Management of ischemic pain with action potential simulation — a case report

Abstract
Ischaemic pain belongs amongst the most difficult to treat pains in palliative care. The pain is frequently severe and resistant to available analgesics. Treatment of this condition is difficult especially when the condition is inoperable. We describe a patient with severe ischaemic pain in the lower leg due to previous vascular problems and superimposed deep venous thrombosis who responded well to action potential simulation (APS), a therapy using microcurrents which resemble the body’s own biocurrents. This technology is frequently used by complementary therapists but is still barely known to evidence-based medicine.

Key words: limb ischaemia, ischaemic pain, arterial emboli, opioids, micro electro therapy, action potential simulation

Introduction
Ischemic pain belongs to the most recalcitrant symptoms in Palliative Care. Fortunately, this type of pain is rare among patients suffering of cancer, but its prevalence is dramatically higher in patients suffering of diabetes and vascular diseases [1, 2]. As a rule ischemic limb pain is severe, highly variable during the day and is accompanied by paleness and a cold feeling in the distal part of extremity. Treatment of this condition is primarily by surgical procedure: a bypass or arterial prosthesis/stent [3, 4]. When this is impossible or contraindicated some improvement can be expected from sympathectomy [5, 6], nerve section [7] or epidural morphine [8] but the evidence for this is weak. Ischemic pain responds poorly to analgesics and this is the reason why different techniques are often tried [9, 1]. In this article we describe a patient with severe ischemic pain in lower leg who responded well to action potential simulation (APS), therapy with micro-currents that resembles body own biocurrents. This technology is frequently used by complementary therapists, but still hardly known to the evidence based medicine [11, 12].

Case report
Mr. K (65) was urgently referred to the pain clinic because of the severe pain in his left lower leg. He had a history of arterial insufficiency and prosthesis in the left popliteal artery and in the past he was using warfarin. However, when he developed oesophageal cancer and became anaemic warfarin was discontinued. Two months later he developed left sided deep vein thrombosis. This
was accompanied by a severe pain in the left leg. Doppler ultrasound investigation confirmed thrombosis in the femoral vein, but also showed no blood circulation beyond the popliteal artery. Fraxiparin was commenced but discontinued because of small brain bleeding confirmed by the brain CT scan. The pain was present during and after therapy with Fraxiparine. At assessment the pain was much worse on movement and the action radius was not more than several meters. He slept poorly, usually sitting in the chair. He used oral morphine (morphine sulphate tablets with controlled release, 100 mg a day and oral morphine sulphate of immediate release several times a day, 15 mg per dose). Unfortunately this medication was not able to influence his pain. He and his surrounding noticed his mental deterioration, depression, sometimes agitation. On examination: multiple venectasies. The left lower leg was swollen and cold around the knee. Palpation was not painful. There were no arterial pulsations palpable starting from the iliac artery in the groin. No bruits. Pulsations on the right leg were normal. On the shin, on the heel and on the second toe of the left leg small areas of necrosis were observed.

The patient was treated using APS technology. An MK2 US two channel machine produced by Tech Pulse Manufacturing Pty Ltd. South Africa was used. Four gel electrodes PALS Platinum Blue 901220, 5 x 5 cm, were placed on both sides of the left upper leg and under his feet. The current intensity was slowly increased with potentiometer until the patient could feel it under the electrodes. The current intensity was then decreased to just below the threshold and the therapy was continued for 8 minutes in one session.

He was swapped to oxycodone controlled release 30 mg bd and oxycodone immediate release 15 mg, paracetamol 1 g and ketamine 30 mg per dose. After the first treatment the patient suffered severe muscle shakes but was not breathless, agitated or had hyperthermia. The therapy was administered usually in the morning and this resulted in 6–7 hours of analgesia. At the later stage the treatment intensity was increased to 8 minutes therapy twice daily. This resulted in much better analgesia, especially at night. He used his breakthrough medication once or twice a day. He felt so much better that he volunteered to undergo radiotherapy again.

Ischemic pain can originate from vascular intraluminal obstruction, usually by thrombo-embolus, external compression by the tumour and dissecting aneurysm. Deep venous thrombosis, like in our case can cause ischemic pain in the situation where the arterial circulation is already compromised. Increased pressure in the limb, due to congestion may especially impair circulation in new collaterals.

Pharmacological treatment of this kind of pain is very difficult. Fraxionated heparine (Fraxiparin) is the treatment of choice as it has a potential of decreasing congestion in the limb and decreasing the pressure [13]. However, in our case this treatment was not effective and needed to be discontinued because of serious haemorrhage.

Another possibility in this treatment is the use of ketamine in combination of morphine. This treatment is sometimes effective at the higher doses and it is frequent that the cognitive functions of the patients are severely compromised. In our case the original treatment with morphine alone was not effective and needed to be changed. We have chosen for oxycodone as this drug has less potential to compromise cognitive functioning [14]. Ketamine and paracetamol were added to the “acute” break through doses and appeared to be adequate.

Important in the treatment is positioning of the patients. Some patients prefer to sit in the chair with their leg supported. A good explanation for this was found by Ubbink et al [15]. Under physiological conditions arterioles respond with vasoconstriction when changing position from supine to sitting. This is probably due to increased sympathetic tonus. However, this mechanism is probably disturbed in patients experiencing ischemic pain and the perfusion of the limb is higher in sitting than in supine position.

The main treatment was by applying a new technology of APS. Action potential simulation simulates the bio-currents. The sub-threshold electric current has as an effect on the synthesis of the ATP by the cells. In that way the cells can be “vitalised” and damages can be repaired. This is very much different to the use of TENS principle, where the electric shocks stimulate nociceptors and close the “gate” in the spinal cord hampering the conveyance of ascending pain impulses. However, comparison of the two methods reveals short term similar results [11]. In our patient APS was effective within several minutes after the first treatment. Apparently the patient experienced during the first night severe shaking without increased body temperature or breathlessness. This reaction, seen very often by the APS therapist, suggest that some toxins are freed from the ischemic area and are absorbed to the circulation.
Discussion

Treatment with APS not only improved pain but also, visibly, improved skin circulation as within several days the necrotic discolorations of the skin disappeared. Potentially the therapy may improve collateral circulation to such an extent that the patient may discontinue it one time.

In the Netherlands and in Poland, APS therapy is known by the physiotherapists. However, in the UK, there is much less interest in development of these kind of methods. Before the method can be used in routine treatments, it should be first validated in different clinical settings, the staff should be trained to use it and cheap devices can be developed and used by the patients themselves. Our case suggests that the method may be useful in some otherwise intractable pain conditions.

However, before we shall conduct clinical trials, we should better understand how the technology is working and what kind of physiological effects are involved. Without this, clinical trials lacking this internal validity may render negative results [16] and the technology will be discarded before it is understood.

References
