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REVIEW ARTICLE

Anaesthesia for chronic spinal cord lesions

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Summary

Increasing numbers of patients with spinal cord injury present for surgery or obstetric care. Spinal cord injury causes unique pathophysiological changes. The most important peri-operative dangers are autonomic dysreflexia, bradycardia, hypotension, respiratory inadequacy and muscle spasms. Autonomic dysreflexia is suggested by headache, sweating, bradycardia and severe hypertension and may be precipitated by surgery, especially bladder distension. Patients with low, complete lesions, undergoing surgery below the level of injury, may safely do so without anaesthesia provided there is no history of autonomic dysreflexia or troublesome spasms. An anaesthetist should be present to monitor the patient in this situation. General anaesthesia of sufficient depth is effective at controlling spasms and autonomic dysreflexia but hypotension and respiratory dysfunction are risks. There is a growing consensus that spinal anaesthesia is safe, effective and technically simple to perform in this group of patients. We present a survey of 515 consecutive anaesthetics in cord-injured patients and a review of the current literature on anaesthesia for patients with chronic spinal cord lesions.

Keywords *Complications*; trauma, spinal cord. *Anaesthesia*.

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Dramatic improvements in the care of spinal cord injury patients have been achieved over the last few decades [1]. The active management of urinary tract and respiratory complications has lead to a decrease in mortality from renal and respiratory failure. Overall mortality decreased from over 80% at the time of World War I to less than 2% by the early 1980s. The implications of increased survival are an increase in the prevalence of spinal cord injuries, an increase in the numbers of patients presenting for elective surgery and an ever increasing number of spinal cord injured patients who develop further medical conditions as the result of normal ageing.

Although the majority of spinal cord injured patients currently undergo elective surgery in specialised spinal units, the appearance of such patients on operating lists of other hospitals is likely to increase. Anaesthesia in the cord-injured patient poses unique difficulties. An understanding of the relevant pathophysiology assists in the provision of safe peri-operative care.

Presentation of cases

Data were collected prospectively on the anaesthetic management of all patients from the National Spinal Injuries Centre at Stoke Mandeville Hospital who presented for surgery between April 1996 and April 1997. Records were obtained for 515 consecutive operations performed on 384 patients. The majority of patients were treated on dedicated spinal injury operating lists and were anaesthetised by a consultant anaesthetist.

Figures 1 and 2 show, respectively, the age distribution of the survey patients and the distribution of level of cord lesions. The largest age group encountered was the 30–50 year olds, although there is a significant (and growing) elderly population. Lower thoracic and lower lumbar lesions were most frequently seen. Many patients have been counted more than once and, as a consequence, these data do not provide a reliable reflection of the demography of spinal cord injury but they do give a

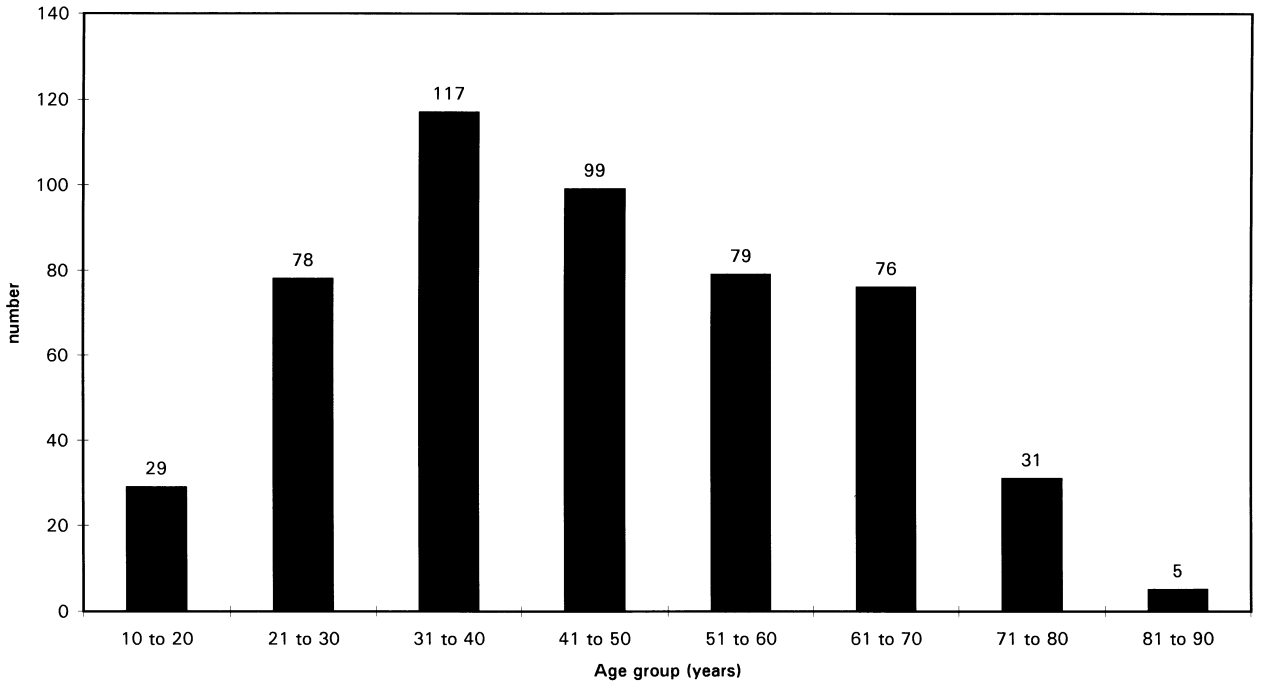

Figure 1 Age distribution of survey patients.

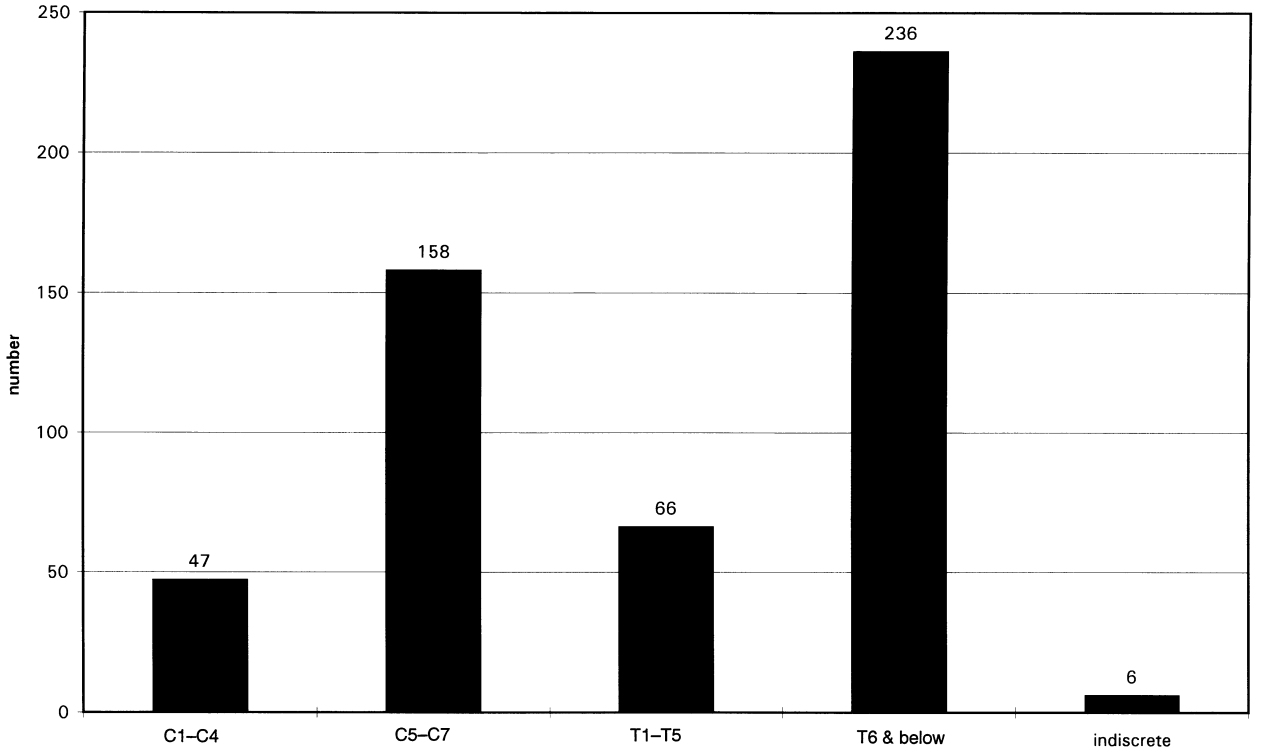

Figure 2 Distribution of injury level of survey patients.

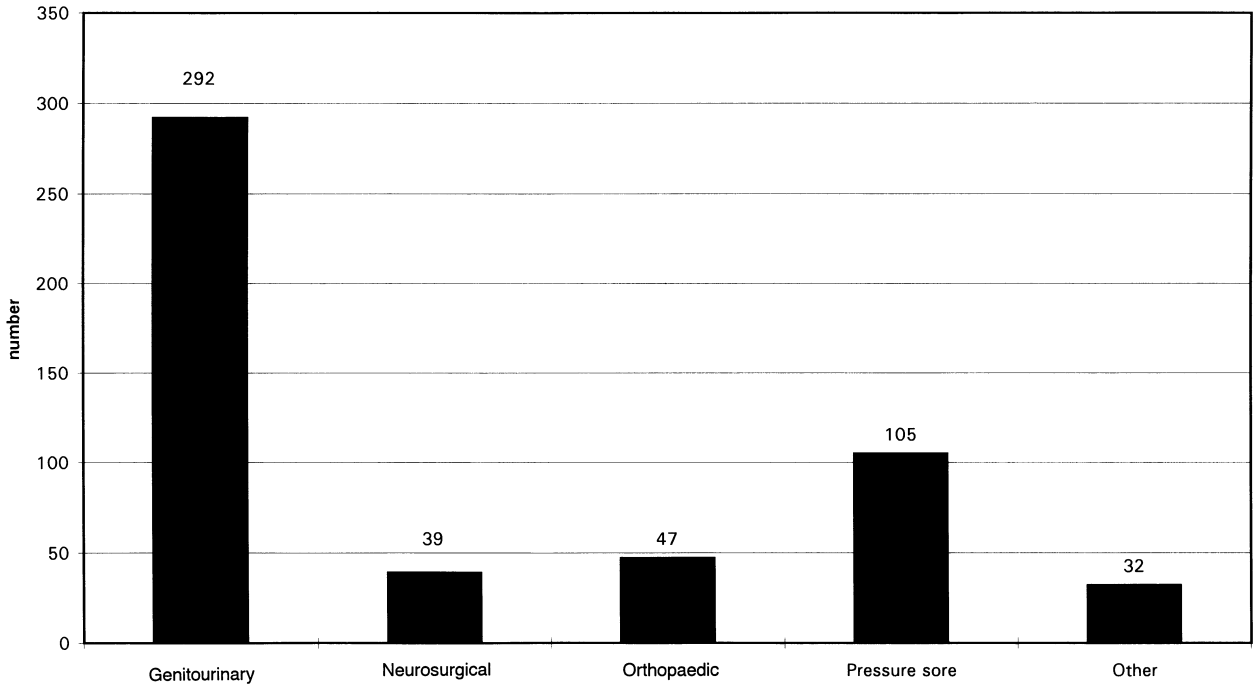

Figure 3 Surgical category for procedures in survey patients.

picture of the population that typically presents for surgery. The distribution of surgical speciality is given in Fig. 3. Urological procedures were by far the most common, followed by surgical treatment of pressure sores. Orthopaedic surgery was carried out mainly for spinal fixation and treatment of fractures sustained at the time of cord injury, although many fractures occurred in patients with long-standing lesions. The vast majority of cases in the neurosurgery category involved insertion or removal of intrathecal baclofen infusion apparatus for the management of spasticity but this category also includes insertion of anterior sacral root stimulators and phrenic nerve pacing. Operations in the 'other' category were a wide variety of procedures including hysterectomy, third molar extraction, laparotomy and electroconvulsive therapy.

Epidemiology

There are of the order of 40 000 spinal cord-injured patients in the UK, of whom 25% are managed from Stoke Mandeville Hospital. In the fiscal year 1994–95, there were 156 new admissions to the National Spinal Injuries Centre: 118 male and 38 female. Figure 4 shows the age distribution of these new patients. The majority of cases are traumatic in origin, with road traffic accident (RTA) being the commonest single cause. Table 1 shows the causes of the new lesions.

Pathophysiology

Spinal shock

At the time of spinal cord injury there is a brief and explosive autonomic discharge resulting from direct compression of descending sympathetic nerves [2], causing severe hypertension and arrhythmias. Intense increases in

Table 1 Causes of spinal cord lesions in new patients referred to The National Spinal Injuries Centre in the fiscal year 1994–5.

Traumatic	
Road traffic accident	38
Fall	29
Sports and recreation	22
Assault and violence	11
Hit by objects	8
Minor or unnoticed trauma (including disc prolapse)	5
Water accidents	2
Unknown	2
Air accident	1
Nontraumatic	
Vascular	15
Tumour of spine or spinal cord	8
Congenital and development	5
Infection	4
Motor neurone disease and demyelination	3
Unknown	2

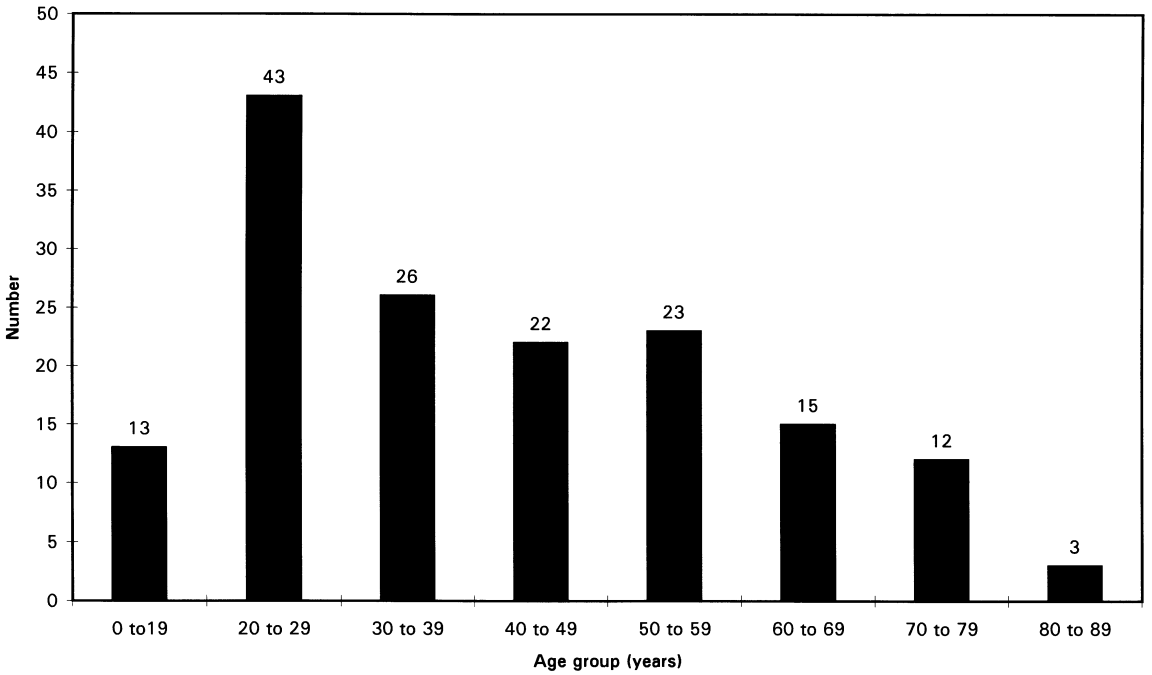

Figure 4 Age distribution of patients referred to The National Spinal Injuries Centre in the fiscal year 1994–95.

afterload may lead to left ventricular failure or subendocardial infarction and may cause disruption of the pulmonary endothelium. This phase lasts a few minutes and is followed by the longer period of 'spinal shock', resulting from the sudden loss of sympathetic discharge.

The term 'spinal shock' was coined in 1941 by Marshall Hall. This phase is characterised by hypotension and bradycardia, due mainly to vasodilation and reduced preload but myocardial dysfunction may also play a part. Muscle flaccidity and loss of reflexes accompany the cardiovascular changes. Vagal parasympathetic discharge continues unopposed. Certain manoeuvres such as intubation or tracheal suction may precipitate severe bradycardia or asystole, although in reported cases this response occurred only when tracheal stimulation occurred in the presence of hypoxaemia [3, 4] and may be prevented by pretreatment with oxygen or atropine [4, 5]. This phenomenon has not been reported outside the phase of spinal shock, although it is clear from personal experience that bradycardia may complicate the peri-operative period in any patient with high spinal cord injury.

The period of spinal shock may last a few days or up to 6–8 weeks after injury, during which changes in neuronal connections occur in the spinal cord distal to the site of injury and lead to the gradual return of sympathetic efferent discharge, along with muscle tone and reflexes. This final phase is therefore referred to as the 'reflex phase'.

Autonomic dysreflexia

Arguably the most important complication of spinal cord lesions, and of particular concern to the anaesthetist, is the phenomenon of autonomic dysreflexia. This condition is characterised by massive disordered autonomic response to certain stimuli below the level of the lesion. The condition was first recognized in 1860 [6]. In 1917, Head and Riddoch [7] recorded the effects of bladder distension on soldiers with spinal cord injury. The review by Colachis [8] is the most thorough evaluation of autonomic dysreflexia to date.

The most common clinical feature is hypertension, though it is not universally seen. Headache is common, as is sweating, which may be profuse and is often seen above the level of injury [7–9]. Flushing or pallor above the level of the lesion and reflex bradycardia are seen in the majority of cases [8]. Other reported features include pupillary changes, Horner's syndrome, nausea, anxiety and penile erections (Table 2). Hypertension in autonomic dysreflexia is paroxysmal and may be severe. Systolic pressure above 260 mmHg and diastolic pressures ranging from 170 to 220 mmHg have been reported [8]. Such hypertensive crises have led to intracranial and retinal haemorrhages, seizures, coma, myocardial ischaemia, pulmonary oedema and death [8].

It is difficult to arrive at a clear-cut definition of autonomic dysreflexia and this may explain variations in

Table 2 Clinical features of autonomic dysreflexia.

Commonly seen	Sometimes seen
Hypertension	Nausea
Headache	Anxiety
Sweating, pallor	Pupillary changes
Bradycardia	Horner's syndrome

the published incidence. Most recent reviews [8, 10, 11] quote an incidence of 85% of those with a lesion higher than the seventh thoracic vertebral level (T7). This figure dates back to unpublished observations by Ernest Bors and was first referred to in Kurnick's classic paper on autonomic dysreflexia in 1956 [12]. Lindan *et al*. [9] defined autonomic dysreflexia as a sudden increase in systolic and diastolic blood pressure with or without headache and cutaneous manifestations such as sweating or pallor. Their survey of a group of 213 patients with complete lesions at T6 or above produced a figure of 60% for women and 46% for men. The incidence is related to level of lesion and the condition affects 60% of patients with cervical lesions but only 20% of those with thoracic lesions. It can occur in patients with incomplete lesions. The onset of symptoms can occur at any time from 3 weeks to 12 years after injury [9, 13, 14].

The neurophysiological changes are the result of both loss of descending inhibition from higher centres and, more importantly, alterations in connections within the distal spinal cord [12, 15–17]. Afferent impulses are carried by fibres which synapse within the dorsal grey matter of the spinal cord at various levels and ascend the dorsal and lateral columns until blocked at the level of spinal cord injury. The terminal boutons of presynaptic fibres divided by the cord transection become disorganised, leading in turn to derangement in neighbouring, intact efferent fibres. Over the weeks following injury, presynaptic boutons multiply, forming chaotic, inappropriate reflexes. Interneurones excited by the afferent inputs synapse with preganglionic sympathetic neurones in the intermediolateral grey column of the cord. This results in a widespread inappropriate sympathetic response which lacks the usual descending inhibitory influences, leading to profound vasoconstriction.

Noradrenaline plays an important role in the pathophysiology of autonomic dysreflexia. Circulating noradrenaline levels decrease acutely after spinal cord injury and the levels remain low, presumably as a result of the reduced sympathetic activity. Increases in plasma noradrenaline levels and in urinary noradrenaline metabolites are observed during episodes of autonomic dysreflexia [8]. However, the levels reached are below those normally seen in nonspinal cord-injured patients and attempts to produce similar blood pressure responses with noradrenaline infusions in normal subjects require plasma levels many times greater than those seen during autonomic dysreflexia [18], suggesting that cord-injured patients are more sensitive to the effects of catecholamines.

A range of stimuli can thus trigger off a mass autonomic response. Bors and French [19] reported that the greatest responses were produced by stimuli with the most caudal root levels below the region of spinal cord injury. This explains why pelvic visceral stimulation is most commonly implicated. Bladder distension is responsible for 75–82% of episodes of autonomic dysreflexia [9, 13]. Other important causes are bowel distension, uterine contractions, acute abdominal pathology, anal fissure and urinary tract infection. Cutaneous and proprioceptive stimuli are less commonly implicated but manipulation of pressure sores, ingrown toenails and even sunburn have been known to trigger the phenomenon [8].

Management of an episode of autonomic dysreflexia should always begin with removal of the precipitating stimulus, if known. This alone may be sufficient and blood pressure often returns to baseline levels immediately. Assuming the upright position will also produce a useful fall in blood pressure [8]. If the cause is not obvious, bladder distension and faecal impaction should be excluded. A urinary catheter should be checked for blockage and urinary tract infection excluded. Tight clothing and footwear should be loosened. If the precipitating cause is still not clear, underlying pathology such as a long bone fracture, pulmonary embolus or acute abdominal pathology should be sought.

In many instances, though, pharmacological intervention is necessary to lower blood pressure. The other manifestations do not normally require drug treatment and usually subside if accompanying hypertension is treated. The attacks tend to be paroxysmal and therefore antihypertensive medication needs to be of rapid onset and short duration. Ganglion blocking drugs such as trimetaphan and mecamylamine are reported to be highly effective agents [8], though they are rarely used today. First-line agents include sublingual nifedipine 10 mg, sublingual or transdermal glyceryl trinitrate and α-adrenergic blocking agents such as phentolamine 2–10 mg [1].

Many other agents have been used in prevention or treatment. Phenoxybenzamine may be used, though in one case a 60-mg dose failed to abolish hypertension during an episode of autonomic dysreflexia [20]. Reserpine may be more reliable because of its nonadrenergic effects. The α-1 adrenergic blocker prazosin has been shown to be effective in prevention [21]. Guanethidine is effective in prevention but is associated with postural hypotension that persists after cessation of the medication

[22]. Calcium channel blockers are used widely in both prevention and treatment. Sublingual nifedipine 10 mg was shown to be effective in the treatment of hypertension during cystoscopy and is effective in prevention when given orally 30 min before cystoscopy [23]. Hydralazine is also commonly used to treat hypertension in autonomic dysreflexia but variable results have been reported. Lindan *et al.* [9] used hydralazine in 26 patients and found a narrow therapeutic range between minimal efficacy and episodes of hypotension. Clonidine is useful where dysreflexia and spasticity occur together. The effects of anaesthesia on the development of autonomic dysreflexia are considered later.

Other cardiovascular changes

Blood volume is often reduced in chronic tetraplegics, sometimes to as low as 60 $ml.kg^{-1}$, though this figure is highly variable and has led some to suggest that blood volume should be measured in these patients before surgery [24]. The changes in autonomic function lead to an abnormal response to the Valsalva manoeuvre. In tetraplegics, an increase in intrathoracic pressure causes a continuous fall of blood pressure with no tendency to plateau [25]. When the intrathoracic pressure is released there is no overshoot in blood pressure [26]. Patients with high lesions are prone to orthostatic hypotension, particularly in the first weeks after injury [5]. Head-up tilt causes a fall in stroke volume and cardiac output. Venous return is impaired and pooling of blood occurs in the lower limbs. Subcutaneous blood flow is reduced and vascular resistance in the limbs increases, in a response which is not abolished by proximal nerve blockade, suggesting a local veno-arterial reflex [27]. Renin levels rise to a greater extent than in normal subjects, leading to release of angiotensin II and aldosterone with consequent salt and water retention. This is independent of sympathetic activity and is probably the result of reduced renal perfusion [27]. Gradual adaptation subsequently occurs and frequent head-up tilting accelerates this process. Tolerance of the upright position probably owes more to changes to the autoregulation of cerebral blood flow than to improvement of postural hypotension [28].

Respiratory changes

Respiratory insufficiency is a common consequence of spinal cord injury. This is mainly the result of muscle weakness, although there are some data to suggest that reductions in respiratory drive and ventilatory response to hypercapnia also occur [29]. There are four groups of muscle which play a part in normal respiration: the diaphragm, the intercostal, the abdominal wall and the accessory group. Patients with lesions at C1 or C2 lose diaphragmatic function and become apnoeic immediately. If they survive, such patients are condemned to lifelong artificial ventilation. A proportion of those with lesions at C3 and C4 will suffer a similar fate, though the precise innervation varies and some will be spared. Some patients with partial phrenic paralysis will regain some function over time because of recruitment. Patients with lower cervical lesions lose intercostal function but frequently compensate by increasing the strength of the accessory muscles, particularly sternomastoid and the scalene muscles. Up to 90% of total ventilation may be contributed by the accessory muscles [30]. Another factor commonly overlooked in cervical injuries is the possibility of associated trauma to the phrenic nerve itself. Carter found that 2% of patients with traumatic tetraplegia develop unilateral diaphragmatic paralysis [31] as a result of other injuries, although this is usually transient and function typically returns within 2–3 months. Lesser degrees of diaphragmatic dysfunction occur more commonly.

Lung volumes reflect these changes. Vital capacity in tetraplegics is often 1000–1500 ml (30% of normal) immediately after injury but subsequent recovery to a value of 2200–2500 ml is usual [32] within 6 months of injury [33]. It is important to be aware, however, that the level of lesion may ascend temporarily over the first few days after injury and respiratory function may deteriorate over this period. Expiratory reserve volume is zero or markedly reduced as a result of loss of active expiration [33]. Paralysis of the abdominal wall is also responsible for the paradoxical effect of posture on respiratory function. In the supine position, the displacement of the diaphragm by abdominal contents permits a greater excursion and forced vital capacity is optimum in the horizontal or 35° head-up positions [34]. Work of breathing is increased [30].

Paralysis of abdominal wall muscles severely impairs the ability to cough. Tetraplegics achieve coughing by contraction of the clavicular portion of pectoralis major and this manoeuvre may be effective enough to cause dynamic airway compression [35]. Compensatory mechanisms are developed such that tetraplegics defend ventilation as well as normal subjects in the face of an expiratory resistance [36]. However, retention of secretions and atelectasis are common and ventilation/perfusion mismatch may occur. About 20% of patients with acute tetraplegia develop hypersecretion of bronchial mucus in the first weeks or months after injury. The mechanism is unknown but may be the result of disturbed neuronal control of bronchial mucus gland secretion [37].

Muscle

Following spinal cord injury, acetylcholine receptors spread from the motor end-plate of affected muscle fibres and cover the whole muscle membrane. When a

depolarising muscle relaxant such as suxamethonium is given, depolarisation takes place throughout the whole muscle. The resulting ionic flux can lead to vast and potentially fatal increases in serum potassium concentrations. This phenomenon was first described in 1970 by Tobey [38], when he published his series of four patients who suffered cardiac arrest after administration of suxamethonium. The patients were all young marines who had become paraplegic in Vietnam between 44 and 85 days previously. Tobey suspected that hyperkalaemia had been responsible for the cardiac arrests and went on to prove his hypothesis by subjecting four more patients to a suxamethonium infusion. In a later study in baboons, John *et al*. [39] showed that the peak increase in serum potassium concentration ($5.5\,mmol.l^{-1}$) occurred at 14 days after injury and the half-peak increase occurred at 8.4 days. Gronert and Theye [40] showed that potassium flux was reduced by precurarisation with gallamine but full paralysing doses were required to prevent it altogether.

Spasticity develops when intact spinal reflex arcs exist below the level of the lesion. Such reflexes appear after the phase of spinal shock, usually in the third or fourth month after injury. Spasms can be provoked by minor stimuli and can be violent enough to throw the patient from a wheelchair. Surgery is a potent stimulus. Spasms can cause injury to limbs and may lead to the development of contractures by preventing ranging exercises. However, the presence of spasticity helps to prevent osteoporosis and muscle wasting and improves venous return. Medical treatment is therefore only indicated where spasms interfere with daily activities. Spasms tend to be more troublesome in patients with incomplete lesions [1, 5]. Treatment involves removal of any precipitating cause and passive physiotherapy. The mainstay of drug therapy is with the γ-amino butyric acid agonist baclofen, which is given orally in the early stages. Increasingly, for patients with persistent troublesome spasms, baclofen is given intrathecally via an indwelling infusion pump. Benzodiazepines and dantrolene are also used occasionally. Kyphoscoliosis and other deformities can occur with asymmetrical or incomplete lesions and may require corrective surgery.

Bone

Bone density is significantly reduced below the level of spinal cord injury. Mineral loss occurs throughout the skeleton, except in the skull. Resorption occurs rapidly and is greatest in the pelvis. Homeostasis is reached after 16 months, at about two-thirds of the original bone mass and close to fracture threshold [41].

Para-articular heterotopic ossification occurs in 20% of cord-injured patients [1]. This is characterised by bone deposition around large joints and is of unknown cause. It presents as a warm swelling of the joint and is treated with passive physiotherapy, etidronate and, in severe cases, surgery.

Temperature

High spinal cord injury impairs normal mechanisms of thermoregulation, both preventing shivering in response to cold and sweating or vasodilation in response to heat. The patient becomes partially poikilothermic and body temperature reflects environmental temperature. In most tetraplegics, body temperature falls, though modest exercise in hot conditions can raise the temperature by up to 2 °C in 30 min [42]. The patient's ability to sense changes in body temperature is reduced. Hypothermia can present with delirium or depressed conscious level.

In high spinal cord injury, the principal thermoregulatory centre, the hypothalamus, is isolated from the majority of mechanisms for heat generation, which should theoretically impair the patient's ability to mount a fever. It is clear, however, that fever is reliably generated in response to infection [43] and considerable controversy has resulted from attempts to explain this observation.

Skin

Decubitus ulcers occur in up to 60% of patients with cervical lesions [44] as a result of immobility, lack of sensation over pressure areas, altered regulation of skin blood flow, muscle atrophy and psychological factors. Left untreated, pressure sores can lead to chronic infection, osteomyelitis, septicaemia and amyloidosis. The presence of an ulcer can trigger spasms or autonomic dysreflexia and is a common reason for surgery in cord-injured patients. Typical management of an established ulcer is in-patient admission and debridement, with a view to surgical closure after 2–3 weeks.

Venepuncture is often difficult in patients with high lesions [45] because the skin is atrophic and hyperaesthetic and has reduced blood flow. Involuntary withdrawal of the hand may occur in response to venous cannulation.

Blood

Anaemia is a common finding in spinal cord injury. In one series, 52.3% of patients had haemoglobin levels less than $13.6\,g.dl^{-1}$ [46]. Most commonly, the anaemia is normochromic and normocytic and is associated with chronic conditions such as decubitus ulcer or urinary tract infection [46].

The risk of deep vein thrombosis formation is greatly increased in the early stages. Untreated, 80–85% of patients will develop a deep vein thrombosis. A variety of prophylaxis regimens are in use; at Stoke Mandeville

patients are given warfarin from day 5 postinjury and the International Normalized Ratio is kept at 2.0–2.5 for 12 weeks.

Genito-urinary

Bladder emptying is dramatically altered by spinal cord injury. Immediately after injury, voluntary control of voiding is lost, the bladder is areflexic and retention of urine occurs. During the reflex phase, reflex voiding develops but detrusor–sphincter dyssynergia is common, whereby both the detrusor muscle and the external sphincter contract simultaneously. This leads to incomplete voiding, high intravesical pressures and vesico-ureteric reflux [47]. Urinary tract infection is common as a result of high residual volumes and the use of urethral catheters. Renal calculi form easily in these conditions. The combination of vesico-ureteric reflux, calculi and ascending infection are a potent recipe for renal failure, which, before the advent of modern urological management, was the most common cause of death in cord-injured patients. Renal amyloidosis resulting from chronic osteomyelitis or decubitus ulcers was once a frequent finding, although is rarely seen today. As a result of postural factors, secretion of atrial natriuretic peptide leads to nocturnal diuresis, which can lead to severe hyponatraemia in some patients.

Bladder management aims to achieve continence, with good reservoir capacity and voiding under control with minimal residual volume. However, it is rare to achieve all these aims. The initial options are intermittent catheterisation, indwelling urethral catheter or indwelling suprapubic catheter. Long-term management options include sphincterotomy, which helps to allow voiding at lower pressures, and anterior sacral nerve root stimulators which provide voluntary voiding with greatly reduced residual volumes. Artificial sphincters and ileal conduits may also be used.

Gastrointestinal

Acute gastroparesis and ileus is common immediately after spinal cord injury and caution needs to be applied with the use of oral fluids in the early stages. Gastric emptying is delayed in patients with high spinal cord injury. Segal *et al.* [48] showed that the mean half-time of gastric emptying in supine tetraplegics was 50.5 min, compared with 10.1 min in controls.

Chronic pain

Chronic pain may complicate spinal cord injury in as many as 60% of cases [49] and is usually difficult to manage. For a thorough account of assessment and management of this problem, the reader is directed to the chapter by Glynn and Teddy [50].

Table 3 Suggested checklist for assessment of spinal cord-injured patients before surgery.

1. **Sensory level compared with surgical field.**
2. **Complete or incomplete lesion?**
3. **Time since injury**: spinal shock or reflex phase? Risk of hyperkalaemic response to suxamethonium 3 days – 9 months.
4. **Previous anaesthetic history**: especially use of 'standby' anaesthesia. Previous anaesthetic records should be located.
5. **Airway and neck movements**: particularly after cervical spine surgery.
6. **Respiratory assessment**: particularly high lesions. History of respiratory tract infections, intensive care unit admissions, etc. Tracheostomy past or present. Vital capacity should be measured in all patients with lesions above C7. When in doubt, chest X-ray and arterial blood gases should be ordered.
7. **Cardiovascular assessment**: baseline blood pressure, heart rate. History of postural hypotension. History of autonomic dysreflexia – suggested by symptoms of headache and sweating when bladder full.
8. **Musculoskeletal**: spasms, contractures, pressure sores.
9. **Medications**: especially anticoagulants, baclofen and dantrolene.
10. **Allergies**
11. **Full blood count**: anaemia is common, especially in the presence of pressure sores or chronic sepsis.
12. **Urea and electrolytes**: to exclude renal impairment.
13. **Liver function tests**: especially where there is chronic sepsis.

Anaesthesia

Assessment

A suggested checklist for the assessment of patients before surgery is given in Table 3.

Standby, local anaesthesia and sedation

The loss of sensation caused by spinal cord injury means that many patients can undergo surgery without anaesthesia and without feeling pain from the operative site. The choice of this technique of 'standby anaesthesia' depends on various factors:

Site of surgery and level/completeness of lesion.

Likelihood of autonomic dysreflexia. Autonomic dysreflexia is more likely in patients with cervical lesions and in those who have a past history of the phenomenon, either during surgery or in daily activities. Autonomic dysreflexia occurs more frequently during urological surgery, and is less common during body surface surgery.

Likelihood of spasms. Patients who are frequently troubled by spasms usually require anaesthesia for control during surgery. Unlike autonomic dysreflexia, spasms are frequently triggered by proprioceptive and cutaneous stimuli.

Willingness of patient. Many cord-injured patients prefer to be asleep for surgery. Intravenous sedation is suitable in some cases.

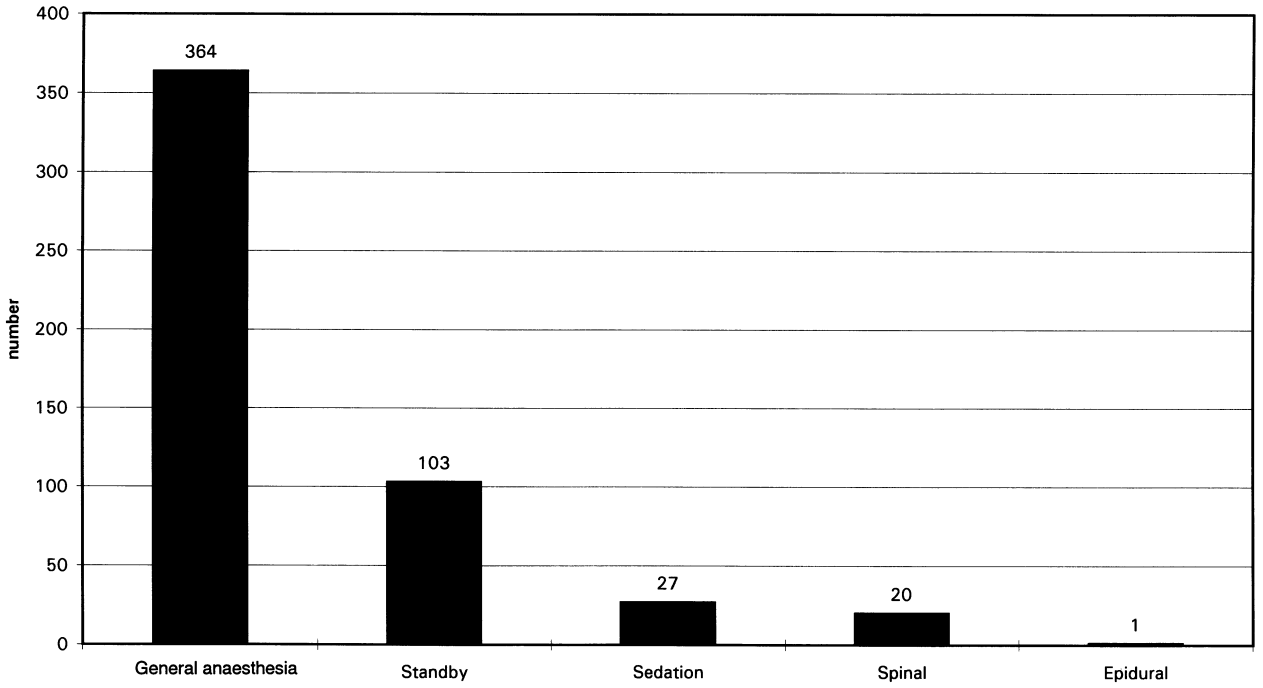

Figure 5 Anaesthetic technique employed in survey patients.

Previous anaesthetic history. Many cord-injured patients have had multiple procedures and may regularly undergo surgery without anaesthesia.

Figure 5 shows the anaesthetic technique employed in the survey patients. Approximately one-fifth of cases were conducted with 'standby' anaesthesia. The use of sedation, local infiltration or topical anaesthesia to prevent autonomic dysreflexia has been disappointing [51, 52]. Local infiltration anaesthesia is useful to allow surgery to take place in areas of incomplete sensory deficit, particularly for body surface surgery, and sedation is sometimes used for reasons of patient preference. Local anaesthetic solutions containing adrenaline should be avoided because of the greater sensitivity of the cord-injured patient to catecholamines. Sedation with benzodiazepines may reduce intraoperative spasticity [5].

For all operations on cord-injured patients, regardless of anaesthetic technique, it is recommended that the usual precautions are taken, namely that an anaesthetist should be present, venous access is secured and monitoring is in use throughout the procedure.

General anaesthesia

Preparation

Premedication may be given according to preference. Those patients with higher lesions may be sensitive to standard premedication doses [45], although delayed gastric emptying may reduce the effect of oral drugs. Sedative premedication is not commonly used at Stoke Mandeville Hospital. In some centres, pre-operative medication is given to prevent autonomic dysreflexia, e.g. oral nifedipine 10 mg, 1 h before surgery.

Monitoring

It is important that basic monitoring devices are applied before induction and remain in place until after recovery. Frequent noninvasive blood pressure measurements should be made during the induction period. Invasive monitoring is not considered necessary on a routine basis at Stoke Mandeville. Central venous pressure measurements are difficult to interpret but pulmonary artery wedge pressure measurement may be a useful guide to fluid management during major surgery in compromised patients.

Drug considerations

There are at present no data on the pharmacokinetics of anaesthetic drugs in spinal cord injury but certain differences may be predicted. Patients with spinal cord injury often have a lower than usual blood volume and a reduced lean tissue mass as a result of muscle wasting. This implies a smaller volume of distribution for intravenous anaesthetic agents and a smaller vessel-rich compartment. This explains the greater observed sensitivity of these patients to intravenous induction agents [45]. The effect is compounded by an absence of reflex sympathetic activity, reducing the ability to compensate for myocardial

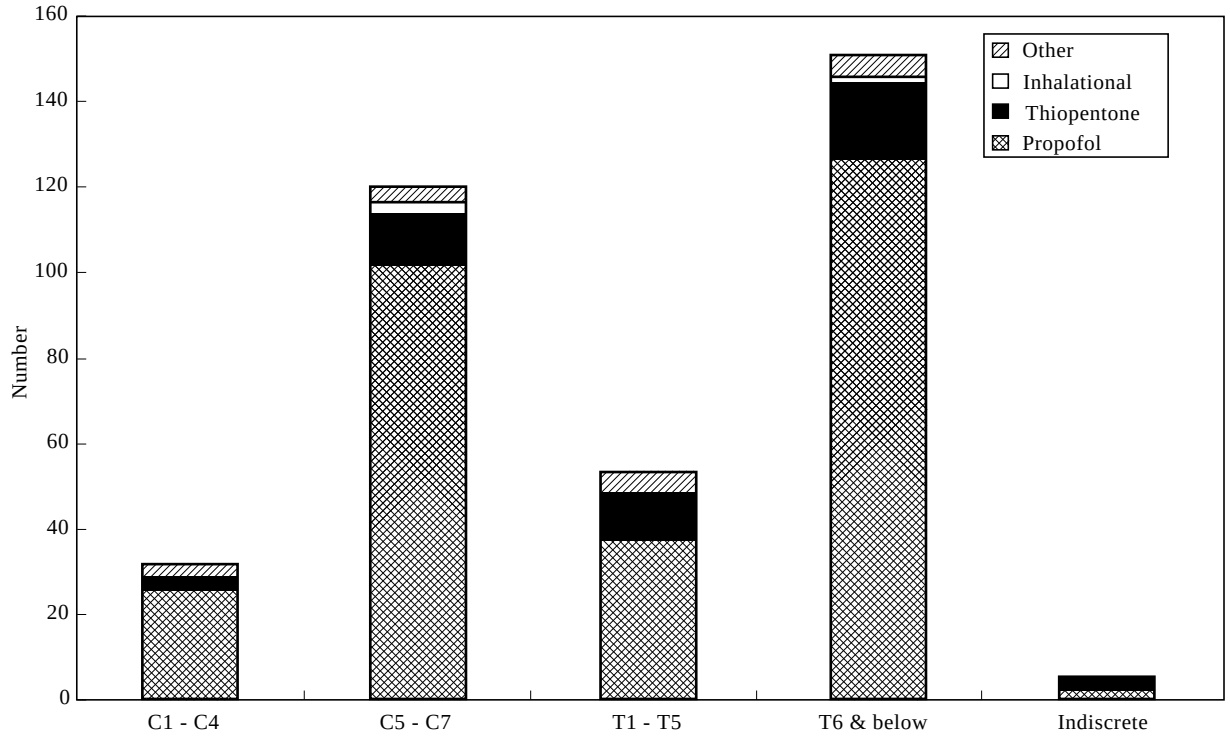

Figure 6 Induction agent used in survey patients receiving general anaesthesia, grouped by level of lesion.

depressant effects. Spinal cord injury is associated with renal impairment, which may result in reduced clearance of some drugs.

There are no special considerations that favour the use of any particular induction agent. However, propofol is firmly established in our practice, being used for the majority of survey cases. Figure 6 shows a breakdown of the agents used in the survey patients by level of lesion. Ketamine carries the theoretical risk of worsening muscle spasm. Papaveretum is avoided in urological surgery in males because of the theoretical risk that papaverine may provoke penile erections [5]. Few data are available on the use of nondepolarising neuromuscular blocking agents. Such drugs are commonly used to facilitate tracheal intubation but their use beyond an initial intubating dose is rarely required [45]. Anecdotal evidence and personal experience suggest that the doses required are smaller in cord-injured patients.

Suxamethonium

Since the appearance of a series of papers in the early 1970s, there have been very few studies on the use of suxamethonium in spinal cord injuries. A particular area of controversy is the duration of denervation hypersensitivity and the point after injury at which use of suxamethonium may safely resume. Of the handful of reported cases of cardiac arrest attributed to suxamethonium-induced hyperkalaemia, the longest period between injury and occurrence was 6 months. In this case, as with many others, the arrest was most likely to be the result of hyperkalaemia, though absolute proof is lacking. In Tobey *et al.*'s study on peripheral nerve injuries, hyperkalaemia occurred up to 192 days after injury [53], although the relevance of this to spinal cord injury is open to question. Published recommendations for the period after which suxamethonium can be safely used include 6 months [40, 45], 9 months [15] and 18 months [10] after injury. There are no hard data on which to base a rigid recommendation for the safe use of suxamethonium. Most authors agree that suxamethonium may be used safely in the first 72 h after injury [39, 40, 45] and few would deny that elective use of suxamethonium is safe after 9 months. Use of the drug between these two points carries an unspecified risk but could possibly be justified in the face of a strong indication. It is worthy of note that in the case reports of cardiac arrest reviewed, all the patients were successfully resuscitated and recovered uneventfully [[54] (two cases), [38] (four cases) [55] (two cases), [56] (one case), [57] (one case), [58] (one case), [59] (two cases), [60] (one case), [61] (three cases)]. It would therefore be unfortunate to allow any spinal patient to die of airway obstruction for fear of causing a hyperkalaemic arrest.

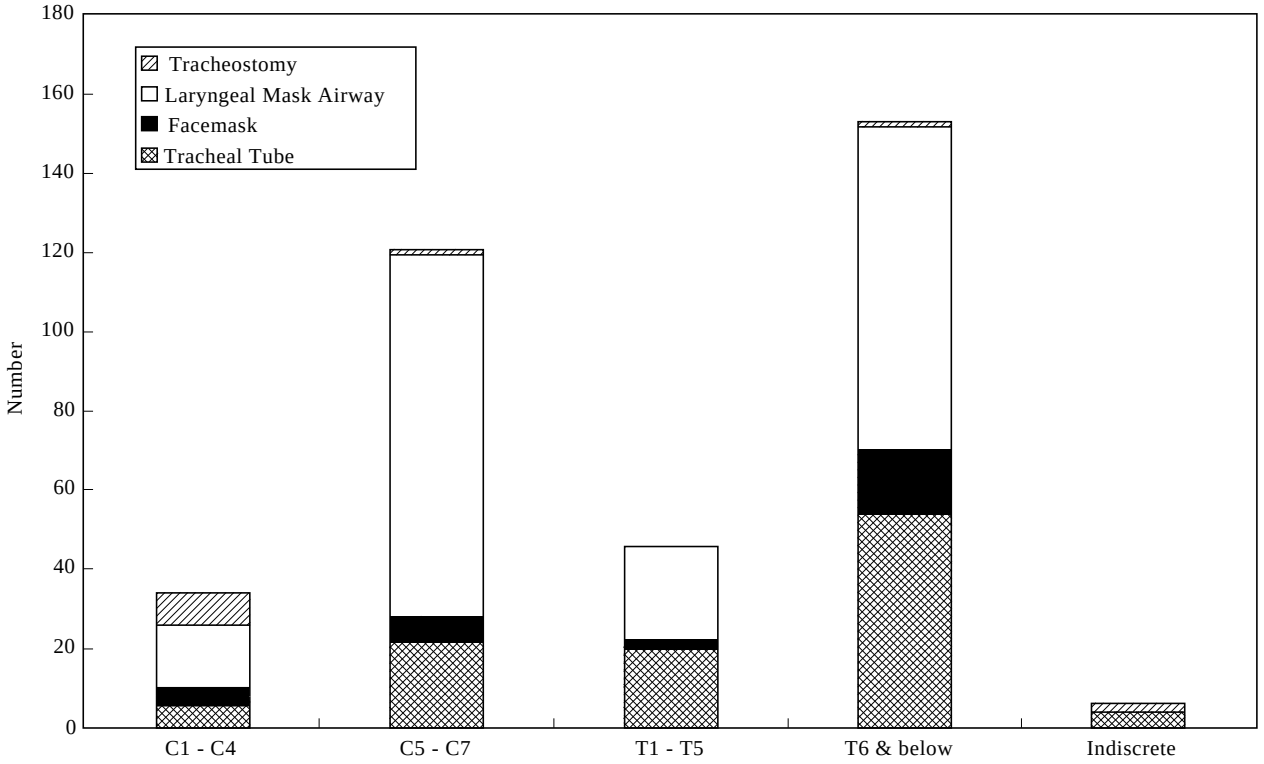

Figure 7 Airway management of survey patients receiving general anaesthesia, grouped by level of lesion.

Induction

A wide-bore intravenous cannula is recommended. It is common practice to give 500–1000 ml of crystalloid before induction to reduce the likelihood and severity of hypotension after induction. Although gastric emptying is slower in cord-injured subjects, no significant increase in the incidence of regurgitation of gastric contents has been demonstrated. Rapid sequence induction is not routinely employed at Stoke Mandeville for elective surgery. Tracheal intubation may be difficult in those with fused cervical vertebrae [10] and bradycardia may occur on intubation, especially during the phase of spinal shock. This can be prevented by the prior administration of atropine [3].

Figure 7 shows the techniques of airway management of the survey patients who underwent general anaesthesia, broken down by level of lesion. In our practice, the choice of airway technique is largely the same as it is for noncord-injured patients. The laryngeal mask airway is the most commonly used technique in all categories. Theoretical reservations that this should lead to an unacceptable incidence of aspiration have not been borne out.

Maintenance

Several authors have pointed out the advantages of intermittent positive pressure ventilation. Schonwald *et al.* noticed a lower incidence of arrythmias during halothane anaesthesia [62] and Goy points to greater overall stability [45]. Many patients with cervical lesions have borderline lung function and hypoxia and hypercapnia are likely if some patients are allowed to breathe spontaneously. However, positive pressure ventilation causes a greater drop in blood pressure in tetraplegic patients, particularly when combined with myocardial depressant drugs like halothane [5]. Spontaneous ventilation is usually appropriate for short operations. A total of 136 patients in the survey received intermittent positive pressure ventilation during anaesthesia. Most of these patients underwent surgery for which intermittent positive pressure ventilation is normally indicated, e.g. abdominal surgery and surgery in the prone position.

Positioning

The cord-injured patient is even more susceptible to the effects of poor positioning. The threat of pressure sores makes generous padding at all pressure points essential. Sheepskins or pillows are preferred [45]. Limbs should be well secured to prevent injury from spasmodic movements.

Fluid management

Many patients have reduced blood volume and fluid preloading may help to prevent hypotension during

anaesthesia. The cardiovascular complications of high spinal cord injury leave the patient unable to compensate for blood loss [45] and all fluid losses should be diligently replaced.

Anticholinergic drugs

It is common practice to give anticholinergic drugs at induction, especially where resting pulse rate is less than 60 beats.min^{-1}. This is highly recommended during the phase of spinal shock when reflex asystole is more common.

Thermoregulation

Cord-injured patients have a greatly reduced ability to generate heat after surgery. For this reason, prevention of hypothermia during surgery is of great importance. Standard warming mattresses are not suitable because of pressure on the skin [45] but humidification, fluid warming, superficial hot air blankets and operating in a warm theatre are all recommended.

Autonomic dysreflexia

In a single case report in 1963, Drinker and Helrich [63] reported the successful use of halothane to control autonomic dysreflexia. Since then halothane has been recommended as the drug of choice for cord-injured patients. However, it is likely that the depth of anaesthesia, not the choice of agent, is responsible for successful control of autonomic dysreflexia [62] and subsequent reports have supported the use of enflurane [62] and isoflurane [8].

An attempt was made to record the occurrence of autonomic dysreflexia during the study period. This was hampered greatly by the absence of an agreed published definition for the phenomenon. Data gathered on the basis of 'we don't know what it is but we know it when we see it' have limited value but in our survey 'mild' or 'severe' autonomic dysreflexia was recorded in 43 cases (8.3%). Of these, all but 10 were urological procedures. There were no instances of autonomic dysreflexia in patients with lesions below T5.

Where autonomic dysreflexia occurs during surgery, management should begin with removal of the precipitating stimulus, if possible. Most episodes appear to be brief and self-limiting and in 21 of our 43 cases no specific treatment was given. Dysreflexia occurring under general anaesthesia is best treated with increasing anaesthetic depth in the first instance. Where drug therapy is required, nifedipine and labetalol are used most commonly, though propranolol, midazolam, spinal anaesthesia and transdermal glyceryl trinitrate patches were all used on survey patients.

Spasms

In survey patients, spasms occurred in response to surgical stimuli in 36 cases (6.9%). Spasms can occur during general anaesthesia if the patient is too lightly anaesthetised and can usually be managed by increasing the anaesthetic depth. Muscle relaxants are rarely required for this purpose.

Penile erections

Penile erections may complicate urethral instrumentation in males. This response may be abolished by deepening anaesthesia or by the careful administration of metaraminol. Ketamine has been used successfully to prevent and treat erections in neurologically normal patients [64].

Recovery

Special attention to body temperature and respiratory function is required after general anaesthesia. Tetraplegic patients are best nursed supine. Autonomic dysreflexia may occur in recovery and may require drug treatment.

Regional anaesthesia

Spinal anaesthesia has been recommended [5, 10, 65], especially for urological surgery. Its advantages are the reliable prevention of autonomic dysreflexia and avoidance of some of the hazards of general anaesthesia. However, it may be impossible to determine the level of the block and it is not known whether the usual dose/response characteristics are seen in spinal cord injury patients. In some patients, the level of the block may be determined by observing the level at which spastic paraparesis becomes flaccid [66] or by the ability to elicit muscle spasms in response to ethyl chloride spray. Loubser *et al.* [67] presented a series of spinal anaesthetics in cord-injured patients with chronic pain. The level of subarachnoid block was determined in the usual way in 14 of their patients who had low or incomplete lesions (Table 4).

Desmond [68] warned of the potential of spinal anaesthesia for causing hypotension in cord-injured patients. However, Barker *et al.* [65] found that spinal anaesthesia was associated with remarkable cardiovascular stability in their series of six patients, using 1.2 ml of hyperbaric cinchocaine. It is likely that the already low sympathetic tone in cord-injured patients explains this observed stability. Broekner *et al.* [69] reported 25 successful cases using 25–50 mg of hyperbaric 5% lignocaine. Technical difficulties may occur, as a result of kyphoscoliosis, previous spinal surgery, inability to flex the spine due to spasms and bony deformities [62, 70], though these problems may have been overemphasized [62]. In Schonwald *et al.*'s series [62], lumbar puncture was possible in all 90 patients with lesions above T5 but proved impossible in three out of 19 patients with lesions below T5. Previous lumbar spinal surgery had been performed in all three patients. Alderson [5] has recommended use of 1.5–2.0 ml of hyperbaric 0.5% bupivacaine for routine use in urological surgery.

The use of spinal anaesthesia is one aspect of practice at

Table 4 Spread of lignocaine subarachnoid block in 21 spinal cord-injured patients [67].

Patient	Level of sensory deficit due to spinal cord injury	Level of sensory deficit achieved with subarachnoid block	Dose of lignocaine (mg)
1	T10	*	100
2	C7	T4	100
3	T8	T6	50
4	L4	T8	50
5	T12	T6	75
6	T12	T8	75
7	T12	T7	75
8	T12	*	100
9	T10	T8	75
10	T12	T10	50
11	T10	T6	75
12	L2	T6	50
13	C6	T4	100
14	T10	T6	75
15	T12	*	100
16	T8	T6	50
17	T4	*	100
18	T6	*	100
19	C6	T4	100
20	C5	†	100
21	C4	†	100

* No subarachnoid block was achieved above the level of cord injury due to spinal stenosis. † Patients with high complete lesions, in whom level of subarachnoid block was not discernible.

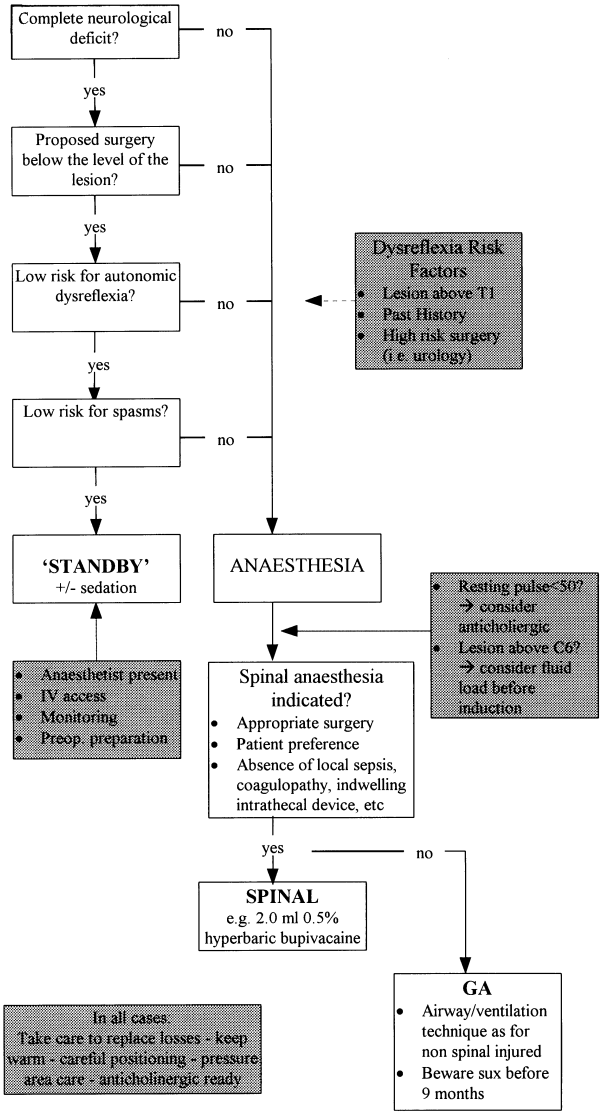

Figure 8 Flow chart for the anaesthetic management of spinal cord injury patients.

Stoke Mandeville that differs from other spinal injury units. In the study period, spinal anaesthesia was used in 20 cases (3.9%), which represents a considerably lower proportion of the total than would be found in many other spinal injury units. Sir Ludwig Guttmann, the founder of Stoke Mandeville's spinal unit, was a firm believer in the avoidance of any intervention that could possibly affect the spinal cord, lest it worsen the long-term neurological outcome. This policy was well known to his patients and even today many of them are suspicious of the offer of an intrathecal injection of local anaesthetic. There is, however, no evidence that spinal anaesthesia in any way affects neurological outcome [10, 62] and practice in this unit is gradually changing.

The use of epidural anaesthesia is widely reported as being less satisfactory. An adequate epidural block should theoretically be as effective as spinal anaesthesia in preventing autonomic dysreflexia but numerous failures have been reported [62, 69, 71]. Failure to block sacral segments and missed segments resulting from distortion of the epidural space are the most likely explanations, together with an inability to assess the block accurately. Another problem is the difficulty of meaningfully assessing the effect of a test dose, which has led to a total spinal in at least one case [62]. Subarachnoid block may be detected in some cases by the disappearance of ankle or knee jerks. Epidural opioids have been used to prevent autonomic dysreflexia with mixed success. Pethidine was used successfully by Baraka [72]. Fentanyl was used by Abouleish *et al.* [73], which initially controlled dysreflexia resulting from uterine contractions but the technique was eventually abandoned in favour of epidural local anaesthetic. The success of pethidine may be related to its local anaesthetic activity. The only common use of epidural anaesthesia in cord-injured patients remains the prevention of autonomic dysreflexia during labour (see below).

Brachial plexus block is a useful means of avoiding general anaesthesia for upper limb surgery. The axillary approach is probably preferable to the supraclavicular approach, given that a pneumothorax is more than usually

Table 5 Spinal cord-injured patients who gave birth at Stoke Mandeville Hospital between June 1994 and November 1996.

Patient no.	Parity	Gestation at delivery; weeks	Level	Lesion	Labour	Epidural	Mode of delivery	Notes
1	0+0	37		tetraplegic	spontaneous	yes	normal delivery	
2	1+1	38	T3	paraplegic	spontaneous	no	forceps	
3	0	36		paraplegic	spontaneous	yes	normal delivery	
4	0	39	C3	tetraplegic	induction	yes	normal delivery	
5	0	39	C6	tetraplegic	induction	yes	emergency Caesarean section	urinary tract infection general anaesthesia
6	2+0	34	T10	paraplegic	spontaneous	no	normal delivery	
7	1+1	37		tetraplegic	none	no	elective Caesarean section	spinal anaesthesia breech presentation
8	1+0	37		tetraplegic	spontaneous	no	emergency Caesarean section	urinary tract infection general anaesthesia
9	0	40	T11	paraplegic	spontaneous	no	normal delivery	
10	0	40	C6	tetraplegic	spontaneous	yes	forceps	
11	0+1	40	T1	tetraplegic	spontaneous	yes	normal delivery	
12	0	40	L1	paraplegic	induction	no	normal delivery	failed epidural

hazardous in cord-injured patients with borderline lung function. In patients with cervical lesions, a nerve stimulator may give neither the sensation of paraesthesia nor the familiar pattern of twitching in the hand, especially if roots of the brachial plexus have been damaged in the original accident.

Figure 8 is a flow chart to assist with the choice of anaesthetic technique in cord-injured patients.

Spinal patients and obstetrics

McCluer *et al.* [74] found that of 227 women with spinal cord injury, 76% of paraplegics and 56% of quadraplegics were sexually active. The incidence of pregnancy in cord-injured women is increasing [75] and maternal age at delivery is falling [76].

Between June 1994 and November 1996, 12 spinal patients gave birth at Stoke Mandeville Hospital. Details of these deliveries are given in Table 5.

Pathophysiology

The physiological changes that occur as a result of spinal cord injury are exaggerated by pregnancy. With high lesions, orthostatic hypotension may be prominent and further reductions in respiratory reserve may occur. Increased minute volume and oxygen demand, along with a reduction in functional residual capacity and poor cough may precipitate pneumonia or respiratory failure. Menstruation often ceases for 6–18 months after injury but usually resumes and fertility in cord-injured women approaches normal [10].

Antenatal complications

Urinary tract infection is extremely common in the pregnant spinal patient and is strongly associated with the use of catheters [76]. Premature labour is commoner in spinal cord-inured patients, particularly with high thoracic and cervical lesions [77–79] and in many cases may be precipitated by urinary tract infection [80, 81]. The anaemia commonly seen in spinal cord injury often worsens during pregnancy and is associated with the development of pressure sores [82]. Such sores can arise after less than 2 h of sustained pressure [75]. Aggressive prevention and treatment of anaemia is recommended [77, 83], with transfusion or iron supplements. The risk of thrombo-embolic complications is further increased during pregnancy [84].

Labour ward

Ideally all spinal cord-injured patients should be seen antenatally by an anaesthetist to discuss intrapartum analgesia and make a respiratory assessment. Patients with spinal lesions below T10 are likely to experience pain during labour and those with lesions between T5 and T10 may at least be aware of contractions. Uterine contractions are a potent trigger for autonomic dysreflexia and the incidence is greater with higher lesions. In many cases, the development of autonomic dysreflexia is the only clue to the onset of labour [73]. Intrapartum morbidity appears to be related to the degree of hypertension. A variety of techniques have been employed to control autonomic dysreflexia in labour, including a range of antihypertensive agents and general anaesthesia, but the measure employed most commonly is epidural analgesia.

Although failures have been reported, a correctly sited epidural block can effectively prevent the development of autonomic dysreflexia for the duration of labour and afterwards. It has been recommended that an epidural is sited before labour in those with high lesions or a history of autonomic dysreflexia [75] and this is the practice at Stoke Mandeville. Siting an epidural catheter may be

difficult because of spasms and disturbed anatomy or difficulties with patient positioning. Monitoring the level of the block is often difficult but the adequacy of the block can be judged by the quality of blood pressure control. Epidural block is not commonly associated with hypotension, provided there is appropriate fluid loading and the block is established slowly [75]. Autonomic dysreflexia may occur up to 48 h after delivery and it is often appropriate to leave the epidural catheter *in situ* for this time [85]. The headache and hypertension of pre-eclampsia may occasionally be mistaken for that of autonomic dysreflexia [84]. If epidural anaesthesia fails to control blood pressure, nifedipine, hydralazine or verapamil may be given. These drugs have no adverse effects on the uterus [86]. As a last resort, general anaesthesia and Caesarean section are occasionally required.

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