I HAVE NO PERSONAL FINANCIAL INTEREST IN ANYTHING I WILL BE PRESENTING
THE CALMARE PAIN SCRAMBLER
3 months (anecdotal) experience treating CORNEAL NEUROPATHIC PAIN

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WHY SHOULD NON-OPHTHALMOLOGISTS BE INTERESTED IN CORNEAL PAIN?

- What happens in the cornea is likely to be mimic neuropathic pain elsewhere.
- The cornea is transparent and accessible to in-vivo confocal microscopy and the imminent introduction of technology that resolves individual cells in the living human cornea.
- It is accessible to topical treatments with minimal systemic spillover.
- Metrics of the results of treatments can be monitored at more fundamental levels.
CORNEAL NOCICEPTOR

- density is 20+ fold greater than in dental pulp
- vulnerable to being exposed to noxious environmental stimuli

THE CORNEA IS THE MOST POWERFUL PAIN-GENERATOR IN THE BODY
AXONOPATHY

SPONTANEOUS (ECTOPIC) PAIN
THE LIQUID CORNEAL BANDAGE

THE COHORT

- Patients with severe refractory corneal neuropathic pain (bathing the cornea in solutions of sub-hypoesthetic concentrations of local anesthetic was ineffective)
- Many were dependent on systemic prescription analgesics

CORNEAL-PROJECTED PAIN ORIGINATING IN THE CENTRAL PAIN PATHWAYS
THE CORNEAL PAIN SIGNALING PATHWAY

A) Physiologic Corneal Pain

- Terminal nerve damage on corneal surface
- To paralimbic region and somatosensory cortex
- Third order neuron

B) Axotomy

- Ascending corneal nociceptive projections
  - Ipsilateral
  - Contralateral
- Descending pain modulation
- Potential sites for nociceptive signal generation or amplification

FIRST ORDER NEURON
SECOND ORDER NEURON
THIRD ORDER NEURON
Neuron crossing over
Trigeminal ganglion
Pons
Periaqueductal gray (PAG)
Thalamus
Amygdala
Trigeminal subnucleus caudalis
54 yr. old American missionary nurse working in Mindanao for 17 years developed unexplained severe unremitting burning corneal pain triggered by severe photophobia OU despite slit lamp-normal eyes. Symptoms included aching head and facial pain that were refractory to topical sodium channel modulators. Health was excellent. Autoimmune markers absent.
CORNEAL NERVES

Patient’s nerves

age matched normal

Cornea Sequence [11], 6/16/2009, OS
#92 / 100: 34 μm

Cornea Sequence [5], 8/1/2008, OS
#40 / 100: 36 μm
62 yo woman with EMS since 1988 developed dry eye-like corneal pain in 2004 that rapidly progressed to level 7-10+ in both eyes and developed burning oral mucosal pain associated with eye pain.

Rx Tegretol 800mg, Lyrica 400 mg., morphine

Scrambler Rx X7 (excellent initial response) followed by 3 pain-free weeks off meds (except morphine). Subsequently resumed meds because of pain recurrence. Pain level too low for Scrambler treatment.

CONCLUSION (n=1): Reduce systemic analgesics to zero if possible by the time of the initial treatment or during treatment. Resume after last treatment.
WHAT DOES THE PAIN SCRAMBLER TARGET?

(A) Physiologic Corneal Pain

- Terminal nerve damage on corneal surface
- To Paralimbic region and Somatosensory cortex
- Ascending corneal nociceptive projections
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- Descending pain modulation

(B) Axotomy

- Potential sites for nociceptive signal generation or amplification
QUESTIONS?

Temporarily reverses maladaptive plasticity
(level of hyperexcitability)

- What determines the durability of its effect?
- What determines its effectiveness, immediate, short-term and intermediate term?
- What is the optimal interval between treatments
  - Initial series?
  - Booster treatments?
- How can we enhance the durability of the effect?
  - Earlier intervention?
  - Is location a variable?
  - Strategy of concurrent systemic anti-nociceptive drug treatment?
CONCLUSIONS

The pain scrambler can be life-changing (and life-saving) for some patients with the most devastating, intractable neuropathic pain involving the innervation territory of the trigeminal nerve.

Clinical experience may advance its utility still further for these patients.