A Case Report: Block Density and its Relationship to Phantom Limb Pain

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Introduction
In 2005, there were an estimated 1.6 million people with limb loss in United States. Of these patients, the incidence of phantom limb pain (PLP) is estimated to be 60-80% (Chaine, 2007). PLP can occur after the amputation of limbs, breast, digit, eye, tongue, nose, teeth, genitals, anus, and ear.

Risk Factors for PLP
- Pre-amputation pain
- Presence of stump pain
- Depression
- Proximal amputation
- Lower limb amputation, Loss of dominant upper limb

Proposed Mechanism of PLP
- Peripheral mechanisms: Stump and neroma hyperactivity
- Central neural mechanisms: Spinal cord sensitization and changes, cortical reorganization and cortical-motor sensory dissociation
- Psychogenic mechanism

Case: 66-year-old gentleman with right ankle osteomyelitis underwent a right below knee amputation

PMHs: Type 2 diabetes, Peripheral Vascular Disease, Congestive Heart Failure, Hypertension, Depression

Septic arthritis and osteomyelitis

Pre-operatively:
Limb pain poorly controlled; Pain = 7/10 VAS.

Limbar Epidural placed immediately pre-operatively
Epidural bolused: 2 x 12ml 2% lidocaine + 1/200k epi

Intra-operatively:
Epidural bolused: 6ml 0.5% bupivacaine + 1/200k epi

Central changes
- Unrinking
- Spraying
- Generalized allodynia
- Vascular remodeling
- Loss of neurons and neuronal function
- Sensation
- Alterations in neuronal and glial activity
- Sensory-motor and sensory-sensory incongruence

Peripheral changes
- Structural changes in neurons and axons
- Synaptic impinquity
- Ephaptic transmission
- Synaptic-effector coupling
- Down- and upregulation of transmitters
- Alterations in chakavehur and transduction molecules
- Selective loss of unmyelinated fibres

Recovery: Upon arrival to PACU, the patient reported severe 10/10 pain located both at the stump and in his phantom foot. The patient described the pain as, “intermittent, zapping, sharp, shooting pain in my ankle.” After 3ml 0.1% bupivacaine (X2) by epidural and 2mg dilaudid IV, his stump pain resolved (0/10 pain); however, the PLP continued (10/10 pain).

- After a bolus of 10ml 2% lidocaine, the patient experienced complete relief of PLP.
- A 0.25% bupivacaine infusion was begun (10 ml/hr).
- PLP returned after the 2% lidocaine wore off.
- A 6ml 0.25% bupivacaine bolus relieved the PLP.

Hospital Post-op:

POD1-3: Epidural 10cc/hr of 0.25% Bupivacaine
POD1: Started on nortriptyline 25mg QHS, gabapentin 100mg TID, and his home dose of Oxycontin 40mg BID (which was stopped on POD2 for somnolence)

POD3-5: Epidural decreased to 6cc/hr
PODS: Epidural discontinued

During this interval, he required supplemental opioids averaging 4 mg of hydromorphone IV per day for rescue and reported intermittent mild to moderate stump pain and occasional mild to moderate phantom limb pain.

POD4:
- Altered sensorium & myoclonus; diagnosed with serotonin syndrome (nortriptyline, trazodone, & home sertraline). Course additionally complicated by UTI and C.Diff

POD26: Stabilized and discharged to a Rehabilitation Hospital.
On discharge, reported that pain was well-controlled and denied PLP.

Outpatient Post-OP:
Continued Oxycontin 40mg BID, hydromorphone PO 2-4mg Q3H PRN, & gabapentin 600mg TID Mild to moderate pain, especially with physical therapy but no PLP reported

Subacute Post-Op:
POD33: Transferred back to MGH 2/2 SOB (acute CHF exacerbation)
POD35: ICU for hypercarbic respiratory failure complicated by AKI, hyperkalemia, and UTI. Stabilized and transferred to floor, overall condition continued to worsen, he transitioned to palliative care and passed away approximately two months after his BKA.

Discussion: This patient had several significant risk factors for developing phantom limb pain including peripheral vascular disease, diabetes, chronic infection leading to amputation, depression, and chronic neuropathic pain in the affected limb. His most significant risk factor is related to his perioperative pain control. Preoperatively, his pain was poorly controlled. His epidual was initially bolused with concentrated local anesthetic intraoperatively. However, a prolonged dosage interval (>2hrs) likely resulted in severe, uncontrolled pain and intense phantom pain. When his epidual was bolused with dilute local anesthetic and opioid (0.1% Bupivacaine w/ 20mcg/ml of hydromorphone), the pain did not improve. Yet when the epidual was bolused with 2% lidocaine (Surgical block), phantom pain sensations immediately ceased.

This case may provide insight into the mechanisms that initiate PLP. Previous research has implicated both central and peripheral nervous system mechanisms mediating PLP, but this case may suggest the afferent sensory input is critical to the initiation of PLP and that the mechanisms responsible for the initiation and maintenance of PLP may be independent. Moreover, because the dilute bupivacaine ameliorated the incision/stump pain, but not the PLP, it suggests distinct afferent pathways for these two pain states.

Conclusions:
1. Perioperative pain control is critical to preventing PLP.
2. The density epidual blockade may play a role in preventing PLP.
3. Further research is needed to understand the mechanisms responsible for the initiation and maintenance of PLP.

References: