Strategies to prevent the transition from acute to chronic pain

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TOPICS

> Conceptual framework
> Risk factors
> Current preventive strategies
> Prospects
Why does pain persist after an acute event?

- Derived from basic research or prognostic studies
- The causal role is undetermined
- Prevention cannot be done on identified determinants

- Persistent tissue damage
- Central neuroplastic changes
- Psychosocial factors
- Genetics
Prognostic studies

> Can identify potential risk factors

> Can establish associations, not causalities
  – Misleading spurious associations due to confounders
    > Coffee consumption and pancreatic cancer – smoking being the true determinant

> Have been invaluable in many areas of medicine
  – Condition: interventions on modifiable risk factors have been shown to improve the outcome
Risk factors

> Chronic pain before surgery
> Psychosocial co-morbidities
> Preoperative opioid intake
> Acute postoperative pain

> Previous functional limitations
> Psychosocial co-morbidities
> Pain at 3 months

> Previous functional limitations
> Psychosocial co-morbidities
> Low general health status

After surgery

Althaus et al, EJP 2014
Sommer et al, Cl J Pain 2010

After traumatic injury

Rivara et al, Arch Surg 2008

After acute LBP

Chou & Shekelle, JAMA 2010
Central sensitization - Rationale

- Peripheral injury cause alterations in central pain processes leading to hypersensitivity
- Central sensitization likely contributes to pain and disability
- Central sensitization may facilitate persistence of pain after the primary injury has resolved

> Does central sensitization in the acute phase predict the occurrence of chronic pain?
Prognostic value of central sensitization (QST) in musculoskeletal pain

> Central sensitization in acute whiplash patients predicted poor outcome at 6 months

  Sterling et al, Pain 2010

> Cold hyperalgesia in acute lateral epicondylalgia predicted persistent pain at 12 months

  Coombes et al, Cl J Pain 2015

> Pain thresholds and altered CPM did not predict transition from acute to chronic low back pain

  LeResche et al, J Pain 2013
  Müller et al, unpublished
Prognostic value of QST for chronic postoperative pain – (1)

> Among different QST, only brush-evoked allodynia was associated with pain 4 months after hysterectomy
  
  *Brandsborg et al, BJA 2011*

> Facilitated temporal summation predicted chronic pain 12 months after total knee replacement
  
  *Petersen et al, Pain 2015 - Petersen et al, Pain 2016*

> Altered CPT, but not other QST, predicted chronic pain 6 months after thoracotomy
  
  *Yarnitsky et al, Pain 2008*
Prognostic value of QST for chronic postoperative pain – (2)

> Pressure pain thresholds before knee or hip replacement did not predict chronic pain 12 months after surgery
  
  Wylde et al, Pain 2015

> The area of pinprick hyperalgesia after sternotomy did not predict chronic post-sternotomy pain at 4-6 months
  

> Pain thresholds and altered CPM did not predict the outcome of low back surgery
  
  Müller et al, unpublished
Altered QST/CPM → Poor outcome

Normal QST/CPM → Good outcome

Altered QST/CPM → Poor outcome

Normal QST/CPM → Good outcome
Relevant questions for clinical use of QST/CPM

> How likely is the occurrence of a poor outcome in patients with altered QST/CPM, compared with the likelihood of a poor outcome in patients with normal QST/CPM?
  – Positive likelihood ratio, should be $\geq 5$

> How likely is the occurrence of a good outcome in patients with altered QST/CPM, compared with the likelihood of a good outcome in patients with normal QST/CPM?
  – Negative likelihood ratio, should be $\leq 0.2$
Do QST / CPM predict transition to chronic pain?

> Results are not consistent
  > Positive and negative findings
  > Predictive QST are not consistent across positive studies
> Most studies did not include potential confounders
> Most studies did not compute likelihood ratios

The current methods:
> May detect pathophysiologic associations between central sensitization and development of chronic pain
> Are unlikely to support clinical decision making
Acute pain and development of chronic pain

Possible causes of this association:

> Severity of the injury, causing severe acute pain and also unable to heal

> Severe acute pain would reflect induction of profound neuroplastic changes leading to persistent pain

> Psychosocial vulnerability would account for both severe acute pain and development of chronic postoperative pain

> Severe acute pain would facilitate psychological morbidities, leading to persistent pain

> Genetic factors would predispose to both severe acute pain and development of chronic postoperative pain (may determine also part the above factors)
Do interventions for acute postoperative pain prevent chronic pain?

Systematic reviews

Regional anesthesia:
> Some evidence that it prevents chronic pain after thoracotomy and breast cancer surgery in 1 out of 4-5 patients treated

Andreae & Andreae, BJA 2013

Pharmacotherapy:
> Modest reduction in the incidence of chronic pain with ketamine (small studies, risk of overestimation)
> The efficacy of gabapentin, pregabalin, NSAIDs, steroid and iv lidocaine is not supported by the available evidence

Chaparro et al, Cochrane 2013
Peri-operative pain prevention programs

Assumptions:
> Modifiable risk factors identified in prognostic studies play a causal role
> Treatments of risk factors improve the outcome

Concept:
> Pre-operative screening and selection of patients at risk
> Interventions in the pre- and acute post-operative phase
> Follow-up and ad-hoc interventions after discharge
Transitional Pain Service
Toronto General Hospital

> Surgical preadmission visit
  - 12.5% of patients identified with a “pain alert” (chronic pain problems requiring daily opioid medication)
  - Assessed after surgery by the TPS
  - Multidisciplinary plan for highly complex patients

> Patients who are not identified prior to surgery are referred to the TPS by the APS or surgical team

> TPS: medication optimization, patient/family education, referrals to behavioral health/ rehabilitation

> Follow-up after discharge at TPS clinic every 2-3 weeks

> Back to primary care 6 weeks to 6 months after discharge

Katz et al, J Pain Res 2015
Preliminary results
Non-randomized

Clarke et al,
Drugs 2015
Program at UW - Harbor View Medical Center

Clinical services
- Pre-anesthesia consults
- Acute pain service
- Transitional care clinic
- Interventional services
- Ambulatory chronic care

Community and primary care support
- Pain specialist in primary care clinic
- Community-based program collaboration
- Tele pain

Clinical operation committee
- Implementation science
- Pain policies
- Quality improvement
- Education for patients and staff
Preoperative care

- Tele-pain consult
- Pain pharmacist
- Pain specialty care
- Pain psychiatrist
- Rehab. psychiatrist
- Addiction support
- Pat. education material
- APS consult alert

Acute periop. care

- Pain specialist
- Pain pharmacist
- Rehab. psychiatrist
- Complement. medicine
- Spiritual care
- Social services
- Addiction support
Transitional pain care
- Pain specialist
- Pain pharmacist
- Rehab. psychiatrist
- Social services
- Addiction support

Post-discharge
(CURRENTLY AVAILABLE FOR SPINE SURGERY)
Follow-up with web-based instrument (Pain Tracker)
1 week, 1 month, 6 month and 1 year
Screening by pre-anesthesia clinic

Only for planned elective surgery

Need for pain expertise if (one or more):
> Morphine equivalent daily dose $\geq 120$ mg
> Opioids plus $\geq 2$ psychoactive drugs
> Methadone use
> Buprenorphine use (in USA used for addiction)
> Prior poor postoperative pain control
> Implanted pain pump or spinal cord stimulator
> Significant psychiatric illness
> Current or recent substance use disorder
Pain behavioral health screen, positive values:
• PHQ-9 (depression) ≥ 15
• GAD-7 (anxiety) ≥ 15
• CAGE-AID (alcohol and drug abuse) ≥ 2
• PCS (catastrophizing) ≥ 20
Pre-anesthesia clinic → Screening positive → Pain behavioral health screen

Screen negative, but either
- MED ≥ 120 mg
- Opioids plus ≥2 CNS drugs
- Prior severe postop. pain

Peri-op. recommendations by PAC

APS assumes care
Pre-operative Pain Care Clinic
- Pre-, intra- and postop. plan
- Recomm. for buprenorphine, naltrexone or IT pump
- Discharge plan – Postoperative Transition Pain Clinic
- Care coordination
- Patient education / expectation alignment
- Community resource identification – Self management programs

Screen positive, or either
- Buprenorphine/Naltrexone
- Intrathecal pump / SCS
- Substance use disorder

Pre-op. Pain Specialty consult

Pre-op. Pain Care Clinic

Behavioral health screen positive

NO
Pre-op. Pain Behavior Pain Clinic
• Address psych. illness / coping
• Medication optimization
• Preop. psychol. interventions
• Patient education / expectation alignment
• Pre-, intra- and postoperative plan
• Care coordination
• Discharge plan – Postoperative Transition Pain Clinic
• Community resource identification – Self man. progr.

Pre-anesthesia clinic → Screening positive → Pain behavioral health screen

Screen positive, or either
- Buprenorphine/Naltrexone
- Intrathecal pump / SCS
- Substance use disorder

Pre-op. Pain Specialty consult

Pre-op. Pain Behavior Pain Clinic
• YES

Behavioral health screen positive

Pre-op. Pain Behavior Pain Clinic → YES
Pre-anesthesia clinic

Screening positive

Pain behavioral health screen

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Peri-op. recommendations by PAC

Pre-op. Pain Behavior Pain Clinic

APS assumes care

Screen positive, or either
- Buprenorphine/Naltrexone
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- Substance use disorder

Pre-op. Pain Specialty consult

Behavioral health screen positive

YES

Pre-op. Pain Care Clinic

NO
The development of effective prevention is hampered by lack of knowledge of the determinants. Risk factors/predictors have been studied. Currently, transition to chronic pain cannot be predicted with sufficient confidence. Regional analgesia and medications may prevent chronic postsurgical pain—but target only part of the problem. Comprehensive prevention programs that account for the complexity of chronic pain are under development. They are promising, but their efficacy has not yet been demonstrated.
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