An Outline of Cauda Equina Syndrome

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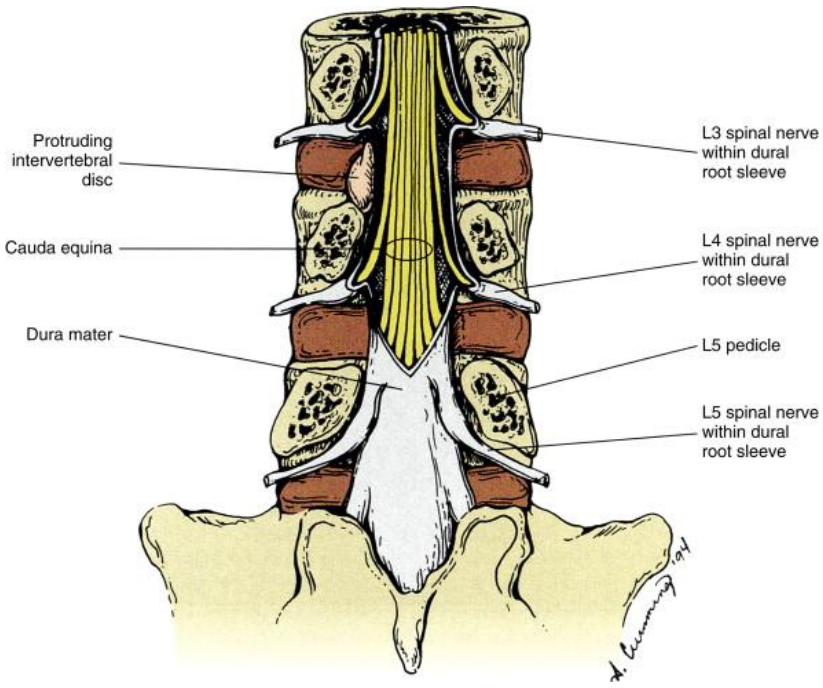
The spinal cord is a direct extension of the brain. It is composed of nerve cells, glial cells (which provide a supporting role but are not active electrically), and nerve fibre tracts. Most of the nerve cells are found in the central grey matter of the spinal cord. The fibre tracts are found towards the periphery of the spinal cord.

There is considerable information processing within the spinal cord. That is, sensory information arrives up sensory nerves, is delivered into the spinal cord, undergoes its first level of processing, and then is transmitted upwards towards the brain via fibre tracts. Similarly, motor instructions from higher centres are transmitted down fibre tracts and onto motor nerve cells on the spinal cord, where the various influences are integrated, and finally a command is sent out (or not) down a motor nerve fibre. The spinal cord is regarded as part of the central nervous system. It does not regenerate after trauma, and has a marked vulnerability to any form of insult such as compression or ischaemia (lack of blood supply).

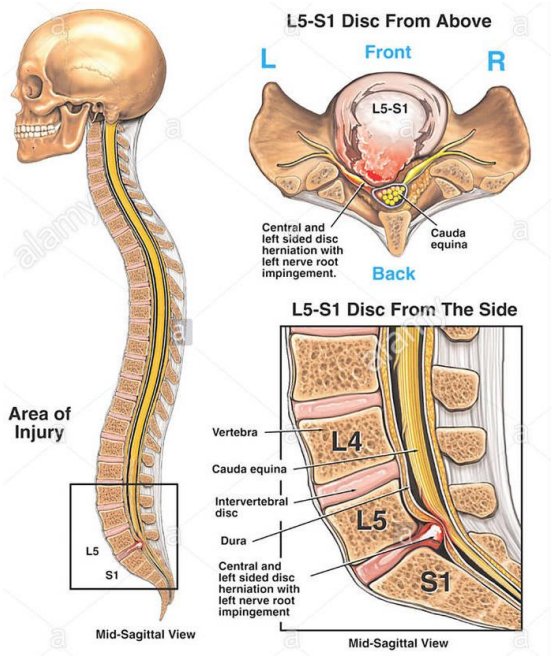
The nerves which leave (or join) the spinal cord, are bundled into structures termed “nerve roots”. Each individual nerve root can be regarded as a collection of further smaller bundles, each of which looked at under a microscope would reveal even smaller bundles. The situation is rather analogous to bundles of electrical wiring that may be fastened together to form a loom, in motor cars or indeed in computer networks. In the human body, the numbers of nerve fibres in each nerve root are measured in tens of thousands.

The actual nerve cells (axons) are fairly constant in size. However, each nerve cell may be increased considerably in diameter (up to 20 times) by a sheath of fatty tissue of insulation known as myelin. Myelinated nerve fibres tend to be rather more robust structures, and seem to be able to resist the effects of compression and ischaemia more than their poorly myelinated or unmyelinated counterparts. This means that even within a single nerve root, there may be a difference in vulnerability of different components of the nerve root to the same damaging influence.

The spinal cord ends in a conical structure termed the “conus medullaris”. Normally, the lowest part of the conus lies at the lower border of the L1 vertebra, or opposite the L1/L2 disc. From the conus, a large number of nerve roots emerge. These nerve roots travel downwards, to leave the spinal column through their respective neural exit foramina. The muscles of the lower limbs, the control of micturition (urination) and evacuation of faeces, and sexual function are all mediated by the nerve roots from L2 downwards. At the L1/2 level (the disc) we would find all the nerve roots from L2 down to S5.



Dorsal (posterior) view of the cauda equina



Axial and lateral views of the cauda equina

Each pair of nerve roots has relatively specific functions. For example, the nerve roots which issue from the L2 and L3 foramina are involved with the control of flexion of the hips, the nerve roots at L5 control dorsi-flexion of the foot, and those at S1 control its plantar flexion. These nerve root values, as they are termed, are not absolute, but they represent where we would find the majority of nerve fibres innervating the corresponding muscles, in the majority of patients. The nerve roots corresponding to the second, third and fourth sacral segments (S2, 3 and 4) are finer, and have a higher proportion of thinner, less thickly myelinated nerve fibres. These nerve roots are particularly vulnerable to compression. They tend primarily to be involved with the control of bladder evacuation, bowel evacuation, and sexual function.

The entire bundle of nerve roots from the bottom of L1/2 (the end of the conus) to the bottom of the sacral canal is termed the "cauda equina", because of its supposed resemblance to a mare's tail. Each nerve root is composed of a dorsal (posterior) sensory part and a ventral (motor) part. The dorsal and ventral roots combine close to the conus, to form the spinal roots from L2 to S5. Therefore in the cauda equina there are nine pairs of spinal roots of any functional importance: L2, L3, L4, L5, and S1, S2, S3, S4 and S5.

Pathophysiology.

But: death is not the mere stoppage of a machine; it is also total ruin of the supposed machinery. Similarly-and this is a lesson which I wish to emphasize as strongly as I can -partial anoxaemia means not a mere slowing, down of life, but progressive and perhaps irreparable damage to living structure.

J S Haldane

A Lecture on the Symptoms, Causes, and Prevention of Anoxaemia (Insufficient Supply of Oxygen to the Tissues), and the Value of Oxygen in its Treatment

Br Med J 1919; 2:65 (Published 19 July 1919)

The nerve roots are damaged by direct compression, and the local release of inflammatory mediators. It is a progressive condition. [1] The final common pathway of damage is ischaemia, due to direct compression and thrombosis of the blood supply[2]. It is unfortunate that the nerve roots of the cauda equina have a tenuous blood supply. The blood vessels are all longitudinal, and run along the length of the nerve roots from above and below. There are no reinforcing branches which enter the nerve roots from the side. Therefore, if the nerve roots are deprived of their blood supply over a significant part of their length, they will suffer irreparable damage; and their function will never recover. It is an unfortunate quirk of Nature that the slender S2-S4 roots, which subserve sphincter and sexual function, are particularly vulnerable to permanent ischaemic damage.

When the nerve roots of the cauda equina are compressed, they are deprived of their blood supply. The initial manifestation will be loss of function. Prompt decompression, and restoration of blood flow, will lead to recovery. On clinical grounds, there is no means of distinguishing between loss of function – which may be reversible – and loss of structure, which is not. It follows that the sooner we restore blood supply – and allow room for swelling – the better the chance of recovery.

Deterioration may be rapid, with the patient progressing from a fully retrievable situation to irreparable damage within hours[3]. In the case of a large prolapsed disc, the fact that the patient develops increasingly severe symptoms and signs does not imply a progressive increase in compression by the pathological lesion. The reason for deterioration, particularly in an acute case, is progressively increasing swelling and ischaemic damage to the cauda equina nerve roots, confined as they are in a very narrow space.

Complete cauda equina lesions.

In practical terms, a complete cauda equina lesion at the L2/3 disc level would spare the L2 nerve roots (which emerge just above the level of the disc), but take out all the nerves below. The patient would be able to flex his hips. However, he would have no sensation from the

inner thigh downwards and there would be no sparing of sensation around the bottom. He would have no useful movement at the knees or ankles. In essence, the patient would be paralysed and wheelchair-bound, doubly incontinent and impotent.

At L3/4 there would be rather more preservation of neurological function. The patient would likely be able to extend his knees, but would again have no control over bladder, bowel or sexual function.

At L4/5 there would be relative preservation of movement of the hips and knees, but there would be loss of foot dorsi-flexion and plantar flexion, and again double incontinence and loss of sexual function.

At L5/S1 a complete lesion would lead to the loss of plantar flexion (weak calves) and loss of bladder, bowel and sexual function. A lesion at the L5 S1 junction that goes downwards and spares the S1 nerve roots (surprisingly common) would affect bladder, bowel and sexual function, but might have little manifestation in the way of weakness in the legs. Even the ankle reflexes (which depend mainly on intact S1 nerve roots) could be preserved. This latter clinical picture is a classic trap for the unwary.

Diagnosis.

Cauda equina syndrome is a spectrum. It is diagnosed by taking a history and examining the patient, suspecting the diagnosis, and confirming it (in modern hospitals) with scanning of the back. The ideal scanning modality is MRI, although CT scanning with intrathecal contrast may be used if MRI is not available. An alternative method is to use the more old-fashioned technique of myelography. Plain radiographs play no part in the diagnosis, unless trauma, malignancy or infection are the precipitating cause.

The problem which arises frequently in medical practice is deciding at which point in this spectrum are a patient's symptoms severe enough to trigger an emergency response? Clearly, from the patient's point of view the earlier the diagnosis is made, the better the outcome. This is now indisputable. Unfortunately, not everybody can have instantaneous access to definitive care. However, because the consequences of missing the diagnosis are so dreadful for the patient, and because the medicolegal implications are so great, the threshold for referral and definitive diagnosis has to be low.

It must be understood that back pain is very common. In any resource-limited health care system, not every patient can have an MRI, CT or equivalent scan. However, any patient who presents to a medical practitioner must have a carefully taken history, and a good neurological examination. In any case of back pain, the doctor must consider whether there are sufficient worrying or sinister features in the clinical history, or on examination, to suggest a diagnosis of spinal cord or spinal nerve root compression. For the purposes of this discussion I will confine myself to the diagnosis of cauda equina compression, although clearly the same considerations apply to lesions higher up in the nervous system.

The practitioner must consider whether the problem could be due to an underlying tumour; an infection; a large disc prolapse into the spinal canal; a blood clot around the spinal canal; or an inflammatory or other process.

Red flag symptoms

It is a general principle in acute neurosurgical problems that symptoms tend to precede signs. Therefore, the practitioner must look for what are termed "Red Flag symptoms". The following is a list of common symptoms which is by no means exhaustive (see Fairbank J ref 11).

Common symptoms are:

1. Bilateral sciatica: Pain in both legs extending below the knees.

2. Weakness in the leg/legs.

3. Numbness in the leg/legs.

4. Any loss of sexual function; erectile disturbance, loss of vulval/vaginal sensation etc.

5. Any complaint of difficulty with micturition (urination) including retention of urine and incontinence.

6. Any complaint of difficulty with anal sphincter control, including difficulty with passing wind safely; i.e being able to detect the difference between wind and a motion.

7. Any complaint or mention of numbness around the genital area, particularly the peri-anal area.

These symptoms may be present to a greater or lesser degree, and in any combination. In addition, in the early stages of cauda equina syndrome, the symptoms may vary both in nature and in severity. In incomplete lesions, these symptoms may fluctuate with time, and the clinical care – and urgency - must be based on their worst manifestation; that is, the clinician must always consider the worst case scenario, and be alert to the possibility of developing cauda equina syndrome. This is particularly so as diagnosis and treatment at the early, incipient or even partial stage leads to a much better outcome.

<u>"Red flag" signs</u>

Red flag features on examination (signs) include bilateral loss of straight-leg raising; weakness in the legs; numbness; bilateral loss of reflexes, loss of sensation, particularly in the "saddle" area, and any loss of anal sensation or anal sphincter tone. Clearly, any patient who has bilateral symptoms and signs is in a much higher risk category for cauda equina syndrome, as it suggests that the compressive lesion is near or in the mid-line, and not off to one side. This represents a much greater threat to distal neurological function.

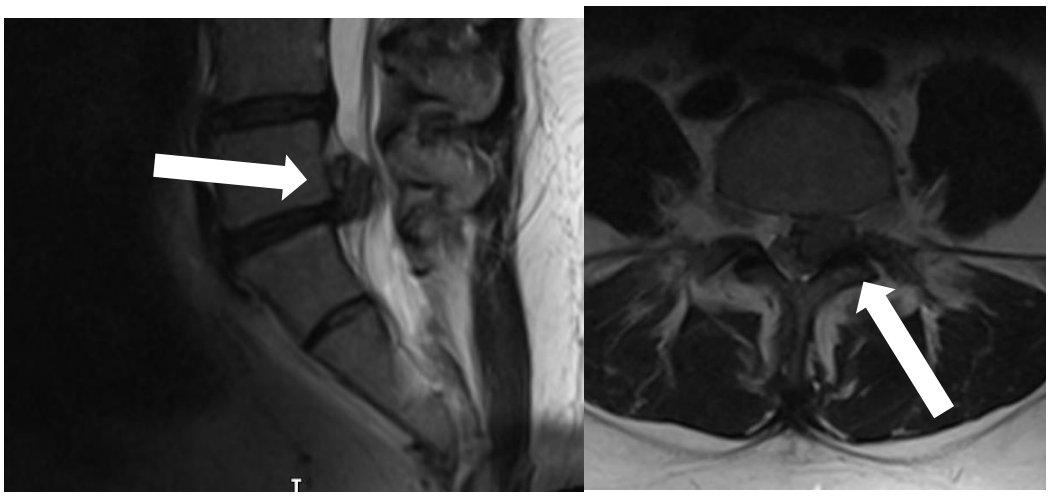
Assessment of residual volumes post voiding is important. The residual volume should be less than 65 mls in younger patients: in older patients 100mls is acceptable. [15]

THERE IS A COMMON MISCONCEPTION THAT A NORMAL NEUROLOGICAL EXAMINATION EXCLUDES AN INCIPIENT, THREATENED OR PARTIAL CAUDA EQUINA SYNDROME. IT DOES NOT. SYMPTOMS ALONE ARE SUFFICIENT INDICATION TO TRIGGER EMERGENCY MANAGEMENT.

Radiological investigation

The diagnosis is confirmed with immediate MRI scanning, as indicated earlier. Cauda equina syndrome would be diagnosed by the presence of acute compression of the nerve roots of the cauda equina, usually manifest by the lack of any spinal fluid visible around the nerve root. **The presence of any combination of "red flag" symptoms and signs, especially when present bilaterally, must trigger an immediate response on the part of the medical practitioner.** This is because the patient could have an incomplete or threatened cauda equina syndrome. Unfortunately, there is a depressing tendency for cauda equina syndrome to be left to progress, usually over a period of hours or days, from an incomplete (almost always retrievable) syndrome to a complete (usually irretrievable) syndrome.

It must be noted that cauda equina syndrome remains a clinical diagnosis. There is a modern tendency to place excessive if not complete reliance upon the MRI scan appearances. The clinician must note that cauda equina dysfunction can occur even when there is cerebrospinal fluid (CSF) visible around the nerve roots in the theca, at the compressed level. It is not necessary for a patient to have a "complete blackout" of the CSF space around the nerve roots for there to be significant cauda equina dysfunction.[7]



MRI scan in a case of cauda equina syndrome from a large disc prolapse. Note that the disc prolapse occupies virtually the entire spinal canal, with severe compression of all the transiting nerve roots at that level.

One feature of the radiological investigations which does not receive its due attention is the anterior-posterior diameter of the spinal canal, and with it its cross-sectional area. It is variable. It does not always correlate with body size. The diameter may vary between 18 mm, to as little as less than 10 mm. Relative stenosis is defined as an anterior posterior diameter less than 12 mm. Absolute stenosis is said to exist when the diameter is less than 10 mm. The importance of this measurement is that a relatively small disc prolapse may seriously threaten the merits of the cauda equina, in a patient with a congenitally narrow canal.

Outcome

The results of the management of cauda equina syndrome illustrate a more general principle in neurosurgery: the harder and longer the nervous system is squeezed, the worse the outcome tends to be in the long term. This principle holds true for all acute neurological conditions that involve compression or distortion of the nervous system, and deprivation of

its blood supply: abscess, tumour, haematoma, swelling, infection or compression by a disc prolapse, to name but a few.

Thus, cauda equina syndrome, when it presents to the medical profession, cannot be regarded as a homogenous clinical entity. Rather, there is a spectrum of severity at presentation. The severity may vary from just enough in the way of clinical features to alert the clinician (such as severe bilateral sciatica with no neurological signs) to bilateral complete foot drops, complete sphincter paralysis, impotence and complete saddle anaesthesia with a patulous anus (no anal tone). It also reflects the sad paradox seen in many acute neurosurgical conditions, which is that the likelihood that the diagnosis will be considered is lowest when the condition is most treatable: and highest when the clinical situation is irretrievable[8,9].

Treatment

When relieving the nervous system of compression due to acute compressive lesions by decompressive surgery, most neurosurgeons would regard one of the prime objectives as the prevention of further deterioration. The general principle would be that "what goes into the operating theatre should come out of the operating theatre". If there is restoration of function, one would regard this as something of a bonus. Preservation and restoration of function is most likely in patients who have been decompressed timeously – and widely.

Therefore, if patients with cauda equina syndrome are to be prevented from being left with long term disability, the sooner it is diagnosed and treated the better[10].

For 8 years I was first the Lead Clinician, and then Clinical Director of neurosciences in my own Unit. The quality standard for the management of cauda equina syndrome I established was based on a simple mnemonic, termed the "Six Ss". These were the actions that were to be taken as soon as the diagnosis was suspected.

1 **Starve** the patient: (may need emergency anaesthesia and surgery)

2 Organise an immediate MRI **Scan.**

3 Administer a large dose of intravenous **Steroids** (may be a tumour or inflammatory condition – and there is little lost by such treatment)

4 Summon a **Surgeon**

5 Get the patient into the operating theatre for **Surgery**

6 All within **Six hours**

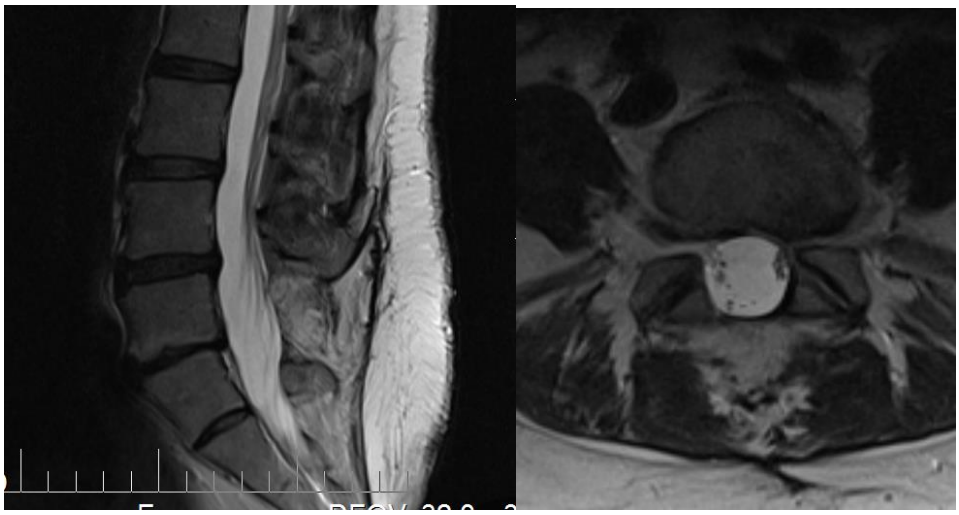
Surgical management.

"There is no place in the management of this condition for the micro discectomy approach that is routine for the radicular pain syndrome as sciatica due to disc herniation. The traditional mid line approach with bilateral exposure of laminae and complete laminectomy should be performed to immediately decompress the cauda equina prior to manipulation the thecal sac in an attempt to remove the disc fragments."(H Bridwell et al., 1997 page 1558)

See also the extract from “A Neurosurgeon’s notebook” by CBT Adams.

In cases of cauda equina syndrome due to a large disc prolapse, it is essential that the decompression is very wide, and done through a full laminectomy – if necessary above and below the level of compression. The nerve roots within the thecal tube will have been severely compressed. They will swell – either immediately, or soon after surgery. They have to be given room, both at the time of surgery, and in the days thereafter.

This is particularly so if the surgeon unintentionally breaches the dura while removing bone, especially early on. A dural breach implies that there is no room for manoeuvre at the level of maximal compression. It is essential that surgeon heeds this warning, and moves the site of bone removal above and below, where the dura will be slacker, and where there will still be a cushion of CSF around the intrathecal nerve roots. Manipulation of the dura at the level of maximal compression is a potent cause for the lack of significant neurological improvement after surgery; a cause of neurological deterioration; and a recipe for potential disastrous surgical trauma. Above all, the surgeon is strongly advised not to patch the dural breach with Surgicel. Surgicel is composed of cellulose; and cellulose swells, particularly when in contact with CSF. Leaving this material behind in the spine is actually expressly forbidden by the manufacturer for this reason: read the accompanying pamphlet!



Post-operative MRI scan from the patient illustrated above. Note the complete laminectomy, and a wide decompression of the dural tube at the previously operated level. The patient made a complete neurological recovery.

Conclusion.

It is in the light of these comments that the previous literature about cauda equina syndrome needs to be judged. This might go some way to explaining the confusion between the different accounts, particularly with regard to delays in diagnosis and definitive treatment (which is almost always a surgical decompression).

From this account of cauda equina syndrome, we can consider any particular case. There are a number of questions that need to be answered.

At what stage did the clinical condition show sufficient worrying features that differentiated him/her from the large number of patients who attend their medical practitioners with simple back and leg pain?

When did he exhibit sufficient clinical features to justify the suspicion of cauda equina syndrome – threatened, impending, incomplete or complete?

What should have been done immediately?

Would such immediate action have resulted in a better clinical outcome?

Finally, is there still the opportunity for spontaneous neurological recovery, or has the "neurological die been cast"?

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Standards of Care for Investigation and Management of Cauda Equina Syndrome

Background

Cauda Equina Syndrome (CES) is a relatively rare but disabling condition which can result in motor and sensory deficits, incontinence of urine and faeces, and loss of sexual function.

Any patient with a possible diagnosis of threatened /partial/complete CES requires urgent investigation.

Presentation

A patient presenting with back pain and/or sciatic pain with any disturbance of their bladder or bowel function and/or saddle or genital sensory disturbance or bilateral leg pain should be suspected of having a threatened or actual CES.

Imaging

The reliability of clinical diagnosis of threatened or actual CES is low and there should be a low threshold for investigation with an emergency MRI scan at the request of the examining clinician and MRI must be available at the referring hospital 24/7.

The decision to perform an MRI does not require discussion with the local spinal services.

The MRI must be undertaken as an emergency in the patient's local hospital and a diagnosis achieved prior to any discussion with the spinal services.

The MRI must take precedence over routine cases and any reasons for a delay or a decision not to perform an emergency scan should be clearly documented.

If MRI is contraindicated, discussion with local spinal services is appropriate.

There are four potential outcomes from the investigation

1. Cauda equina compression confirmed leading to immediate referral to an appropriate surgical service.
2. Cauda equina compression excluded but a potential structural explanation of pain identified. This should precipitate appropriate advice about potential future cauda equina symptoms and may include referral via local spinal pathways during working hours.
3. Non-compressive pathology may be identified (e.g. demyelination) which should precipitate referral to the appropriate service.
4. No explanation of the patient's symptoms may be apparent. An appropriate plan for further management is required and may include a cervico-thoracic MRI and referral to continence services.

Surgery

Nothing is to be gained by delaying surgery and should be undertaken at the earliest opportunity, considering the duration and clinical course of symptoms and signs, and the potential for increased morbidity while operating in the night. We do not consider that there is anything in the literature that justifies contravention of this principle and reasons for any delay in surgery should be clearly documented.

Post-Operative Care

All patients with ongoing sphincter disturbance should be promptly referred to local continence services which may include colorectal and urological services or spinal cord injury services.

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