Summary
A case of repetitive epileptic fits in a post stroke patient after transcutaneous electrical nerve stimulation (TENS) is presented. It seems more likely than not that the TENS triggered the repetitive fits in this patient. Although the risk of this adverse effect seems to be small, it should be borne in mind when TENS is used in a post-stroke patient. Since TENS is now used commonly in post-stroke patients, this problem is worthy of further study.

Keywords
Transcutaneous electrical nerve stimulation, stroke, epilepsy, adverse effects.

Introduction
During the last 15 to 20 years, transcutaneous electrical nerve stimulation (TENS) has been used successfully for pain relief,1-3 particularly for diseases of musculoskeletal origin.4,5 TENS has also proved effective for the treatment of angina pectoris,6 dysmenorrhea,7-9 leg ulcers,10 and to control gastric acid secretion.11 In addition there is evidence that electrical stimulation either as electroacupuncture12-15 or TENS16 produces a beneficial effect in patients suffering from strokes.

The mode of action of TENS is still not completely understood. Nevertheless, there is strong evidence that TENS excites Aβ (touch) fibres which pass to the dorsal horn of the spinal cord where they activate interneurons which release the inhibitory neurotransmitter γ-amino butyric acid (GABA).17,18 The latter causes presynaptic inhibition of C-fibre nerve endings and thereby blocks the onward transmission of incoming nociceptive (pain) impulses to the brain. Other mechanisms are probably involved but these have yet to be elucidated.

TENS is normally regarded as a very safe treatment with a low incidence of adverse effects, few of which are serious. Contraindications are also few; nevertheless TENS should be avoided in patients with a pacemaker (especially a demand pacemaker) and electrodes should not be positioned in the anterior triangle of the neck, as stimulation of the carotid sinus nerve may cause acute hypotension.17,19 During pregnancy, it is unwise to place the electrodes over the uterus, except during delivery when TENS has been used to reduce labour pains (electrodes applied to two levels of the lumbar region).20

I believe it is important to report this case because repetitive fits after TENS treatment have not previously been described in the literature, and particularly because TENS therapy is used increasingly in the rehabilitation of stroke patients.

Case History
A 34-year old man consulted the author with a right-sided motor and sensory deficit after an ischemic stroke four months earlier. The deficit was accompanied by spasticity and hyperreflexia. The infarction was within the left posterior frontal, superior temporal and ganglionic regions. Two weeks prior to the consultation, the patient felt faint and had experienced pins and needles in the right side of the face. At the same time he became aware that his speech was slurred. He was admitted to the hospital where he was prescribed sodium valporate 200 mg three times a day.
Initially I treated the paralysed arm using two methods. First I treated it with electroacupuncture (2 Hz) on a weekly basis for 6 weeks; and this was not associated with any adverse effects. The equipment used was IC-1107+, ITO Co. Ltd., Japan. Electrodes were placed on the acupuncture site GB21, located on the top of the trapezius muscle and on the acupuncture point LI4, located in the web between the first and second metacarpal bones. The stimulation was set to create visible muscle contraction without creating pain. The patient was also started on TENS therapy (RDG Tiger Pulse Model 120z) on the paralysed arm for 30 minutes three times a day. The patient was instructed to increase the current until visible muscle contractions occurred in the arm. The stimulator was adjusted to deliver pulsed stimulation in very short bursts of high frequency stimulation (100 Hz) presented at low frequency (2 Hz). The stimulation current was adjusted to between 15 and 30 mA. Self-adhesive electrodes were placed at the same sites as mentioned above.

TENS produced no improvement, so the patient was instructed to increase the treatment periods to one hour four times daily. At the same time, the patient was instructed to increase the strength of stimulation to the highest acceptable level without producing pain. The following day the patient developed a typical tonic/clonic seizure and was admitted to the local hospital where the dosage of sodium valproate was increased to 500 mg twice a day.

On returning home, the patient continued to use TENS treatment with increased duration and strength of stimulation and he developed another epileptic fit. The following day the patient made another attempt to use TENS, which resulted in the third fit. The treatment was abandoned, and he suffered no further fits, a situation which did not change over the following three months.

Discussion
It is not uncommon for patients to develop epileptic fits after a stroke21 and one might argue that the fits reported in this case were coincidental. Against this view is the fact that the patient developed epileptic fits on three consecutive days after TENS treatment, whereas after the treatment had been terminated, no further fits were reported during the following three months. Therefore, it seems more likely than not that in this patient the fits were a direct consequence of the TENS treatment.

This case report raises a number of important questions, particularly the mechanism whereby TENS may have induced the epileptic fits. One could speculate that the release of opioid peptides, for example endorphin, by pulsed TENS (it is not released by continuous TENS) could have interacted in some way with the patient’s anti-epileptic medication. To the best of my knowledge no studies are available to clarify this issue. Moreover, there is no known interaction between morphine and anti-epileptic medication.22

Even a minor stroke damages or destroys a number of neurones in the brain and it seems likely that these changes may increase the sensitivity of other parts of the brain to excitation by various means. For example, it is known that after strokes the sensitivity to temperature may be altered.23 Thus, it seems possible that the sensitivity of neurones to electrical stimulation may also change and this might account for the production of fits in the patient reported here. On the other hand, strokes occur commonly, whereas this is the first time, to the best of my knowledge, that fits associated with TENS have been reported after a stroke. Furthermore, since TENS is used commonly after strokes this further suggests that the phenomenon is not only uncommon but also that post-stroke changes in sensitivity to electrical excitation are also uncommon.

During the first three weeks of treatment the patient used the TENS equipment for half an hour three times a day and during this period no adverse effects were seen. But after increasing both the number of treatments and also the duration and strength of stimulation, it was then that repetitive fits occurred. One might argue that some critical threshold had been exceeded either
due to the prolonged treatment time or to the increased strength of shocks. It seems less likely that the changes were due to the increased treatment time because it is common practice for some patients to use their TENS machines for several hours daily.24

One could argue that the increased strength of shocks was the reason for the fits but this is not very likely because the pain induced would have had a self-limiting effect. Finally, one might argue that the type of stimulation used (pulsed or burst) might have been the trigger factor. Three different forms of TENS stimulation are available:

(i) continuous high frequency/low intensity stimulation (100 Hz);
(ii) continuous low frequency/high intensity stimulation (2 Hz); and
(iii) intermittent pulsed or burst stimulation which utilises both high and low frequencies at high intensity.

Forms (ii) and (iii) are associated with the release of opioid peptides and therefore it seems possible that it was this factor that was associated with the production of repetitive fits.

After cessation of TENS treatment and during the following three months, the patient did not develop further fits. This may have been:

a) as a consequence of cessation of TENS treatment;
b) due to the increased dose of sodium valporate prescribed; or
c) due to coincidence.

On the basis of the arguments advanced above, (a) seems the most likely explanation.

It is puzzling that the phenomenon has not been described previously in the literature and a number of reasons for this may be considered. It is possible that post-stroke fits triggered by TENS occur but with a very low incidence. Alternatively, it is possible that the problem occurs frequently but because about 20% of patients have fits after a stroke,25 those associated with the use of TENS are assumed to be solely the result of the stroke. Thus it is reasonable to speculate that fits in stroke patients may be due to the stroke but might be due to TENS treatment where this has been employed.

Many patients are unconscious after a stroke and the mortality is high. There is evidence that the amount of recovery that follows a stroke may be enhanced by treatment with acupuncture and/or TENS. This paper presents details of a stroke patient in whom TENS probably induced epileptic fits by some mechanism. Whereas post-stroke TENS therapy is used widely, the present case report is, to the best of the author’s knowledge, the first in which it seems likely that strong stimulation with pulsed or burst TENS caused epileptic fits. Therefore, on present evidence the risk of this adverse effect of TENS is likely to be very low but nevertheless should always be borne in mind when TENS therapy is used after a stroke. The problem is worthy of further study.

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I would like to thank Professor JW Thompson, Newcastle, England for constructive comments to the paper.

Reference list


**Commentary**

**Barbro Johansson**

Experimental data have shown that cortical networks adjacent to an area of focal brain ischaemia are hyperexcitable. This is due to an altered ratio between excitation and inhibition, which to a large extent depends upon a reduction of GABAergic inhibition.1 In the mouse, the perifocal hyperexcitability can be demonstrated one to three days after the ligation (onset of ischaemia), and reaches a maximum 28 days after the lesion, but is still seen one year later.2

The patient’s facial paraesthesiae and slurred speech two weeks before the treatment were probably the result of such hyperexcitability. Clearly, the very intense TENS treatment, which is likely to increase the release of excitatory substances, might further alter the balance between excitation and inhibition. The risk might vary depending on the location of the infarct, with cortical infarcts having a higher risk than subcortical infarcts. A recent comparison between acupuncture (including electroacupuncture), low frequency high intensity TENS and a presumed subliminal high frequency TENS did not show any difference in outcome in a randomized controlled multicenter trial on 150 stroke patients.3 In this commentator’s opinion, therefore, very intense TENS treatment should no longer be used.

**Reference List**


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Stimulation (TENS) in a post-stroke patient

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doi: 10.1136/aim.19.1.46

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