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Brief Commentary

Hypodermis Tension Loop: A New Preventative Measure for Lead Migration in the Morbidly Obese

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Electrode migration/displacement is reported to be the most common complication of spinal cord stimulator (SCS) implantation, with the literature reporting incidences from 13.2% to 22.6%. There have been numerous publications describing techniques preventing lead migration, with most involving tying leads to skin and fascia for trial and permanent leads, respectively. However, few have addressed how to prevent migration in the case of hypermobile tissue seen in the morbidly obese. We describe the creation of subcutaneous tension loops to prevent lead migration.

Key words: Spinal cord stimulator, trial, lead migration, subcutaneous tissue, hypodermis, tension loop, morbidly obese

We present a case of a 40-year-old morbidly obese woman, where lead migration was prevented using a novel technique of hypodermal tension loop creation.

Objectives

We demonstrated that the amount of lumbosacral subcutaneous tissue directly contributes to the likelihood of electrode migration during percutaneous trial implantation . The authors describe the first report of a hypodermis tension loop to prevent lead migration in the morbidly obese.

Methods

A 43-year-old morbidly obese woman (BMI > 60) with chronic, intractable, lower back pain (LBP) s/p L3 to S1 laminectomy/fusion and failed back surgery syndrome (FBSS) underwent SCS trial implantation (1). On follow-up imaging, 3 vertebral body lead migration was appreciated despite lead anchoring with anchor/suture, surgical taping, and standard superficial tension loop (2). SCS leads were placed using standard percutaneous technique and sutured to the skin using the Medtronic® anchor system. Final electrode placement was verified using fluoroscopy and paresthesia pattern prior to discharge. The patient experienced low back and leg paresthesias for 5 hours following the procedure, after which lumbar paresthesias decreased and leg/groin increased. Three level lead migration was identified on follow-up examination where superficial anchor-lead complex was unchanged. Repeat stimulator trialing was scheduled due to submaximal pain coverage during initial trial. Electrode migration without lead-anchor disruption suggested subdermal or epidural cause. The authors surmise the focus of the migration occurred within the hypodermis, because it is the most deformable location within the lead tract (1).

A repeat stimulator trial was performed one month after the initial procedure using entry, position, and anchoring techniques indicated above, but a hypodermal tension loop was created to counteract the compressible nature of the subcutaneous tissue (Fig. 1). Two 14-gauge 7-inch standard introductory Medtronic® needles were used to enter the T12/L1 epidural space on opposite sides of the corresponding spinous processes. Sixty cm standard Medtronic ® leads were

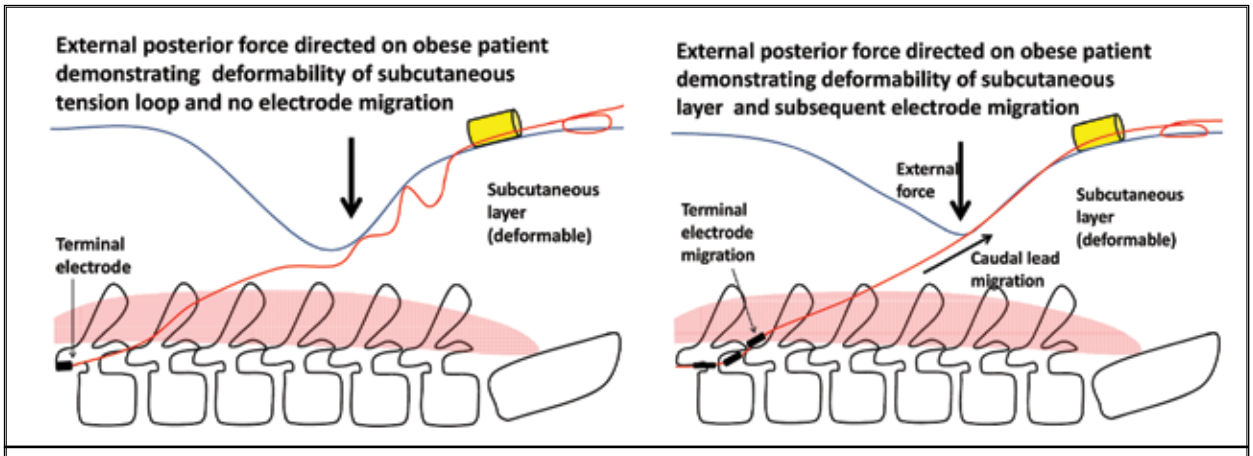

Fig. 1. *Diagram of hypodermal tension loop created to counteract the compressible nature of the subcutaneous tissue.*

introduced to the superior endplate of the T7 vertebral body using a standard approach. Dorsal and midline placement were confirmed. Interrogation produced lumbar and bilateral leg paresthesias. The needles were removed and cables were advanced under low resistance through existing subcutaneous tunnels, while fluoroscopy simultaneously imaged the inferior electrodes and subcutaneous portion of the leads to ensure electrode stabilization. Lead advancement was stopped when medium resistance was felt at the dermal tip of the introducing lead and when subdermal lead buckling occurred. Leads were advanced at approximately 1 mm/sec to prevent subdermal cable kinking which could damage the internal wires. Approximately 2 cm of lead advancement achieved this result (3).

Results

No change in bracing/activity was prescribed relative to the first trial.A 5-day post stimulator trial demonstrated non-appreciable lead migration but did display hypodermal tension loop deformation, suggesting the internal tension loop absorbed external forces (Figs. 2-4). The patient maintained excellent pain relief throughout repeat trial. Subsequently, she underwent successful surgical implant.

Conclusion

In some cases SCS lead migration is due to buckling of dorsal subcutaneous tissue, especially in the morbidly obese. Mainly comprised of loose connective and fatty tissue, this layer is subject to deformation, thus leads that penetrate this space are subject to the same (4,5). Positions that increase force on lumbar soft tissue such as sitting with lumbar support and lying supine are likely contributing factors (6). We demonstrate that creation of a subcutaneous tension loop can prevent lead migration by absorbing the forces on the surrounding tissue.

Discussion

Lead migration typically occurs when there is no intra-spinal lead fixation point and a force is placed on the lead that is greater than the weakest extra-spinal fixation point along the lead track. This is the case in most percutaneous SCS trials and early stage implants (7-10). Subcutaneous tissue is a weak fixation point and we propose that patients who have more of it have inherently less lead stabilizing ability than patients without it. Furthermore, it has been shown that changes in body habitus, such as increased abdominal girth or weight gain following SCS implantation, may cause electrode migration or breakage (11). Published studies have demonstrated a correlation between obesity and SCS complications (12).

In the obese population it is notable to mention that the needle-lead complex must initially pass through substantial subcutaneous tissue, thus making these individuals more prone to lead migration following large spinal movements. Conversely, in patients with low/normal BMIs, the needle-lead complex is passing through far more muscle tissue, which better encapsulates and withstands the same posterior forces of spinal movement that would cause migration. Rigid lumbar bracing is commonly applied post-procedure to prevent lead migration and minimize truncal motion, but it may also limit migration by preventing the buck-

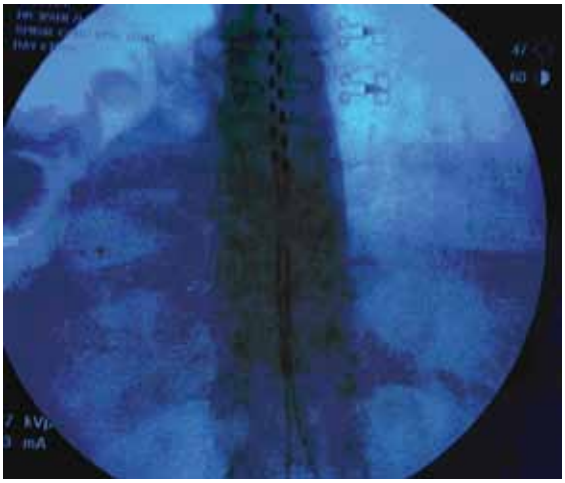

Fig. 2. *Immediate post trial lead positioning with tension loops.*

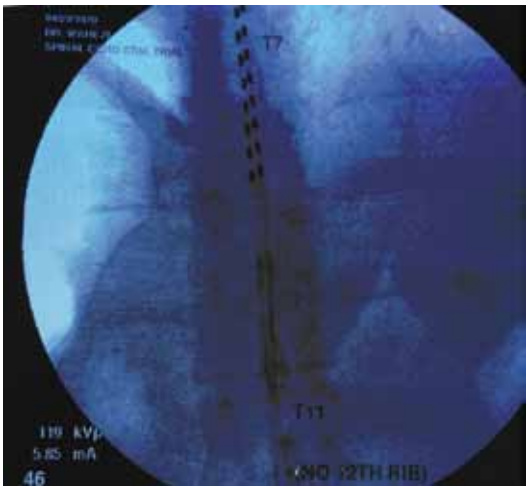

Fig. 3. 5 Day post trial image demonstrating minimal lead migration.

ling of soft tissue by outside forces. This strategy is excellent for lead protection after implantation, but may be a confounding variable during SCS trials. We argue that, in select cases, the creation of a hypodermis tension loop produces subcutaneous lead freedom, minimizing electrode movement. Thus, adding an extra level of diagnostic utility to SCS trials.

Current limitations of this study are that a larger patient population is needed to assess the efficacy of the hypodermis tension loop and its potential to limit electrode migration. Moreover, this new technique may not be applicable to patient populations who have a lower BMI/subcutaneous tissue. The creation of multiple subcutaneous loops increases lead deformation while limiting electrode migration; this deformation, especially at the most mobile segments of the lead, may subject the internal wires to fracture. More trials should be performed to validate this technique. However, in this report, the patient served as their own control, and no changes were made to the trial revision other than the creation of the hypodermal tension loop. This limited variability in practice favors implementation of a hypodermal tension loop in common day practice.

We suggest that addition of this new approach to standard anchoring techniques can benefit practitioners and patients because of its technical simplicity, time and cost effectiveness, and favorable outcomes.

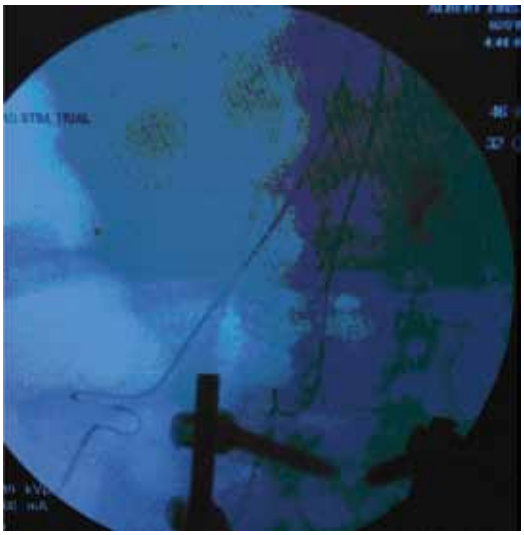

Fig. 4. Demonstration of subcutaneous tension loops.

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