ABC of Spinal Cord Injury

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AT THE ACCIDENT

Spinal cord injury is a mortal condition and has been recognised as such since antiquity. In about 2500 BC, in the Edwin Smith papyrus, an unknown Egyptian physician accurately described the clinical features of traumatic tetraplegia and revealed an awareness of the awful prognosis with the chilling advice: "an ailment not to be treated." This view prevailed until the early years of this century. In the first world war 90% of patients suffering a spinal cord injury died within one year of wounding and only about 1% survived more than 20 years. Fortunately, the vision of a few pioneers—Guttmann in the United Kingdom together with Munro and Bors in the United States—has greatly improved the outlook for those with spinal cord injury, although the mortality associated with tetraplegia was still 35% in the 1960s. The better understanding and management of spinal cord injury have led to a reduction in mortality and a higher incidence of incomplete spinal cord damage in those who survive. Ideal management now demands immediate evacuation from the scene of the accident to a centre where intensive care of the patient can be supervised by a specialist in spinal cord injuries.

At present the annual incidence of spinal cord injury within the United Kingdom is about 20 per million of the population. In recent years there has been an increase in the proportion of injuries to the cervical spinal cord and this is now the commonest indication for admission to a spinal injuries unit.

Although the effect of the initial trauma is irreversible, the spinal cord is at risk from further injury by injudicious early management. The rescue services must avoid such complications, in the unconscious patient by being aware of the possibility of spinal cord injury from the nature of the accident and in the conscious patient by suspecting the diagnosis from the history and basic neurological assessment. If such an injury is suspected the patient may be handled correctly from the outset.

Causes of spinal injury—New patient admissions to Duke of Cornwall Spinal Treatment Centre 1984-5

Road traffic accidents

Car
Motorcycle
Pedestrian

55%

Motorcycle injuries constitute 31% of total admissions, twice the incidence due to car accidents.

Domestic and industrial accidents

Falls down stairs
Falls from scaffolding
and ladders

22%

Criminal assault 5%

Injuries at sport

Diving into shallow water
Rugby
Horse riding
Hang gliding
Others

18%

Management at the scene of the accident

With the recent proliferation of immediate care schemes there has been an increasing tendency for doctors to be called to the scene of accidents, particularly when ambulance staff are anxious about moving an unconscious patient or one complaining of pain in the neck or back.

The unconscious patient

If the patient is unconscious a brief description of the accident may be obtained from witnesses and the mechanism of injury suggested by the distribution of bruises, abrasions, or other wounds—for example, wounds of the forehead often accompany hyperextension injuries of the cervical spine. It should be assumed that the force that rendered the patient unconscious has injured the cervical spine until proved otherwise by radiography of its entire length. Until then the head and neck should be carefully placed and held in the neutral (anatomical) position and stabilised by gentle longitudinal traction. If gross spinal deformity is left uncorrected and splinted the cervical cord may sustain further injury from unrelieved compression. The patient should then be placed supine, taking extreme care that unnecessary movement does not occur in any part of the spine, so that resuscitation can be started immediately if necessary. The prone position is unsatisfactory as it may severely embarrass respiration, particularly in the tetraplegic. The standard recovery (semi-prone) position necessitates cervical rotation and is therefore also contraindicated. However, if vomiting occurs the patient may have to be turned on his side with the head supported in the neutral position.

The airway must be protected in unconscious patients. At the very least debris and dentures must be removed from the mouth, an oropharyngeal airway inserted, and oxygen given. Suction should be avoided if possible in the tetraplegic since it may stimulate a vagal reflex, aggravate pre-existing bradycardia, and occasionally precipitate cardiac arrest (a problem to be discussed later). With care, endotracheal intubation is usually safe in patients with injuries to the spinal cord, although there is a small risk of exacerbating an extension injury of the cervical spine during the normal manoeuvre required for this. Patency of the airway and adequate oxygenation must take priority in the unconscious patient, but whether intubation is performed at the scene of the accident or later, in the hospital receiving room, depends on the patient's respiratory state and the ability of the attending doctor. Endotracheal intubation may be facilitated by an assistant who prevents rotation, exerts gentle traction on the neck, and ensures that it is flexed or extended no more than is necessary.

Blind intubation techniques should not be undertaken since the insertion of an orotracheal tube in this way requires expertise and "blind" insertion of a nasotracheal tube is normally performed with the neck rotated. Oesophageal obturator airways may be inserted incorrectly, do not maintain patency of the respiratory tract below the hypopharynx, and, unless the stomach can be aspirated, may pose a risk of oesophageal rupture if the patient tries to vomit. In future flexible fiberoptic instruments may provide the ideal solution to the problem of difficult intubation in patients with a cervical injury.

Once the airway is protected intravenous access should be established in case cardiopulmonary support is required, but the clinician should remember that in uncomplicated cases of spinal cord injury the patient may be hypotensive and easily overinfused. If respiration and circulation are satisfactory the patient can be examined briefly where he lies. A short general examination should include measurement of pulse and blood pressure, brief assessment of level of consciousness and pupillary responses, examination of chest and abdomen for signs of gross trauma, and passive limb movement to detect any obvious fracture or dislocation. Spinal cord injury may be suggested by spinal deformity, diaphragmatic breathing due to intercostal paralysis (seen in patients with tetraplegia or high thoracic paraplegia), and flaccidity in the paralysed limbs.

Suction: beware of vagal reflex stimulation

Clinical features of spinal cord injury

- Pain in the neck or back, often with radiation due to nerve root irritation
- Sensory disturbance distal to neurological level
- Weakness or flaccid paralysis below this level

The conscious patient

In the conscious patient resuscitative measures should again be given priority. At the same time a brief history can be obtained which will help to localise the level of spinal trauma and identify other injuries which may further compromise the nutrition of the damaged spinal cord by producing hypoxia or hypovolaemic shock. A short general examination should be undertaken and a basic neurological assessment made by asking the patient to what extent he can feel or move his limbs. The diagnosis of spinal cord injury rests on the symptoms and signs of pain in the spine, sensory disturbance, and weakness or flaccid paralysis.

Analgesia

In patients with low spinal cord injuries narcotic analgesics are usually safe, but in cervical and upper thoracic injuries, where ventilation is already impaired, they should be avoided if possible, particularly as chest injuries often coexist. If pain is uncontrollable without their use then the dose administered must be kept to a minimum to control symptoms without causing respiratory depression. In practice pentazocine is relatively effective and has been found to be safe.

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The cold water tank that feeds the boiler of my domestic hot water supply contains a considerable growth of algae. A plumber advised me that it was not practicable to be rid of this growth in such a tank as if a new tank were fitted it would be similarly infested within a month or two. Is there a health hazard here?

The algal growth in the cold water tank is probably *Chlorella* or a related species. The organism gains access to the system through the potable water supply, which may contain low concentrations of such algae. Chlorination of the system at concentrations of 2 parts per million as free chlorine for 24 hours should be sufficient to kill the algae. If the tank is uncovered then this might provide another means of entry for the algae, as well as providing access for light, which is essential for continued growth. Covering the tank is therefore also necessary. *Chlorella* and related algae do not produce any toxins that cause disease in man, although abortion and death in cattle have been described.—D BAXTER, lecturer in community medicine, Manchester.

To what extent should a family history of hypertension and stroke be taken into account when deciding what oral contraceptives a woman should use?

The history requires clarification, especially regarding strokes, which are a common cause of death in the elderly. Much more notice should be taken of arterial disease occurring in a first degree relative under the age of 45 than a precisely similar history in older or more distant relatives. The former would indicate referral, when possible, for plasma lipid studies.¹ If these showed an atherogenic lipid profile, that would be an absolute contraindication to the combined oral contraceptive and also, in my view, to any prolonged use of the progestagen only pill. If the patient had a normal lipid profile and was not herself hypertensive, however, either type of oral contraceptive might be prescribed at the patient's request, with full monitoring. In the case of the combined oral contraceptive special attention should be paid to regular measurements of blood pressure, three monthly at first and never less frequently than every six months—with a low threshold for recommending transfer to another method should it arise. This extra concern would be for fear of haemorrhagic stroke, in the light of the family history, particularly if she was a cigarette smoker or suffered from migraine. Indeed, she should be warned that the occurrence of any focal symptoms in association with the headaches—symptoms that might be interpreted as caused by transient cerebral ischaemia—would indicate stopping the method at once and taking immediate medical advice.² In the absence of these problems, however, she could continue to use the method to the usual upper age limit for smokers

and non-smokers respectively.¹ The progestagen only pill could be used even if the patient were mildly hypertensive, since in the absence of oestrogen progestagens appear to have no effect at all on blood pressure.³ It would be a particularly appropriate choice in an older woman.—J GUILLEBAUD, senior lecturer in gynaecology, London.

1 Guillebaud J. The combined pill. In: Loudon NB, ed. *Handbook of family planning*. Edinburgh: Churchill Livingstone, 1985:58-61 (table 4.2).

2 Bickerstaff ER. *Neurological complications of oral contraceptives*. Oxford: Oxford University Press, 1985:84-6.

3 Wilson FSB, Cruickshank J, McMaster M, Weir RJ. A prospective controlled study of the effect on blood pressure of contraceptive preparations containing different types and dosages of progestagen. *Br J Obstet Gynaecol* 1984;91:1254-60.

If a neonate is infected with hepatitis B virus from a hepatitis B surface antigen positive, hepatitis B e antigen positive contact, the infant has a 95% chance of becoming a carrier of HBsAg. If the same neonate is immunised with vaccine there is an almost equal chance that the child will become immune. Why does HBsAg on its own elicit an immune response in neonates yet fail to produce the desired response when present with intact virions?

Most babies born to HBsAg, HBeAg positive mothers in high risk areas are initially IgM anti-HBc negative and first develop HBsAg in serum three months or more after birth.^{1,2} Infection is therefore considered to occur at or shortly after birth.^{1,2} The presentation of HBsAg in microgram quantities in adjuvant (vaccine) at this time is more likely to stimulate an anti-HBs response than the exceedingly small amounts presented in the few virions that infect the neonate. The large amounts of HBsAg released after incubation at around three months in the unvaccinated baby probably overwhelm the immune system and produce "high zone" tolerance. A similar mechanism may explain the impaired anti-HBs response in chronic HBsAg carriers.³ The priming of anti-HBs in the vaccinated child may prevent tolerance and allow more effective elimination of infected cells early on.—A L W F EDDLESTON, professor of liver immunology, London.

1 Beasley RP, Hwang LY. Postnatal infectivity of hepatitis B surface antigen-carrier mothers. *J Infect Dis* 1983;147:185-90.

2 Anonymous. Prevention of perinatally-transmitted hepatitis B virus infection [Editorial]. *Lancet* 1984;i:939-42.

3 Dusheiko GM, Hoofnagle JH, Cooksley WG. Synthesis of antibodies to hepatitis B virus by cultured lymphocytes from chronic hepatitis B surface antigen carriers. *J Clin Invest* 1983;71:1104-13.