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A Selection of Abstracts from the Conference (10 Years of Spinal Research)

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Epidemiology of road accident: spinal injury survey

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Two hundred and two cases of severe spine and cord injuries were admitted to various hospitals in New South Wales during 1977 and 1978.

First aid at the site of accident was either inadequate or not given. One man ambulance and private vehicles were used in 43 cases. The mean time from notification of the ambulance to admission to the hospital of so-called first admission was 2·6 hours. Following admission to the nearest hospital the surviving patients (132) underwent 155 transfers to other hospitals. The minor country hospitals acted as qualified first aid stations only. All but 28 patients were eventually transferred to multi-disciplinary hospitals for definitive treatment; of the 28 not so transferred 24 died.

The time lag, from the time of accident to definitive treatment at a multi-disciplinary surgical centre or spinal unit, amounted to a mean 22 hours in this series.

The lack of qualified first aid at the site of accident, the long hours of and inadequate conditions in transport for definitive treatment, the unnecessary multiple inter-hospital transfers, are all perfectly preventable factors and may have accounted for or contributed to at least one half of all deaths and to secondary cord damage in the surviving patients in this series.

Reduction of the time lag between the accident and definitive treatment to the unavoidable minimum would save many lives and prevent many a neurological deficit. To achieve such reduction integration of ambulance and multi-disciplinary hospital services into efficient trauma centres is most urgently required and long overdue.

The contribution of neuropathology to the restorative neurology of spinal cord injury: acute, subacute and chronic. A review of existing and possible new interventions

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The neuropathology of a large number of patients who died with spinal injury provides a basis for 'restorative' neurological considerations including neurophysiological and artificial methods. In a study of roadside deaths it was found that in many subjects the spinal cord either escaped injury altogether or was only partially damaged. This finding emphasises the need for great care in the immediate management and adequate resuscitation of such patients. The preliminary results of experimental work conducted by Professor Wise Young of New York University on cats indicates that the salvage rate of the long tracts can be improved using methyl prednisolone, naloxone, gangliosides or pulsed magnetic energy given 4 hours after injury. The results of controlled studies on patients are now anxiously awaited.

In the subacute stage post injury it is well known that the neurological state of the

patient may seriously deteriorate in the period 6 weeks to 4 months after surgery. Although the cause of this deterioration is not known it is possibly related to the Wallerian degeneration and other sequential neuropathological changes. There may also be a neurophysiological explanation. Isolation from higher centres and abnormal inputs at the spinal level may change the reflex patterns and connections so that previous 'useful' functions are lost. Therefore physiological interventions utilising the property of neuronal plasticity need to be developed to prevent these changes.

In the chronic stage there are known causes for late deterioration. Examples are spinal stenosis, neuroma of nerver roots, post traumatic syringomyelia, atheroma of the aorta and segmental arteries and arachnoiditis of the cauda equina. Epidural electrical stimulation of the spinal cord may be beneficial in the late stages. Functional electrical stimulation of muscles may also be useful. Bladder capacity can also be improved by electrical stimulation of the detrusor muscle or the pudenal nerve.

Of great theoretical interest in the future is the possible application of molecular neurobiology to the problem of spinal trauma. It is likely that the genes responsible for growth, regeneration and maturation of the central nervous system will be identified some time in the future and it may follow that autografts could be successfully applied for the replacement of the lost spinal tissue. It is possible that such grafts could also be 're-educated' when the molecular basis of plasticity becomes known.

These concepts are doubtless 'futuristic' but are presently justified as they define the eventual objective of our endeavour and thus serve as a guide for research.

Spinal injuries in rugby football: vital statistics over the last 10 years

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Rugby football as a total contact sport is a game with recognised hazards. By the middle of the 1970s it had become evident that these included serious neck injuries such as had been identified in American gridiron football. In 1975 cases were reported in a number of countries and between 1976 and 1978 10 serious spinal cord injuries were sustained in football in New South Wales. The apparent causes were collapse of scrums and incorrect or illegal tackling. Despite various preventive measures and outlawing of high and 'spear' tackles a further spate of these injuries occurred in 1982 and 1983 in rugby union in Australia and most other major rugby countries were also reporting similar disturbing findings. Subsequently the International Rugby Football Board changed or approved variations of the Laws to 'depower' and stablise the scrum and prevent 'pile-ups' after tackles. Such modifications together with a great awareness of the risks appear to have been effective and last month in London representatives of the other Board countries all reported a significant drop in the incidence of serious injuries. A retrospective survey of 45 football injuries admitted to the Spinal Injuries Unit of the Royal North Shore Hospital between 1968 and 1985 has recently been carried out by Dr John Yeo and his staff providing valuable information about the incidence and mechanism of these injuries and their prevention.

AIDS paraplegia: infection or myelopathy

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Rapidly developing evidence for functional and structural similarities between elements of the immune system and the central nervous system perhaps make it less than surprising that the HTLV-111 virus displays tropism for not only the T-4 inducer cells of the

immune system but also a number of cells within the central nervous system and perhaps the peripheral nervous system. It has been known for many years that brain cells and certain T-cells share membrane antigens and this is particularly true of suppressor T-cells and oligondendrocytes.

Brain tissue obtained from patients with AIDS who developed unexplained dementia have been studied by numerous techniques that have provided evidence to suggest (a) that brain tissue is heavily infected with the HTLV-111 virus in such cases; (b) that the virus actively replicates in the central nervous system and (c) that there is a considerable amount of unintegrated viral DNA present in infected cells. Suspensions of brain tissues from patients who died of AIDS with central nervous system complications when injected intravenously into chimpanzees or rhesus monkeys lead to rapid infection of the T-cells of these animals.

All levels of the neuraxis may be affected by primary infection with the HTLV-111 virus. The tropism for cells of the nervous system allows neurological infection and development of symptoms before immunosuppression is present. The initial symptoms of infections with the HTLV-111 virus may therefore involve neurological damage.

As clinicians become better at managing the opportunistic infections that develop following the onset of significant T-cell depression, as many as 25% of patients are dying a neurological death. Neurological complications in AIDS are predominantly from central nervous system infections, followed by neoplasms, vascular pathology, peripheral neuropathy and miscellaneous conditions such as retinopathy, aseptic meningitis, mass lesions, polymyositis and demyelination.

The pathology associated with the encephalitis caused by the HTLV-111 virus is diffuse and nonspecific. In the grey matter, one notes a marked accumulation of microglial cells and the development of paravascular nodules. Brain biopsy material frequently reveals the presence of cells that morphologically appear to be infected with cytomegalovirus, but it has been surprisingly difficult to culture this virus from brain tissue, suggesting that the morphological appearance may be due to a virus other than CMV. All areas of the brain may be involved, but there is a marked predominance for pathological changes to occur in the brain stem. Characteristically, the changes provoke no local inflammatory response. In the white matter of the brain, microglial nodules accumulate, reactive astrocytes are frequent, and demyelination may occur.

There is a broad clinical spectrum associated with AIDS encephalitis. Initially, patients may present with subtle cognitive changes, malaise, lethargy, loss of libido, and depression. The clinician may assume that these changes are caused by a reactive depression to the diagnosis of infection with the AIDS virus. However, such changes are more frequently symptomatic of central nervous system infection.

Withdrawal from business associates and friends, and a slow but sometimes rapid deterioration in higher central skills are characteristic. Progression to death usually occurs slowly, but may occur within weeks with increasing dementia and confusion being associated with 'neon light consciousness', in which higher centre skills appear and disappear in brief cycles. Examination of the cerebrospinal fluid frequently shows nothing more than a mild pleocytosis. The glucose level may be low and the protein concentration may vary. Recently we have observed oligoclonal banding in a number of cases. CT scans usually show marked cerebral atrophy as the major feature of this form of the disease and rapid changes in the CT scan can occur.

Multiple tract degeneration has now been reported in a number of cases. Such widespread involvement may precede or follow the development of dementia. Spastic quadriparesis has been reported and this can develop very rapidly. Symmetrical lesions are not uncommon.

Peripheral neurophathy is also recognised as a prominent feature of infection with the HTLV-111 virus. Characteristically, this neuropathy is very painful, involves a symmetrical distal sensory loss and features damage to large and small fibres. Ankle reflexes may be significantly reduced. Examination of the cerebrospinal fluid has usually been normal in such cases. EMG shows a distal sensory motor neuropathy, usually with minimal evidence of denervation. When the peripheral neuropathy symptoms of partial paralysis occur early in the course of HTLV-111 infection before significant im-

munosuppression has occurred, there is a chance for improvement in the neurological problem. As it is not at all certain that patients who develop neurological complications in Category B or C AIDS will go on to develop the fatal form of the illness, they should be given the benefit of the doubt regarding prognosis and all efforts at symptomatic relief and rehabilitation seem warranted at this time.

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N.M.R. = No more radiology?

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Nuclear Magnetic Resonance (NMR) or Magnetic Resonance Imaging (MRI) is a new method of imaging the magnetic environment of protons using resonant radiofrequency pulses in the presence of an intense and homogeneous magnetic field.

Ionising radiation is not used and there are no hazards, except to patients with cardiac pacemakers or intracranial aneurysm clips.

Advantages of MRI in spinal cord imaging include safe, atraumatic examination, and the ability to provide direct sagittal, coronal or axial plane images.

Using surface coils to provide high resolution and thin slices, MRI is more sensitive than CT/myelography in the diagnosis of spinal cord syrinx, tumour, atrophy and myelopathy.

MRI is as accurate, and less invasive than CT/myelography in diagnosing extrinsic spinal cord compression from metastases, infection or tumour.

MRI shows promise in assessment of the traumatised spinal cord; in the acute stage (transection, oedema or haemorrhage); in subacute syrinx formation; and in chronic atrophy. Maintenance of a patient with acute or multiple injuries within an MRI unit does pose problems and is more difficult than with CT.

The technique is capable of showing cord grey and white matter structure and of demonstrating cord demyelination in multiple sclerosis.

There are problems in the performance and interpretation of spinal MRI such as cerebrospinal fluid pulsation artefacts and the 'enlarged but normal central spinal cord canal'.

In the future, it may even be possible to examine safely and non-invasively the blood flow and metabolism of the spinal cord invivo by using Xenon CT and Magnetic Resonance Spectroscopy.

Evoked potentials in spinal injury

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In keeping with the emphasis of this meeting on recent and future developments, this paper will briefly review two techniques for assessing the function of spinal cord pathways. These are (i) somatosensory evoked potentials, which depend on transmission in ascending sensory pathways, and (ii) percutaneous electrical stimulation of motor cortex and descending motor pathways.

Somatosensory evoked potentials (SSEPs). Electrical stimulation of a peripheral nerve produces a synchronised neural volley that can be recorded using an averager at a number of sites along the peripheral and central pathway, up to and including the primary somatosensory cortex. Virtually any accessible nerve can be used. Routinely this laboratory chooses one of those listed in Table I, unless there is a clinical reason for favouring some other nerve.

Table I Somatosensory Evoked Potentials

Nerve	Nerve roots	Spinal pathway	Final pathway
Median [wrist] (Mixed)	C6,C7,C8,T1	Posterior columns	Medial lemniscus
Ulnar [wrist] (Mixed)	C8,T1	Posterior columns	Medial lemniscus
Posterior tibial (Mixed)	Mainly S1	Posterior spinocerebellar tract	Medial lemniscus
Sural (Cutaneous)	Mainly S1	Posterior columns	Medial lemniscus

The afferent fibres responsible for the evoked potentials are large myelinated fibres innervating skin and muscle. Small afferent fibres that subserve nociceptive and thermal sensations and which relay to anterolateral spinal pathways (the spinothalamic system) may be activated by the electrical stimulus but generally make no detectable contribution to the recorded potentials because (i) their conduction velocities are so low that the small potentials are too dispersed by the time their activity reaches the spinal cord, and (ii) the larger, faster afferents produce long-lasting potentials that may be superimposed on any small-fibre potential. The activity of large myelinated afferents is transmitted by pathways in the posterior quadrants of the spinal cord. For upper limb nerves the posterior columns transmit the afferent volley, be it purely cutaneous or mixed cutaneous and muscle afferent. The situation is different for lower limb nerves: cutaneous afferent volleys traverse the posterior columns but group I muscle afferent volleys traverse the posterior spino-cerebellar tract to the cranio-cervical junction where a collateral to nucleus Z relays the afferent activity to the medial lemniscus. With mixed nerves (such as the posterior tibial at the ankle, the tibial in the popliteal fossa, or the peroneal at the fibular head) the afferent volley comes from both skin and muscle but the evoked cerebral potential is determined predominantly (perhaps exclusively) by the muscle afferent component of the volley.

The neural volleys may be recorded at a number of sites using surface electrodes or subcutaneous needle electrodes (Table II). At neurosurgical and orthopaedic operations the evoked potentials can be recorded from the same sites using the similar recording electrodes. Alternatively, epidural electrodes can be inserted above and below the area of interest to record directly from the spinal cord, and this technique is probably more sensitive (see below).

Table III lists the areas in which SSEPS may be of value. Lesions within the somatosensory pathway may produce a reduction in amplitude and a prolongation in latency of

Table II Somatosensory Evoked Potentials

Upper limb nerves
Axilla
Erb's point
C1 Spinous process
Parietal cortex
Lower limb nerves
L1 Spinous process
Parietal cortex
At operation
From the above sites, e.g. scalp
Directly from cord using epidural electrodes

Table III Value of the SSEP

Diagnosis
Demonstration confirmation of an abnormality
Localisation of an abnormality
Prognosis
Head injury – value established
Spinal injury – value less certain
Monitoring
I.C.U.
Spinal operation

potentials recorded central to the lesion. For example, with lower limbs inputs a spinal lesion might prolong 'central conduction time': the interval between spinal entry and cortical input. With upper limb inputs, a cervical spinal lesion would produce abnormality in the B-C interval and brain stem or higher lesions abnormality in the C-D interval. With head injury, prognosis is worse the greater the abnormality in the C-D interval. With spinal injury there must, of necessity, be some correlation between degree of abnormality and outcome, but this has not been established as conclusively or as reliably as for head injury, probably because SSEPs provide no information about sensory pathways traversing the anterior quadrants of the spinal cord or about descending motor pathways.

In the Intensive Care Unit SSEPs may allow an objective assessment of patients whose illnesses or therapy interfere with clinical examination. SSEPs are also used in orthopaedic and neurosurgical operations on the spine and spinal cord to monitor cord integrity on a moment-to-moment basis, thus obviating the wake-up test often used in scoliosis surgery. The limitations of SSEPs are (i) they monitor the function of only specific sensory pathways in the posterior quadrants, and (ii) they cannot allow immediate detection of an adverse change because averaging is required to define the small potentials. In this regard epidural recording electrodes have advantages over the conventional recording electrodes and sites in Table 1 because the epidurally recorded potentials are bigger and require fewer averaging sweeps. In addition a higher stimulus rate can be used.

Motor pathway stimulation. Using single high-voltage electrical pulses of brief duration it is possible to activate central nervous system pathways directly. Such pulses applied to the scalp over the motor cortex activate corticofugal (corticospinal and possibly other) pathways and can produce a contraction in relevant muscles, particularly if they are already being contracted by the subject. With comparable stimulation of the spinal cord, descending motor pathways can be activated directly and stimulation at the cervical and lumbar enlargements will activate motor axoms as they pass from the ventral horn to

the ventral root. It is therefore possible to estimate central conduction time for descending corticospinal pathways from cortex to segmental level.

To some extent the usefulness of this technique is limited by (i) the need to maintain a background contraction of the responding muscle if the cortically-induced twitch contraction is to be strong, and (ii) repetitive volleys produced in corticospinal axons by strong stimuli. These limitations are less important when the technique is used for operative monitoring using epidural electrodes. The descending volley in spinal motor pathways is quite large and can be identified in single sweeps: few responses, if any, need be averaged.

The advantages of this technique for monitoring spinal cord function are (i) the lag between the occurrence of an insult and a recognisable change in the neural volley is less, (ii) the motor pathways being monitored are more vulnerable to traction-induced cord ischaemia than sensory pathways in the posterior quadrants, and (ii) outcome is correlated more with motor function. However, with the same epidural electrodes it is possible to record both ascending somatosensory and descending motor volleys, and to do both will probably come to be seen as optimal monitoring in the next decade.

The case for operative reduction and fixation of vertebral fractures and dislocations

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In the past two decades there have been major technical advances in instrumentation for the treatment of scoliosis. In the U.S.A. in particular, this has often been indiscriminately applied to the treatment of patients with spinal injuries. Indications for surgery became blurred; surgical intervention was often unjustified. The indications for surgery remain the same as always – failure of conservative management. That is, failure to reduce a fracture or dislocation to an acceptable position, failure to achieve long term stability, and sometimes, failure to achieve the optimal neurological outcome. Particular indications are seat belt injuries with significant kyposis and/or facet joint dislocation, shear fractures with inherent instability, burst fractures with partial or progressive neurological damage, some rotational fracture dislocations, and iatrogenic instability, particularly following laminectomy. In general, open reduction should be accompanied by local fusion, and by stable fixation to achieve early rehabilitation. Operative intervention to achieve early rehabilitation in situations where ultimate stability can be achieved by conservative means is probably never justified.

Depression, suicide and euthanasia in the acute phase following spinal cord injury

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It has commonly been assumed that most patients with traumatic paraplegia or quadriplegia eventually experience depression. Depression has been regarded by many as a normal and even inevitable phenomenon, part of the phasic response to spinal cord injury. Studies systematically assessing the incidence of depressive illness have challenged these beliefs by demonstrating that not all patients with spinal cord injury experience significant depression.

We undertook a pilot study which included all patients consecutively admitted to the Spinal Injuries Unit of the Austen Hospital between 1/10/84 and 28/2/86 who had not sustained a significant head injury. Each of 98 patients were interviewed by the Spinal

Injuries Unit liaison psychiatrist within 2 weeks of admission. A current diagnosis of depression or other psychiatric illness was made following detailed clinical interview, mental status examination and completion of the 17-item Hamilton Depression Rating Scale. Patients were observed for the duration of their hospital admission by members of the multi-disciplinary treatment team and re-assessed by the psychiatrist if they showed any signs of depression. All psychiatric diagnoses were made according to criteria specified in the 3rd edition of the *American Psychiatric Association's Diagnostic and Statistical Manual of Mental Disorders* (DSM-111). Eleven patients met the DSM-111 criteria for major affective disorder.

Patients were predominantly male (N = 10) and ranged in age from 19–70 years old. Six patients were aged less than 40 years while 4 were greater than 60 years. Six were quadriplegic and 5 paraplegic. This approximates to the usual ratio of quadriplegic to paraplegic patients admitted to the unit. Time of onset ranged from 2 weeks to 5 months after the spinal injury. One of the 11 patients had a past history of bipolar affective disorder. This patient's spinal injury was the result of a failed suicide attempt made at the time of a major depressive episode.

The diagnosis of depression in the physically ill is difficult. Many of the biological symptoms of depression, such as sleep disturbance, somatic pre–occupation, loss of sexual interest, and appetite disturbance with or without weight loss and fatigue, may occur in the non-depressed spinal injured patient. The diagnosis of a depressive illness is made by the presence of a qualitative and persistent lowering of mood, accompanied by loss of interest in self, environment and family, difficulty concentrating/thinking/making decisions, anxiety, irritability, feelings of worthlessness/hopelessness, fear of losing one's mind or losing control, difficulty doing anything because it's too much trouble, crying a lot, thinking of suicide or wanting to die or suicide attempt.

The wish to die is frequently expressed by patients in the early stages of treatment and rehabilitation following spinal cord injury. Generally 'I would rather be dead' means 'I don't want to be paraplegic or quadriplegic'. Suicide ideation at this time fluctuates and is marked by ambivalence. Persistent suicidal ideation may be seen in those patients who fail to adjust to spinal cord injury. Successful adjustment and rehabilitation involves several psychological tasks including maintaining a sense of personal worth, keeping distress within manageable limits, maintaining or restoring relations with other significant people, enhancing the prospects for recovering bodily parts and developing a personally valued and socially acceptable lifestyle. Failure in these tasks may be due to factors in the individual patient, the family or the social environment, including the spinal injuries unit.

All 11 depressed patients in our study were treated with antidepressant medication and supportive psychotherapy. Antidepressant drugs used were mianserin and nomifensine. All were significantly improved with medication. Alleviation of anxiety was evident within the first week of treatment but significant improvement in depression did not occur until 3–6 weeks after commencing treatment.

Failure to recognise a depressive illness, or the incorrect assumption that depression is a 'normal response' or 'phase of adjustment' to spinal cord injury may adversely influence the short and long term rehabilitation of the spinal cord injured patient. Disinterest, anergia and lack of motivation are common depressive symptoms and may account for lack of progress in physiotherapy and occupational therapy and for failure to achieve full potential in rehabilitation. Irritability and aggression may result in the patient being inappropriately identified as a 'problem' and being scapegoated by other patients and staff. Marital and family problems occurring as a result of a patient's depression may be wrongly linked casually to the spinal injury. Suicide may occur in the depressed spinal injured patient just as in many other patients with a depressive illness.

Given the prevalence of depression in the general population and the multiple stresses and life changes caused by spinal cord injury it is not surprising to find that a significant number of patients with spinal cord injury suffer a depressive illness at some point. Early assessment of the patient, the family, and the social situation allows identification of those at risk for the development of a depressive illness. Careful assessment does allow differentiation of a depressive illness from other causes of dysphoric mood. It is likely

that those units who find all patients experience depression fail to differentiate the various causes of dysphoric mood, while those who find no depression simply fail to recognise its presence. Both these situations result in failure to treat a reversible illness and may produce many adverse sequelae including suicide.

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A practical approach to functional electrical stimulation – balancing the case

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In the spinal injuries unit of the Royal North Shore Hospital of Sydney, we have used functional electrical stimulation (FES) in programmes for patients with complete and incomplete spinal cord lesions. The candidates for this treatment, suffering from incomplete spinal cord lesions, begin the programme of strengthening 3 to 4 weeks after the injury. The FES is used to enhance recovery in conjunction with conventional physiotherapy. For patients with complete lesions, upper motor neurone in type, the program involves muscle strengthening so that standing, and later walking may be attempted. All patients have their own FES equipment for use at home and return to the unit at least every 2 weeks for reassessment.

This FES programme is presented to the patient as an exercise programme rather than a technique to achieve functional gait. Initially the daily programme will involve 2 or 3 hours of stimulation and exercise routine in order to maintain muscle bulk.

There are tremendous psychological benefits for the paraplegic casualty, previously confined totally to his wheelchair. Osteoporosis may be more readily controlled and circulation generally improved with the prevention of trophic skin ulceration.

Functional electrical stimulation miracle walking in the 80s

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Although the 'Golden Age of Electrotherapy' was in the latter part of the last century we may be in the midst of a renaissance. In the last 40 years there has been a resurgence of interest in the use of electricity in rehabilitation, especially in the enhancement of ineffective motor function, largely as the result of better understanding of neuromuscular function and technological advances in stimulating nerve mediated functions.

Application of functional efferent electrical stimulation (RES) to control muscle function relies on excitation of preserved motor nerve fibres innervating contractile muscle. There is a postulated afferent stimulation of spinal reflex mechanisms. The results of this are difficult to ascertain as it is overshadowed by muscle contraction and the consequent inflow from muscle receptors and Golgi tendon organs. Nevertheless there is a facilitation of synergistic muscles and inhibition of antagonists probably via afferent stimulation of the group 1a fibres. It has also been noted, that as well as immediate improvement of movement and other motor reflux activity such as clonus, there have

been lasting beneficial effects in these areas after cessation of stimulation. These are difficult to explain.

There are a number of critical factors determining successful percutaneous neural excitation. These are impedance, size and orientation of electrodes, and parameters of the electrical stimulus.

In general, FES can be used in situations where the patient demonstrates difficulties with voluntary movement. These arise after a stroke, head trauma, spinal cord injury, some lower motor neuron disorders such as incomplete brachial plexus lesions and Guillain-Barre syndrome, and following joint replacement surgery and joint and tendon repair. We have used it with good effect in gait training of above knee amputees.

Generally it is used to maintain or increase range of motion, to strengthen muscle, to facilitate voluntary motor function, to inhibit spasticity and to provide a functional 'orthosis'. FES requires intact nerve excitability. Absolute contraindications include the presence of a demand pacemaker, and an open wound or recent scar in the vicinity of the stimulated field.

It is important not to confuse FES with the classical forms of electrotherapy applied directly to muscle or consider it as a mere orthotic aid. This form of stimulation appears to influence the central organisation of motor activity and may result in improved muscular performance. Applied FES gives an immediate substitute for impaired function and enables the patient to repeat the pattern many times, possibly improving the organisation of movement.

There is an ambitious development of multichannel systems with programmed stimulation sequences. The goal researchers are seeking is a functional orthosis to improve ineffective movement and in particular to enable spinal man to walk. The main barrier is not so much the technical aspects of the hardware, but rather the interfacing of man to machine.

An overview of neuropathic bladder dysfunction by electrostimulation

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Disorders of both the storage and voiding functions of the bladder associated with neuropathy are amenable to manipulation by training, drugs, surgery and electrostimulation. The success of each of these treatment modalities depends upon careful patient selection.

By utilising different types of electrostimulation, it is possible now to achieve inhibition or stimulation of detrusor contractions, improvement in bladder sensation and sphincter competence and to modulate detrusor sphincter co-ordination with various degree of success. The historical evolution of attempts to achieve either urinary continence or electromicturition by such techniques extend over the past 20 years.

In the neuropathic bladder, failure of bladder emptying due to sphincter dyssynergia causing outlet obstruction or to destrusor failure or to a combination of both factors results in considerable morbidity and mortality. Electrostimulators applied to the bladder muscle, splanchnic nerves, sacral segmental nerves, sacral anterior roots and the conus medullaris have met with mixed success in improving vesical emptying.

Recent research has implicated varying degrees of subtle neuropathy in the aetiology of sphincter weakness incontinence. The treatment of this type of incontinence by electrostimulation of the pelvic floor and urethral sphincter muscles antedated this discovery by 20 years.

Current research into the modulation of detrusor hyper-reflexia by acupuncture indicates that there may be a future for this type of treatment in the management of reflex and urge incontinence.

Whilst it is now possible to modify many aspects of vesical and urethral function by electrostimulation, many of the techniques are still largely experimental and of unknown long term effect.

Careful patient selection and a multi-disciplinary team approach are essential to the satisfactory utilisation of such techniques in current clinical practice. Much further research is required before optimal and predictable long term results can be guaranteed.

Initial experience with the anterior sacral root stimulator

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The patient, a male, was rendered a complete T9 paraplegic in 1982 at the age of 25. At his initial discharge his bladder was emptied by tapping the lower abdomen to produce reflex detrusor contraction, and by automatic micturition. In addition, he was performing intermittent clean self catheterisation twice daily because of high residual urines (200 ml). Urodynamics during his first admission showed poor detrusor contraction and external sphincter spasm.

Urodynamics two years later showed well co-ordinated detrusor activity and voiding with peak pressure of 40–50 cm of water and a prompt flow rate. Little obstruction was present at the external sphincter. Residual urine was approximately 40 ml.

Residuals remained high on intermittent clean self catheterisation and an external sphincterotomy was performed on 15.6.83. At operation, grade 3 trabeculation of the bladder was noted; a submeatal stenosis was incised and an external sphincterotomy performed. Residuals remained at approximately 200 ml. Urodynamics in October 1983 showed weak detrusor contraction with peak pressure of 20 cm of water with voiding and no obstruction at the external sphincter.

During 1984 and the first half of 1985 the patient suffered one or two urinary tract infections per month. These were generally asymptomatic but characterised by cloudy, discoloured and offensive urine. In June 1985 he was admitted for assessment including urodynamics. He was suitable for the insertion of an anterior sacral root stimulator and this was performed on 26.6.85 with the invaluable help of Dr D. Thomas, Urologist, from Sheffield, United Kingdom.

The operation took 7 hours of operating time, required the use of the operating microscope and was performed under cover of gentamicin and flucloxacillin. The former was continued for one week post-operatively and the latter for two weeks. Urine was clear of infection at the time of the operation.

Stimulation using the stimulator was first performed on 28.6.85. All leads were shown to function and there was a non-specific bladder pressure rise and partial penile erection. On day five, stimulation produced voiding with some penile tumescence and a detrusor pressure of more than 60 cm of water. Urodynamics performed at three weeks and seven weeks showed residual usines between 20–30 ml.

The patient continued to suffer urinary tract infections at a rate of approximately one a month until given a six week course of antibiotics. There have been no urine infections in the last 4 months. During this latter period residual urines have decreased to 10 ml with an occasional one of 30 ml. The patient no longer does intermittent clean self catheterization.

The patient now stimulates his bladder with a weak or low strength stimulus of the S3 leads. The intermittent effect is obtained by moving the transmitter head away from the receiver at regular intervals. There is no response to stimulation of the S2 or S4 nerves. Good intermittent voiding is achieved with gradual development of a penile erection, through the bladder is completely empied before the erection is complete.

Urodynamics at the end of March 1986 showed complete emptying. Bladder capacity has improved slightly and voids are mostly of about 450 ml. The patient has not to to be continent, as the toilets at work are not accessible. He therefore continues to wear an external collecting device.

There is extension and abduction of the legs with stimulation. This has not changed at all during the post-operative period. His spasm pattern has not otherwise been affected by the operation.

In summary the patient has achieved an excellent result in that he no longer has frequent urinary tract infections. Residual urines have decreased from 30–40 ml to zero on urodynamic studies and from 30 ml by intermittent catheterization to approximately 10 ml. The incidence of urinary tract infection has declined probably due to the reduction of residual urine volume.

The lax anal sphincter and spinal disease; a new electrophysiological approach to investigation

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At St Mark's Hospital, during the past 8 years, we have investigated the cause of idiopathic anorectal incontinence, and also of genuine stress incontinence of urine, using a variety of different neuropathological and electrophysiological techniques. These investigations have revealed that this socially devastating disability is commonly due to damage to the innervation of the pelvic floor sphincter muscles caused by injury sustained during childbirth. In addition, similar cumulative injury to this innervation can arise from recurrent stretch-induced injury to these nerves during straining at stool in people with intractable constipation and abnormal bowel habit.

In order to establish these causative relationships we first examined muscle biopsies from the external anal sphincter, puborectalis and levator ani muscles in autopsied normal subjects and in patients with faecal incontinence. These investigations showed that there was denervation and reinnervation of these muscles, especially of the puborectalis and external anal sphincter muscles. We then adapted the technique of single fibre electromyography to this problem. This technique enables the extent of reinnervation in a muscle to be assessed quantitatively in a relatively simple fashion, by measuring the fibre density in the muscle defined as; the mean number of muscle fibres innervated by a single motor unit calculated from 20 separate recordings. The latter reflects the packing density of muscle fibres in motor units and this is an index of reinnervation. In patients with incontinence the fibre density was increased, and we continue to use this measure as a routine test in our laboratory. Similar abnormalities have been found in the striated muscle of the periurethral sphincter muscle in women with genuine stress incontinence of urine.

We also examined the nerve fibres innervating the external anal sphincter muscle in women with incontinence and found that there was loss of myelinated nerve fibres and fibrosis in the terminal twigs of this innervation.

We recorded the terminal latency in the inferior rectal branches of the pudendal innervation of the external anal sphincter, called the pudendal nerve terminal motor latency (PNTML). A similar method of intra-rectal stimulation of the pudendal nerves was devised to measure the perineal nerve terminal motor latency (Per NTML), by recording the response in the periurerthral triated sphincter muscle with surface electrodes mounted on a Foley catheter. With these methods we showed that there was slowing of motor conduction in the terminal portions of these nerves. An increase in the PNTML was particularly a feature of faecal incontinence of stress type, and an increased Per NTML was a major feature of patients with stress incontinence of urine, without faecal incontinence. When both types of incontinence co-existed there was marked abnormality in both these terminal motor latencies. Further, these abnormalities could be correlated with injury in childbirth. In a prospective study we correlated these electrophysiological abnormalities with prolonged labour, with forceps delivery, with the size of the fetal head and with multiparity. There are additional correlations, especially in incontinent women who have never experienced childbirth, with severe and intractable constipation, and with straining at stool due to abnormal bowel habit, whether congenital or acquired.

Most women with incontinence develop the disorder in middle life or later. In an investigation of elderly people we found that ageing was associated with electrophy-

siological evidence of denervation in the external anal sphincter of a degree sufficient to carry a significant risk of incontinence. However, this could not always be explained by the causative factors discussed above. We therefore considered the possibility that an additional factor leading to weakness of the pelvic floor sphincters might be important in these patients and, particularly, that there might be damage to this innervation from age-related lumbosacral spondylosis with encroachment on the exit foramina of the ventral roots.

In order to evaluate this possibility we used the method introduced by Merton and Morton for stimulation of the brain. We stimulated the lumbosacral nerve roots in the cauda equina at the L1 and L4 levels. We measured the latencies to the pelvic floor sphincter muscles (especially to the external anal sphincter, puborectalis and periurethral striated urinary sphincter muscles) from these two sites and used the difference between the results at the two stimulus levels to any single muscle as a measure of conduction in the S2 and S3 nerve roots in the cauda equina between the L1 and L4 levels. In about 15% of patients with genuine stress faecal or urinary incontinence there was slowing of conduction between these two vertebral levels. The validity of these new electrophysiological investigations has been tested in relation to the radiological features in a series of patients presenting with typical clinical features of cauda equina disease.

By utilising a similar non-invasive transcutaneous electrical stimulation technique it is possible to excite the descending motor pathways in the spinal cord and to measure the latency to the striated sphincter muscles. In this technique the conduction time in this spinal motor system presumed to represent conduction in the corticospinal tracts, is assessed between stimulation sites at the C6 and L1 vertebral levels. Conversion of this latency difference into conduction velocity gives a convenient correction for height. Using this method the motor conduction velocity in the human spinal cord was found to be 67 m/s (SD 9 m/s). This is about 10 m/s faster than conduction in the cauda equina nerve roots. In a further extension of this technique we have recorded the response in the external anal sphincter and periurethral striated sphincter muscles from cortical stimulation, at the vertex. This latency, of about 25 ms, is of course dependent on body height.

By adopting different recording sites, the central and peripheral latencies in different upper and lower neuron pathways may be selectively studied, and these techniques hold great promise in the clinical investigation of the motor system in a wide range of different disorders. For example, slowing of conduction in the central motor pathway in the brain has been demonstrated in multiple sclerosis, and we have found marked slowing of motor conduction velocity in the spinal cord in this disease and in radiation myelopathy. We have also used this technique to study conduction in the motor system in motor neurone disease, and have found that motor conduction is slowed in the spinal cord in some cases. Although we have not yet had the opportunity to use this method in the evaluation of the descending motor pathways in the spinal cord in spinal trauma it seems likely that this method will be of value in the assessment of the presence of residual function in this system.

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Issues in male fertility and sexuality

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Increased awareness among health care professionals and consumer groups that sex plays an important role in adjustment to disability developed in the late sixties and seventies. This stimulated research into the sexual response as well as into the social and psychologic aspects of sexuality in spinal cord injured people.

There is wide agreement that the provision of information regarding the individual's prognosis and remaining sexual function after spinal cord injury is important in sexual adjustment. This can only be done after careful medical examination and in the presence of a stable lesion. However, the patient should feel, fairly early on, that he or she is free to ask questions and discuss fears and problems. The ability of the patient to participate in the counselling process and accept information will depend upon the degree of their adjustment to the profound change in their body and self image as well as their changed socio-economic status and their increased dependency on others. The patient's age and sexual experience should also be considered in designing a counselling programme. If a spouse is available, counselling should always involve the spouse.

Society still places a great value on sexual potency in the male. This attitude is often reflected in staff of a spinal cord injuries unit, particularly nursing staff. Several investigators have found that nursing staff place greater importance on the return of sexual function than on return of bowel/bladder function and mobility. This is unlike the patients who usually rank sexual function as the last in order of importance in that list.

Emphasis must be placed on non-genital forms of sexual gratification as well as on the psychological needs for emotional support and closeness that are important in the couple's

relationship. Emphasis must also be placed on the patient's remaining assets so that he or she learns to concentrate on those rather than on the disability.

Provision of information regarding practical aspects of sexual contact such as what to do with urinary drainage equipment and advice regarding bowel and bladder care is obviously also important.

The literature on sexual response of the spinal cord injured male is extensive. Unfortunately, most of the data has been collected by survey or interview. Little attempt has been made at physiological observation or even corroborative interview of the spouse. There are also wide differences in the definition of such basic terms as 'erection', 'ejaculation' and 'incomplete lesions'. Interpretation of the data is therefore difficult. However, some general trends emerge.

Psychogenic erections in patients with upper motor neurone lesions are rare and probably do not exist. The majority of males with cervical lesions and thoracic lesions retain reflex erectile potency. This declines significantly in lesions below T7. Erection may not be of sufficient duration for intromission. Of interest is the significant number (up to 40%) of men with lower motor neurone lesions who retain psychogenic erectile potency in the absence of reflex erections.

Ejaculation in men with complete spinal cord injuries is rare, occurring in about 7% and is more common in those with lesions below T7.

In recent years, emission of sperm has been stimulated in paraplegic men by a number of means including electro-ejaculation, application of a vibrator to the glans penis, and injection of intra-thecal prostigmine. The last technique has been associated with at least one death due to dysreflexia but recent successful 'ejaculation' with subcutaneous prostigmine has been reported. Unfortunately, the quality of semen obtained by these means is poor. With rare exceptions, the sperm count is depressed, the sperm mobility is poor and there is a high incidence of abnormal forms.

Testicular biopsy in men with upper motor neurone lesions usually reveals retarded spermatogenesis with normal or hyperplastic leydic cells. Interstitial fibrosis is also seen occasionally. On the other hand, men with lower motor neurone lesions usually reveal normal spermatogenesis. The aetiology of these abnormalities is unclear. Depression of plasma testosterone level in the first 6 weeks after injury has been reported by several investigators and suggested as a factor. Plasma testosterone levels may also fall in patients after major surgery and in burn victims. In a group of 23 of our own patients, similar levels of depression were seen in patients with complete lesions and incomplete lesions and in those with a vertebral fraction and no loss. It is likely, therefore, that the depression of testosterone levels is a normal reaction to stress.

Raised scrotal temperature has been suggested as a possible cause for retarded spermatogenesis, though firm evidence is lacking. It is probably that chronic infection and lack of drainage constitute other factors leading to reduced fertility, as well as factors such as retrograde ejaculation, the effect of urine contamination of semen and the effect of the various drugs used by our patients.

The importance of improving the quality of the ejaculate is obvious. Although there is some evidence that quality does improve if a regular pattern of ejaculation can be established, the difficulty in obtaining ejaculation for many of our patients remains.

Let's have a baby—collection of semen

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This paper and video presentation illustrates methods used to collect semen from spinal cord injured males at the Princess Alexandra Spinal Injuries Unit of the last 10 years.

Currently we are using modifications of the techniques described by Professor Giles Brindley. The three methods of semen collection including the use of the Matoba ME25 Vibrator, a locally manufactured electro-ejaculation device incorporated in a plastazote

digital extension, and a two finger prostatic massage. The techniques are used independently or in combination.

Twenty four patients were tested, and a total of 31 semen collections were made from March 1985 to March 1986. Ten patients were tested during their acute admission. Five of these had complete lesions and 5 were incomplete. Only 1 of the patients with complete lesions and 2 of the patients with incomplete lesions produced semen specimens showing evidence of sperm. No tests were performed on patients who were still in spinal shock, since I have been singularly unsuccessful with this group in the past.

Fourteen patients were tested as outpatients. Six of these sustained complete lesions and 8 incomplete lesions. Three of the patients with complete lesions and 5 of the patients with incomplete lesions produced specimens with 5 to 10 motile sperm per high power field. Two of the patients with complete lesions and 1 of the patients with an incomplete lesion have subsequently been successful in impregnating their spouses.

Of the 13 patients with cervical lesions, 3 had complete lesions and 10 were incomplete. Five of the patients with incomplete cervical lesions produced specimens, one of which resulted in a pregnancy. Amongst the 11 paraplegics there were 9 with complete lesions and 1 with an incomplete lesion that produced specimens, and 2 of these from patients with complete lesions resulted in pregnancy.

One patient with a T12 flaccid paraplegia and with significant wasting of the lower limb musculature was able to obtain semen.

The length of time following injury once out of spinal shock before attempting to collect semen does not appear to influence the chance of success. In fact, it would seem that the most successful are those patients who are 3 to 4 years post injury.

No patient actually ejaculated normally with any of the methods used. No patient sustained an erection throughout the procedure.

All patients were first tried on the Matoba ME 25 Vibrator. Before attempting the trial, the bladder was drained either by tapping or by catheter, and if his lesion was above T5 the patient's blood pressure was checked. The patient was place on a supine position and the vibrator, set at high speed, was used over the glans penis. The most sensitive area is generally found to be the central surface over the frenulum, and within 2 to 3 minutes it is generally evident whether there is likely to be a successful result or not. Strong abdominal contractions and spasm in the lower limbs is generally a good sign. If there is no response, two finger prostatic massage generally produces one to two drops of fluid suitable for microscopic examination. If nothing as achieved, usually week later I try the electro ejaculator, built to Professor Brindley's specifications. The distal end of the digital extension contains a 1/2 cm diameter electrode, and this is used to explore the ventral surface of the sacrum. Once satisfactory contractions of the gluteals and pelvic floor have been elicited, surged rhythmical contractions are maintained for 5 to 10 minutes. If the patient has some sensation, he is given the controls and can guide the electrode to the area that feels most satisfactory. Although it is rare to get any form of ejaculation from this procedure, two finger prostatic massage will generally give a result suitable for examination. If the patient's blood pressure rises above 200 systolic, or he complains of a headache, I abandon the trial. To date, on two occasions only has this been a problem, and in both situations it occurred when using the electro ejaculator. One patient with an incomplete lesion found the electro ejaculator and the vibrator were too uncomfortable.

The uncertainty of fertility is a problem that is often seen but is seldom solved. The present regimen has enabled us to divide patients into those who might have a chance of fatherhood and those that appear to have none.

At 18, most males seem to be primarily interested in erections and fertility is considered a complication, but by the age of 25 the urge to procreate seems to be more dominant. It is perhaps for this reason that many of the younger patients show little interest in semen collection trials and are much more concerned when it is suggested that urological procedures may result in the loss of erections.

When a couple present wishing to try for a pregnancy, I usually give the vibrator a trial and discuss the process of artificial insemination. If the trials are successful, they obtain a vibrator and use it at home. The semen is collected in a 2 ml syringe and

injected into the vagina. The wife keeps her buttocks elevated on a pillow for 1 hour after the insertion. For optimal success, it is best to pinpoint the most fertile period and patients are encouraged to try to cover this period with three to four inseminations over five days. Regular ejaculation seems to improve the quality of semen. Motility reduces more rapidly in paraplegics than normals, with survival times of sperm of greater than 2 hours being rare in vitro.

If a couple have no success, we usually refer them to our local Fertility Clinic, and they go through further trials. Usually these culminate in artificial insemination, using an anonymous donor's semen. The fact that they have tried to collect the semen themselves seems to make the concept of artificial insemination more acceptable.

Children, marriage and marital outcome in the spinally injured

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The impact of Spinal Cord Injury involves the psyche and social functioning of the patient on many levels. The effects on marriage, both pre-existing and subsequent, could therefore be expected to be significant, especially because of the high incidence of impaired sexual function.

The incidence of marriage breakdown may also be an indicator of the level of adjustment to the altered circumstances after spinal cord injury, and therefore of the effectiveness of the non-physical aspects of rehabilitation.

The incidence of marriage breakdown may also be an indicator of the level of adjustment to the altered circumstances after spinal cord injury, and therefore of the effectiveness of the non-physical aspects of rehabilitation.

A simple questionnaire was applied randomly to patients attending for outpatient or inpatient care at the Spinal Injuries Unit at the Austin Hospital, Victoria. Patients who had not been married or had not lived a de facto relationship either before or after the injury or who were unable to comprehend the questionnaire because of intellectual impairment or language difficulty were excluded.

One hundred and fifty-seven completed questionnaires have been obtained so far. There are 122 males and 35 females, with 82 (67%) of the men and 19 (52%) of the women being paraplegic, and 40 (33%) of the men and 16 (48%) of the females being tetraplegic. Complete lesions were present in 51 of the male paraplegics and 10 of the female paraplegics and in 13 of the male tetraplegics and 4 of the female tetraplegics. Average age at injury was 29 years for men and 36 years for the women. The average age for the men completing the questionnaire was 43·8 years for the men and 47 years for the women.

Of the 122 men in the study, 66 had married before their injury; of these, 44 marriages are still together. Two had been widowed, and 7 separated before their injury. Of the 57 married at the time of SCI, 13 marriages broke up after the injury. There are children in 39 of the marriages which are still together, and in 9 of those which broke up after the injury. None of the men whose marriages failed after the injury were tetraplegics.

Twenty-one of the 35 women had been married before their injury. One marriage ended in divorce and the patient remarried before her injury. Five women were widowed before or at the time of their injury. Of the 15 remaining marriages, 5 ended after injury; 10 are still together.

The average time before marriage break-up after injury was 5·4 years for the men and 3·0 years for the women.

There were 26 de facto relationships (defined as living together in a non-casual relationship) before injury, involving 7 women and 12 men. Four of 9 relationships involving men, and 2 of 4 involving women, present at the time of injury, broke up after injury. There are children in 6 of the 7 relationships which are still intact.

The number of marriages after injury was 41 involving 37 men and 4 women. Of these, 33 involving 30 men and 3 women, are still intact, after an average of 11 years. One man and one woman were widowed, and 6 men separated or divorced. There are

one or more children in 20 of the marriages which are still intact, and only one in those which are not.

A total of 22 de facto relationships were entered into by 17 persons (12 men and 5 women) after injury, of whom 7 men and 2 women are still present after an average of 3·3 years. No children were born to any post-injury de facto couple.

Of the 41 couples who married after the injury, 12 had a total of 27 children by natural means; 9 were born to 6 couples using artificial insemination of donor semen, and 8 children were adopted by 4 couples. One couple had 1 child by adoption, and 2 naturally.

Eleven children were born to 5 women after injury. One, whose husband is a paraplegic, by artificial insemination of donor semen, the others by natural means.

Seventy nine men were able to achieve erection, 47 describing the erection as strong and 13 as weak. Forty seven of these men claimed some psychogenic control. Sixty seven of the men had experienced coitus. One additional man had a penile implant.

Of the 35 women 18 had experienced coitus. A hysterectomy had been performed on 11 women.

It would not be surprising if a spinal cord injury sustained by one partner had a disastrous effect on many marriages and de facto relationships. A number of studies have however shown that the rate of breakdown of marriages contracted before the event does not differ significantly from that in the general community, while those marriages that occur after the injury tend to survive rather better. An exception is the study by Guttman, who found the rate of breakdown in his population to be significantly higher than the general population.

The Institute of Family Studies in Melbourne report that an average of 35% of marriages in Victoria ended in divorce in 1985 at an average of 10·5 years after marriage and that there were children in 61% of these marriages.

Our figures of 25% of marriage breakdown after spinal cord injury indicate a rate below that of the state average though our sample is small and incomplete.

It may be that the stability of the marriages is a reflection of the efforts increasingly made by the staff of the spinal unit to involve significant others as much as possible in the treatment, and if necessary, in the care of the patient. As much information as possible is given to them regarding the injury and likely outcome from the time of the admission, using both family meetings and individual counselling where appropriate. Patients are followed up frequently during their first year as an outpatient by the outpatient clinics and by a visting nurse experienced in spinal injury care and in sexual counselling.

In talking to the patients concerned I gain the impression that the circumstances arising from the injury to one partner caused both partners to look more consciously at their relationship than is commonly the case in marriages. This reinforces the concept that a spinal cord injury provokes a family crisis which needs to be treated. Certainly the opportunity to discuss the relationship with supportive counsellers helps the people concerned to take stock of their feelings and their committment to each other, and to cope more effectively with the tensions that arise in any marriage.

The rate of relationship breakdown may also be an indication of the degree to which the patient copes with the disability. It may in the future be possible by correlating this information with other measures such as the re-admission rate for preventable complications such as pressure sores, to obtain an index for self audit of the effectiveness of the non-physical rehabilitation in the spinal unit.

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Pharmacological implications of lioresal research: where do we go from here?

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In the early 1950s, Gamma Aminobutyric Acid was identified in the central nervous system as an inhibitory neurotransmitter. Oral and parenteral administration of this agent were found to be without significant effect because of its highly polar and hydrophilic nature. The addition of lipophilic side chains increased its fat solubility and beta-parachlorphenol gamma aminobutyric acid (Baclofen) was found to be an inhibitor of spinal reflex activity by both the oral and parenteral route.

Baclofen (Lioresal in its marketed form) is rapidly and completely absorbed in the gastro-intestinal tract. It is excreted in the urine and faeces, the majority in unchanged form and the rest as a de-aminated product. Virtually complete elimination is seen after 24 hours, with a half life of about 3 to 4 hours.

The majority of side effects of Baclofen have been transient and usually consist of disturbances in mental function such as nightmares or alteration in personality. Reduction in dose or frequency of administration usually eliminates these effects and allows continuation of the drug. Patients who are old and who have multiple sclerosis appear to be more prone to these side effects which may occur at dose levels of 30–40 mg daily. High doses and rapid increases in dose are inadvisable in these patients.

No serious long term sequelae have been reported in over 10 years of use of the drug. Overdose results in loss of conciousness, respiratory depression, flaccidity and fixation of pupils. Treatment is supportive and no permanent sequelae of overdose has been reported.

Baclofen has its effect in the spinal cord. It directly or indirectly produces pre-synaptic inhibition of afferent terminals. Although chemically it resembles GABA, its mechanism of action appears to be different. It has been suggested that the (L) isomer of Baclofen is responsible for almost all of its reflex depressant properties, and that it binds to GABA B receptors, restricting calcium influx into pre-synaptic terminals and consequently depressing evoked transmitter (probably glutamate) release.

Baclofen has also been shown to have an analgesic effect. This may be due to antagonism of substance P, or perhaps again secondary to binding of (L) Baclofen to GABA-B receptors.

An analgesic effect may be expected to reduce spasticity by diminishing afferent input to the alpha motor neurone.

Clinically Baclofen is excellent for the management of spasticity. It reduces bladder outlet obstruction and improves bladder training and the development of a balanced neurogenic bladder.

In those patients who are young and not suffering from a demyelinating disease, our usual practice is to commence with 10 mg 4 times daily, increasing gradually to 25 mg 4 times daily. This is effective in about 80% of patients. If spasticity is not sufficiently reduced, an increase of dose to 150 mg daily may be made with relative safety. In rare instances, a dose of 200 mg daily has been employed with more obvious side effects. If nightmares are a problem, the nocturnal dose may be reduced. Often a reduction of individual dose but an increase in frequency of administration to 4 hourly will lessen side effects and maintain a useful therapeutic action.

Reports are now appearing of the intrathecal use of Baclofen for management of tetanus, for short term reduction of severe spasticity and for long term management of refreactive hypertonicity via depot injection techniques. This is obviously a promising technique for those patients who suffer from gross spasticity unmanageable by conventional techniques.

Baclofen is arguably the most beneficial pharmacological agent to have emerged in the last ten years for patients with spinal spasticity.

The long term functional results of conservative and operative management in complete paraplegics with spinal cord injuries between T10 and T12

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There is considerable controversy over the early management of people sustaining unstable fracture or fracture dislocation to thoraco dorsal spine with complete paraplegia. Laminectomy has now been abandoned in most centres as it does not improve neurological recovery and enhances instability. However, with improved techniques of internal fixation, open reduction and internal fixation using a variety of different techniques is now becoming more popular and advocated in the world literature.

This study analysed 87 athletes attending the World Paraplegic Games at Stoke Mandeville in 1984. All athletes sustained complete neurological lesions between the level of T10 and L2. Clinical details were obtained from each athlete. Assessment then consisted of measurement of spinal movement in the sagittal plane using spondylometers, and movement in the rotation plane using a rotometer. Body trunk strength was measured with each athlete in their own wheelchair using a myometer (Penny and Giles). This gave a recording in kilograms force. Trunk balance was assessed on both a static and dynamic basis. The age, sex, follow-up and body weight for both spinal fusion and conservative groups were similar.

The results demonstrated that the conservatively treated paraplegics had a mean of 12 degrees more sagittal plane movement than their comparable spinal fusion group. The conservative group had a mean of 25 degrees more trunk rotation than the fusion group.

There was no difference in flexor trunk power measured with the myometer between the two groups, but when extensor power was measured it was found to be 25% less in the spinal fusion group. There was no difference between the two groups when tested for static dynamic trunk balance.

Two other athletes who had spinal fusions from the high thoracic region down to the sacrum, are presented separately. Although both were neurologically T10 lesions their function was so poor that they were unable to compete in the same athletic class and competed against those with a much higher neurological deficit (above T5).

The results of this study demonstrate that spinal fusion, particularly over multiple segments in complete paraplegics has a deleterious effect, not only on spinal movement, but also on body trunk strength.

It is concluded that surgery should be reserved for specific cases and that any spinal fusion necessary should be over as short a segment as is feasible. Long fusions to the sacrum are condemned, and in the young, growing, traumatic paraplegic every effort must be made to prevent spinal deformity as the surgery in these cases often has to be extensive.

Patterns of re-admission to a spinal unit

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The Spinal Injuries Unit of the Austin Hospital treats virtually all spinal cord injuries arising in Victoria, Tasmania, and the Riverina area of New South Wales. It assumes life-long follow-up of the patients treated. Over the last 30 years, about 2500 patients have been treated and about 1000 of these are still under regular review. The majority of patients living in the catchment area who develop significant complications or who require hospitalisation for unrelated causes are readmitted to the Austin Hospital for treatment. Over 1300 consecutive readmissions for a total of 514 patients occurring between

1.1.79 and 30.6.85 have been analysed. Readmission diagnosis, sex, age and duration of stay were recorded. While most of these patients were readmitted on only one or a few occasions, 10% had six or more radmissions. Recurrent pressure sores in combination with social and psychiatric problems appeared to be the reason for readmissions for some of these patients.

The study showed that a relatively small number of patients used a disproportionately large amount of the available resources. In many cases the problems leading to readmission could be considered avoidable. A pressure clinic has been established, and psychosocial and psychiatric support services have been improved. It will be interesting to see if these services reduce the 'avoidable' readmission rate.