

Unfortunately, the incident reported by Drs Behura and Ahuga could not be investigated fully as the product sample was not made available to us for examination. However, by the nature of the product design, we can only conclude that either the valve was incorrectly positioned within the catheter housing or, once the valve was opened, by injecting through the side port, it was prevented from closing correctly.

More than 1 billion BD Venflon™ have been used over the years with few similar reported complaints. In cases where we have had the opportunity to observe the actual product in question, we have found particles of glass retained between the sealing surface of the valve and the body of the catheter housing, creating a canal sufficient to permit leakage. In these cases, our investigations have identified that the infusate used was drawn into a syringe from a glass ampoule.

BD recommends best practice for drawing-up infusates from a glass ampoule should always be to use a BD Blunt Filter Needle or other commercially available equivalent filter device.

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The lidocaine patch: a useful addition to the pain clinic armamentarium

We report the successful use of lidocaine patches in two patients with neuropathic pain. A 39-year-old woman attended the pain clinic with a 5-year history of burning pain and paraesthesia in the sole of her left foot. Surgery for presumed Morton's metatarsalgia had proved ineffective. The 2 cm² painful area overlying the surgical scar demonstrated altered sensation, allodynia, cyanosis and reduced skin temperature suggestive of complex regional pain syndrome type 1. After numerous other interventions the patient was prescribed Lidoderm® patches (Endo Pharmaceuticals Inc., Chadds Ford, PA) applied to the painful area. At review she reported significant pain

relief, saying that the area felt 'numbed' with the additional benefit that the patch physically protected the sensitive skin. She continues to use the patches as her sole form of pain relief 1 year later. A 27-year-old woman was referred to the pain clinic with an 18-month history of postherpetic neuralgia in the left T7 dermatome. Pain was described as a continuous burning sensation with associated marked allodynia. Application of Lidoderm patches to the painful area helped significantly, particularly reducing the allodynic component of her pain syndrome.

Both patients had neuropathic pain that proved resistant to treatment with normal analgesics, tricyclic antidepressants and anticonvulsants, but responded to topical lidocaine. The pathophysiology of neuropathic pain is complex, but includes neuronal injury leading to the abnormally functioning sodium channels [1, 2]. Therefore treatment with a neuronal membrane stabiliser such as lidocaine would be a rational choice. Unfortunately, the use of parenteral lidocaine is limited by toxicity and oral lidocaine congeners such as mexiletine, while less toxic, have unproven efficacy and frequent side-effects [3]. The Lidoderm patch allows topical application of lidocaine 5%. The patches are licensed in the USA for the treatment of postherpetic neuralgia and are available in the UK if imported. Penetration of lidocaine into intact skin after application of a Lidoderm patch is sufficient to produce an analgesic effect, but not enough to produce a complete sensory block. The patches measure 10 cm × 14 cm and consist of an adhesive material (containing the lidocaine) on a non-woven polyester felt backing. Up to three patches may be applied at any one time, for up to 12 h in a 24-h period. They may be cut to size or shape as necessary. If applied to intact skin, as recommended, only 2–3% of the dose applied is absorbed; peak plasma levels of lidocaine average 0.13 µg.ml⁻¹ (toxic level 5 µg.ml⁻¹) [4].

A small number of studies have shown the benefit of Lidoderm patches in postherpetic neuralgia [5, 6]. There are also several anecdotal reports of their use in osteoarthritic knee pain and

myofascial trigger points. We are not aware of any studies comparing Lidoderm patches with antidepressants or anticonvulsants in the treatment of neuropathic pain. Adverse effects are rare, mainly involving skin reactions at the site of application. For the UK market, cost remains the major disadvantage: the cost of 30 patches is approximately £190, and they must be imported from the USA.

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- 4 Endo Pharmaceuticals. Lidoderm® Prescribing Information. <http://www.endo.com>.
- 5 Rowbotham MC. Lidoderm patch: double-blind controlled study of a new treatment method for post-herpetic neuralgia. *Pain* 1996; **65**: 39–44.
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Mirror box therapy for complex regional pain syndrome

A 63-year-old woman presented with swelling and reduced range of movements in the right upper limb following a fracture of scaphoid bone. She was diagnosed with complex regional pain syndrome (CRPS I). Simple analgesics and neuropathic medications were of no benefit, and sympathetic blocks were equally ineffective.

Following a study course at the International Association for the Study of Pain conference in Sydney, Australia, in 2005, she was commenced on mirror box therapy (Fig. 4), first described by Ramachandran in the treatment of phantom limb pain [1]. The patient had an immediate and dramatic improvement in the movement of the affected limb and reduction in pain scores of more than 50%. Following this trial in the outpatient department, she was then issued with a mirror box to take home.

Ramachandran proposed in patients with phantom limb pain that there is disruption in the normal interaction between motor intention to move a limb and proprioceptive feedback. They speculated that visual feedback might interrupt this pathological cycle. Mirror box therapy for CRPS is thought to work by providing such a visual feedback: by moving the unaffected limb in front of the mirror there is cortical reorganisation of the sensory homunculus within the parietal cortex. Whether these changes in the cortex are a primary or secondary effect of CRPS is still unknown. Mirror box therapy may provide a quick, cheap, effective and relatively painless form of management for a condition that is very resistant to conventional therapeutic strategies.

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Figure 4 Mirror box in use.

Reference

- 1 Ramachandran VS, Rogers-Ramachandran D. Synaesthesia in phantom limbs induced with mirrors. *Proceedings of the Royal Society of London – Series B: Biology Sciences* 1996; **263**: 377–86.

Adrenal crisis presenting as an acute abdomen

We report an interesting case of an 'acute abdomen'. A 63-year-old female was admitted to the Emergency Department with a brief history of severe diarrhoea and rectal bleeding. Colonoscopic findings were normal. Over the next 24 h she developed lower abdominal pain with distension and became hypotensive. Her blood biochemistry results were sodium 133 mmol.l^{-1} , potassium 5.3 mmol.l^{-1} , urea 10.1 mmol.l^{-1} and creatinine $186 \text{ }\mu\text{mol.l}^{-1}$. Suspecting peritonitis, surgeons performed a laparotomy which revealed a large quantity of uninfected peritoneal fluid, but no perforation of a viscus. Microbiological screening did not reveal any infection. In spite of all measures, she developed multi-organ failure and died a week later.

Her past medical history included a head injury 35 years previously and she was maintained on carbamazepine and sodium valproate for post-traumatic seizures. She was hypothyroid on thyroxine supplementation. About 10 years prior to this admission she underwent

Keller's operation on her foot and post-operatively had severe seizures and high fever. A few months later she underwent appendectomy and had a difficult postoperative recovery with seizures and hyponatraemia (113 mmol.l^{-1}), which was attributed to the syndrome of inappropriate ADH secretion secondary to her anti-epileptic medications. On another occasion she was admitted with tonsillitis and developed features of septic shock with a high fever and persistent hypotension requiring fluids and antibiotics. Her only uneventful hospital visit was for an orthopaedic procedure during which she was given a methyl prednisolone injection into her shoulder joint.

At post mortem examination, despite a prolonged and extensive search, the adrenal glands were not identified. The thyroid gland was small and the pituitary fossa was considerably enlarged and the pituitary gland was largely replaced by non-functioning fibrous and calcified tissue. On review it appears complications during her previous hospital admissions could have been manifestations of adrenal insufficiency. The only uneventful hospital stay was when she had a steroid injection in her shoulder, which incidentally provided the steroid coverage during the stressful period.

In this case, the adrenal failure could have been the result of the head injury she sustained several years earlier, resulting in damage to the pituitary gland. Against this possibility is the enlarged pituitary fossa found at autopsy. She had also suffered from unexplained episodes of headaches, perhaps due to a necrosed pituitary tumour? Histological examination of the pituitary gland revealed calcified and atrophic tissues. Similar cases have been described in the past where the cause was attributed to autoimmune adrenalitis [1]. Acute gastrointestinal symptoms and persistent unresponsive hypotension should raise the possibility of adrenal insufficiency. As our case demonstrates, missing this diagnosis can lead to unnecessary surgical exploration.

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