UNIVERSITY *of* WASHINGTON

Strategies to prevent the transition from acute to chronic pain

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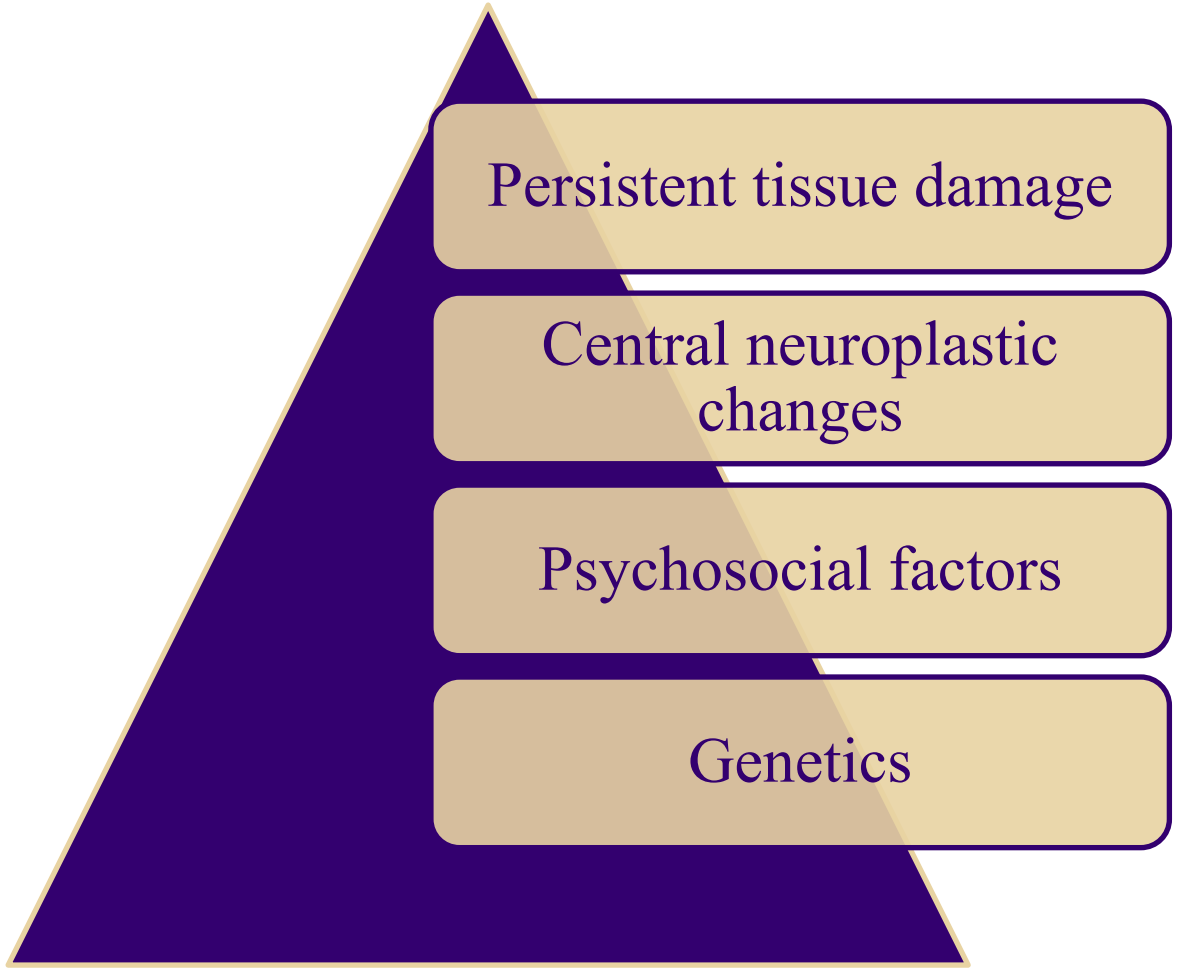
TOPICS

- 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**

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

After surgery

Althaus et al, EJP 2014
Sommer et al, Cl J Pain 2010
Rozet et al, A&A 2014

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

After traumatic injury

Rivara et al, Arch Surg 2008

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

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  Müller et al, unpublished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**

  Setälä et al, Acta Anesthesiol Scand 2016

- **Pain thresholds and altered CPM did not predict the outcome of low back surgery**

  Müller et al, unpublished

Poor outcome

Good outcome

Altered QST/CPM

Normal QST/CPM

Good outcome

Poor 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 **≥ 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 **≤ 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

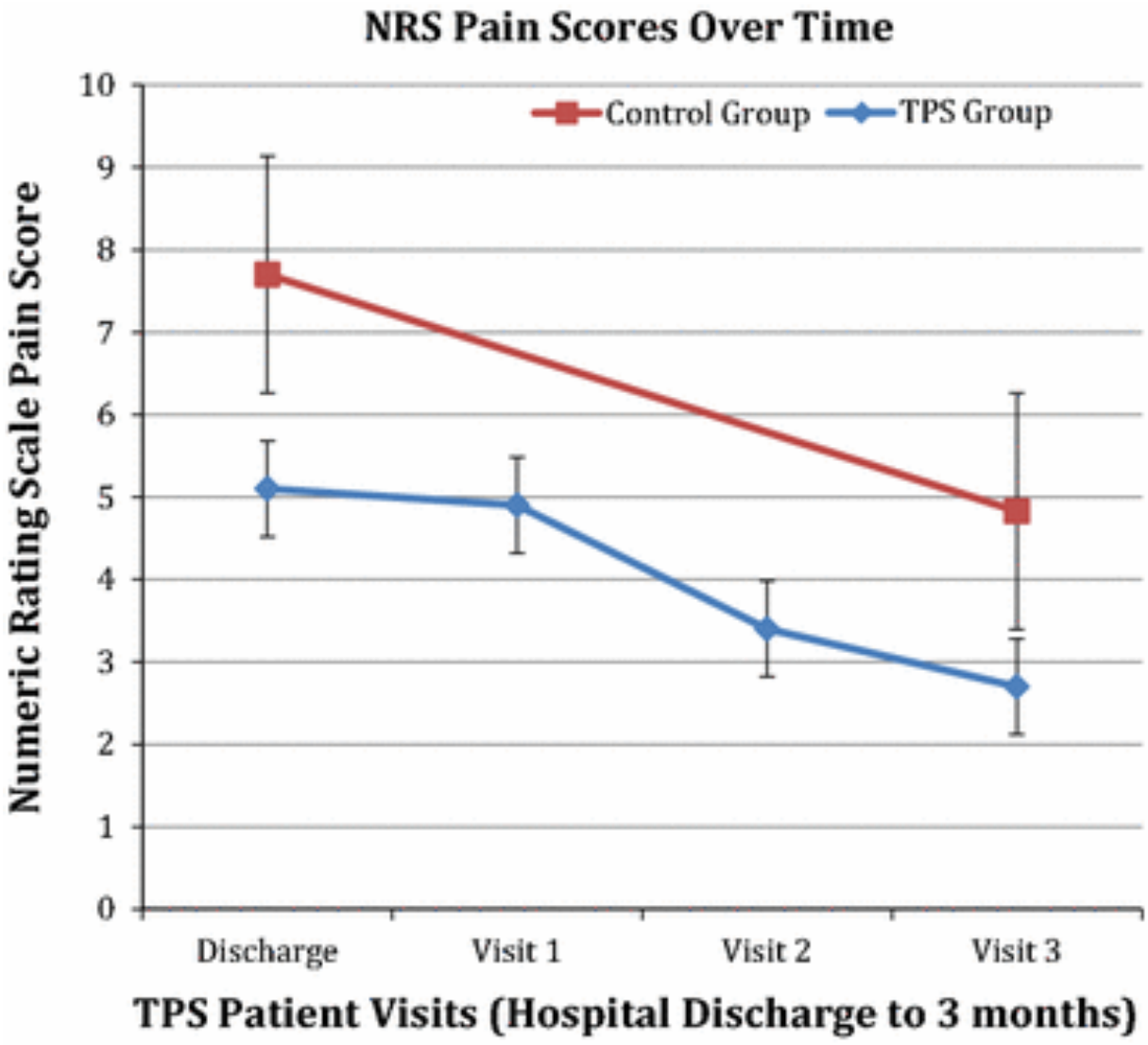
> Follow-up and ad-hoc interventions after discharge

Transitional Pain Service

Toronto General Hospital

Katz et al, J Pain Res 2015

- 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



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

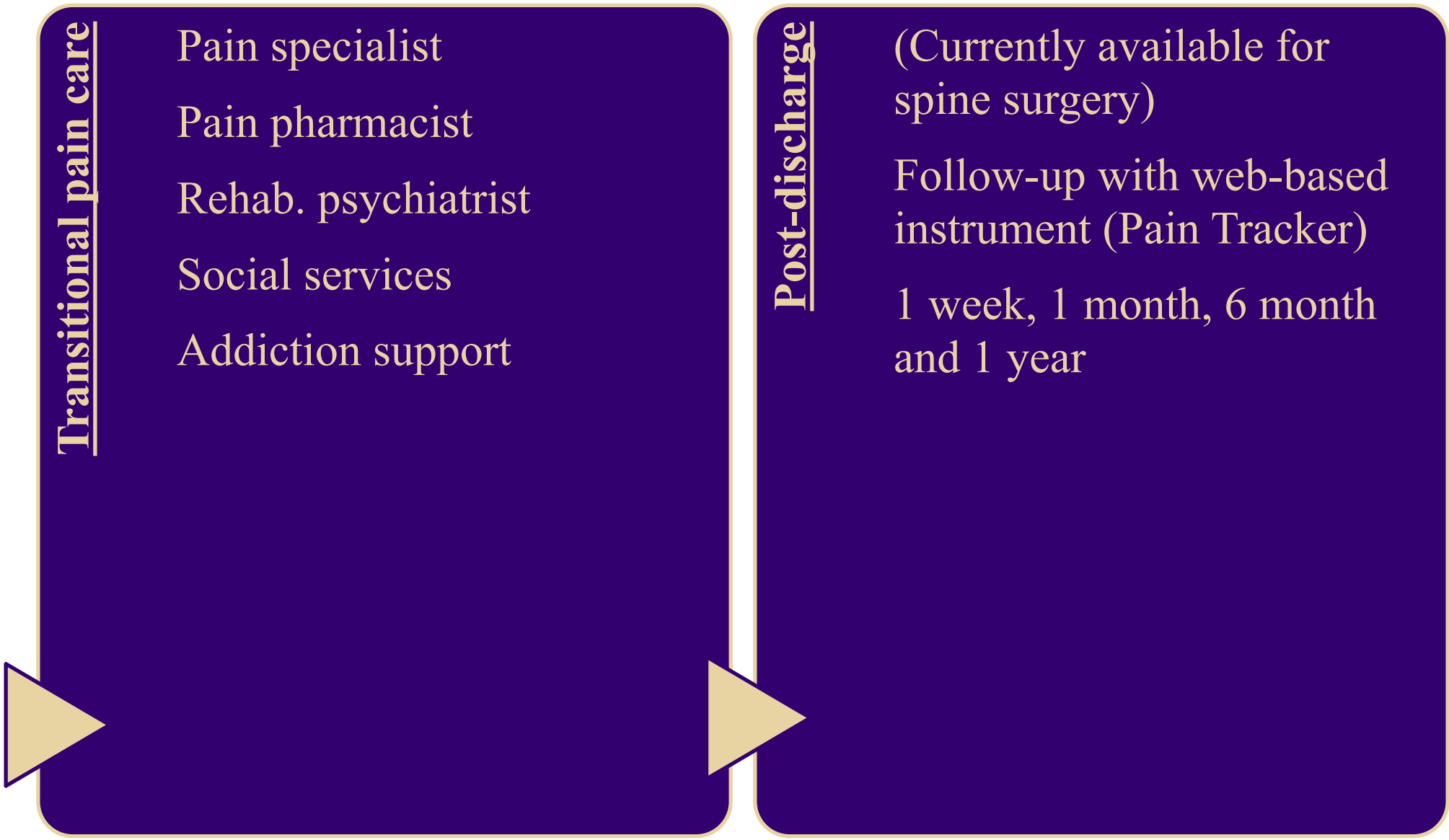
Community and primary care support

- 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

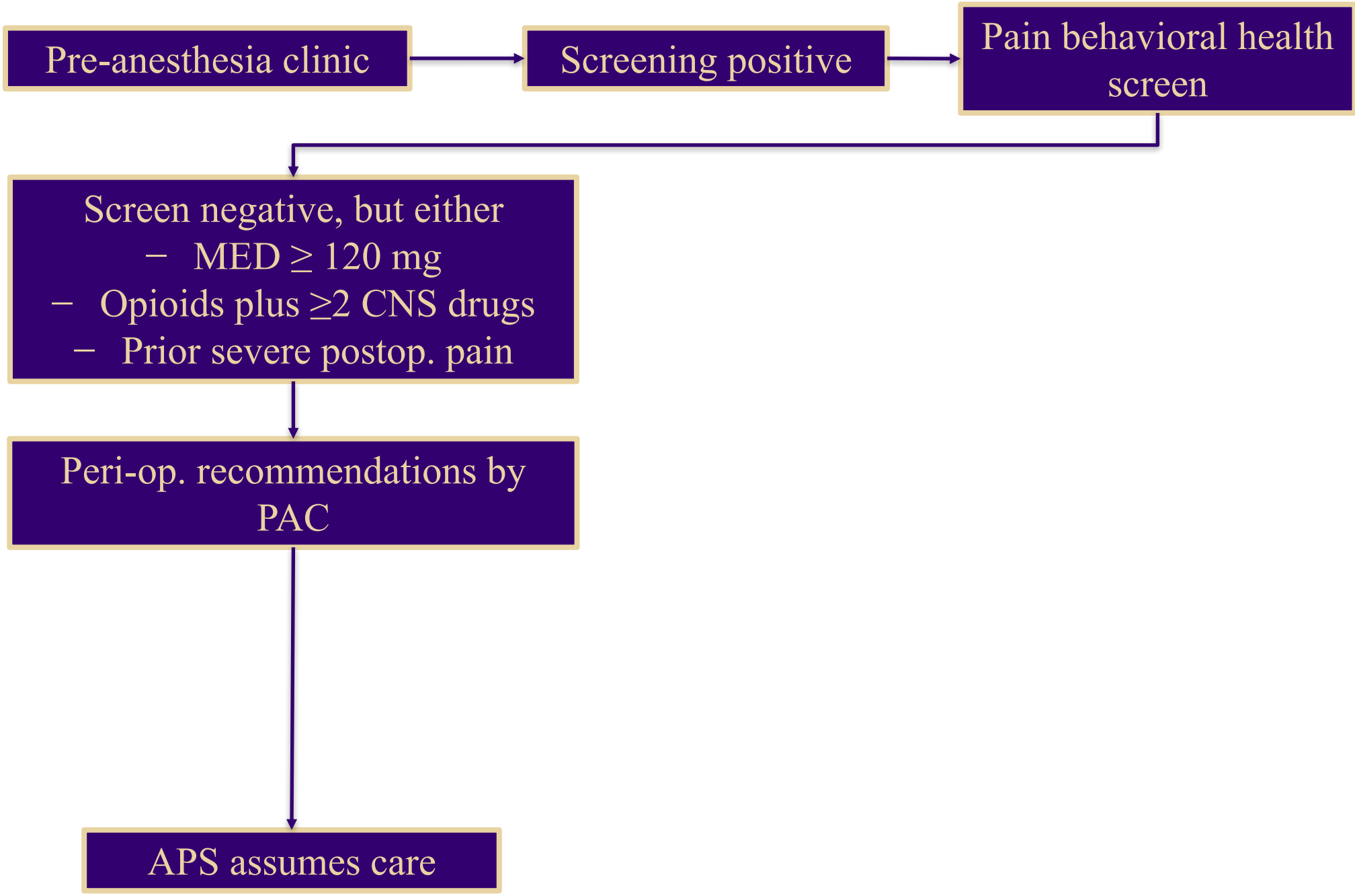
Only for planned elective surgery

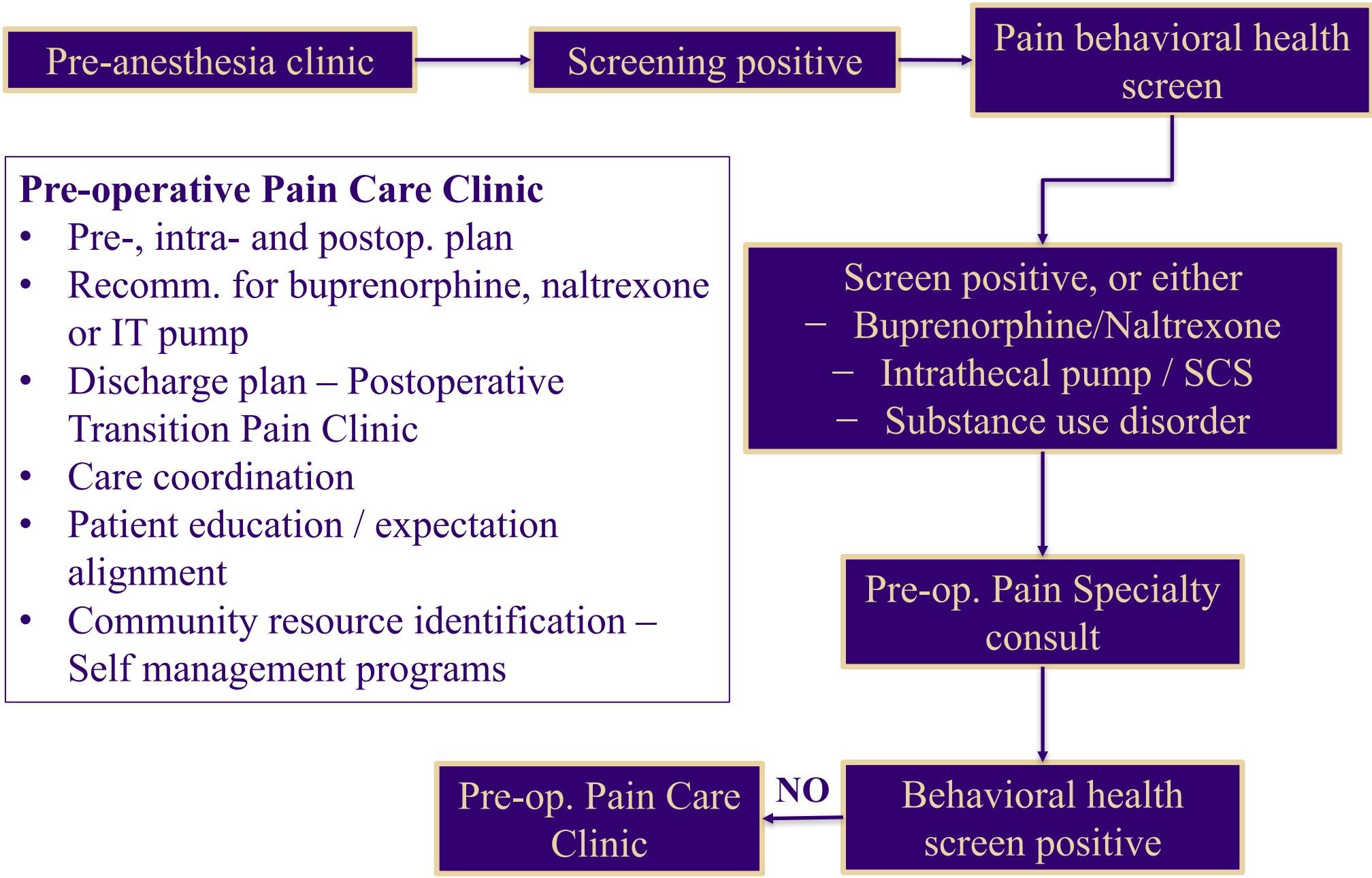
Need for pain expertise if (one or more):

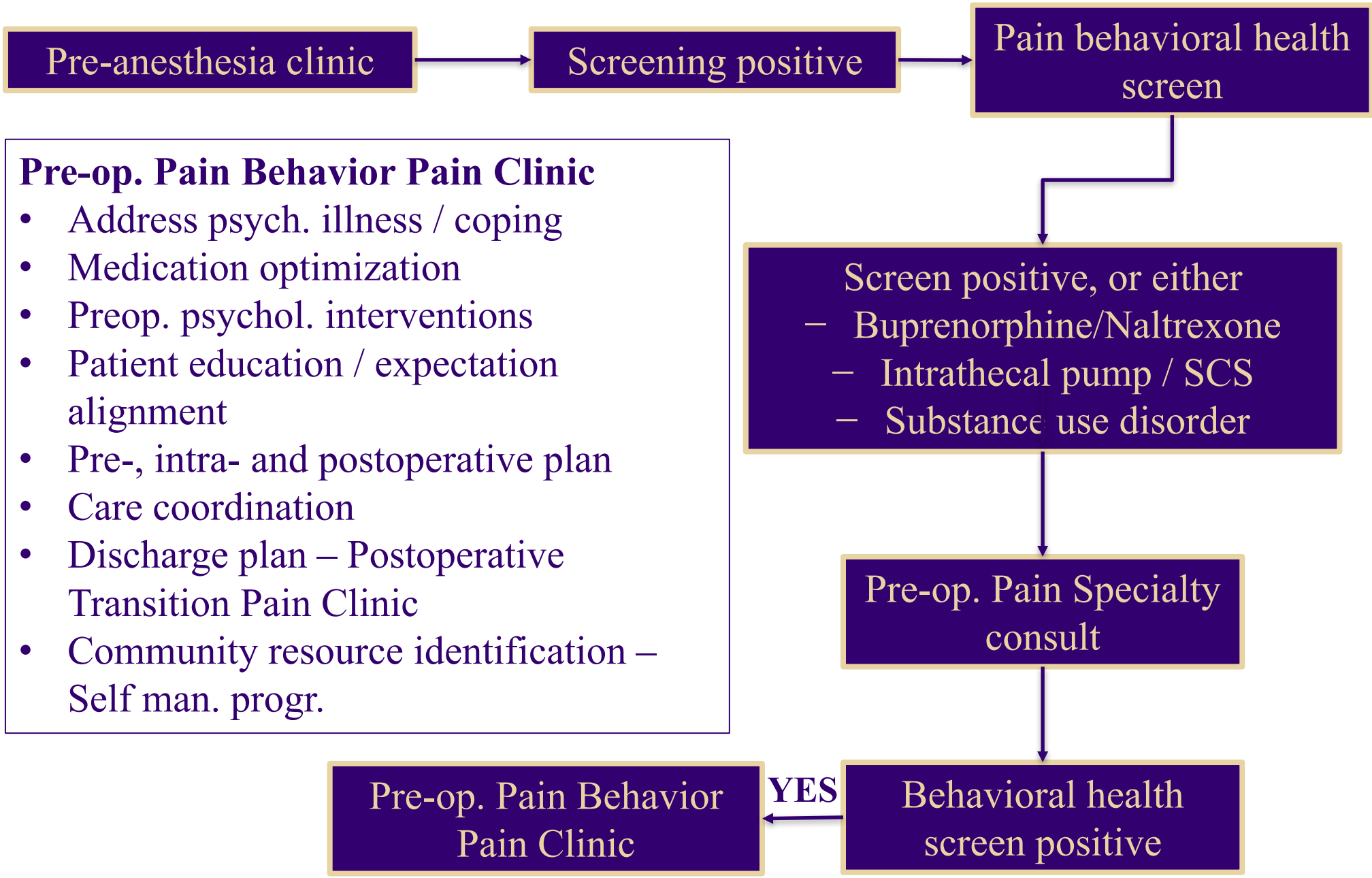
- Morphine equivalent daily dose ≥ 120 mg
- Opioids plus ≥ 2 psychoactive drugs
- Methadone use
- Buprenorphine use (in USA used for addiction)
- Prior poor postoperative pain control
- Implanted pain pump or spinal cord stimulator
- Significant psychiatric illness
- Current or recent substance use disorder

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-anesthesia clinic
Screening positive
Pain behavioral health screen
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
Behavioral health screen positive
NO
Pre-op. Pain Care Clinic


Pre-anesthesia clinic
Screening positive
Pain behavioral health screen
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.
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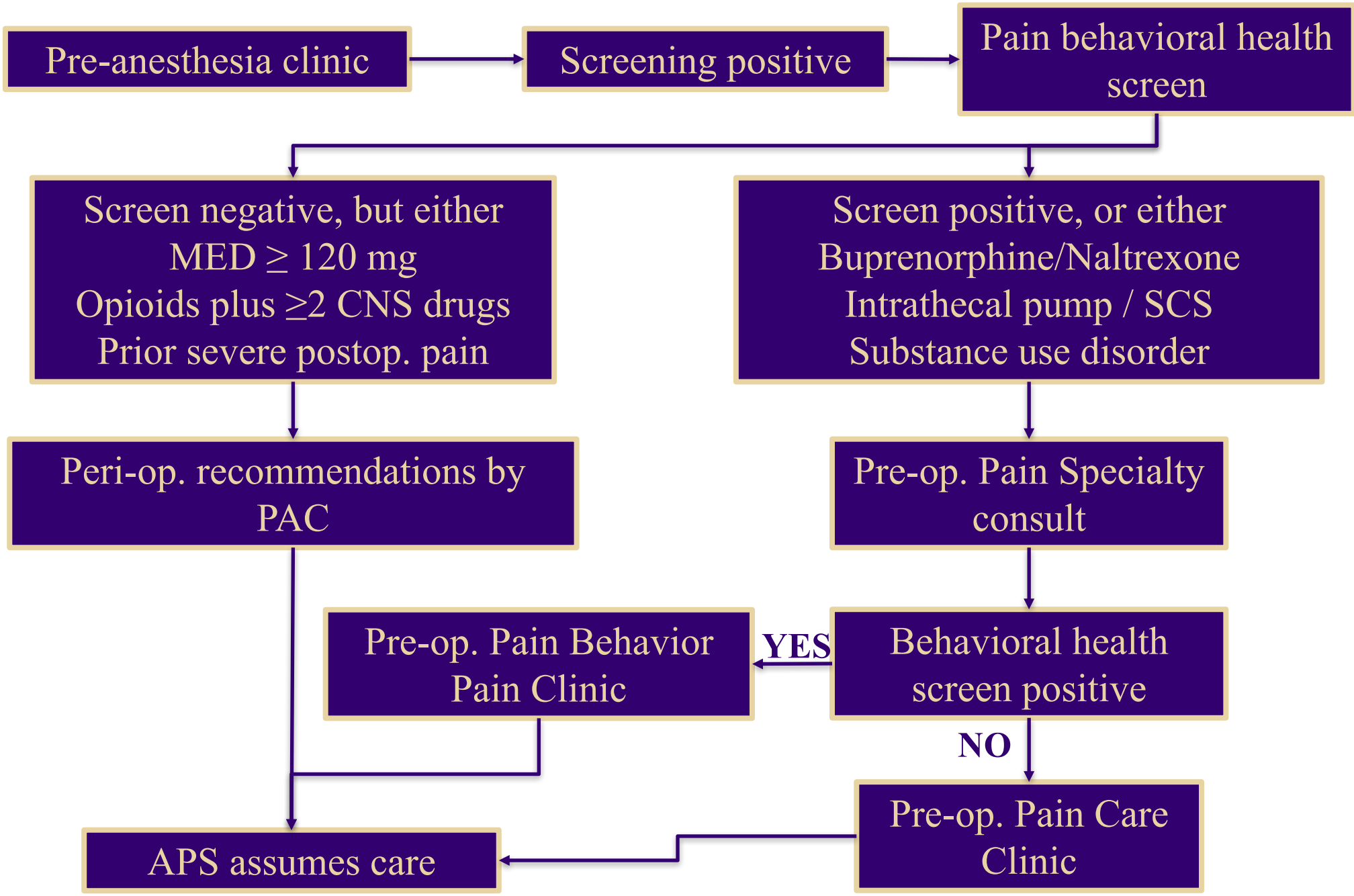
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YES
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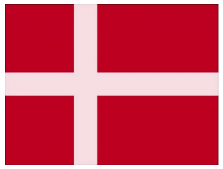
NO
APS assumes care
Pre-op. Pain Care Clinic

TAKE-HOME MESSAGES

- 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 post-surgical 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

Acknowledgments



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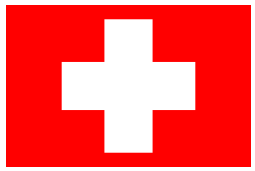

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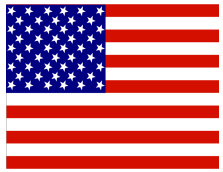

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