Review Article

Chronic postsurgical pain after cardiac surgery: A narrative review

ABSTRACT

Chronic postsurgical pain (CPSP) is a prevalent and debilitating sequela of cardiac surgery, exerting a profound impact on patients' quality of life, functional recovery, and healthcare systems. Its pathophysiology includes complex mechanisms, including peripheral and central sensitization, neuroplastic alterations, and inflammatory pathways, influenced by demographic, psychological, and perioperative factors. Inadequate management of acute pain is a critical contributor to its development. This review examines the etiology of CPSP, presents key risk factors, and critically evaluates pharmacological and nonpharmacological interventions. Particular attention is devoted to the role of regional anesthesia techniques and emerging preventive and therapeutic strategies, highlighting the necessity of multidisciplinary, evidence-informed approaches to address this persistent clinical challenge.

Key words: Cardiac surgery, chronic postsurgical pain, regional anesthesia

Introduction

Chronic postsurgical pain (CPSP) after cardiac surgery is a significant and often under-recognized clinical issue that can profoundly affect patients' quality of life and functional recovery. While advances in surgical techniques and perioperative care have improved patient outcomes, the persistence of pain beyond the typical healing period poses a substantial burden on both patients and healthcare systems. CPSP is particularly relevant in cardiac surgery, where the invasiveness of procedures like sternotomy, thoracotomy, and rib retraction increases the risk of long-term pain syndromes.

In 2019, the International Association for the Study of Pain redefined CPSP. According to this definition, CPSP is characterized as pain that develops or worsens after a surgical procedure, persists for at least 3 months, and is distinct from any pain experienced before the surgery. This pain is localized to the surgical site and cannot be explained

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by other causes, such as cancer recurrence or infection. The new ICD-11 classification provides a clearer framework for identifying CPSP, recognizing it not merely as a symptom but as a secondary disease.[1]

Estimates suggest that over 50 million surgical procedures are performed annually worldwide, underscoring the potential magnitude of the CPSP burden. Approximately 10% of surgical patients experience prolonged pain, with 1% suffering from severe CPSP.[2] The incidence of chronic pain can persist up to 48 months post surgery. Globally, more than 2 million open-heart surgeries are conducted each year, with over 300,000 patients reporting pain persisting for at least 1 year post surgery. Following cardiac surgery, the incidence of pain at the third postoperative month is approximately 29%, decreasing to 18% at 2 years and 15% at 1 year.[3] Notably, a recent study reported that the prevalence of chronic neuropathic pain after cardiac surgery is approximately 14.7%, underscoring the potential neuropathic component of CPSP and the need for targeted therapeutic approaches to address this specific pain subtype.[4]

Given these significant statistics, it is crucial to investigate the factors contributing to CPSP and its long-term effects on patients' quality of life. The ongoing multicenter CPSP-Cardiac study investigates the incidence of CPSP after cardiac surgery and its impact on quality of life. Recruiting 1176 patients undergoing median sternotomy, the study assesses both acute and chronic pain. Primary outcomes include CPSP incidence at 3 months, while secondary outcomes examine opioid use, psychological health, and patients quality of life.[5]

The clinical relevance of addressing chronic pain in cardiac surgery patients cannot be overstated. Chronic pain not only impairs physical and psychological wellbeing but also increases healthcare utilization, reduces work productivity, and places a financial strain on healthcare systems. Given the increasing volume of cardiac surgeries worldwide, the prevention, timely identification, and effective management of CPSP have become pressing public health priorities. Despite the availability of various treatment modalities, a comprehensive understanding of the underlying mechanisms and evidence-based strategies for prevention and management remains limited.

The primary objectives of this review are to provide an in-depth exploration of the mechanisms underlying CPSP after cardiac surgery, identify key risk factors, and evaluate the effectiveness of current and emerging management strategies. The review aims to bridge the knowledge gap by presenting an evidence-based narrative on the etiology, prevention, and treatment of chronic pain in this unique surgical population.

Mechanisms of Chronic Pain

Chronic pain mechanisms encompass peripheral sensitization, central neuroplasticity, supraspinal changes, and inflammatory pathways after tissue injury.

Peripheral sensitization occurs after nerve damage, surgical incisions, or tissue injury, leading to heightened sensitivity and reduced activation thresholds in nociceptive neurons.[6] This process involves changes in ion channels like voltage-gated transient receptor potential vanilloid (especially TRPV-1) channels, sodium channels, and calcium channels, which regulate pain sensitivity and nerve excitability. These alterations result in mechanical and thermal hypersensitivity and contribute to chronic pain development.[7]

Central sensitization involves enhanced activity in nociceptive pathways due to increased membrane excitability, synaptic efficacy, and reduced inhibition, demonstrating the neuroplastic potential of the central nervous system.[8] This process is significant in chronic pain, including postoperative pain following cardiac surgery. Possible mechanisms include glutamate-mediated activation of N-methyl-D-Aspartate (NMDA) and amino-3-hydroxy-5-methyl-4-isoxazole propionate (AMPA) receptors, intracellular calcium increases, and post-translational modifications that sustain synaptic changes.[9] Dysfunction in descending pain modulation pathways, particularly changes of serotonergic and noradrenergic secretion, exacerbates pain perception.[10] Structural changes in brain regions like the anterior cingulate cortex and prefrontal cortex contribute to persistent pain.[11]

Chronic pain is maintained by inflammatory mediators, cytokine release, and glial cell activation.[12] Neurogenic inflammation, driven by cytokines (e.g., TNF-alpha, IL-1 beta), neuropeptides (e.g., substance P, calcitonin gene-related protein (CGRP)), and chemokines, lowers nociceptor thresholds and sustains peripheral sensitization.[13] At injury sites, an "inflammatory soup" of peptides, lipids, and neurotransmitters promotes nociceptor activation and pain signal transmission to the central nervous system (CNS).[14]

In the CNS, glial cells like microglia and astrocytes release proinflammatory mediators, reactive oxygen species, and brain-derived neurotrophic factor, enhancing neuroinflammation, synaptic plasticity, and central sensitization.[15] Excess glutamate activates NMDA and AMPA receptors, while substance P and CGRP intensify nociceptive signaling. Reduced gamma-aminobutyric acid levels disrupt inhibitory balance, leading to sustained hyperexcitability.

Chronic pain is associated with altered connectivity in supraspinal regions like the anterior cingulate cortex, insula, and prefrontal cortex, which show structural and functional changes such as gray matter reductions.[11] Dysfunction in descending pathways from the periaqueductal gray to the rostral ventromedial medulla impairs inhibitory control while increasing excitatory signaling in the spinal cord. Disruptions in serotonin and norepinephrine levels amplify pain and reduce endogenous pain inhibition.[16]

Risk Factors

Patients with CPSP experience health-related chaos due to the feeling of despair caused by pain and a decrease in quality of life. Beyond pain, this situation impedes critical postoperative recovery and prevents return to work and normal daily life, resulting in functional disability. Adequate understanding and management of perioperative pain constitutes a fundamental strategy for the prevention of CPSP after cardiac surgery. Figure 1 illustrates anesthesia- and surgery-related risk factors during both the preoperative and postoperative periods.

Preoperative risk factors

Risk factors for CPSP include younger age, female gender, preexisting pain, higher body mass index, and history of osteoarthritis.[3,17] The presence of a number of psychological disorders, such as preoperative anxiety, depression, post-traumatic stress disorder, and pain catastrophizing, and the use of antidepressants have been identified as risk factors for chronic pain.[18,19] In addition, some studies have shown that genetic predisposition, polypharmacy, living alone, frailty, and low mental quality of life are associated with an increase in the frequency or severity of chronic pain.[20-22]

Intraoperative risk factors

Anesthesia-related risk factors

Opioids, and particularly ultra-short-acting remifentanil, play a role in the development of opioid-induced hyperalgesia and subsequent increased risk of CPSP.[23,24] A perioperative opioid sparing strategy using analgesic alternatives, regional analgesia techniques, and multimodal analgesia may reduce the risk of developing CPSP and additional opioid-induced side effects.

Surgical risk factors

Emergency surgery and previous cardiac surgery are shown as risk factors for CPSP. The type of cardiac surgery (e.g., coronary artery bypass grafting, valvular surgery or combined surgery), surgical technique and duration of surgery, and use of the internal thoracic artery are thought to be risk factors for the development of CPSP, probably related to variables such as the type of retraction of thoracic structures and the amount and duration of stretching.[22,25,26]

Postoperative risk factors

Intense acute postoperative pain (especially the first 5 days) is a risk factor for CPSP to be severe and extensive.[3,17] Besides resternotomy shortly after the original surgery is shown as a CPSP risk factor,[25] it is mentioned that sternal wound complications, ranging from superficial infections to sternal instability and mediastinitis, increase the risk of CPSP.[22]

Pharmacological treatments: Opioids, NSAIDs, anticonvulsants, and antidepressants

CPSP after cardiac surgery presents a particularly complex clinical challenge, necessitating a multimodal and individualized pharmacological approach. Efforts often involve the use of systemic pharmacologic therapies. A recent meta-analysis highlighted the focus on agents

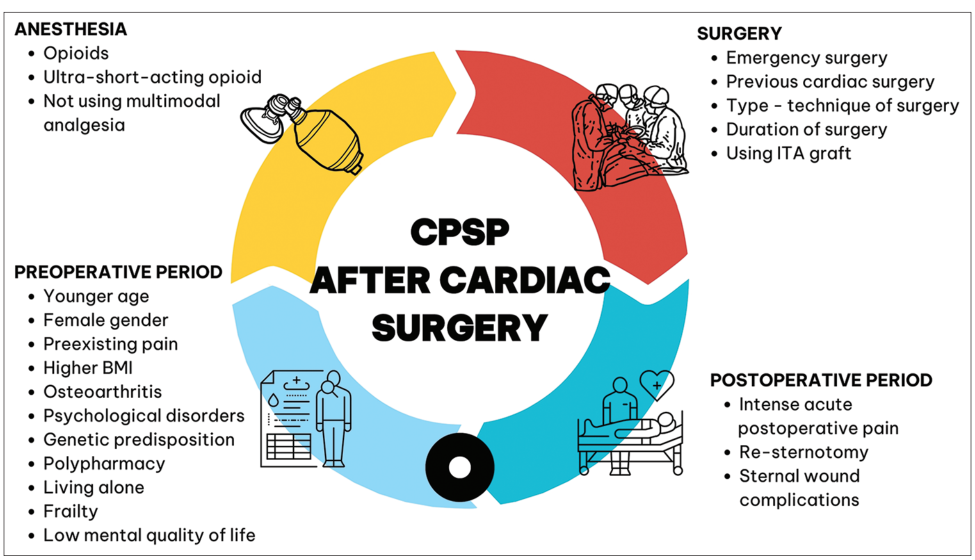

Figure 1: Risk factors for chronic postsurgical pain after cardiac surgery

such as ketamine, pregabalin, gabapentin, intravenous lidocaine, nonsteroidal anti-inflammatory drugs (NSAIDs), and corticosteroids for CPSP prevention.[27]

Intravenous ketamine infusion at subanesthetic doses, particularly as an adjunct to opioid-based analgesic regimens, has shown efficacy in managing acute postoperative pain.[28] Although ketamine is considered one of the most promising agents for CPSP prevention, a meta-analysis including 27 studies found no significant effect of ketamine on the prevalence of CPSP, regardless of the outcome timing, duration of drug administration, or type of surgical procedure.[27]

Gabapentinoids, including gabapentin and pregabalin, have been considered for the management of neuropathic pain and are sometimes utilized in the prevention or treatment of CPSP.[29] However, a meta-analysis that included 18 studies reported no significant effect of gabapentin on pain outcomes assessed at 3 and 6 months postoperatively. Also, pregabalin did not significantly reduce CPSP overall. Interestingly, subgroup analyses within this meta-analysis revealed a statistically significant reduction in CPSP with pregabalin in specific surgical populations. For instance, in patients undergoing cardiac surgery, three trials demonstrated a risk ratio (RR) of 0.25 (95% CI 0.13 to 0.50) at 3 months.[27] These findings suggest that while gabapentinoids may not universally prevent CPSP, pregabalin could offer benefits in selected patient populations and surgical contexts, warranting further investigation into its targeted use.

Intravenous lidocaine administration during surgery has been demonstrated to reduce the incidence of CPSP in multiple meta-analyses; however, these studies are predominantly limited to noncardiac surgeries.[30,31] This finding is promising for the management of chronic pain following cardiac surgeries as well. Nonetheless, it is emphasized that higher-quality evidence is required to establish definitive and robust conclusions.[30]

Beyond those, the use of NSAIDs has been evaluated, and it is recommended to administer paracetamol and NSAIDs during both intraoperative and postoperative periods. However, the use of COX-2 inhibitors is not recommended.[32] Although no meta-analysis has been published specifically addressing chronic cardiac pain, it has been reported that the use of NSAIDs for 24 hours or less can reduce the incidence of CPSP at 12 months.[27]

Regional anesthesia techniques: Thoracic epidurals, paravertebral blocks, or novel blocks

Since successful analgesia management shortens the postoperative recovery period and reduces undesirable conditions such as pulmonary complications, postoperative pain management after cardiac surgery is very important.[26,33] Regional anesthesia techniques have an important role in both the prevention and treatment of CPSP.[32,34,35] Regardless of the type of surgery performed, median sternotomy/valve surgery or minimally invasive, regional anesthesia techniques are being used effectively in the treatment of CPSP, along with the widespread use of ultrasound in anesthesia and pain medicine departments.[34,36] Regional anesthesia has several advantages in managing CPSP, addressing the limitations of systemic analgesics and improving patient outcomes. Regional anesthesia techniques like neuroaxial techniques, nerve blocks, and fascial plane blocks often reduce or eliminate the need for high-dose opioids. A recent meta-analysis demonstrates that for the management of acute postoperative pain, fascial plane blocks, particularly the erector spinae plane block (ESPB), are significantly more effective than placebo, as indicated by a substantial reduction in 24-hour opioid consumption.[37] Furthermore, regional anesthesia promotes better functional outcomes by reducing the impact of persistent pain on daily activities.

Neuroaxial techniques

Thoracic epidural analgesia (TEA) and paravertebral blocks (TPVB) are the main and gold standard techniques to provide thoracic analgesia after cardiac surgery. These two techniques may also be used in the treatment of CPSP. There are clinical studies reporting that TEA provides effective analgesia and reduces opioid requirements after cardiac surgery. Similar to TEA, TPVB can be successfully performed in the treatment of sternotomy pain and thoracic analgesia.[36,38-40] However, TPVB and TEA have the risk of epidural hematoma, cord injury, and pneumothorax.[41] Therefore, clinicians have preferred ultrasound-guided fascial plane blocks in recent years.

Erector spinae plane block

Although the exact mechanism of action is somewhat controversial, the clinical and radiologic studies suggest that ESPB provides successful thoracic analgesia.[34,41,42] The advantages of ESPB are that it is performed with ultrasound guidance and the transverse process is an anatomical natural barrier in front of the pleura. Local anesthetic injected in the fascial plane between the erector spinae muscle and the underlying transverse process both spreads in the anterior posterior way and blocks the ventral/dorsal rami. ESPB provides multidermatomal blockade of the hemithorax with a single injection.[34,41] There are studies and case reports indicating that ESPB is effective in the treatment of both acute and chronic pain after cardiac surgery.[43,44] An important advantage of ESPB in the treatment of chronic pain is that a catheter can be inserted into this area.[45]

Parasternal blocks

Parasternal blocks are performed in the parasternal region and are of two types, superficial and deep parasternal intercostal plane blocks (PIPB), depending on whether they are performed superficially or deeply on the intercostal muscles.[34] They provide sensorial blockade in the T2-T6 dermatomes. Randomized clinical studies have shown that the effectiveness of superficial and deep PIPBs is similar.[46] PIPBs cannot cover the dermatome area below the T6 level. A novel block, rectointercostal block, can be used in patients who develop CPSP due to epigastric drains used in open cardiac surgery performed with median sternotomy.[47,48]

Interpectoral (IPP) and pectoserratus plane blocks (PSP)

IPP and PSP block (formerly known as PECS II) is a pectoral region block. This technique covers T3-T6 intercostal nerves, intercostobrachial nerve, long thoracic nerve, and medial/lateral pectoral nerves. IPP and PSP block is an anterior chest wall block, and it is performed by administrating the local anesthetic solution to the surface and deep to the pectoralis minor muscle. This may be performed for the anterior and lateral chest wall analgesia in CPSP patients.[34] It may be useful for epically minimally invasive cardiac surgery (MICS).

Superficial and deep serratus anterior plane blocks (SAPB)

The SAPB is a lateral chest wall block, and it covers the lateral branches of the T3-T9 intercostal nerves. The local anesthetic is administrated to the surface or deep of the serratus anterior muscle. Based on the dermatomal coverage, it is more suitable for pain management after MICS. In patients who develop CPSP after MICS, SAPB may be preferred depending on the dermatome area.[34]

The literature evaluating the effectiveness of regional anesthesia techniques in managing chronic pain following cardiac surgery remains limited. The CPSP-Cardiac study holds significant promise as a comprehensive investigation into the impact of regional techniques on chronic pain, offering valuable insights into this underexplored area.[5]

Nonpharmacological Approaches to Prevent Chronic Pain

Nonpharmacological methods, in addition to pharmacological treatments, are known to play an important role in reducing chronic pain after cardiac surgery. These methods provide a holistic approach to treatment by targeting both the physical and psychological dimensions of pain.[49] However, the literature presents varying results regarding the effectiveness of these methods.

Although supported by low-quality evidence, it is thought that physical activity and exercise can improve physical function and quality of life by reducing the intensity and frequency of pain.[50] Transcutaneous Electrical Nerve Stimulation has been shown to reduce pain scores on the second and third days after surgery in patients who underwent thoracotomy for malignant lesions as well as to improve their long-term quality of life.[51] Psychological interventions hold significant importance in the management of chronic pain after cardiac surgery. Clinical hypnosis stands out as a method among these interventions, reducing the intensity, impact, and analgesic requirements of pain by inducing changes in the sensory, emotional, and cognitive dimensions of pain. Furthermore, cognitive-behavioral therapy and progressive relaxation techniques are other effective psychological approaches that can contribute to chronic pain management during this process.[52,53]

As mentioned in PROSPECT recommendations, massage therapy and music therapy are suggested as complementary approaches to pharmacological interventions. Studies have shown that hand massage significantly reduces postoperative pain and improves pain scores compared to placebo interventions (e.g., simply holding the patient's hands). Foot massage, Swedish massage, personalized massage, and general massage have also been reported to have positive short-term effects on pain scores. However, objective measures such as analgesic requirements, length of hospital stay, and functional recovery have not been consistently reported in all studies.[32] Considering that even patients under general anesthesia can perceive environmental stimuli, the use of music therapy in operating rooms and postanesthesia care units is recommended. Music therapy can positively contribute to the recovery process by reducing pain scores. In addition to massage, methods such as osteopathic manipulative treatment, heat therapy, cold therapy (cryotherapy), and acupuncture can be effective in managing postoperative pain. In critically ill cardiac surgery patients, nutritional support, sleep, and rest are important elements supporting pain management.[32] Each of these complementary methods can be incorporated into an integrated treatment plan to enhance the effectiveness of pharmacological approaches and support the recovery process for patients.

Preventive Strategies

Preoperative assessment and counseling

A thorough preoperative evaluation is essential to reduce the risk of CPSP in cardiac surgery patients. Tailored strategies based on these assessments can optimize patient outcomes.

Preoperative strategies

Key demographic and clinical risk factors, such as advanced age, female gender, high body mass index, chronic pain history, and comorbidities (e.g., diabetes, osteoarthritis), should be identified. Frailty assessment is also critical, given its strong correlation with CPSP development.[3,17,21,54]

Psychological risk factors, including anxiety, depression, post-traumatic stress disorder, and pain catastrophizing, should be systematically assessed.[3,54] Incorporating a pain psychologist or integrative pain management team can support patients with significant psychological risks. Additionally, patient education about postoperative pain expectations and management strategies has been shown to improve satisfaction and reduce pain severity.[3,55]

Intraoperative interventions

Effective intraoperative pain control is vital to prevent the progression from acute to CPSP.[54,55] The use of remifentanil should be avoided due to its association with opioid-induced hyperalgesia and increased CPSP risk. Instead, opioid-sparing approaches should be prioritized, including multimodal analgesia and regional anesthesia techniques such as thoracic paravertebral blocks, parasternal blocks, and truncal fascial plane blocks.[41] Adjuvant therapies, including ketamine, gabapentinoids, and acetaminophen, may further enhance pain control.[56] Although minimally invasive surgical approaches facilitate faster recovery, they may still be associated with significant postoperative pain.[57] Minimizing sternal retractor trauma and using prophylactic sternal plating in high-risk patients may mitigate CPSP.

Postoperative care

Enhanced Recovery After Surgery protocols specific to cardiac surgery aim to integrate multiple objective variables to optimize patient outcomes and implement strategies to prevent the development of chronic postoperative pain.[58] Early implementation of integrated postoperative multimodal analgesia with opioid-sparing regimens, removal of chest drainage tubes on the first postoperative day, early mobilization, removal of sternal wires following complete healing, and incorporation of interdisciplinary pain management approaches in patients with baseline pain are critical components.[56,59]

Chronic Pain in Pediatric Patients

Advances in pediatric cardiac surgery have significantly improved surgical outcomes, making CPSP a more significant issue in this patient group with a long life expectancy.

Prevalence

Taking into account all surgical interventions, the prevalence of CPSP has been shown to be similar to that in adults, ranging between 20 and 40% approximately 1 year after surgery in children, adolescents, and young adults.[60] However, for cardiac surgery, there is very limited information on the prevalence of CPSP in children and a lower incidence compared to adults is suggested. In a study conducted by Lauridsen *et al*.[61] involving 171 children aged 0–12 who underwent cardiac surgery, the frequency of pain observed after 3 months was reported as 20%, with pain localized to the scar region associated with sternotomy.

Risk factors

Age, psychosocial behavior, preexisting pain, postoperative pain intensity, functional status before surgery, type of surgery, and parental factors have been identified as potential risk factors for the development of CPSP in children. However, the extent to which psychosocial factors contribute to CPSP has not yet been determined. Most studies agree that as existing anxiety, depressive symptoms, sleep quality, and pain catastrophizing by parents are strongly associated with CPSP.[62]

Mechanism

It is necessary to understand the mechanism that leads to CPSP in order to prevent or minimize it; however, sufficient knowledge to explain it in children is currently lacking. Similar to adults, mechanisms in children are thought to involve inflammatory changes mediated by neurotransmitters, receptors, cytokines, genetic factors, and modifications in gene expression, as well as structural and functional changes in the brain associated with the chronic pain.[63]

Prediction and prevention strategies

Strategies to predict and prevent CPSP should encompass preoperative, intraoperative, and postoperative interventions. Although proper management of acute postoperative pain reduces CPSP risk in adults, this has not been demonstrated in children. Furthermore, no specific anesthetic or perioperative analgesic technique has been identified that has been shown to prevent CPSP. The results of studies on the effects of preoperative anxiety of the child and the parents on the development of CPSP remain controversial. Recent publications have found insufficient evidence to support a strong relationship between perioperative anxiety and CPSP in pediatric populations. However, other psychological factors such as anxiety, sensitivity, and catastrophizing may potentially predict pediatric CPSP.[64]

Perioperative analgesic approach

In pediatric cases, current analgesia protocols are predominantly designed based on the type of surgery rather than individual needs and are typically limited to the early postoperative period. Currently, no analgesia regimen is known to prevent the development of CPSP in children. However, it is accepted that an effective pain control that is individualized and supported by opioids, regional methods, and adjuvants will contribute to the prevention of CPSP.[63]

Treatment

The treatment of chronic pain in children is a multidisciplinary approach, including psychological, physical, or occupational therapy as well as other supportive services such as acupuncture, massage, nutrition, or school-based services.[60] Evidence regarding pharmacological agents for chronic pain treatment in children is limited, with relatively few low-quality studies available. Consequently, data extrapolated from adult populations are often relied upon. Although the evidence for gabapentin or pregabalin, tricyclic antidepressants, and serotonin reuptake inhibitors is weak, they have been shown to be effective in some cases.[65] Acetaminophen, ibuprofen, and other nonsteroidal anti-inflammatory drugs are frequently used, while opioids may be an option for treating severe pain.[66] Nonpharmacological methods have also been suggested to be as beneficial as pharmacological approaches, though this has not been specifically studied for cardiac surgery.

Future Insights and Innovations

Although recent studies have primarily focused on procedure-related causes of chronic postsurgical pain after cardiac surgery—such as transitioning from conventional to minimally invasive techniques or using strategies like regional anesthesia and multimodal drug therapies—it is equally important for surgeons, anesthesiologists, pain specialists, and potentially neuroscientists to focus on patients who experience this pain more frequently or more intensely. A comprehensive, patient-centered preoperative assessment, covering aspects such as pain catastrophizing, pain tolerance, and biomarkers related to chronic pain, should guide the development of strategies to prevent and reduce this condition.

When approached from the perspective of anesthesiologists, our role involves both predicting individuals who are likely to experience significant pain during preoperative evaluations and developing preventive strategies for patients prone to chronic pain. At present, the most prominent focus in this regard, as mentioned earlier, is studying the effectiveness of regional anesthesia strategies. These studies should encompass blocks applied at all levels and layers, from neuraxial to parasternal, as well as combinations of techniques that may be insufficient on their own and require complementary methods to enhance their effectiveness.

The establishment and widespread adoption of transitional pain centers, which aim to manage postoperative pain and prevent its progression to chronic pain, would represent a crucial step in preventing and reducing chronic pain following cardiac surgery, which is the focus of this article. These centers rely on a multidisciplinary approach, involving anesthesiologists, physiotherapists, psychologists, nurses, and other specialists as needed.[67,68] Setting up transitional pain centers in major cardiac surgery units, enabling real-time sharing of data with clinicians, and ensuring their rapid dissemination could be among the most significant steps toward achieving this goal.

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Conflicts of interest

There are no conflicts of interest.

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