Success rates of regional anaesthesia depend on a number of factors that fall under the mantle of infrastructure. Programmes that report high success rates with regional anaesthesia provide an environment that is conducive to performing high quality regional anaesthesia. Enthusiastic teachers, block rooms, support from surgeons and nursing personnel and planning are important components of a successful programme. Teachers should de-emphasise the success/failure

component of regional anaesthesia and concentrate more on providing a good experience for patients and learners. More and more evidence is accumulating that regional anaesthesia, by itself or in combination with general anaesthesia, not only provides improved pain control but also reduces morbidity and mortality.[79] One must always have a contingency plan in the event of failure of regional anaesthesia, including being prepared to administer general anaesthesia if necessary. Serious problems are more likely to occur when the anaesthesiologist is forced to administer general anaesthesia in a rushed and uncontrolled fashion.

Finally, it is important to assess the adequacy of a block well in advance of the start of surgery; it is not appropriate to await the patient's response to the surgical incision. This kind of cavalier approach to regional anaesthesia is unacceptable and has led to legal action[80,81] and jeopardises the use of these valuable techniques in anaesthesia.

5. Preoperative Considerations in Regional Anaesthesia

Safe regional anaesthesia begins when one first encounters a patient who is a potential candidate. Patients must be made aware of the more common possible complications of a given technique and it is unnecessary to concern them about rare complications. Thus, patients should be informed of the risk of headache and urinary retention, if spinal anaesthesia is used for a hernia repair, but not necessarily about spinal haematoma and cord compression.

Not all patients are suitable candidates for regional anaesthesia. Absolute contraindications include the following:

- patient refusal;
- coagulopathy [although an International Normalised Ratio (INR) of ≤ 2 is acceptable for ophthalmic procedures];
- infection at the site of injection;
- allergy to local anaesthetics.

Relative contraindications include:

- anxiety states;
- schizophrenia;
- neurological disease;
- comatose states;
- sepsis;
- anatomical anomalies.

All patients should have a thorough preoperative evaluation. The anaesthesiologist should carefully review the list of medications the patient is receiving and be particularly aware of anticoagulant medications, which are sometimes prescribed preoperatively. β-Blockers, antihypertensives and diuretics, alone or in combination, can augment cardiovascular changes that usually occur with CNB.

6. Monitoring Regional Anaesthesia

There is a general tendency to 'drop one's guard' when dealing with conscious patients and this is referred to as 'vigilance decrement'.[82] Monitoring requirements for regional anaesthesia should be just as stringent as those required for general anaesthesia.

Many regional anaesthesia procedures are performed such that the operator does not always have a full view of the patient's face (e.g. when performing spinal and epidural blocks). 13% of patients have a great fear of needles and vasovagal episodes are not uncommon when such individuals are approached with a needle.[83] Therefore, it is very important to have an assistant observing the patient at all times. The electrocardiogram and pulse oximetry are essential monitors while performing regional anaesthesia. A baseline blood pressure reading should be taken before performing the block. When the regional anaesthesia procedure is complete, the remaining monitors are attached. End tidal CO_2 monitoring is usually not used in conscious patients; however, there are special nasal prongs available that facilitate this.

Recovery from regional anaesthesia must also be carefully monitored. Discharge from the recovery area requires evidence of regressing sensory and motor blockade and stable vital signs. Local anaesthetic infusions are now routinely used in many medical centres around the world. Every effort should be made to minimise motor blockade in these cases for 2 reasons. First, to allow ambulation and second, to allow early diagnosis of com-

pression in the epidural or subarachnoid space should bleeding occur. Patients should be instructed to report back pain or increasing motor blockade. Patients receiving local anaesthetic infusions should have motor function of the lower extremities checked every 4 to 8 hours.

In our opinion, epidural catheter sites should be inspected on a daily basis for signs of inflammation or infection and catheters removed after 5 days unless there is some compelling reason to retain them. Epidural catheters should be carefully labelled and every effort must be made to avoid injecting substances that may be toxic to the central neuraxis.

7. Perioperative Management of Regional Anaesthesia Cases

The anaesthesiologist's responsibility for cases performed under regional anaesthesia begins when the patient agrees to the technique and continues until all of the effects of that intervention have regressed. Urinary retention tends to occur when spinal or epidural opioids are used, and therefore catheterisation is routinely required. Other features such as postdural puncture headache (PDPH), backache, bladder dysfunction and persistent numbness may not manifest themselves until 1 or 2 days after the procedure. However, responsibility for these inconveniences still remains with the anaesthesiologist. It is our duty to inform patients of these problems in advance and to advise them what to do in the event of such problems.

Sedative drugs are often administered to the patient, prior to performing the block, to reduce anxiety and improve their comfort. Agents such as benzodiazepines, opioids, propofol and thiopental may be used alone or in combination. However, they must be given with care. A patient given an excess amount of sedation may no longer be able to inform the anaesthesiologist when they experience paraesthesia or pain as the block needle is advanced or during injection of the local anaesthetic. A degree of airway obstruction may also result in an oversedated patient, leading to hypercarbia and possibly hypoxia.

In a recent study by Fanelli et al.,[84] 25% of patients stated that they would not have a PNB again because of the discomfort experienced. Therefore, although we strongly discourage the performance of regional techniques in the comatose state, we do encourage effective sedation. Ideally, we should administer sedation cautiously and maintain verbal contact with the patient.

Ideally, all patients should be visited postoperatively; however, in modern practice, most minor surgical procedures are performed on an ambulatory basis, and therefore a telephone call to the patient on the first postoperative day is a reasonable alternative. Patients undergoing supraclavicular blocks should be warned about the risk of pneumothorax and advised what to do if symptoms occur. Patients should be warned about the risk of burns (e.g. from radiators), or pressure on desensitised areas when sensory anaesthesia persists beyond discharge.

8. Complications of Intravenous Regional Anaesthesia

Intravenous regional anaesthesia (IVRA) is one of the oldest techniques in anaesthesia. Bier[85] first described IVRA in 1908. The technique as Bier described it did not have much appeal because it required an intravenous cutdown. Holmes[86] reintroduced the technique in 1963 using a percutaneous intravenous approach. IVRA is now widely used for minor upper and lower extremity procedures all over the world. The technique is very easy to perform but there are a number of recognised risks.

An intact tourniquet is essential in order to establish and maintain IVRA. Pressures of 200 to 250mm Hg or 150mm Hg above systolic blood pressure have been recommended.[87,88] The most common complication is local anaesthetic toxicity due to faulty tourniquet technique. Auroy et al.[28] reported seizure activity in 2.7 per 10 000 cases. Seizure activity may occur in 4 ways. First, inadequate exsanguination before inflation of the tourniquet allows one to exceed the tourniquet inflation pressure during the injection, allowing escape of the local anaesthetic solution into the circula-

tion. Secondly, interosseous escape of the local anaesthetic can also occur during injection. Thirdly, accidental or premature deflation of the tourniquet (within 15 minutes) allows the local anaesthetic to enter the circulation and finally, if an excessive dose of local anaesthetic is injected, toxicity may occur on release of the tourniquet, even following an appropriate inflation period. The tourniquet is usually applied to the upper arm or thigh; however, tourniquets have been applied to the forearm and lower leg to reduce the quantity of local anaesthetic required. Tourniquet application is less reliable in the distal portion of the extremity because of the changing diameter of the limb. The risk of toxicity is much greater in lower limb surgery where larger quantities of drug are required and there is much more leakage of local anaesthetic. This results in a higher incidence of inadequate blocks compared with upper limb IVRA.[89]

Seven deaths were reported in the UK between 1979 and 1983 when bupivacaine was used for IVRA; as a result, it is no longer approved for this use.[90] Lidocaine 0.5%, free from preservatives, is one of the most frequently used local anaesthetics for IVRA; the recommended dose is 3 mg/kg. One of the best tolerated local anaesthetics for IVRA is prilocaine because of its favourable pharmacokinetics; however, it is no longer available in many countries. A preservative-free form of chloroprocaine was recently introduced in Europe and has many potential benefits, especially with regard to toxicity.[91]

Compartment syndrome has been reported following IVRA of both upper and lower extremities[92,93] Hypertonic saline was mistakenly used as a diluent for the local anaesthetic in one of these cases. Long bone fractures of the forearm or leg are at increased risk of compartment syndrome and IVRA should not be used. Severe ischaemia of the upper extremity has been reported in at least 1 case following IVRA in an otherwise healthy young female: the aetiology was unclear.[94] Some of the possibilities include excessive tourniquet pressures, allergic reactions, undiagnosed Raynaud's disease, sickle cell disease, intra-arterial injection, or a drug administration error. Venous thrombosis is a recognised complication of tourniquet application. There are some anecdotal reports of subclavian steal syndrome following sudden loss of resistance in the upper extremity, leading to transient cortical blindness.[95]

In summary, the risk of serious complications following IVRA is very low. IVRA is ideally suited for upper limb surgical procedures lasting 30 to 45 minutes. Patients presenting for IVRA should be questioned about sickle cell disease, a history of Raynaud's and allergies to local anaesthetics. It is also advisable to avoid IVRA in traumatic injuries of the extremities. The limb must be thoroughly exsanguinated prior to injection of the local anaesthetic. Appropriate doses of preservative-free local anaesthetic should be used. One should always have dedicated intravenous access to inject other medications if required.

9. Complications of Brachial Plexus Anaesthesia

Brachial blockade is one of the most interesting blocks in regional anaesthesia. The axillary approach is by far the most popular among anaesthesiologists. It is also safer and is associated with fewer complications. Supraclavicular blocks, on the other hand, appear to have a higher success rate.

The incidence of seizure activity with brachial blocks is one of the highest of all the commonly performed regional techniques: Brown et al.[29] reported an incidence of 0.2% following all brachial blocks. Upon reviewing these data more closely, the incidence of seizures was very low in axillary blocks and 7 to 8 times higher with supraclavicular blocks (1.2 per 1000 *vs* 7.9 per 1000, respectively).

Pulmonary complications are uncommon following brachial plexus blocks and are confined to supraclavicular approaches. The incidence of clinically significant pneumothorax following supraclavicular brachial plexus anaesthesia varies between 0 and 6.1%.[96,97] Phrenic nerve paresis is very common following supraclavicular blocks, but patients rarely become symptomatic.[98] However, one should avoid supraclavicular techniques

in patients with moderate or severe impairment of pulmonary function and it is prudent that blocks should not be performed bilaterally.

Permanent neurological injury following brachial plexus blockade is rare. Neuropraxia lasting up to 3 months has been reported in about 2% of cases.[99] Auroy et al.[28] reported a 1.9 per 10 000 incidence of permanent injury following PNBs (which included brachial plexus anaesthesia) and found that either pain or paraesthesia was experienced in all cases.

The majority of serious permanent brachial plexus injuries have been reported following the supraclavicular approach. An interscalene block, performed with an 8cm needle, resulted in a permanent deficit at the C8/T1 level.[100] In another instance, permanent neurological deficit resulted following the interscalene approach where the block was performed after the patient was anaesthetised.[101] Bashein et al.[102] reported a case of permanent phrenic nerve palsy following an interscalene block and Winnie[103] has described an anecdotal report of a Brown-Sequard syndrome following attempted interscalene block using a spinal needle. It can be seen that in most of these cases of nerve injury involving the brachial plexus, the usual standard of care was not achieved.

The risk of vascular compromise appears to be greater with axillary blocks. There have been reports of venous aneurysm[104] and arterial compromise,[105] which required surgery. There is also a case of temporary ischaemia of the upper limb when the transarterial approach to the brachial plexus was used.[106]

Horner's syndrome (pupillary constriction, ptosis, enopthalmos, loss of sweating) is frequently observed following supraclavicular approaches to the brachial plexus[107] and patients should be informed of this temporary distortion. Hoarseness may occur if the local anaesthetic spreads to the recurrent laryngeal nerve. There are a number of other less common complications following brachial plexus block, including bronchospasm, haematoma formation, auditory impairment, total spinal/epidural block and carotid compression.

In summary, brachial plexus blocks are challenging but serious permanent complications are rare. Complication rates are higher with blocks above the clavicle. Most of the serious nerve injury cases reported involved deviations from the usual standards recommended. Axillary blocks appear to be safer overall, but supraclavicular blocks should be selected when an axillary block will not suffice, for example in shoulder surgery.

10. Complications of Peripheral Nerve Blocks

Most of the complications of PNBs are shared in common, although brachial plexus blocks have been dealt with separately in section 9 because many of the complications are unique. PNBs are performed by blindly injecting local anaesthetic drugs in the vicinity of neural targets using paraesthesia and nerve stimulation methods to locate a given peripheral nerve. The incidence of neural injury following PNBs is in the range of 1 to 2%. Most of these injuries are neuropraxias with the vast majority recovering within 3 months. Anaesthesiologists readily attribute these injuries to needle and injection damage whilst performing the block. There is considerable speculation about the aetiology of nerve injury following PNBs: a recent study by Fanelli et al.[84] reported that the most consistent variable associated with postoperative neuropathy was tourniquet pressures greater than 400mm Hg. Deliberate intraneural injection of local anaesthetics consistently damages nerves and should be avoided,[108] since pressures generated during an intraneural injection can be as high as 700mm Hg.[109] The cutting edges of the needle can damage nerve fascicles and the concentration of the local anaesthetic and additives may also contribute to the damage in these nerves.

A recent study by Auroy et al.[28] reported an incidence of serious nerve injury following PNBs of 1.9 per 10 000. Two factors were consistently associated with these injuries: pain on injection and paraesthesia. We must accept that needles used to perform regional anaesthesia can damage nerves. Should we use long or short bevelled needles or non-

cutting needles with a side hole? There are conflicting reports in the literature about these issues.[110,111] However, whatever needle design we use, we should heed the advice of one of the pioneers of brachial plexus anaesthesia, Kulenkampff,[112] who said, 'to avoid injury to the brachial plexus, only a very fine needle should be used.'

The risk of local anaesthetic toxicity is greater when performing PNBs because much larger doses of local anaesthetic are used.

The excellent analgesia and degree of motor block that the use of PNBs can provide has led to some concern about the late diagnosis of deep venous thrombosis arising from immobilisation and compartment syndrome. It should be pointed out that compartment syndrome is a clinical diagnosis, not based solely on a complaint of pain, and furthermore it is unlikely that neural blockade would mask an ischaemic pain as evidenced by the fact that tourniquet pain is a frequent accompaniment of upper and lower extremity regional anaesthesia.

11. Complications of Ophthalmic Regional Anaesthesia

Ophthalmic regional anaesthesia is practiced by both anaesthesiologists and ophthalmologists. It is an ideal technique for ophthalmic surgery since many of the patients are elderly with comorbid disease. Ophthalmic regional anaesthesia has a very steep learning curve and is among the most satisfying regional techniques performed because the success rate is so high. However, there are some serious risks involved.

From time to time patients taking anticoagulant medication present for cataract surgery. It is now considered acceptable to perform retrobulbar and peribulbar blocks in such patients, provided the INR does not exceed twice the normal value.[113] Surgery should be postponed in severely hypertensive patients because of the risk of retrobulbar haemorrhage.

11.1 Haemorrhage

The risk of retrobulbar haemorrhage varies depending upon the experience of the person performing the block. Edge and Nicoll[114] reported an incidence of 0.44% in 12 500 cases, whereas Hamilton[115] reported only 1 case in 20 000 blocks. The severity of the haemorrhage varies depending upon the origin of the bleeding; arterial bleeding is most dangerous because tamponade can occur, leading to ischaemia of the globe – lateral canthotomy may be required to relieve the pressure. The site of injection is also important; vascular structures are larger in the apex of the orbit; the upper, nasal area is particularly vascular and should be avoided. Therefore the use of small gauge disposable needles (25 gauge), less than 31mm in length, are now recommended to reduce the risk of haemorrhage.[116,117]

11.2 Brain Stem Anaesthesia

Brain stem anaesthesia results from local anaesthetic spreading directly to the brain from the orbit. The incidence is reported to be 1 per 350 to 1500.[118] Symptoms first appear within about 2 minutes of the injection. Maximum effects are visually observed within 20 minutes and recovery occurs in 2 to 3 hours. The symptoms of brain stem anaesthesia vary tremendously. Symptoms and signs that may be observed include:

- confusion;
- shivering;
- convulsions;
- paralysis;
- loss of consciousness;
- apnoea;
- hypotension;
- bradycardia;
- nausea/vomiting.

The treatment will vary depending upon the symptoms. Surgery should be postponed and the patient should be observed and treated appropriately in a recovery area.

Brain stem anaesthesia is less likely to occur if shorter needles (<31mm) and small doses of local anaesthetics are used.[117]

11.3 Globe Perforation

Blind insertion of a needle into the orbit is associated with a risk of penetrating the globe. The risk varies with the skill and knowledge of the operator, the site of injection and the axial length of the globe. One series reported an incidence of 0 per 2000[119] and another an incidence of 1 per 12 000.[116] Patients with an elongated eyeball (>26mm), such as myopic patients and those with retinal detachment or requiring refractive surgery, are more susceptible to perforation of the globe. Myopic patients with staphyloma are particularly vulnerable.[120] The axial length of the globe is often available preoperatively, particularly in patients presenting for cataract surgery.

All needles should be directed tangentially and with the bevel facing the globe. One must attempt to visualise in the 'mind's eye' the equator of the globe and avoid repositioning the needle until the equator is past. Pain or resistance to needle advancement may herald perforation of the sclera.

11.4 Myelotoxicity

The myelotoxic effects of local anaesthetic drugs have already been discussed in section 2.3. Direct injection of these drugs into the highly sensitive eye muscles can permanently damage them. The inferior rectus appears to be particularly vulnerable.[121,122] Resulting damage can result in permanent diplopia.

11.5 Miscellaneous Complications

Globe ischaemia, optic nerve and facial nerve damage and oculocardiac reflex are less frequent complications of ophthalmic regional anaesthesia.

In summary, knowledge of anatomy, gentle needle handling and experience help reduce complications in this important area of regional anaesthesia.

12. Complications of Central Neural Blockade

Spinal and epidural anaesthesia remain the mainstay of regional anaesthesia. Recent data from Auroy et al.[28] indicates that these 2 techniques account for close to 70% of all regional blocks performed in approximately 103 000 cases. Fortunately, serious permanent injury directly associated with CNB is rare. The risk of systemic toxicity is nonexistent with spinal anaesthesia and 0.01% with epidural anaesthesia.

12.1 Postdural Puncture Headache

PDPH is one of the most common complications reported with these 2 techniques. Advances in needle design and gauge have dramatically reduced the risk of PDPH associated with spinal anaesthesia to close to 1%, even in obstetric patients.[123,124] The incidence following epidural anaesthesia in obstetric patients is also around 1%.[125] However, the incidence of PDPH has been reported to be as high as 37%,[126] rising to 70% if the Tuohy needle inadvertently breaches the dura.[127]

A number of factors influence the incidence of PDPH, and they are listed in table III.

The smaller gauge (25 to 29 gauge) pencil point needles certainly reduce the incidence of PDPH, but they may be technically more difficult to use. They require the use of introducer needles since the smaller, more flexible, needles tend to deviate from the intended target when advanced through the tissues, resulting in multiple attempts. As a consequence of this narrow gauge, several seconds may elapse before CSF is visible in the hub, making identification of the subarachnoid space more difficult. These needles are expensive and some would argue that their use should be reserved for those at greatest risk for PDPH (patients aged <50 years, women, obstetric and ambulatory patients).

Even though enormous strides have been made to reduce the risk of PDPH, there is no room for

Table III. Factors linked with postdural puncture headache (PDPH)

Strong links	Weak links
Needle gauge	Angle of needle
Age	History of PDPH
Gender	State of hydration
Pregnancy	Duration of recumbency
Bevel design	Local anaesthetic
Bevel orientation	Preparation (solution)

complacency. PDPH is very incapacitating to patients and can increase the length of stay in hospital. Therefore, every effort must be made to reduce this complication.

A lengthy treatise on PDPH is beyond the scope of this article and can be obtained from textbooks.

12.1.1 Treatment

Treatment for PDPH includes:

- hydration;
- analgesics;
- recumbency;
- blood patch;
- saline patch;
- caffeine;
- corticotropin (ACTH);
- surgery.

More than 50 different treatments have been recommended to treat PDPH. Often simple measures such as hydration (oral/intravenous), oral analgesics and bed rest are sufficient. But if this fails, then the most reliable method is 'the blood patch', first described by Gormley[128] more than 40 years ago. About 20ml of autologous blood is drawn under sterile conditions and slowly injected into the epidural space at/or below the level of the puncture. The volume of injectate varies from patient to patient, up to a maximum of 20ml, and the endpoint for injection is a complaint of discomfort in the back. Smaller volumes are required in the thoracic region,[129] and a lumbar blood patch has been used to successfully treat cervical dural puncture.[130] Patients should remain recumbent for at least 2 hours following a blood patch and should not be involved in any strenuous activity for 24 hours.[131]

Complications have been reported with the blood patch: 2 cases of lumbovertebral syndrome,[132] various cranial nerve palsies[133] and 1 case of cauda equina syndrome in a patient who had 6 blood patch procedures.[134]

Most PDPHs respond to the blood patch; however, occasionally patients will not improve. We arbitrarily recommend no more than 3 blood patches on any 1 patient, with recalcitrant cases necessitating complete neurological workup. If the diagnosis is still considered to be PDPH, bed rest for 1 to 2 weeks is recommended. If the headache persists despite these measures, the patient may require a laminectomy and suture of the dura mater. Fortunately such incidences are rare. The use of magnetic resonance imaging may be helpful in establishing the diagnosis, as it can demonstrate the extradural accumulation of cerebrospinal fluid.[135]

12.2 Backache

Backache is a major concern of patients and the public at large following CNB.[6] Although it is likely that epidural and spinal needles contribute to some of the reports of back pain following CNB (albeit rarely), for example, disc lesions have been reported following spinal anaesthesia and lumbar puncture,[136,137] there are other causes of backache following regional anaesthesia that should be considered. The use of some local anaesthetic agents may be a factor. For example, the addition of EDTA to chloroprocaine was clearly linked with backache following epidural anaesthesia.[19] The injection of relatively large volumes of local anaesthetics into the epidural space has also been implicated in the production of back pain.[138,139]

Epidural analgesia and anaesthesia is often deemed to be the cause of *post partum* backache. However, 1 study demonstrated that the incidence of back pain, 2 months after delivery of a baby, was the same regardless of the anaesthesia technique[140] and 2 studies from the UK showed that there was no association between epidural analgesia and the incidence of new backache *post partum*.[141,142]

The most consistent and perhaps the most important factor associated with back pain following surgery is the duration of surgery, irrespective of the anaesthetic technique. The incidence rises from 18% with surgery lasting less than 1 hour to 50% when surgery lasts 4 to 5 hours.[143]

12.3 Transient Neurological Symptoms

Transient radicular irritation, now more commonly referred to as TNS, is a relatively new symptom complex that occurs following spinal anaesthesia. The name of the symptom complex suggests

a neurological origin but the typical symptoms are more suggestive of a musculoskeletal or myofascial origin and for that reason are described in this section.[144,145]

When Rigler et al.[59] reported 4 cases of cauda equina syndrome in patients given continuous spinal anaesthesia with 5% hyperbaric lidocaine, it raised concerns that lidocaine may be toxic to neural tissue. In 1993, Schneider et al.[20] published the first report of transient neurological toxicity in patients given a 'single shot' spinal using 5% hyperbaric lidocaine, again raising the possibility of a drug-related neurotoxic effect. Thus, the great debate began: are the symptoms due to drug toxicity or are they musculoskeletal in origin?

Typically in TNS, there is complete recovery from the spinal block. The patient complains of pain or dysaesthesia in the back and buttocks, within 24 hours, radiating to the legs, often cramping in nature and lasting up to 72 hours. The pain can be treated with simple analgesic medication. In contrast to patients with a cauda equina syndrome, there is no sensory or motor deficit and no bladder or bowel involvement.

It was thought that the greater baricity, mass of drug (100mg) and osmolarity associated with 5% hyperbaric lignocaine were causative factors. However, case reports and prospective studies have shown that TNS occurs with various concentrations of lidocaine, from 5 down to 0.5%,[20,146-150] with a dose as low as 40mg[151] and with both hyperbaric and isobaric solutions.[20,146-150] In addition, TNS has been described with other local anaesthetic agents and the use of vasoconstrictors may be a factor.[152,153] The incidence of TNS varies from 10 to 37% with 5% hyperbaric lidocaine,[20,146] 0 to 3% with bupivacaine,[152,155] 30 to 37% with mepivacaine[154,155] and 6.8% with tetracaine.[153]

Patient position has also been implicated as an aetiological factor.[20,146,152] The lithotomy position can cause stretch of the dorsal lumbosacral nerve roots, compromising perfusion to the neural tissue, making the nerves more susceptible to a toxic insult.

Comorbid disease may also play a role. In an *in vitro* study in diabetic and nondiabetic rats, lidocaine was applied to neural tissue resulting in significantly greater injury in the diabetic rats.[156] A woman with type 1 (insulin-dependent) diabetes mellitus developed TNS after a spinal anaesthesia with 1% isobaric lidocaine (40mg), following an arthroscopy.[151]

It is hard to imagine that when an agent such as lidocaine has been used for 50 years, in thousands of spinal anaesthetics, a problem related to toxicity would not have been identified earlier. Perhaps the symptoms were disregarded? Whatever the aetiology of TNS, should we continue to use lidocaine for spinal anaesthesia? It does not seem logical to prolong the duration of lidocaine (the safety of which is in debate) by the addition of epinephrine, when there is a suitable alternative available, namely bupivacaine.

In summary, there are some known causes of back pain following epidural and spinal anaesthesia. When these techniques are competently performed, it is highly unlikely that back pain or injuries occur as a result of trauma from the needle. The most consistent link with back pain in anaesthesia and surgery is the duration of surgery.

12.4 Disturbances of Micturition

One of the most common adverse effects of CNB is urinary dysfunction, and it is also one of the least studied complications of regional anaesthesia.[157] Concerns about urinary dysfunction are often overlooked because patients may require urinary catheterisation for other reasons. It takes up to 8 hours to regain full function of the detrusor muscles and even 1 episode of excessive distension of the bladder may have long term damaging effects on the ability to micturate. The problem becomes abundantly apparent when ambulatory patients undergo CNB.

In 1 study, 90% of patients administered relatively large doses of epidural morphine developed urinary retention, and another group given epidural bupivacaine had a 60% incidence.[158] A 1 to 3% incidence was reported by Breivik et al.[159] in pa-

tients receiving an epidural infusion of bupivacaine and fentanyl. This reduction in the incidence of urinary retention can perhaps be explained by the greater lipophilicity of fentanyl.

Bladder catheterisation should be performed routinely in all patients administered epidural or spinal opioids, in elderly patients and in those with known urinary dysfunction. The risk of urinary retention is a major inhibiting factor towards the use of spinal and epidural anaesthesia in an ambulatory setting.

12.5 Cardiovascular Disturbances

Hypotension is an expected cardiovascular event following either spinal or epidural anaesthesia. The onset and magnitude of hypotension is usually more marked with spinal anaesthesia. The expected drop in blood pressure following CNB is usually about 20% and is predominantly due to arterial and arteriolar vasodilation secondary to sympathetic blockade.[160] A decline in blood pressure of >30% is usually associated with a fall in cardiac output.[161] The key factor in sustaining normal cardiac output is maintenance of venous return: elevation of the lower extremities (10°) and of the head (5°) can maximise venous return during spinal and epidural anaesthesia. The reduction in blood pressure associated with CNB is generally more profound in elderly and hypertensive patients.

Bradycardia frequently occurs following CNB, the decline in heart rate usually being greater following spinal anaesthesia. The aetiology of this bradycardia is not completely understood. The most logical explanation following spinal anaesthesia is interruption of the cardiac sympathetic fibres (T1 to T4), allowing vagal tone to predominate. However, there are many examples of bradycardia when the cardiac sympathetics are intact. A reduction in venous return can result in a bradycardia by a number of mechanisms. There are vagally mediated receptors in the right atrium that, when stretched less, produce a decrease in heart rate.[162] In the left ventricles there are receptors that, when stretched, inhibit sympathetic activity and increase vagal tone.[163] A paradoxical form of this reflex may result in profound bradycardia during CNB – a relatively empty ventricle coupled with a vigorous ventricular contraction causing profound bradycardia, which may progress to full cardiac arrest.[164]

12.6 Cardiac Arrest

We do not normally link cardiac arrest with regional anaesthesia. In 1988, the Closed Claims Study alerted us to the dangers of cardiac arrest associated with spinal anaesthesia.[165] There were 14 cardiac arrests in otherwise healthy patients undergoing relatively minor procedures. The outcome was very poor: only 1 of the 8 survivors recovered sufficiently to live independently. The aetiology of cardiac arrest was not clear, but a number of patients were heavily sedated, possibly contributing to airway obstruction and subsequent hypoxaemia and hypercarbia. In 50% of cases, bradycardia was the first sign of a problem. Cardiac arrest occurred on average 30 minutes after the spinal injection and epinephrine was not administered in the majority of patients until 7 minutes had elapsed. Animal studies have shown that during 'total spinal' anaesthesia, coronary perfusion pressure falls below that required for successful resuscitation. It was also demonstrated that epinephrine can increase coronary perfusion pressure above that critical threshold.[166]

Information gleaned from the Closed Claim Study was very important. It was evident that cardiac arrest could occur with little warning, even in healthy patients under appropriately conducted spinal anaesthesia. There should be no delay in treating bradycardia and if and when cardiac arrest occurs, epinephrine should be used without delay and in large doses. An additional 30 cases of cardiac arrest following both spinal and epidural anaesthesia have been added to the Closed Claims database and further analysis of these cases shows a strong link with a sudden vagal stimulation linked with surgery, a medication or a position change.[167] The Closed Claims Study is limited by the lack of a denominator; however, it did draw our

attention to a serious but treatable problem, and for that reason was highly important.

A recent report by Auroy et al.[28] showed that the incidence of cardiac arrest under spinal anaesthesia was 6.4 per 10 000, 6 times higher than that reported with epidural anaesthesia and other regional techniques ($p < 0.05$). However, it should be pointed out that although this study was prospective, it was not randomised, therefore it is likely that the severity of illness was higher in the spinal anaesthesia cohort.

Known risk factors associated with cardiac arrest under CNB include:

- hypovolaemia;
- increased vagal tone;
- fixed cardiac output;
- sick sinus syndrome;
- reduced venous return;
- sepsis.

Hypovolaemia is a wellknown risk factor for cardiovascular collapse under CNB. Kennedy et al.,[168] investigating the effect of hypovolaemia in volunteers, demonstrated profound changes in cardiovascular parameters following haemorrhage and 1 patient experienced a vagal arrest. Increased vagal tone from any cause can induce cardiac arrest. Fixed cardiac output states such as mitral stenosis, aortic stenosis and cardiac tamponade require high atrial filling pressures in order to maintain cardiac output, and therefore any interference with venous return results in a profound fall in cardiac output. Patients with sick sinus syndrome are prone to develop bradycardia and cardiac arrest during CNB.[169] Finally, sepsis may be a contributing factor to cardiac arrest following CNB.

In summary, spinal anaesthesia is a very simple technique to perform but produces rapid and profound changes in physiology, which if not appropriately managed result in major circulatory changes that can be life threatening even in healthy patients. In the words of Nicholas Greene, 'the sine qua non of safe spinal anaesthesia is maintenance of venous return.'[170]

12.7 Neurological Injury

12.7.1 Permanent Neurological Complications

Serious permanent neurological injury following CNB is fortunately rare; however, when it does occur it sometimes generates more publicity and adverse comment than even a fatality. Furthermore, the courts are more likely to find blame in serious neural injury cases even when standards of care are met.[80,81] Anaesthesiologists are easy targets for blame in these cases, when in fact other causes are more likely. There is a paucity of prospective data on this topic in the literature.

Vandam and Dripps[136,171-174] reported one of the most thorough studies of neurological injury following spinal anaesthesia in a series of articles published in the early 1950s. They reported 1 case of serious permanent injury following 10 098 spinal anaesthetics, which was eventually shown to be unrelated to the technique. Massey Dawkins,[27] in a retrospective study from 1969, reported a 0.02% incidence of permanent damage following epidural anaesthesia in a series of 32 718 epidurals. More recently, Dahlgren and Törnebrandt[175] reported a 0.07% incidence of permanent neurological injury following CNB in a study involving 17 733 patients that was both prospective and retrospective. There were more serious permanent injuries in the epidural group (0.1%) than in the spinal group (0.03%).

This unexpectedly high incidence of serious permanent injury generated quite lively debate among anaesthesiologists in Scandinavia. Renck[62] suggested a number of possible reasons to explain the greater incidence. First, the incidence of risk factors for permanent injuries may have been increased in the population studied. Secondly, the methods of assessment used may have been flawed and thirdly, clustering of rare events in small populations and inclusion of all types of neurological complications may have artificially inflated the incidence of complications.

The most recent study of permanent neurological injury was reported by Auroy et al.[28] in France. This is the largest prospective study of regional anaesthesia complications ever reported, with over

100 000 cases. Serious neurological injury (defined as an injury lasting longer than 3 months) was 3 times more common following spinal anaesthesia than epidural anaesthesia (0.01 *vs* 0.003%); this is in direct contrast with that observed by Dahlgren and Törnebrandt.[175]

One must be cautious when interpreting these data because although the study by Auroy et al.[28] was prospective, it was not randomised and it is likely that the spinal anaesthesia cohort had a greater number of risk factors than the epidural group.

12.7.2 Transient Neurological Complications

Transient neurological complications are quite common following CNB. Vandam and Dripps[136] reported an incidence of 0.8% following 10 098 spinal anaesthetics; cases in which traumatic lumbar puncture had occurred were excluded. Symptoms consisted mostly of paraesthesiae in the perineum and legs and usually had a lumbosacral distribution. The authors concluded that these symptoms were linked with the local anaesthetic solution. Continuous spinal anaesthesia was used in a small number of cases in this series, but the incidence of transient neurological deficit (TND) was proportionally much higher in that group, suggesting the possibility of a mechanical aetiology for the symptoms.

The incidence of TND associated with spinal anaesthesia in the series of Auroy et al.[28] was 0.04% (20 cases of 40 640). The incidence of TND associated with epidural anaesthesia is lower; Massey Dawkins[27] reported an incidence of 0.1% in a series of 32 718 cases and Auroy et al.[28] an incidence of 0.01%.

It appears that the incidence of both transient and permanent neurological injury is still very low, but there is little room for complacency when dealing with vulnerable structures like the spinal cord and nerve roots. Risk factors for neurological injury were reviewed by Renck[62] and are listed in table IV.

Table IV. Risk factors for neurological injury after central neural blockade

Vascular pathology
Prolonged continuation of block
Anatomical distortion
Patient position
Polyneuropathy
Catheter technique
Bleeding disorders
Multiple attempts
Diabetes mellitus
Inexperienced personnel
Anticoagulant medication
Surgical issues
Hypotension
Idiopathic

12.7.3 Avoidance of Neurological Injury

Procedure-oriented anaesthesiologists sometimes neglect to question patients about potential risk factors for neurological injury. Patients who have a neurological disease require a thorough history and careful neurological examination if CNB is contemplated. We must carefully review the list of medications the patient is receiving, paying particular attention to anticoagulant medications and the timing of doses. We must also insist on recent laboratory values reflecting the patient's coagulation status before proceeding with CNB. Strict sterile technique is strongly recommended when performing CNB.

It is a good idea to use a marking pen to indicate key landmarks. It is most important to identify Tuffier's line when performing spinal anaesthesia; failure to do so has resulted in spinal cord puncture.[176] Patients with anatomical abnormalities, for example kyphoscoliosis, ankylosing spondylitis and Klippel-Feil syndrome, can present technical challenges and the procedure must not be continued if problems are encountered. Epidural anaesthesia is best avoided in patients with a history of spinal stenosis,[57,177] since even small volumes of local anaesthetics produce marked increases in pressure in these cases, interfering with perfusion of neurological structures.

Multiple attempts at needle placement are not only uncomfortable for the patient, but also increase the risk of damage to neural structures. There are very few cases that absolutely must be performed under regional anaesthesia, and if nec-

essary there should be no hesitation in abandoning the procedure in favour of general anaesthesia.

The hyperlordic position favoured by some surgeons exposes patients to increased risk of neurological damage regardless of the type of anaesthesia used and must be strongly discouraged.[177,178] CNB should be avoided in patients so positioned, in order to avoid further confusion should neurological injury occur.

12.8 Coagulation Disturbances

It is generally agreed that regional anaesthesia is contraindicated in anticoagulated patients. Indeed this was one of the undisputed absolute contraindications to regional anaesthesia. We have made some concessions to this embargo in recent years, including performing ophthalmic blocks in anticoagulated patients, and now many anaesthesiologists feel comfortable administering epidural and spinal anaesthesia in patients on low dosage heparin.

Our surgical colleagues prescribe a number of agents to reduce the risk of thromboembolic complications related to surgery. The fact that these agents can reduce morbidity and mortality by up to 70% leaves little doubt of their clinical benefit.[179,180] However, the use of such drugs (low dosage unfractionated heparin, LMWH and warfarin) can influence and affect our anaesthetic technique. The main concerns relate to an increased potential for bleeding during the perioperative period and an increased risk of vertebral canal haematoma in association with CNB.

Vertebral canal haematoma is a medical emergency and if unrecognised, will lead to spinal cord compression. It is a rare event and the majority occur spontaneously, with a reported incidence of 1 per 1 000 000 population per year.[181] Tryba[182] estimated the risk of vertebral canal haematoma to be 1 in 150 000 after epidural block and 1 in 220 000 after spinal block when he reviewed a large series (1.5 million patients) of epidural and spinal blocks.

Vandermeulen et al.[183] identified 61 cases of vertebral canal haematoma related to CNB between 1904 and 1994. Of these, 42 had identifiable coagulation abnormalities or risk factors. The block was noted to be technically difficult or 'bloody' in 30 patients and required multiple attempts in 12. 32 patients had an epidural catheter placed, spinal bleeding occurred in 15 immediately after the catheter was removed and in 9 cases there were therapeutic plasma concentrations of heparin. Overall, 53 of the 61 patients had an identifiable abnormality of either haemostasis or technique.

It can be clearly recognised that the main risk factors for vertebral canal haematoma are the presence of a coagulopathy, a difficult/bloody technique, the presence of an epidural catheter and the timing of its removal in relation to the administration of anticoagulant therapy.

The introduction of LMWHs such as enoxaparin highlighted again the problem of performing central blocks in the presence of drugs influencing coagulation. It has a much longer half-life (4 to 6 hours) and greater bioavailability (90%) than unfractionated heparin.[184] The recommended dose is 20mg once daily in Europe but 20 to 30mg twice daily in North America. This difference in practice is reflected in the number of reports of vertebral canal haematoma, and was examined by Tryba and Wedel,[75] who estimated the incidence of haematoma to be 1 in 2 250 000 in Europe and 1 in 14 000 in the US.

Antiplatelet drugs such as aspirin do not increase the risk of haematoma;[185,186] however, there is a suggestion that nonsteroidal anti-inflammatory drugs and heparin should not be given together in patients receiving CNB.[187]

There are European and US guidelines available to address the above problems.[182,188] They relate to the timing of the block in relation to the administration of thromboprophylactic agents, as dictated by pharmacokinetic factors. Blocks should not be performed within 4 hours of low dosage heparin; the drug can be given 1 hour after the block has been carried out (although the consensus statement of the American Society for Regional Anesthesia suggests that there is no problem performing a CNB in the presence of 'low dosage' heparin[189]). An intravenous bolus of heparin (for ex-

ample as used in vascular surgery) can be safely given 1 hour after the block has been performed.[190] With regard to LMWH, if the first dose is given postoperatively, then the time interval should be at least 8 hours and likewise, epidural catheters can be removed 8 to 12 hours after the last dose. Patients on anticoagulant drugs can have a CNB provided the drug has been discontinued and blood parameters have returned to normal.

Careful attention to these guidelines and avoidance of a 'difficult' block (something that is not always predictable) will minimise the risk of bleeding and haematoma formation. Obviously cooperation and liaison with surgeons is desirable. Finally, it should not be forgotten that spinal and epidural blockade *per se* can reduce the risk of thromboembolic disease.[191-195]

13. Complications of Combined Spinal-Epidural Anaesthesia

Combined spinal-epidural anaesthesia (CSE) has gained popularity in the past 15 to 20 years. It provides rapid onset of anaesthesia and analgesia with the ability to extend both the duration and extent of the block. A double-space or single-space (needle through needle) technique may be used. CSE has been used for orthopaedic, vascular and gynaecological procedures, but has gained most popularity in obstetric practice for both labour analgesia and anaesthesia for caesarean section.

There are a number of different commercial kits available for the needle through needle technique. They include those with a long spinal needle and those that have made modifications to the Tuohy needle, so that it has a 'back eye' or 2 lumens.

Many of the complications that occur with this technique are common to spinal and epidural anaesthesia (PDPH, hypotension, neuropraxia), and discussion of these may be found in a recent review article.[196] There are, however, a number of features unique to CSE that are worth discussing.

13.1 Failure

Failure of the spinal component has been reported to be as high as 25% when using the needle through needle approach.[197] Using the double space technique, the incidence was reduced to 4%.[198] In a more recent study, Albright and Forster[199] reported a failure rate of less than 1% in a series of 6000 patients. There are a number of factors that may explain the failure of a needle through needle approach, including deviation of the spinal needle from the midline, using a needle that is too short, the use of saline to identify the epidural space which may be mistaken for CSF, poor 'feel' when dura mater is punctured, and patient position.[196,200]

13.2 Metal Particles

There has been some concern that the passage of the spinal needle through the curved tip of the Tuohy needle may result in metal particles being introduced into the subarachnoid space.[201-203] Indeed Eldor and Guedj[204] have suggested that metal particles are a cause of aseptic meningitis. However, Carrie[205] stated that if the spinal needle is introduced perpendicular to the bevel of the Tuohy needle, contact between the 2 needles is minimal. Herman et al.[206] concluded that metal particles were not created when the spinal needle was passed through the epidural needle. However, they found that magnetising the epidural needle resulted in metal flakes/filings standing up on the bevel and that they were not seen in the washings. Likewise, a study by Hargreaves[207] concluded that there was no evidence to support the production of metal particles as a result of the needle through needle technique.

Therefore, although there appears to be the potential to introduce metal into the subarachnoid space, it has not been shown conclusively.

13.3 Catheter Migration

The risk of the epidural catheter breaching the dura via the puncture created by the spinal needle has been a concern. There are a number of case reports where this has occurred or at least suspected.[208,209] Holmström et al.[210] performed epiduroscopy in cadavers and found that it was impossible to force an 18 gauge epidural catheter through

the dural hole created by a 25 gauge spinal needle. However, if the dural hole was created by a Tuohy needle, then 45% of catheters were able to pass.

13.4 Infection

A number of cases of meningitis associated with CSE have been reported, all in the obstetric population, although it is possible that this reflects that CSE is used most commonly in this group.[211-213] The majority of reports have been of bacterial meningitis, which were recognised and treated promptly. There was one case of aseptic meningitis, considered to be related to skin disinfectant. There are 2 case reports in the literature of epidural abscess complicating CSE.[214,215]

Therefore, as with all regional anaesthesia techniques, meticulous attention to asepsis is mandatory.

13.5 Cardiac Arrest and Death

There has been 1 report of cardiac arrest, successfully resuscitated, associated with CSE for caesarean section.[216] A diagnosis of peripartum cardiomyopathy was made; the role of CSE is uncertain. But it should be noted that a relatively large dose of local anaesthetic was used, with a resulting high block.

In the most recent confidential inquiry into maternal deaths in the UK, only 1 death was directly related to anaesthesia.[8] This occurred in a patient who had received CSE. There were a number of points illustrating that management of both the CSE and resuscitation was substandard. Epidural drugs (alfentanil, clonidine, bupivacaine) were administered shortly after the spinal component was performed and in relatively large doses. The patient developed a high block and consequent respiratory difficulties, hypotension and, eventually, cardiac arrest.

13.6 Overview

CSE appears to be safe and efficacious in the provision of rapid pain relief in labour and allows flexibility in the subsequent management of labour and operative delivery. Care should be taken when performing the procedure and in the drugs administered.

14. Evaluation of Neurological Injury Following Regional Anaesthesia

Anaesthesiologists are easy targets for blame when neural deficits are reported postoperatively, especially if associated with regional anaesthesia. Since regional anaesthesia may involve blind insertion of needles towards neural targets, the burden lies with the anaesthesiologist to prove that damage was not caused, which makes neural injury a difficult problem to deal with. In reality, of course, the *res ipsa loquitar* doctrine does not readily hold and therefore physicians are obliged to maintain a very open diagnostic mind when dealing with these challenging cases. Patients may have pre-existing neurological disease that we are not aware of; anomalous anatomy may compromise the circulation to nerve tracts and of course, causes of neural injury related to anaesthesia must be included. Intraoperative positioning may compromise neural functions in some cases – improperly positioned retractors may damage nerves. In the obstetric population, 'obstetric palsies' can occur even in the absence of regional anaesthesia.

When a neurological injury is diagnosed postoperatively, we must obtain a thorough history and perform a complete physical examination. The anaesthesiologist all too often acts as a spectator in these cases, when in fact we have far more information concerning preoperative and intraoperative events than do neurologists. Symptoms and signs of compression of the spinal cord must be dealt with urgently (within 6 to 12 hours), otherwise permanent paraplegia or quadriplegia may result. We must work as a team and strive to arrive at a diagnosis before serious permanent injury occurs. Diagnostic tools should be used judiciously, to help us arrive at a correct diagnosis and this is where we must rely on our neurology and radiology colleagues to guide us. Electrodiagnostic and imaging techniques have taken the guesswork out of many

diagnostic dilemmas, allowing us to make a quick and precise diagnosis.

15. Conclusion

Significant advances have been made in regional anaesthesia in the past 100 years. The introduction of catheter techniques paved the way for improved regional anaesthesia conditions in the operating room and also allowed anaesthesiologists to extend their expertise into the postoperative period. We have better needles, catheters and local anaesthetics and we have more knowledge about the optimum use of these techniques in the perioperative period. The benefits of regional anaesthesia far outweigh the risks and we need to find better ways to teach regional anaesthesia.

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