DRG Stimulation: Evolution or Revolution in Neurostimulation?

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Introduction:

> What is Neurostimulation?
> The use of pulsed electrical energy to control pain
> At the beginning it included things like:

1882: The “Faradic Electrifier,”

Late 1800s: The Gaiffe TENS unit

1919: The Electreat

Eventually included:

> Peripheral nerve and deep brain stimulators, TENS units
> Traditional (Tonic) Spinal Cord Stimulator (SCS), High-frequency (HF) SCS, Burst SCS and Dorsal Root Ganglion Stimulators (DRG)
How Does it Work?

- Conventional/tonic SCS:
  - 1965: Gate Control theory
    - Mid 1960s: Peripheral nerve stimulators
    - 1972: Dorsal column neurostimulators were first marketed to neurosurgeons in the United States
  - Favorably alters the local neurochemistry → suppresses the hyperexcitability of wide dynamic range interneurons
  - Activation of supraspinal circuitry
  - Restoration of a favorable oxygen supply and demand balance
Programming - SCS/DRG

- **Amplitude**
  - Intensity/strength of each individual pulse
  - Controlled by voltage (V) or current (ohms)

- **Pulse width**
  - Duration of a pulse (msec)

- **Rate (frequency)**
  - Pulses per second (Hz)

- **Electrode selection**
  - Complex topic for **Tonic**, less complex for **HF**

2015 Burst FDA Approval:

2015 HF FDA Approval:

2016 DRG FDA Approval:

<table>
<thead>
<tr>
<th>Programming (very rough numbers)</th>
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<td>Amplitude mA</td>
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<td>Burst</td>
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<td>HF</td>
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<td>DRG</td>
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How Do the High-Density Patterns Work?

- **HF**
  - Multiple hypotheses

- **Burst**
  - 1970s - BURST TENS existed.
    - Was applied more for nociceptive pain types.
  - Was developed to mimic nature
  - Additionally, works on the medial spinothalamic pathway

- **DRG**
  - Works on the DRG (first-order sensory neuron)
Major Studies

- 2008: PROCESS Study (18)
- 2010: Burst SCS: Toward Paresthesia-Free Pain Suppression (19)
- 2013: Burst SCS for Limb and Back Pain (15)
- 2015: Tonic versus HF SCS: Response to HF in Tonic Non-responders (10)
- July 2016: SENZA-RCT 24mos follow-up (7)
- March 2017: Burst or HF (10 kHz) SCS in FBSS Patients With Predominant Back Pain: One Year Comparative Data. (5)
- Apr. 2017: ACCURATE Trial (2)
- Sept. 2017: SUNBURST Trial (17)
PROCESS Study (18)

- Prospective, Multicenter RCT
- Compared Tonic SCS + Conventional Medical Management (CMM) and CMM alone
- FBSS over 24 months
- Tonic SCS + CMM (52 assigned → 42 @24mos)
- Primary Outcome: >50% leg pain relief
  - 48% of Tonic SCS subjects [using SCS as final therapy]
- Secondary Outcomes at 2 years:
  - No statistically significant improvement in back pain relief
  - Improved health-related QOL, and functional capacity over CMM only.
- Conclusions:
  - Tonic SCS were effective for neuropathic radicular pain
  - Are they only effective for neuropathic radicular pain?
Burst SCS: Toward Paresthesia-Free Pain Suppression (19)

- SCS electrodes implanted for neuropathic pain in 12 patients via laminectomy
- During external stimulation, the patients received BOTH:
  - **TONIC**
    - VAS
      - $t = 0$
      - Axial: 4.42 (improvement of 1.83)
      - Limb: 3.13 (improvement of 4.41)
    - Paresthesias: 92% - relatively stable at 1 year.
  - **BURST**
    - VAS
      - $t = 0$
      - Axial: 1 (improvement of 5.25)
      - Limb: 0.25 (improvement of 7.29)
    - $t > 1$ year (average follow-up 20.5mos)
      - Axial: (improvement of 3.7)
      - Limb: (improvement of 5.15)
    - Paresthesias: 17% - relatively stable

- Compared to **Tonic**: ↓Paresthesias, ↑Analgesia (including axial pain), Better Sensory and Affective scores on McGill Short Form.
- The effects were sustained.
Burst for Limb & Back Pain (15)

- Randomized, placebo-controlled trial comparing burst (x1 week), tonic (x1 week) and placebo (x1 week)
- 15 consecutive pts. Primarily FBSS. Paddles placed via laminectomy.
- Primary Outcomes:

  - **Burst**
    - %Improvement of VAS Overall: 55%
    - %Improvement of VAS Back: 51%
    - %Improvement of VAS Limb: 53%
  
  - **Tonic**
    - %Improvement of VAS Overall: 30.9%
    - %Improvement of VAS Back: 30.3%
    - %Improvement of VAS Limb: 51.5%
  
  - **Placebo**
    - %Improvement of VAS Overall: 10.9%
    - %Improvement of VAS Back: 18.9%
    - %Improvement of VAS Limb: 11.7%

- Secondary outcomes

  - Pain Vigilance Questionnaire and Awareness Questionnaire
    - Burst improved attention to pain and pain changes. Tonic and placebo worsened these measurements.
  
  - Encephalogram:
    - Burst stimulation activates the dorsal anterior cingulate and right dorsolateral prefrontal cortex more than tonic stimulation.
Tonic vs Burst: Responders & Suppression (14)

- 2 centers (1 Belgium, 1 Netherlands), Retrospective, 102 pts.
- All neuropathic pain - mostly related to FBSS or DMPN
- 1st group = Tonic failures (24), 2nd group = Tonic responders (78)
- Switched them all to Burst
- Overall NRS: Baseline 7.8 → Tonic 4.9 → Burst 3.2
- Tonic SCS Failures
  - 62.5% responded to Burst → 43% pain suppression
- Tonic SCS Responders
  - 50.6% suppression w/ Tonic → 73.63% suppression with Burst
  - 94.87% had a better effect on burst suppression
- Conclusions:
  - If a pt failed Tonic, there’s a good chance they might still respond to burst
Tonic vs HF: Responders & Suppression (10)

- Prospective RCT
- 22 (primarily FBSS) non-responders to Tonic
- Randomized to 1kHz stim (HF) or Tonic for 3 weeks → 1 week washout → switched to the other programming for 3 weeks.

Results
- 95% (21) improved with HF (average NPRS reduction = 2.41)
- 41% (9) improved with Tonic (average NPRS reduction = 0.32)

Conclusions:
- If a pt failed Tonic, there’s a good chance they might still respond to HF
**SENZA Trial (7)**

- Prospective RCT w/ 24-month follow-up.
- Comparison of **HF** and **Tonic** SCS
  - For chronic BACK and LEG pain
    - Pre-trial VAS >5 for both back and leg pain
- Assigned randomly (1:1) to receive:
  - HF (101→trial→90)
  - Tonic (97→trial→81)
- Responder rate (defined as >50% back pain reduction from baseline):
  - **3 months:**
    - HF
      - 84.5% for back pain
      - 83.1% for leg pain
    - Tonic
      - 43.8% for back pain
      - 55.5% for leg pain
  - **24-months**
    - HF
      - 76.5% for back pain
      - 72.9% for leg pain
    - Tonic
      - 49.3% for back pain
      - 49.3% for leg pain
SENZA Trial (7) - Cont’d

- Also, less disability and increased satisfaction with HF

Conclusion:
- Long-term superiority of HF to Tonic
- No Paresthesias
- Worked for back pain & leg pain
HF vs Burst (5)

- Prospective, single-center
- Comparison of Burst and HF - f/up 3-14mos
  - FBSS w/ >/=70% back pain +/- leg pain
  - Assigned in alternating non-randomized fashion (1:1) to receive:
    - HF (8→trial→6) - Percutaneous leads
    - Burst (8→trial→8) - Paddle leads via laminectomy

**VAS-Back (***)**
- HF: 8 (+/-0.63) to 3.5 (+/-3.27)
- Burst: 8 (+/-0.76) to 1 (+/-1.41)

**VAS-Leg**
- HF: VAS-leg not reported
- Burst: Increased 18% from baseline
  - VAS-leg 3.6 to 1.5
  - Decreased 50.2% from baseline

**BDI (***)**
- Baseline -100% had moderate depression (BDI 20-29)
  - HF: 33% had improvement from moderate to minimal.
  - Burst: 75% had improvement from moderate to minimal.

**PSQI (***)**
- Baseline: 93% had PSQI score >5 (poor sleep quality)
  - HF: 50% PSQI <5
  - Burst: 88% PSQI <5

*** = NOT statistically significant!
HF vs Burst (5) - Cont’d

Conclusions:

- Burst (*w/ paddle leads) and HF (*w/ percutaneous leads) are effective with a slight superiority for subjects using burst (*w/ paddle leads) in a small sample study in patients w/ FBSS
  - Statistically significant:
    - Burst > HF reduction in leg pain
  - Non-statistically significant
    - Burst > HF
      - Reduction in back pain
      - Improvement in depression
      - Improvement in sleep
  - Burst may be more stable with time (paddle leads?) - although SENZA-RCT seems to indicate that HF is also stable with time
ACCURATE Trial (2)

- Prospective, multicenter, randomized comparative effectiveness trial over 12 mos
- Comparison of DRG vs Tonic
  - Lower extremity CRPS 1 or 2

Assigned randomly (1:1) to receive:
- DRG (73→trial→61)
- Tonic (73→trial→54)

- Responder rate (>50% pain reduction)
  - 3 months
    - DRG - 81.2%
    - Tonic - 55.7%
  - 12 months
    - DRG - 74.2%
    - Tonic - 53.0%
- DRG
  - Greater improvements in quality of life
  - Greater improvements in psychological disposition.
  - Less postural variation in paresthesia,
  - Reduced extraneous stimulation in nonpainful areas
ACCURATE Trial (2) - Cont’d

- Paresthesias
  - 3mos
    - DRG: 15.3% (Just in their pain site: 84.7%)
    - Tonic: 35.2% (Just in their pain site: 64.8%)
  - 12mos
    - DRG 5.5% (Just in their pain site: 94.5%)
    - Tonic 38.8% (Just in their pain site: 61.2%)

- No difference in device-related and serious adverse events

- Conclusions
  - More targeted therapy (if they have paresthesias - likely just at the pain site)
  - Greater improvement in QOL, psychological disposition
Prospective, randomized multicenter study.

Successful tonic trial → 100 subjects were randomized to Burst or Tonic for the first 12 weeks, and then the other stimulation mode for the next 12 weeks.

Subjects then used their choice of Burst or Tonic and were followed for one year.

- 70.8% preferred Burst over Tonic (p < 0.001).
- Preference was sustained through one year:
  - 68.2% preferred burst
  - 23.9% preferred tonic
  - 8.0% had no preference.

Conclusions: 23.9% preferred Tonic (because of paresthesias?)
Burst - Advantages vs Limitations

Advantages:

- More stable with time than HF (2017 HF vs Burst)
  - Contradicted by 2016 SENZA-RCT
- Likely better for nociceptive pain than tonic and possibly HF (2017 HF vs Burst)
  - Contradicted for HF comparison by 2016 SENZA-RCT
- Subparesthetic (compared to Tonic)
  - ~17% with paresthesias
- Some pts like paresthesias (more versatility in reprogramming with burst)
- Paddle and percutaneous leads are available
- Improved mood and sleep (vs HF and possibly DRG)
  - Not statistically significant but there is a physiologic mechanism for it

Limitations:

- More paresthesias than HF (17% paresthesias vs none)
- Higher amplitude signal required than DRG
- High current demand.
- Presumably:
  - More positional variations in stimulation than DRG
HF - Advantages vs Limitations

Advantages:
- Seems to be stable with time (SENZA Trial)
- Works well for nociceptive and neuropathic pain (SENZA Trial)
- No paresthesias

Limitations:
- Very limited programming
- Higher amplitude signal required than DRG
- Very high current demand - need rechargeable pulse generators; May need daily recharging.
- No paddle lead available
- Trend towards less improvement in mood/sleep (than Burst)

Presumably:
- More positional variations in stimulation than DRG
DRG - Advantages vs Limitations

- **Advantages:**
  - Theoretically could be more effective in historically challenging pain targets (w/ **Tonic**) such as the foot or chest wall
    - Dorsal CSF diameter is largest at T5. Spinal nerves at the end of the spinal cord float freely with the CSF.
    - Whereas **HF** and **Burst** (like Tonic) theoretically may be ineffective
  - Much more likely to report paresthesias just in their painful site
  - Less energy requirements
  - Less postural instability
    - Limited mobility of the **DRG** relative to the spinal cord, ↓CSF volume at the **DRG**

- **Limitations:**
  - 1 lead per 1 **Dorsal Root Ganglion** limits the applicability
  - 5%-15% paresthesias (ACCURATE Trial)
  - No paddle leads
  - No strong evidence that it covers nociceptive pain
  - Longer procedure (107mins vs 76mins - ACCURATE Trial), more leads
    - More non-serious procedure-related AEs in the **DRG** arm (46% vs 26% - statistically significant)
    - Significantly more fluoroscopy time
Conclusions at this Point?

- **Burst** and **HF** are superior to **Tonic**
  - And if **tonic** fails it’s reasonable to try **Burst** or **HF**
- **DRG** is superior to **Tonic** for neuropathic pain that covers a limited number of dermatomes
- **Burst** and **HF** cover nociceptive pain well (better than **Tonic**)
- Some patients like paresthesias
Other Factors in Choosing Neurostimulation

- Features of the battery/energy usage?
  - Chargeable vs rechargeable?
  - How often do you have to recharge?
- MRI-safe?
- Do you need paddle leads?
- Any additional features?
  - Activity tracker
- Support staff
  - Quality of the reps
- Size of the implantable
The Future

- More head-to-head studies
- Efficacy in different neuropathic and nociceptive conditions
- Efficacy in different regions of the body
- What factors predict who will respond better to HF vs Burst vs DRG or even tonic?
- Better programming
- Cost-effectiveness
- Mechanisms of action - not well understood
References - Page 1


References - Pictures/Diagrams/Tables

- Pictures 1,2,3,4 from
  - http://www.burtonreport.com/infspine/nhistneurostimparti.htm

- Picture 5 and 11 from

- Picture 6,7,8,9,12 adapted from:
DRG
Target for Neurostimulation

- DRG contain cell bodies of primary sensory neurons
- DRG Type A: touch, proprioception, vibration
  - large
- DRG Type B: nociception
  - small
- Participate in signaling process
- sense and manufacture molecules
- modulate sensory processing
The Dorsal Root Ganglion in Chronic Pain and as a Target for Neuromodulation: A Review

Neuromodulation: Technology at the Neural Interface
29 OCT 2014 DOI: 10.1111/ner.12247
DRG Anatomy

- Sensory root connect to cell bodies via T junction
- DRG sits in neural foramen
- Surrounded by Glial Cells
- Neurons in DRG have receptors for neurotransmitters
The Dorsal Root Ganglion in Chronic Pain and as a Target for Neuromodulation: A Review

a. Pseudounipolar neuron

b. Unipolar neuron
The Dorsal Root Ganglion in Chronic Pain and as a Target for Neuromodulation: A Review

T-junction acting as a barrier to AP propagation to DH

T-junction acting as a low pass filter of AP propagation to DH

T-junction acting as a propagator of APs

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The Dorsal Root Ganglion in Chronic Pain and as a Target for Neuromodulation: A Review
DRG Anatomy

- Surrounded by Satellite Glial Cells
- Receptors to various neuroactive chemicals
  - e.g. bradykinin, ATP, cytokines
- Respond to environmental change
- Probable participation in signal processing and transmission in DRG
- ? Role in neuropathic pain
- morphological and biochemical change after nerve injury
DRG and Neuropathic Pain

- Injured DRG neurons may become hyperexcitable
- Injured Peripheral afferent fibers can lead to hyperexcitability in axotomized DRG neurons
- With injured PAF electrical impulses may originate in DRG and can be blocked with lidocaine
- Na channels
- Neuropathic Pain may involve not just PAF injury but involvement of SGC, Schwann cells, spinal microglia, peripheral immune system
- Ectopic DRG firing may lead to central sensitization and allodynia
DRG and Gene Expression

- Up regulation of neuropeptides, receptors, ion channels, signal transduction molecules, synaptic vesicle protein etc seen in DRG after peripheral nerve injury consistent with changes in gene expression in animal model.
- May alter function of cell body
- May play role in development of neuropathic pain
DRG and Ion Channels

- DRG express several Sodium Channels
- Channels may change expression and gating properties after PAF injury. May lead to spontaneous activity and burst firing, the electrical signature of neuropathic pain
- Suggested that electrical stimulation may alter sodium channel gene expression. ? Return to normal
DRG and Ion Channels

- Potassium Channels
- PAF injury can lead to reduction in K channel subunit expression in DRG neurons, probable role in hyperexcitability
**DRG and Ion Channels**

- Calcium
  - Increased Ca currents in periphery may lower nociceptive threshold
  - PAF injury may lead to decrease in Ca currents in primary afferents
  - Inflammation may alter Ca channels in DRG (rat model)
- Electrical stimulation of DRG may modify burst and tonic activity in pseudopolar cells
- Electrical field stimulation of DRG increases Ca influx and reduces multiple action potential frequency and conduction velocity; neuronal excitability
Role of DRG in Pain

- DRG not a passive conduit for afferent transmission
- Cellular changes can occur in DRG with PAF injury
Electrical Stimulation of DRG

- Possible impact of stimulation on growth factors in DRG which can impact the development of neuropathic pain
- Also stimulation may impact immune system response to PAF injury and therefore development of chronic pain
- Stimulation may lead to modification of tonic and bursting neurons in DRG which may be abnormal with PAF injury
Hypothesized Mechanisms of Action of Dorsal Root Ganglion Stimulation

- **Modification of growth factor release**  Release of abnormal growth factors and inhibition of normally produced growth factors within the DRG resulting from PAF injury may be modified by direct or indirect electrical stimulation of the DRG, resulting in a decrease in chronic pain.

- **Reversal of cytokine release**  We have seen that PAF fiber injury leads to activation of microglia within the DRG, which, in turn, leads to a cytokine cascade. This cytokine cascade leads to inflammation and neuropathic pain. Electrical stimulation of the DRG reverses activation of microglia within the DRG, thereby reversing the abnormal cytokine cascade that leads to the development of neuropathic pain.

- **Downstream and upstream effects**  It is proposed that electrical stimulation of the DRG results in chronic pain relief through its downstream effects of vasodilation and stabilizing the sensitized peripheral nociceptors and its upstream effects of deactivating sensitized wide-dynamic-range neurons within the spinal cord and turning off brain centers that are turned on by PAF injury and inflammation.
Hypothesized Mechanisms of Action of Dorsal Root Ganglion Stimulation

- Rectification of electrical activity patterns: It is proposed that electrical stimulation of the DRG may alter abnormal patterns of electrical activity of DRG neurons resulting from PAF injury, thereby decreasing chronic pain.

- Reversal of genetic changes: It is proposed that electrical stimulation of the DRG reverses nonphysiological early and late genetic changes that result from PAF injury.
Hypothesized Mechanisms of Action of Dorsal Root Ganglion Stimulation

- Down-regulation of abnormal ion channels and restitution of normal ion flux We have also seen that there are changes to Na+, K+, and Ca++ ion channels and ion current flux resulting from injury and inflammation that lead to increased excitability of the periphery, the DRG, and the spinal cord. We have also seen that electrical stimulation has effects on the immune system, Ca++ currents, and Na+ channels. Therefore, it is proposed that electrical stimulation of the DRG reverses the production of abnormal Na+, K+, and Ca++ channels and reverses abnormal flux of Ca++ ions.

- Filtering of electrical impulses The T-junction of the DRG neuron can either act as an impediment to electrical impulses from the nociceptor to the dorsal root entry zone, participate in the propagation of the electrical pulse, or act as a low-pass filter to electrical information from the periphery.
DRG Stimulation
Animal

- Ganglionic Field Stimulation to rat DRGs
- Measured by action potential #, conduction velocity, AP propagation failure measured pre and post GFS
- GFS reduced neuronal excitability demonstrated with reduction in # neurons which could generate AP or multiple AP and a reduction in conduction velocity

Koopmeiners et al Neuromodulation 2013
DRG Stimulation
Clinical

- Deer et al 2013
- N = 10
- Trunk and limb pain
- Trial DRG stimulation
- f/u 3 to 7 days
- Pain reduction, improvement, medication use, adverse effects
A Prospective Study of Dorsal Root Ganglion Stimulation for the Relief of Chronic Pain
A Prospective Study of Dorsal Root Ganglion Stimulation for the Relief of Chronic Pain

Overall VAS Pain Scores

Baseline  Peak Cp  FU1  FU2  FU3

0  10  20  30  40  50  60  70  80

VAS Score (mm)
A Prospective Study of Dorsal Root Ganglion Stimulation for the Relief of Chronic Pain
A Prospective Study of Dorsal Root Ganglion Stimulation for the Relief of Chronic Pain

- Results
  - average 70% reduction in VAS
  - 8/9 patients >30% pain reduction
  - 7/9 reduced pain medication
  - No adverse effects
A Multicenter, Prospective Trial to Assess the Safety and Performance of the Spinal Modulation Dorsal Root Ganglion Neurostimulator System in the Treatment of Chronic Pain

- Liem et al 2013 Neuromodulation
- N = 32
- 6 month f/u
- Pain rating reduced by 58%
- 50% or greater pain reduction
- 57% back pain, 70% leg, 89% foot
- Discontinuation of DRG stimulation led to return of pain
- Parethesia did not change with change in position
A Multicenter, Prospective Trial to Assess the Safety and Performance of the Spinal Modulation Dorsal Root Ganglion Neurostimulator System in the Treatment of Chronic Pain
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Neuromodulation: Technology at the Neural Interface
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Neuromodulation: Technology at the Neural Interface

A Multicenter, Prospective Trial to Assess the Safety and Performance of the Spinal Modulation Dorsal Root Ganglion Neurostimulator System in the Treatment of Chronic Pain

Neuromodulation: Technology at the Neural Interface
One-Year Outcomes of Spinal Cord Stimulation of the Dorsal Root Ganglion in the Treatment of Chronic Neuropathic Pain

Authors

Liong Liem MD et al 21 August 2014

- N = 51
- 76.5% good outcome with average pain relief 74%
- Nonresponders average pain relief 5%
One-Year Outcomes of Spinal Cord Stimulation of the Dorsal Root Ganglion in the Treatment of Chronic Neuropathic Pain

Neuromodulation: Technology at the Neural Interface
21 AUG 2014 DOI: 10.1111/ner.12228
One-Year Outcomes of Spinal Cord Stimulation of the Dorsal Root Ganglion in the Treatment of Chronic Neuropathic Pain
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![Bar chart showing outcomes of spinal cord stimulation]

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One-Year Outcomes of Spinal Cord Stimulation of the Dorsal Root Ganglion in the Treatment of Chronic Neuropathic Pain

![Graph showing changes in pain levels over time.](http://onlinelibrary.wiley.com/doi/10.1111/ner.12228/full#ner12228-fig-0005)
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Liong Liem MD et al 21 August 2014

- Adverse Events
- N = 86
- Temporary motor stimulation n = 12
- CSF leak, headache n = 7, infection n = 7
- Lead revision 4
- Explants 7
Questions ?