

## The Skeletal Biomechanics of Pressure Ulcer Prevention by Muscle Activation

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### **Abstract**

*Pressure ulcers are common and debilitating wounds that arise when immobilized patients cannot shift their weight. "BION™ Active Seating" aims to prevent pressure ulcers by using chronically implanted, wireless microstimulators (BIONs) to shift a paralyzed subject's weight, and build up gluteal muscle volume and blood circulation. Traditionally gluteus maximus has been considered an important actuator for hip extension to relieve seated pressures via neuromuscular electrical stimulation. We computed the moments of gluteus maximus relative to the sciatic hip extensors (hamstrings), using a model based on anatomical relationships and rigid body mechanics. Surface stimulation experiments were used to validate these predictions and to identify promising stimulation sites and strategies for such treatment. We recognize the value of gluteal stimulation for reducing disuse atrophy and improving circulation, but we demonstrate that it is neither required, nor desired, to achieve hip extension when seated. Instead hamstring stimulation is required, and is capable of providing sufficient hip extension to relieve hydrostatic pressures in the soft tissues under the ischium.*

### **1 Introduction**

Pressure ulcers (PUs) are a debilitating pathology resulting from pressure and shear in the soft tissues of immobilized patients. Blood vessels become occluded and the soft tissues they supply necrose. About 30% of immobile patients develop PUs. The multitude of preventive therapies that exist stand as ironic testament to their own inadequacies. Current treatments require prolonged passive tissue load reduction, often together with surgical repair. Even with these aggressive and expensive treatments, at costs of \$50-80K per incident, recurrence rates are as high as 60% in the first year after surgery [1,2].

Able-bodied people do not get PUs because they can voluntarily contract their muscles, thereby shifting their weight while activating trophic mechanisms that maintain muscle bulk, strength, and circulation. The value of neuromuscular electrical stimulation (NMES) for PU prevention (PUP) has been described [3,4]. Proposed mechanisms include weight shifting to relieve and improve pressure distribution, increasing muscle volume (padding), and increasing vascularity. So why is NMES not in regular use to achieve this in paralyzed patients? One reason is that the neural-electronic interfaces that are currently available are unsuitable for use by most patients. Current modalities involve surface stimulation, percutaneous leads, and implanted stimulators with leads. Surface stimulation is difficult to employ chronically, percutaneous leads are invasive, and implantable stimulators are even more so. Their limitations may have driven researchers to target more accessible, but not necessarily ideal, muscles such as gluteus maximus.

NMES with BIONs (wireless, injectable microstimulators) can be used to activate strong muscle contractions and produce skeletal motion, with associated increases in muscle bulk (hypertrophy), strength, and metabolic capacity; hence counteracting the three major etiological factors of PUs (immobility, soft-tissue atrophy, and hypoxia) [5]. We plan to implant BIONs alongside the inferior gluteal and proximal sciatic nerves in patients who are already undergoing gluteal rotation flap surgery for PU reconstruction; offering us control of the gluteus maximus and hamstring hip extensors respectively. We have modeled the skeletal biomechanics of this system to quantify the value of gluteus maximus (GM) stimulation relative to sciatic extensor (SE) / hamstring stimulation, and have used surface stimulation to validate our findings and identify promising stimulation sites and strategies for such treatment.

## 2 Methods

### 2.1 Modeling Of Skeletal Biomechanics:

**Rigid Body Mass Analysis:** The biomechanics of selective muscle stimulation in a paralyzed system have been reconsidered from first principles using rigid body analysis (MSC. visualNastran 4D, MSC Software, CA). A unilateral hip torque motor was set at 63Nm to model isolated hip extension. This was derived assuming the knee was fixed at the seat edge, weight was evenly distributed, and aiming for a scenario of extreme unloading (with the trunk raising 30° in 0.5s, and taking 0.2s to attain this angular velocity). The value correlates well with experimentally derived torques [6].

**Muscle Moment Analysis:** Surface stimulation experiments (2.2 and 3.2 below) demonstrated that SE stimulation decreased ischial pressure by 26%, but GM stimulation *increased* ischial pressure by 16% [7]. Two questions arose: 1) was the traditional reliance on, and inclusion of, GM stimulation the most appropriate means to achieve PUP?; and 2) if one wished to avoid the increased pressures associated with GM stimulation, could SE stimulation alone provide sufficient torque to adequately shift weight? Relative hip extensor capabilities of SE and GM groups were compared, using anatomically accurate models of muscle origin/insertion and wrapping in SIMM (MusculoGraphics, Inc.,

CA) (Table 1). The model's hips and knees were flexed to 90°, with thighs neutral at 0° of external rotation and 0° of adduction. Force (N) and moment arm (m) data were collected for each muscle across its full range of motion (ROM) in flexion-extension, internal-external rotation and abduction-adduction, to derive the torque / moment data (Nm) for each at maximal activation. The moments of muscles in each group were summed to plot GM total moment and SE total moment vs. ROM in each axis.

### 2.2 Surface Stimulation Validation

Transcutaneous stimulation was employed to validate the modeling data described above. Surface electrodes were used to stimulate GM, SE and quadriceps in 2 studies totaling 11 normal subjects, as described elsewhere [7].

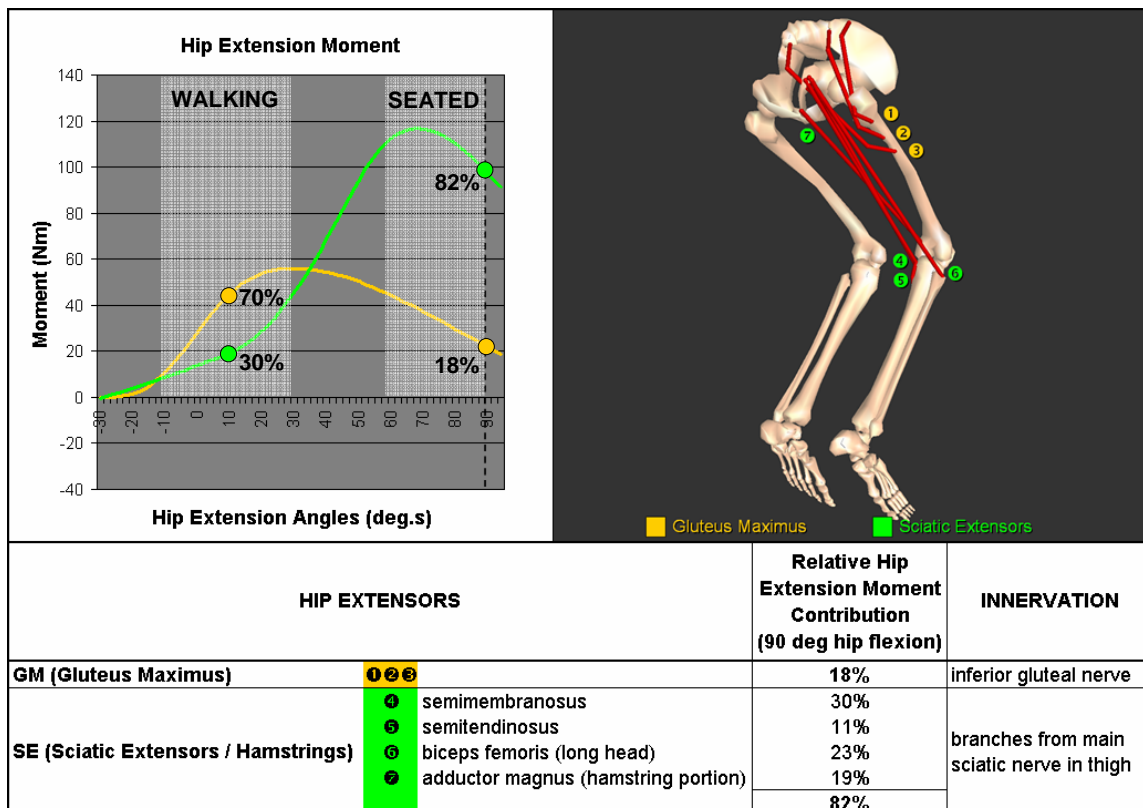
## 3 Results

### 3.1 Modeling Results:

**Rigid Body Mass Analysis:** The model indicated that our calculated torque requirement of 63Nm for an extreme unloading scenario should sufficiently achieve such unloading, and also suggested that pressure relief would be achieved not only ipsilaterally, but to some extent contralaterally too.

**Muscle Moment Analysis:** Data in Table 1 confirm the dominant contribution of GM to hip

Table 1: Hip Extensors and their Innervation [8].



extension during walking when the hip is in a relatively extended position (+30° to -10°) [9]. However, when seated at 90° of hip flexion, over 80% of the extension force originates from SE, and not from GM stimulation (vs. only 30% at 10° of hip flexion); this relationship persists over as much as 20° of hip extension / unloading. Over the range of seated postures, SE stimulation provides about four times more extension torque than GM stimulation (~100-120Nm vs. ~20-40Nm).

### 3.2 Surface Stimulation Results

SE stimulation produced a significant reduction in seated interface pressures as our models predicted (-26% ipsilaterally), together with a mean reduction in total contact area of 25%. GM stimulation actually increased the recorded seating pressures +16% ipsilaterally, with less reduction in contact surface area (mean -10%). The distribution of pressures suggests that the bulging of the stimulated muscle served to concentrate seating pressure in a smaller region ipsilaterally, even slightly off-loading the contralateral buttock (-4%). Conversely, SE stimulation reduced seating pressures both ipsilaterally and contralaterally (-8%) while increasing pressure under the distal thigh. Such weight shifts to the thighs are indicative of successful unloading at the buttocks, and are of little concern. The distal thighs have no bony prominences, are not at risk for PUs, and are the normal areas to which mobile individuals transfer body weight.

### 4 Discussion and Conclusions

We have shown that, seated, SE stimulation provides 82% of the total hip extension torques and decreases ischial pressure by 26%, while GM stimulation provides only 18% of hip extension torque while increasing ischial pressure by 16%. These biomechanical relationships appear to have been overlooked when proposing actuators for weight-shifting. Further, it is reassuring to note that SE stimulation alone will provide sufficient extension torque to adequately unload the buttock tissues (provides ~100Nm while only ~60Nm is required for complete unloading). Using GM stimulation for extension seems likely to produce large moments in abduction (9.9Nm) and external rotation (7.1Nm) compared to the extensor moment (21.7Nm). SE stimulation offers a much larger extensor moment (97.3Nm) with only a small degree of internal rotation (2.6Nm), but with a relatively

large adduction moment (52.2Nm). Adduction is less of a mechanical problem because it can be stopped in the midline by the contralateral limb, perhaps with commercially available foam spacers to avoid abrasions.

These data suggest that even tetanic activation of a healthy GM muscle is unlikely to provide sufficient hip extension to elevate the ischium and reduce seated pressures sufficiently to avoid hypoxic damage. Disuse atrophy in chronic patients will further reduce the efficacy of this strategy. GM stimulation is likely, however, to reduce disuse atrophy and improve circulation, and this mechanism may account for the favorable results reported clinically for GM stimulation. GM and SE stimulation seem likely to be useful for muscle conditioning when the patient is non-weightbearing. In order to produce hip extension while seated, GM stimulation is neither required, nor desired. Selective stimulation of SE should achieve pressure relief under both ischiae in the seated patient, but it remains to be demonstrated if this can be achieved without adverse mechanical, sensory or reflexive effects in the various patients who suffer from PUs.

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### Acknowledgements

This research has been funded predominantly by the Alfred E. Mann Institute at The University of Southern California, Los Angeles, CA 90089, USA; and in part by the National Science Foundation through the Engineering Research Center for Biomimetic MicroElectronic Systems.