of laxatives, and overflow from faecal impaction—may present as incontinence with a normally functioning anal sphincter.[2] Even people with poor functioning of the sphincter can obtain much symptomatic benefit from measures to firm up the stool. If an underlying cause for diarrhoea is not found dietary advice can be very helpful, bearing in mind that a diet rich in fibre may exacerbate incontinence. Occasionally, antidiarrhoeals such as codeine phosphate or loperamide may be required.

In a few patients these measures will be insufficient, and referral for testing of anorectal physiology and endo-anal ultrasonography becomes necessary. These investigations provide objective measures of sphincter pressures, anorectal sensation, rectal capacity,[7] and sphincter defects.[8] A continence adviser can then help most patients by providing support and advice on diet, lifestyle, and pelvic floor exercise. Thereafter pelvic floor physiotherapy and biofeedback—in which patients are taught effective muscle contraction in response to information from a manometric or electromyographic rectal probes—are the mainstays of therapy.[9]

In unresponsive patients with a noteworthy sphincter defect, surgical repair will produce a good outcome in 60-90% of patients, but unfortunately the results deteriorate with time.[10] When surgical repair fails or is inappropriate and where biofeedback is ineffective three options remain for severely symptomatic patients—anal encirclement, sacral nerve stimulation, and diversion.

Encirclement is achieved by means of muscle, and the dynamic graciloplasty, which involves a gracilis muscle wrap augmented by electrical stimulation, is perhaps the most successful. Another approach is the artificial sphincter, which consists of a fluid filled cuff implanted around the anal canal. However, these are specialised techniques which, although they can restore continence, often result in difficulty in passing stools and in morbidity.[2]

Sacral nerve stimulation (or neuromodulation) is a relatively new technique for treating faecal incontinence and is used in patients with an intact or repaired sphincter complex.[11] It involves placing a percutaneous electrode in a sacral foramen (usually S3), and if a two week test period is successful a permanent pulse generator can be implanted. Recent studies suggest that notable improvement can be achieved with little morbidity, and this procedure looks set to have a major impact.[12] Despite these new treatments, the role of faecal diversion should not be ignored. If all else fails quality of life with a stoma can be excellent for the unfortunate patient with refractory incontinence.

Although faecal incontinence is responsible for a great deal of misery and social isolation it lacks a high profile and vocal advocates. It is amenable to treatment that often can be delivered in primary care. Moreover, those unresponsive to simple measures should be given the opportunity for assessment by specialists and appropriate treatment.

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Tetanus in injecting drug users

The latest Clostridium infection to threaten injectors in Britain

Clostridium infections in injecting drug users are of continuing concern for clinicians in emergency departments and intensive care because infected patients may present unwell and deteriorate very quickly without appropriate management. In 2000, an epidemic of necrotising fasciitis resulting in overwhelming sepsis and sudden death in British and Irish injecting drug users was caused by organisms such as *Clostridium novyi*. These flourish in the anaerobic environment of wounds associated with injecting into skin or muscle ("popping"). This may be done inadvertently or deliberately by injecting drug users who cannot find a vein—women, older users, and those with a longer injecting history are at greatest risk.[1] Botulism due to the systemic effects of toxin released from *C botulinum* in wounds in injecting drug users continues to be seen in Britain[2] and elsewhere, particularly in California where it is linked with locally available "black tar" heroin.[3] Patients typically present with diplopia, dysarthria, dysphagia,

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descending flaccid paralysis without sensory symptoms, followed by diaphragmatic paralysis. Early recognition and treatment with antitoxin, antibiotics, and prolonged ventilation can result in full recovery.

First described in subcutaneous morphine abusers in the late Victorian era,[4] the high mortality of tetanus associated with injecting drugs was defined in Chicago 50 years ago before routine vaccination was introduced.[5] Of the 22 patients with tetanus, 12 were injecting drug users and they all died, whereas death occurred in four patients with other types of wound. The association with skin and muscle popping was also recognised decades ago in the United States,[6] where injecting drug users accounted for 19 (15%) of 130 recent cases overall and 45.7 % (16/35) of Californian cases,[7] compared with only two of 175 cases of tetanus in England and Wales between 1984 and 2000.[8]

Something changed in Britain in 2003. At least 24 cases of tetanus occurred in injecting drug users, 23 of them clustered between November 2003 and April 2004, with two deaths.[9] The cause of this increase remains unexplained. A single fatal case has been reported in the Netherlands.[10] Patients presented to centres all over mainland United Kingdom, usually with severe disease requiring prolonged sedation and ventilation. Most had low serum antitoxin antibody levels and no documented history of complete tetanus vaccination.[9]

Tetanus usually presents with a stiff neck and back, difficulty in opening the mouth progressing to difficulty in swallowing, facial and limb muscle spasms, and profuse sweating. Such patients rapidly progress to respiratory embarrassment, which is the usual cause of early death if not managed aggressively. Milder forms with focal spasm also occur but are uncommon in injecting drug users. Symptoms are caused by toxin binding to and inhibiting the γ-aminobutyric acid mediated spinal inhibitory neurons, increasing both spinal and autonomic transmission and muscle activity. No definitive laboratory test exists, but a clinical diagnosis is supported by culture of *C tetani*, detection of circulating tetanus toxin, and absence of tetanus antibodies. Most patients have had contaminated soft tissue wounds from injecting, from which a variety of anaerobes including other Clostridia have been cultivated as well as *C tetani*.

Early recognition and treatment are essential to reduce the mortality from 100% to below 10%. Early intubation and assisted ventilation with prolonged intensive care are usually required, details of which have been reviewed.[11 12] Wounds should be debrided thoroughly, and systemic metronidazole is probably preferable to penicillin, which has γ-aminobutyric acid agonist activity that may potentiate symptoms. Human tetanus immunoglobulin should be given as soon as possible. The evidence for its use, the route used, and the dosage is poor, but a single dose of 5000-10 000 units is usually given intravenously. The intravenous preparation has to be obtained by courier. The intramuscular preparations, usually used for tetanus prophylaxis after a tetanus prone wound has occurred, may be used instead, but this requires large intramuscular injections (1 ml typically contains only 250 units), and such preparations should not be given intravenously.[13] Past meta-analyses, which do not support the intrathecal administration of human tetanus immunoglobulin, have been challenged by a recent prospective study in Brazil showing benefit over the intramuscular route.[14]

Assisted ventilation and paralysis may be required for several weeks. Paralysing agents such as pancuronium can worsen sympathetic overdrive, and magnesium infusions are used to try to reduce this. Autonomic instability leads to wild swings of heart rate and blood pressure that are difficult to control even with short acting β blockers and that can lead to sudden cardiac death typically in the second or third week.

Clinicians should consider the systemic effects of apparently trivial wound infections in injecting drug users who present with unexplained collapse, sepsis, or odd neurological symptoms, which might otherwise be dismissed as the direct results of drug intoxication. Suspected cases of tetanus or botulism should be notified immediately to the local Health Protection Agency (HPA) unit, which can advise on public health measures and specialist laboratory diagnostic support available from the HPA's specialist and reference microbiology division (www.hpa.org.uk).

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