

# Systematic Review Article: Strategies for the prevention of postoperative chronic pain: Perioperative pain management after total joint replacement: a systematic review


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## ABSTRACT

**Objective:** Postoperative chronic pain (CPSP) is a common complication in musculoskeletal surgery. Physiotherapists usually treat patients with pain complaints before and after musculoskeletal surgery. The purpose of this article is (1) to identify the nature, process and importance of CPSPs; (2) expresses the need to understand and solve the group's difficulties. We use a full collaborative transformation model to analyze pain processes and pain management strategies. **Method and materials:** This study is a systematic review. **Results:** Physicians who understand how inflammation alters the function of the immune system can select pain targets for their patients. Clinicians should not assume that patients on multidrug therapy have a medical problem. Instead, the current approach is to manage pain using preventive strategies to reduce the severity of postoperative pain and minimize the development of CPSP. **Conclusion:** The role of biological, surgical, psychological, and patient factors in the transition to chronic pain requires further research to better understand their relationship to pain. Serious illness and drug use. Appropriate assessment and management of risk factors for CPSP requires a team discussion to describe the patient's experience of postoperative pain and to assess the pain that occurs during surgery.

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## Introduction

**M**ost patients undergo musculoskeletal surgery and return to their basic activities or enhanced activity levels within a few months.

However, a significant percentage of people still have significant pain after surgery, and up to 30% may develop chronic pain syndrome (CPSP). These statistics are alarming due to the increasing demand for total hip pain, and for patients with severe pain and disability due to knee replacement surgery and osteoarthritis, this treatment will reduce pain and improve function [1]. Recently, great attention has been paid to the difficult question of how to manage the pain of these patients. Total hip and knee arthroplasty (THA and TKA) is associated with severe pre- and postoperative pain [2-4].

Poor postoperative pain may increase the risk of delayed and/or functional impairment and the development of chronic conditions. Due to significant advances in surgical and orthopedic techniques, total hip arthroplasty (TJA) has become the treatment of choice for patients with hip or knee osteoarthritis to reduce pain and improve joint function. National Institutes of Health and several Canadian federal and provincial groups have determined that TJA or THA is a safe and effective procedure for people with knee or joint pain and disability. reduce discomfort [5-7]. During the decade from 1991 to 2000, the number of primary TKA and TKA procedures more than doubled in the USA, UK, and most Western countries. However, it was confirmed that up to 33% of patients reported functional impairment. improvement and 10% to 30% of patients reported no improvement (or worsening) of pain after TKA or TKA [8-10].

This article describes effective strategies used to manage pain and thereby promote good functioning after surgery. Postoperative assessment and pain scores alone are insufficient to understand the severity of postoperative pain and its relationship to the development of CPSP [11]. The necessity of a collaborative team to participate in the patient's physical recovery and dynamic pain assessment

(motivational) is also discussed. Many doctors hope to reduce the development of CPSP by controlling the severity of pain [12]; Unfortunately, data to support this idea is scarce. Finally, we discuss the underlying physiological mechanisms and risk factors that contribute to the development of chronic pain after surgery.

## Physiology of Postoperative Pain

Classical pain is defined as a neuronal signal that reaches the brain through nociceptors in the spinal cord resulting from a peripheral injury or from the absence of any event. Nociceptors Sensory neurons convert negative physical stimuli into electrical signals (i.e. action potentials), which are then sent via the spinal cord to the location of the negative energy and applied to the brain. 26 However, this theory of pain perception fails to integrate the role of the cerebral cortex in sensation and control [13].

After surgery, inflammatory mediators released from damaged tissue initiate an inflammatory cascade. This inflammatory response lowers the threshold and activates the response of nociceptors (sensory receptors of C and A $\delta$  fibers) to material that is then used by the damaged tissue, a phenomenon known as peripheral sensitization. The neurophysiological response to an injury, including surgery, can be protective (ie, limit pain) and accelerate recovery [14-16].

It is now known that the sensitive process may occur more frequently because of the "attack" that makes the nociceptors respond to surgery. The term "central sensitization" refers to changes in the response of central neurons (eg, the dorsal horn of the spinal cord); Features of central sensitization include responses to activation, contraction, enlargement, and self-activity after injury. All of these can increase postoperative pain [17-19]. The main mechanisms responsible for the development of CPSP have not been identified; however, it has been shown that blocking or reducing peripheral and central sensitivity can inhibit the process that leads to the development of CPSP. Peripheral and central sensitization mechanisms, which are the first defense, may

not breathe, have harmful consequences, and may become more resistant to treatment [20].

Postoperative pain is usually caused by two different mechanisms: nociception (C fibers and A $\delta$  primary sensory receptors detect swelling). Chronic pain such as neuropathic pain caused by direct injury to nerves (eg. For example, nerve cut), also in the postoperative period. Although most neuropathic pain resolves over time, in some cases, neuropathic pain caused by surgery can develop into chronic neuropathic pain that is often resistant to treatment. These injuries cause long-term changes in brain sensitivity that reduce the intensity of subsequent stimuli required to cause pain (eg, the patient's pain threshold decreases) [21].

It is now accepted that anesthesia can reduce but not completely prevent signal transmission from the periphery to the afferent lesion dam, spinal cord, and brain. Also, opioids may not provide bone density to prevent heart attacks. What these findings mean for patients receiving intraoperative anesthesia is that the sensitization process of dorsal horn neurons, although unconscious, is not affected by the use of anesthesia or opioids. For many years, the main treatment for postoperative pain has been opioid-based analgesia [22].

### Multimodal Analgesia

With advances in the understanding of the pathophysiology of pain, multimodal analgesia has become the standard of practice for the management of moderate to severe pain after orthopedic surgery. This application includes the use of different classes of pain relievers with different administration methods to (1) provide optimal therapy at rest and during exercise, (2) reduce opioid consumption, and (3) reduce adverse side effects [23]. A number of clinical studies have demonstrated the efficacy of multidrug therapy; however, the positive results have not translated into clinical practice. Although monotherapy may cause a central nociceptive effect, multimodal analgesic therapy is more effective and generally has fewer side effects than high-dose, single-dose opioid therapy. As discussed below, clear evidence from clinical trials indicates that many

pharmacological treatments using combinations of analgesics each have different mechanisms of action and are effective in blocking multiple devices/receptors.

For example, the results of two recent studies show that pain severity can be effectively managed after TKA or TKA using a variety of treatments. 44,48 Gabapentin, nonsteroidal anti-inflammatory drug multimodal pain therapy (Celebrex), acetaminophen and local anesthetics are effective in controlling perioperative pain and reducing perioperative opioid use. More data are needed to determine whether multimodal analgesic treatments are compatible with regional anesthesia, an anesthesia that affects most patients in the body. and/or peripheral nerve blocks - reduce recovery time for patients and/or increase amount Good recovery for patients after orthopedic surgeries such as THA or TKA.

### Prophylactic methods to reduce postoperative pain and chronic pain

#### Preemptive analgesia

Preemptive analgesia is defined as preoperative antinociceptive therapy that prevents surgery-related central sensitization and postoperative pain severity. 49 The classical design for assessing allergic reactions requires that both groups receive the same treatment before and after surgery; The difference between the two groups is the duration of analgesic administration according to the incision. This finding suggests that dysfunction of the body's nociceptive dam contributes more to central tenderness and pain than postoperative nociceptive dam. It was assumed that the pre-operatively treated group would have fewer patients than the post-operatively treated group [24].

This view of preemptive analgesia is local and narrow, but, in part because we know that (1) factors other than the peripheral nociceptive dam are associated with the incision and that subsequent harmful intraoperative events can lead to death, Min and (2) preoperative, intraoperative, and operative We do not know

which peripheral input causes post-operative central tenderness and pain [25].

### Prophylactic Analgesia

A comprehensive approach to the prevention of postoperative pain has been developed that aims to minimize the immediate and long-term effects of unwanted perioperative afferent inputs. The purpose of preventive analgesia is to reduce the effects of peripheral nociceptive inputs associated with poor preoperative, intraoperative and/or postoperative events/stimuli, not in the relative duration of anesthesia or analgesia [26-28]. These stimuli cause peripheral and central sensitization leading to postoperative pain and the need for analgesics; Preventing sensitization will reduce pain and the need for treatment. Prophylactic analgesia occurs when postoperative pain and/or anesthesia decreases during the observation period of the target drug (mean 5.5 half-lives of the target drug). This requirement ensures that the observed results are not an optimization. Until recently, clinical trials have begun to examine the impact of perioperative interventions well beyond the surgical era (eg, the quest for prevention). Long-term follow-up of patients weeks, months, or years after surgery is necessary to assess the preventive effects of perioperative interventions [29].

### Analgesics commonly used for perioperative pain management

#### Opioids

Opioids such as fentanyl, morphine, meperidine, hydromorphone, and oxycodone are widely used worldwide for postoperative pain management. Opioids remain the mainstay of perioperative analgesia, as they are best for moderate to severe pain and have been shown to be effective in many situations, most likely because they work in, on, and around the spine. Perioperative pain management often relies on opioids as the primary anesthetic [30]. However, as noted above, opioids are generally ineffective for severe exercise-related pain and have short-term side effects (such as pressure, vomiting, sedation, itching, constipation, urination, and difficulty breathing), patients. back. factor. Studies have shown that treatment-related

opioid tolerance may occur during the period of intraoperative use. The development of intraoperative tolerance can reduce the postoperative analgesic effect of opioids, delay hospital discharge, and lead to long-term development that can eventually lead to physical dependence and dependence [31].

Many post-operative pain strategies used by anesthesiologists rely on other classes of drugs to reduce the amount of opioid analgesics taken by the patient. Adherence to multiple treatments at baseline may reduce the side effects of opioid analgesics.

#### NSAIDs Drugs

The anti-inflammatory properties of NSAIDs are due to their anti-inflammatory properties in inhibiting prostaglandin synthesis. The synthesis of prostaglandins is important for the production of inflammatory states, which depends not only on the production of prostaglandins in the pain zone, but also on the action of prostaglandins synthesized in the brain [32]. Prostaglandins are derived from arachidonic acid and are released from phospholipids in cell membranes by the action of the phospholipid A2 (PLA2) enzyme. Cyclooxygenase (COX) catalyzes the first two events of the prostaglandin pathway. The identification of two COX subtypes, COX-1 and COX-2, has led to significant efforts to determine the relative role of each subtype in prostaglandin production under certain conditions<sup>62</sup> and to develop COX-2 selective inhibitors, because COX-2 is responsible for inflammation and pain [33].

The discovery that the inflammatory cascade caused by tissue damage can be reduced peripherally and attenuated at the spinal cord level has increased the use of NSAIDs as an aid in postoperative pain management. Oral and intravenous NSAIDs have greater bioavailability than rectal administration; therefore, previous management is better after surgery. <sup>65</sup> However, NSAIDs are not widely used because of their potential side effects such as abdominal pain or bleeding, kidney failure, and platelet inhibition. Obviously, NSAIDs should be used with caution in patients with a history of renal



failure, sepsis, end-stage liver disease or heart disease. Evidence suggests that selective NSAIDs (eg. For example, ketoprofen / ibuprofen) are safe for stomach ulcers. Regarding bone healing, animal data suggest a link between bone loss and NSAID use,<sup>68</sup> but clinical data do not support this link. A 2-year randomized study showed that celecoxib (Celebrex) for 3 weeks postoperatively did not affect bone healing after TKA. More research is needed on the safety of NSAIDs to evaluate the effect of reducing the inflammatory process on other tissues, such as muscle and tendon repair, and the coordinated change of functional outcomes after treatment.

While it appears that pain reliever should improve the tissue repair process, there is conflicting evidence from animal models of skeletal muscle and muscle injury and treatment, it is difficult to consider long-term antibiotic use beneficial, especially after the menstrual period. In an animal study in which non-selective NSAIDs were used to block long-term inflammation during and after the perioperative period of the disease, researchers found support (holding on the lower limbs) and increased behavior. A steady increase in activity indicates that the barrier is working well; It is not known whether similar results will occur in humans. COX-2 selective inhibitors are designed to target the COX-2 enzyme while protecting the COX-1 enzyme. The only COX-2 drug available in North America is celecoxib (Celebrex) [34-36].

Studies have shown that COX-2 selective inhibitors have an analgesic effect similar to non-selective NSAIDs, but do not affect platelet function. This reduces the risk of perioperative bleeding and means that COX-2 selective inhibitors can ensure safety first. It is particularly important in the joint population that COX-2 selective inhibitors do not affect bone healing. However, its effects on muscle and tissue repair (important for full recovery after surgery) are unknown [37].

Selective NSAID (eg. For example, non-selective or COX-2 specific) are often determined by factors such as drug formulation availability, cost, and patient tolerance. In the post-operative setting, the addition of NSAIDs to post-operative

care is often influenced by the severity of the patient's pain. Arthroplasty pain is defined as mild to severe pain within 48 hours of surgery. 1 Selective COX-2 inhibitors continue to exert beneficial effects on allergy and anti-inflammatory drugs while protecting patients from the effects of joint replacement. COX-1 agent. Celecoxib appears to be the most effective drug in multidisciplinary treatment after orthopedic surgery [38].

### Acetaminophen

Acetaminophen is a commonly available over-the-counter antibiotic and antiseptic; It is very safe and is usually prescribed for mild to moderate pain. The most common symptoms are headache, migraine, fever, flu, toothache, toothache, muscle and joint pain, and neuralgia. The anti-inflammatory effects of acetaminophen are weaker than those of NSAIDs; The lack of anti-inflammatory effects of acetaminophen may reflect low-level effects of prostaglandin synthesis produced by the drug. Conversely, acetaminophen has been shown to block the effect of endogenous pyrogens on the thermoregulatory center of the brain by blocking the production and release of prostaglandins in the central nervous system.<sup>88</sup> Some researchers have reported differences between occupations (eg. For example, the COX-3 pathway). The COX-3 pathway will strongly inhibit the synthesis of prostaglandins in the central hypothalamus, reduce prostaglandin E in the cerebrospinal fluid, and finally produce analgesic and antipyretic effects. Research is still ongoing to clarify the COX-3 mechanism of action of acetaminophen [39].

While the safety profile of acetaminophen is excellent, overdose or overdose is not uncommon. Adults or children under 12 are recommended to take 500 mg to 1 gram of acetaminophen every 4-6 hours, up to 4 grams over a 24-hour period. Although elevations in liver enzymes have been reported with long-term use of high doses, side effects rarely occur with the drug. Number to Treat (NNT) is 2.2 when acetaminophen is combined with codeine.

Comparative studies comparing acetaminophen with NSAIDs show mixed results; 96 authors

concluded that the combination of these drugs is more effective for postoperative pain than either drug alone. The efficacy of acetaminophen in the treatment of mild to moderate pain and its relatively low side effects (no gastrointestinal or platelet aggregation effects) explain why it is still used as part of post-treatment multimodal therapy [40].

### N-Methyl-D-Aspartate (NMDA) antagonists

NMDA antagonists have been the focus of intense research for the discovery of centrally located receptor-ion channel complexes and play an important role in pathological pain induction and maintenance of receptor-ion channel complexes. In particular, ketamine and dextromethorphan have been studied for their ability to provide postoperative pain relief. Low-dose ketamine has been shown to reduce the need for opioids, prolong the duration of opioid/epidural analgesia, and reduce pain. Ketamine was originally developed as a general anesthetic; However, its perioperative use is decreasing due to significant psychological complications. Recently, drug consumption is low (from 0.25-0.5 mg/kg as an initial bolus, then 50-500  $\mu\text{g/kg/hr}$ ) has been suggested as an adjunct to postoperative analgesia and reduction of exogenous opioid-induced hyperalgesia. A protective effect after TKA (more than 5.5-fold increase in mobility observed with ketamine use, more than half of life expectancy [41].

Patients receiving continuous injections of ketamine (3  $\mu\text{g/kg/min}$ ) up to 48 hours after induction of anesthesia) - Surgery, 6 days post-surgery then showed that it improved the best knee flexion. This improvement in movement was maintained at 6 weeks and 3 months after surgery compared to patients receiving placebo. Ketamine and dextromethorphan are not used as part of a multidisciplinary treatment regimen, although there is evidence that NMDA antagonists reduce post-opioid requirements and acute pain in many other surgical populations. One of the medical problems with its use is the lack of oral formulations of ketamine [42]. An oral formulation of dextromethorphan is already available in some

countries; Doctors recommend using oral medication to maintain NMDA receptor blockade after leaving home. 108 More research is needed on the efficacy of dextromethorphan for out-of-hospital use as an adjunct to pain.

### Anticonvulsants

Gabapentin and pregabalin are considered  $\alpha 2\delta$ -subunit voltage-dependent calcium channel blockers, but their mechanism of action is not fully understood. Gabapentin, a standard analog of GABA, has entered clinical practice as an anti-inflammatory drug; A new  $\alpha 2\delta$  ligand, pregabalin, has been introduced as a therapeutic agent for neuropathic pain. Recent studies have shown that gabapentin increases tonic inhibitory current in rat hippocampal neurons. Efforts continue to elucidate the central role that gabapentin may play in the mammalian central nervous system. It is likely that peripheral and central mechanisms mediate the therapeutic effects of these drugs [43].

Over the past 8 years, several studies have examined the efficacy and effectiveness of gabapentin in the treatment of post-traumatic stress disorder. The surgeries studied include abdominal or abdominal surgery, musculoskeletal surgery, 119-126 head and neck surgery, thoracic surgery, varicocele surgery, thoracic surgery and more recently cardiac surgery. In these RCTs, there was usually a single dose of gabapentin prior to surgery. Only the four trials mentioned above failed to demonstrate postoperative pain reduction or reduced opioid-sparing effects. Recently, Mattison et al. Proven efficacy of pregabalin in reducing opioid consumption after THA. Several meta-analyses have confirmed that these antidepressants reduce opioid use and postoperative pain scores [14].

Most studies to date have compared gabapentin with placebo. Gilron et al. and Turan et al. published studies compared gabapentin and celecoxib in combination with each drug alone. Both studies showed similar results: The group that received multiple interventions showed better opioid savings and pain relief than the single-drug group. Similar results have been shown for the efficacy of multi-dose

chemotherapy after THA or TKA. A recent study showed that gabapentin was maintained for 4 days postoperatively by peripheral nerves and celecoxib was effective. This combined treatment resulted in an increase in knee flexion during rehabilitation [15].

One of the challenges of this experiment is the use of negative outcomes (eg, multiple movements) that are not necessarily related to functional ability (eg, ability to climb stairs). Osteoarthritis (OA) is the most common cause of TKA, which makes climbing stairs and walking more difficult than other diseases. As one of the main goals of TDA is to improve physical function, this provides an ideal model to study the effect of gabapentin on functional performance. Although many trials have shown a reduction in exercise due to diabetes, it is still unknown whether this reduction translates to faster recovery and/or improved work performance. Future studies should evaluate the effect of gabapentin and pregabalin on recovery after TJA in the context of multimodal analgesia. Results from these tests should include self-reported and physical activity measures such as the 6-minute walk test, the timed walk test, and the timed exercise ladder, all of which have been shown to be reliable and sensitive to changes in the TJA population [16].

### Local anesthesia

Local anesthesia has been used for hundreds of years to provide anesthesia. Today, local anesthesia remains the most important tool in reducing pain, opioid consumption and opioid side effects. 151 Regional anesthesia (use of local anesthetic) is an important part of orthopedic anesthesia. As part of anesthetic care, local anesthesia has been shown to facilitate rapid movement and hospital discharge. local anesthetic (eg. For example, bupivacaine and lidocaine) are popular sites for use, often in the intraspinal (spinal/epidural) and some joints.

Spinal/Epidural anesthesia has become the anesthetic of choice in lower extremity joint specialists. These centers have created special areas where patients are removed from surgery (spinal/epidural) and peripheral nerves

(femoral and/or sciatic nerve/catheters) before entering the operating room. This is one of the main factors driving an increase in surgery and reduced waiting times for surgery in Ontario.

Prior to the introduction of pain management techniques, anesthesiologists faced the challenge of preparing regional anesthesia for patients undergoing orthopedic surgery, mainly because time in the office was high. The addition of a preoperative field of anesthesia allows for intrathecal/spinal anesthesia and regional anesthesia in patients undergoing major orthopedic surgery. The need for reduced waiting times and increased surgical procedures has led to strong collaboration between anesthesiologists, physiotherapists, nurses, and surgeons to improve the safety of patients undergoing THA or TKA. Intra-articular injection of local anesthetic has been shown to improve pain scores, reduce opioid consumption, and promote recovery of function and range of motion after knee arthroscopy. Although the duration of pain relief may be shorter with peripheral nerve catheters and neuraxial analgesia, these techniques are used in outpatient orthopedic surgery (eg. For example, arthroscopy). Ongoing studies are investigating the effectiveness of intra-articular injections in THA and TKA patients.

### Regional anesthesia service after musculoskeletal surgery

#### Single peripheral nerve blocks

Femoral and sciatic nerve blocks after TKA. The femoral nerve innervates the anterior compartment of the knee joint, and the sciatic nerve innervates the posterior compartment. Pain behind the knee can only be a problem in patients who get a femoral artery, so some doctors say it's still good pain to give double block after TKA. Popliteal nerve blocks (for the sciatic nerve passing below the knee) used in foot and ankle surgery provide complete pain relief with minimal side effects. The duration of the pulses can vary.

#### Continuous Peripheral Nerve Catheters

By placing a catheter in the perineural space, the duration of anesthesia can be extended and the

amount of blockage during anesthesia can be better controlled. Most studies comparing this approach to direct anti-inflammatory (PCA) control in patients have shown better allergy and analgesia at rest and at work, and better analgesia after knee flexion and early hospital discharge. Unfortunately, the increased cost of this procedure and the lack of expertise in peripheral nerve catheter placement often limit its use. When using peripheral blood vessels (injections or catheters), it is important that the anesthetist and physiotherapist participate in the discussion to achieve healing goals and not delay healing due to too much energy [17].

### Postoperative Fear, Anxiety and Pain

Biopsychosocial models now suggest that fear and anxiety-based pain models are important for the development of pain, but this has not been adequately studied. Recognizing the importance of pain-related fear and anxiety in the transition to chronic pain, this section explains how these processes affect acute and chronic pain and how they can affect the body in patients after surgery.

The most common emotions expressed by patients preparing for major surgery are fear and anxiety. These are two very different theories, although they have many similarities. The events and conditions that led to them and the concepts associated with these emotions are often similar; One of the main differences between fear and anxiety has to do with the physical relationship to the threat: fear is defined as an emotional response to an existing and ongoing threat, when stress is the result of potential or potential. Therefore, the behaviors accompanying fear and anxiety are different; avoidance and bad behavior relate to the past and the future, respectively. Avoidance behavior is often caused by stress. Many patients worry that exercise will cause relapse and pain. For example, pain in the days and weeks after surgery can inhibit movement; Deep breathing, coughing, laughing and getting out of bed may be painful depending on the location of the surgery. In anticipation of pain, many patients fear the results of exercise and avoid exercise altogether. It is important to understand the

personal meaning of fear or anxiety. Patients may believe that if they sit or move, their stitches will break and the wound will open; they may also be concerned that these activities will cause permanent damage. Other patients worry that exercise will make the pain worse; In severe illness, they may feel helpless or dependent on medical care. Concerns about exercise after surgery are based on guidelines that tell patients that exercise causes pain [48].

However, misinterpretation of activities and pain from danger can lead to avoidable behaviors that result in weakness, pain and disability. It is important that the physiotherapist and anesthesiologist work together in order for the patient to understand the difference between trauma and injury and to reduce the pain that may occur.

Although avoidance through social withdrawal and inactivity can reduce pain, especially in the early stages, these behaviors can be harmful over the long term. This negative belief includes the expectation that certain physical activities (such as physical activity, work) will affect pain. Avoidance behaviors (such as grooming, cruelty, social withdrawal) may be part of coping with ongoing pain or suspected trauma; However, some of these behaviors may also represent a negative way of dealing with life's problems. Therefore, poor people may not be able to avoid bad jobs or housework, marital discord, and difficult relationships. Pain behaviors that have the dual purpose of avoiding thinking about pain and avoiding other negative life events can lead to pain for many negative reasons [4].

The patient's fear of avoidance, negative beliefs and negative expectations of needing activities that reduce pain through avoidance. As pain progresses, the frequency of avoidance behavior tends to increase, and therefore pain behavior and pain intensity increase. This conflict leads to lower self-esteem and more avoidance. For this reason, patients report that they gradually lose control of their pain when they try to control their behavior by preventing their pain from going away. As patients gain a more accurate assessment of the relationship between pain and behavior, self-management strategies that focus



on negative emotions and negative behaviors can be used to increase self-efficacy and reduce pain. Although the use of cognitive coping strategies during acute illness may be beneficial, the use of these strategies harms mental health, leading to more illness and disability. Preventive strategies will be implemented soon after injury as they can reduce pain and reduce the risk of further injury causing severe pain [10].

But in retrospect, these strategies fail because they encourage continued isolation, failure, and lack of effective testing. Recent controlled studies and experimental data show that fear-related pain can be effectively treated with in vivo interventions in which patients experience fear-inducing and subsequently body movements. The results showed reduced perceptions of severe injury, severe pain, and disability. To our knowledge, there are no studies evaluating the efficacy of this treatment in patients with severe or chronic post-surgical pain.

### **Transition from pain to chronic pain after surgery**

The incidence of CPSP varies with the type of surgery, but even the lowest estimate is high. 178 The data show the 1-year incidence as 10% and 10%. This rate increases to 15% after modified radical mastectomy and to 61-70% after open surgery. Identifying the factors that cause the transition from acute, time-limited pain to chronic pathological pain is crucial if we want to provide effective prevention. Research aimed at identifying potential long-term adaptations is at an early stage. Most studies are cross-sectional and cross-sectional and aim to identify factors associated with the development of CPSP; some studies have used longitudinal models to determine the temporal sequence of CPSP development.

Finally, many studies in the anesthesia literature have attempted to control the risk of complications by using drugs that block postoperative pain or prevent the intervention of neurologists and doctors after surgery. According to a recent review, studies have confirmed previous suggestions that chronic pain can be reduced or prevented with

antibiotics. Intensive perioperative multimodal therapy; However, other studies have failed to show this effect. Currently, predictors of transition to CPSP are disaggregated by biological factors (eg, young age and gender); operation; cleaning; and individual or patient differences [51].

### **Surgery and CPSP**

The following procedures are associated with prolonged pain probability: increased operative time, low (high) surgery, open (laparoscopic or arthroscopic) surgery, hernia repair, pericostal (and intracostal) suturing, and nerve pain. during surgery. It is unclear whether the above conditions are associated with the development of chronic pain. Often there seems to be more injury in these situations; they specifically point to brain damage as a possibility, but this needs to be proven.

### **Psychosocial Factors and CPSP**

Patients experience fear and anxiety about surgery and many issues such as the hospital environment, discharge, treatment procedures, pain, uncertain diagnosis, and postoperative pain during and after the surgery. Pain is caused by many factors, including culture, meaning of the situation, changes in perception and influence, perception of control, instructions and placebos, fear and anxiety [42].

It has been shown that preoperative anxiety and fear-based states are associated with (1) postoperative pain and analgesic consumption and (2) the development of CPSP. Other research has shown that certain mental disorders (for example, cognitive impairment measured after hospital discharge) predict disability band after CPSP and disease by itself – present or past. This appears to be true for all types of surgery regardless of time.

The presence or severity or duration of previous pain is a risk factor for postoperative pain, days and weeks of pain, and prolonged post-operative pain. Not only preoperative pain intensity, but also pain intensity days and weeks after surgery predicted the development of CPSP (with the exception of Katz etc.). Nothing can influence the

development of future pain problems more than the pain itself. One thing to consider, however, is the level of pain that can be predicted. Many competing theories explain why pain occurs, including those that suggest a cause or effect of pain; More research is needed to evaluate these theories and the role of pain and other factors in the development of CPSP [43].

### Summary

While biological (female and adolescent), surgical, psychiatric, and patient factors have been reviewed above, their role in growth and disability in the CPSP process has not been established. If a relationship is just a relationship, it is likely to have risks and consequences from one or more factors. The importance of creating these social contexts is clear; once some risks are identified, they will be modified or removed to reduce the risk of CPSP development and their impact on related projects will not affect the development of CPSP.

### Clarifying the importance of outcome measures

The main outcomes of clinical trials are usually pain severity (or presence/absence) and medication use. It is difficult to find more studies to measure the results. Recommendations for primary monitoring and primary outcomes in clinical trials include routine measurements (for example, comparison of pain and antibiotic use) and affective disorders, mental, emotional, and physical changes.

Evaluation of other activities and side effects during surgery can help predict the severity of postoperative pain, postoperative complications, and the risk of developing CPSP. Understanding the relationship between a patient's pain and function and minimizing the development of CPSP is important, especially for patients who have had TKA or TKA.

Patients with severe osteoarthritis often work regularly for months or years and experience severe pain at night after menstruation. Because OA has no cure, treatment strategies focus on reducing symptoms (i.e. pain and swelling) while maintaining good function.

TJA has become the treatment of choice for patients with severe OA. While the results of TJA are generally considered excellent, the reality is that 10% to 30% of patients still have pain and approximately 20-33% of patients do not report pain. Pain improves even 1-2 years after transplant. With ongoing illness, health problems will develop in the future, perhaps these statistics are not surprising. Identify individuals at risk for CPSP and identify the cause of back pain (ie transmission of mechanical resistance is neurological).

These measures include auditory, physical, biomechanical, psychological, and physiological measures, as well as improving the quality and quantity of pain, and have been studied after TJA without the use of OA and joints. Canada's aging population means the TJA process will take years. Reducing the proportion of patients who continue to develop CPSP after TJA requires concerted effort, particularly by anesthesiologists, physical therapists, and orthopedic surgeons.

### CONCLUSION

The transition from postoperative pain to pathological chronic postoperative pain (CPSP) is a complex and poorly understood process involving biological, psychological, and social factors. The effects of surgery and the need for early recovery and good performance compete with the benefits of perioperative prophylactic multimodal analgesia. These issues should be considered along with a combination of pre-existing conditions and circumstances, psychological and emotional, and social environments to determine the natural course, severity, frequency, duration, and risk of developing CPSP. Psychotherapy has proven effectiveness for other chronic conditions; however, measures to identify people at risk of developing CPSP and other CPSP prevention and treatment methods have not received the attention they deserve. The development and evaluation of various pain management strategies and their implications for surgery and management in patients requiring THA or TKA has been long overdue.

The use of supplements/caregiver programs should not be considered as an indicator of other diseases or “chronic problems” in the patient; instead, there must be a way to be effective and able to reduce long-term poverty and meet the costs of disability. The collaborating team will be able to explore new data and develop methods that can help predict how long patients will recover from surgery and which patients will continue to improve. Encouraging collaboration between anesthesiologists, surgeons, neurologists, and physical therapists will provide a solid foundation for the development of future teams to investigate and manage long-term pain and function after major orthopedic surgery.

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