The progression from acute to chronic pain

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Purpose of review

The persistence of pain after surgical procedure or trauma has become a major focus of interest and its prevention now represents a challenge as an indicator of quality of healthcare. The only way to develop effective strategies to prevent the development of chronic pain is to better understand the mechanisms involved in the progression from acute to chronic pain, with the aim to target high-risk patients and to adapt perioperative management.

Recent findings

Several important risk factors involved in chronic pain development after tissue injury have been identified, underlining how much the phenomenon is complex and multifaceted. Recent studies have highlighted some important points on which to focus, mainly during the acute and subacute postinjury periods. These studies promote a more dynamic approach related to a better evaluation of acute pain resolution using individualized pain trajectories, the assessment of endogenous pain-modulatory processes and individual's psychological flexibility when facing physical threat and pain.

Summary

A dynamic view of both physiological and psychological response of an individual after injury (trauma, surgery) should improve our ability to target predisposed patients who might develop persistent pain. We should then be more able to provide those patients with the most appropriate preventive management.

Keywords

acute postoperative pain, central sensitization, chronic postsurgical pain, psychosocial vulnerability

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Introduction

Acute pain consecutive to tissue injury (trauma, surgery) is one of the most frequent reasons why patients are seeking medical care. Whether most of the patients will recover and return to their normal life, some others will suffer chronic pain and long-lasting disabilities. In Europe, the prevalence of chronic pain reaches 20% [1] with trauma and surgery accounting for 15% of the cases. Among the patients admitted to trauma hospital after severe injury, 15% will report severe persistent pain [2]. After surgery, the incidence of chronic pain [3–5] varies between 15 and 60% (with severe disabling pain ranging from 4 to 10%), in relation with the procedure although less invasive procedures like inguinal hernia, cesarean section and cosmetic surgery may carry a non-negligible risk (Table 1). Chronic pain is a complex problem, multifaceted, with multifactorial cause. However, patients who attribute their pain to a specific cause like trauma or surgery seem to suffer higher emotional distress and higher pain than others whose pain has an insidious or spontaneous onset [23].

For several years, the anesthesiologists have been questioning the long-term impact of perioperative management and seriously considering their role in preventive medicine [24]. The persistence of pain after surgical procedure, trauma or ICU stay has become a major focus of interest and its prevention represents a challenge as an index of healthcare quality. The only way to develop effective strategies aimed to prevent the development of chronic pain is to better understand the mechanisms involved in the transition from acute to chronic pain [25,26•]. We will then be able to target predisposed patients and to provide them with the most appropriate management. The review focuses on chronic postsurgical pain (CPSP) and examines the recent developments, including predictive tools and therapeutic strategies, for some of the well-known factors thought to be involved in the progression from acute to CPSP.

Severe postoperative pain

Poorly relieved acute pain is commonly mentioned as a striking risk factor in CPSP development. Although

retrospective studies may suffer a possible bias related to pain recall, several prospective studies have also underlined the link between the severity of acute pain and persistent pain after trauma [2] and after various procedures ranging from childbirth [17] to major surgery [3–5,25]. Today, despite the availability of multiple analgesic drugs and techniques, relieving acute postoperative pain specifically movement-associated pain remains a challenge in some 30% of the patients [22]. However, the discrepancy between the prevalence of severe postoperative pain currently reported and that of CPSP may lead us to consider the fact that some patients presenting with severe acute pain will never develop persistent pain (Table 1). The question is how to target those who will be at risk for CPSP.

Key points

- Chronic pain is a complex and multifaceted phenomenon. The major risk factors involved in the progression from acute to chronic pain are well known. However, they do not apply to all the patients, while we need to target predisposed patients.
- Novel approaches focusing on a dynamic view of both acute pain resolution and individual's physiological (endogenous pain-modulatory processes) and psychological (pain flexibility) reactivity when facing pain should help to target high-risk patients.
- The subacute pain period, a neglected area of investigation, remains a 'grey zone' which might play a major role in the progression from acute to chronic pain for some patients and deserves further study.

Postoperative pain evaluation

We do not have a dynamic view of postoperative pain evolution. First, it is interesting to point out that only 40% of clinical trials currently assess postoperative pain with mobilization which is associated with functional recovery [27]. Second, a longer assessment of postoperative pain relying on the development of pain trajectories (from day 1 to days 5–6) as proposed by Chapman *et al.* [28••] should allow us to characterize individual's postoperative pain and thereby to identify abnormal acute pain resolution. Looking to the pain trajectory pattern, specifically to the slope, that is, the rate of pain resolution, 25% of patients have a flat slope and 12% show a positive slope (i.e. increase of pain). That lets us suppose that 37% of patients are living with unresolved postoperative pain at day 6 after surgery and perhaps later [28••].

Neuropathic pain component

The nature of CPSP still remains poorly defined, either ongoing inflammatory reaction or neuropathic origin; iatrogenic neuropathic pain caused by incision and tissue exposure is being thought to be the most common cause of CPSP [3]. Using adequate tools, that is, quantitative sensory testing and specific questionnaires, several studies report a major neuropathic component in CPSP and not only after limb amputation [10,13,18] (Table 1). However, not all lesions in the somatosensory system lead to neuropathic pain. Persistent sensory dysfunctions can be highlighted in pain-free postsurgical patients (e.g. 20% postherniotomy [14], 43% postthoracotomy [29] and 37% after breast augmentation [19]). Further, cofactors like poor capacity of nerve regeneration (suspected genetic origin), decreased pain tolerance (altered endogenous pain processing) and particular psychological profile play a major role as shown in the 33% of patients who developed CPSP after nerve repair [30]. Every injury including nerve trauma may trigger a specific inflammatory-immune profile, partly determined by genetic components. A recent study has implicated perioperative immune and inflammatory responses into postoperative peripheral neuropathies not related to mechanical damage [31]. After lateral thoracotomy, both the incidence (5 vs. >40%) and the intensity of CPSP are decreased in lung transplanted patients by comparison with patients

Table 1 Progression from acute pain to chronic postsurgical pain after various surgical procedures

	Severe acute pain (at 24 h)	Subacute pain (from day10 to 6–8 weeks)	Chronic pain (from 3 to 12 months)	Neuropathic component of CPSP
Limb amputation [6,7]	[30%]	50–75%	50–85%	>80%
Thoracotomy [8,9]	[30%]	39–50%	16–21%	>46%
Breast cancer surgery [10]	[30%]	16%	47%	65%
Major abdominal surgery [11,12•]	[30%]	18%	7–14%	?
Craniotomy [13]	20%	6%	7–29%	25%
Inguinal hernia [14,15]	7%	14%	12%	80%
Knee arthroplasty [16]	44%	16–52%	13%	?
Hip arthroplasty [16]	40%	20%	12%	?
Cesarean delivery [17,18]	17%	9–16%	4–10%	53%
Cosmetic breast surgery [19,20]	[30%]	25–32%	6–14%	38%
Cosmetic chest surgery [21]	[30%]	25%	14%	?

For acute pain, 30% is mentioned by default as the incidence of severe acute postoperative pain reported currently in the literature [22]. For chronic pain, the table mentions the incidence in general but not severe pain which concerns 4–10% of the patients reporting persistent pain [3]. CPSP, chronic postsurgical pain.

operated for lung cancer [8]. Such observations question the role of immune response in CPSP and the benefit of immunosuppressive therapy as preventive strategy in surgical procedures which carry a high risk for nerve damage. Regarding the aforementioned findings, a neuropathic risk factor should be more seriously considered in the acute postoperative period, specifically in patients with severe postoperative pain who fail to respond to classical analgesic treatments [32]. Although data are scarce, a prospective study reported 3% postoperative patients managed by an Acute Pain Service diagnosed with neuropathic pain [33]. During their follow-up, 78% of these patients suffered persistent pain at 6 months and 56% at 12 months. Using adequate questionnaires aimed to diagnose neuropathic pain, another study revealed an incidence of 8% for neuropathic pain immediately after thoracotomy [9]. These findings argue for earlier appropriate diagnosis and therapeutic intervention concerning postoperative neuropathic pain.

Central sensitization, hyperalgesia and pain

Nociceptive inputs from injured tissues trigger a prolonged and usually reversible increased state of nervous system hyperexcitability referred to as central sensitization [34•]. This amplification of neural signaling within the central nervous system elicits pain hypersensitivity which translates into clinical hyperalgesia, mainly evoked by mechanical stimuli. Central sensitization participates to postoperative pain, perhaps enhancing the pain experienced by the patient although the relationship between central sensitization and acute postoperative pain level is unclear [35]. Hyperalgesia is a natural phenomenon aimed to facilitate tissue healing [5], but it may become maladaptive when persisting beyond the natural recovery period. Sustained central sensitization is thought to be one of the mechanisms underlying the development of persistent pain after injury [5,34•]. Preclinical studies have undercovered various physiological mechanisms involved in central sensitization phenomenon [36]. For a long time, medical care givers have focused on preventing those central neuroplastic changes to occur, sincerely believing their treatments might stop the development of persistent pain, unfortunately with inconstant clinical results. First, the extent and the duration of central sensitization, thereby its importance in pain chronicization process, may highly differ among individuals even after similar injury. Recent preliminary results show that, after limb amputation, prolonged and individualized (from 4 to 83 days) perineural infusion of local anesthetic allows us to significantly reduce the incidence of phantom pain (16% instead of the 67% usually reported with shorter lasting therapeutic interventions) [6].

Second, the mechanisms involved in central sensitization, which result from the balance between endogenous inhibitory and excitatory processes, may also differ among patients. Interindividual differences in the modulation of endogenous pain perception and modulation place patients at more or less risk to present with severe acute pain or chronic pain [37]. Secondary hyperalgesia, that is, mechanical hypersensitivity in uninjured tissues surrounding the wound, has caught particular attention as a surrogate measure of central sensitization after surgery [24,32]. The extent of secondary hyperalgesia correlates with the risk for CPSP as demonstrated after major abdominal surgery [11,12•]. However, all individuals presenting with secondary mechanical hyperalgesia at 24–48 h after surgery will not develop CPSP (e.g. after major abdominal surgery, 32–36% of patients show mechanical hyperalgesia surrounding the wound but only 7–11% will report CPSP [11]; similar findings exist for cesarean delivery). Evaluation of patients with and without postherniotomy pain also reveals a role of central sensitization in persistent pain. CPSP patients show a high incidence of contralateral groin pressure hyperalgesia (26%), that is, mirror image of pain mediated by central mechanisms, a feature which is not found in patients without pain [14,38]. Further, increased pain from repetitive tactile stimulation, that is, temporal summation, a surrogate measure of wind-up pain which is highly suggestive of altered central nociceptive processing, is found in 51% of CPSP patients but only in 15% (and at low intensity) of pain-free patients [14,38]. Clearly, central sensitization plays a role in a subpopulation of CPSP patients.

Individual's state of endogenous pain-modulatory processes

Preoperative assessment of pain sensitivity may predict to some extent the degree of postoperative pain and the probability to develop persistent pain [39•]. Recent interesting developments have focused on dynamic test paradigms – in contrast with static tests like pain threshold – designed to measure endogenous pain processing and to depict individual's pain-modulation capacity [40••,41]. Early promising results show that patients with poor inhibitory systems (i.e. bad conditioned pain modulation) are more susceptible to develop CPSP after thoracotomy [42] or major abdominal surgery [12•]. Moreover, patients with enhanced excitatory processes (i.e. positive temporal summation) have higher postoperative pain and are more prone to CPSP [12•,43]. Future therapeutic developments should be aimed to either reinforce weak inhibitory processes or to block excitatory processes according to the patient's preoperative assessment of endogenous pain-modulatory processes.

Preoperative chronic pain

Preoperative pain found in more than 50% of patients undergoing surgery [22], either at the operative site or elsewhere, is a well known risk factor for both severe postoperative pain and CPSP [3–5,25,26•]. Chronic pain

unrelated to the surgical site may be characterized by an underlying state of pain amplification, that is, central sensitization, as observed in patients suffering idiopathic pain disorders, for example, fibromyalgia, irritable bowel syndrome, chronic headaches, back pain, among others [44]. Those patients show altered pain sensitivity and modulation (suspected genetic origin) and are prone to both exacerbation of their underlying pain condition after surgery and higher acute pain at incision site. Preoperative pain at the surgical site may or may not be associated with plastic changes into the central nervous system. Preoperative groin pain does not seem to be related to a state of central sensitization which might support the development of CPSP postherniotomy [15,45]. In contrast, preoperative pain associated with severe osteoarthritis [46] or with shoulder impingement syndrome [47] involves for some patients a state of central sensitization associated with temporal summation, referred to as pain and hyperalgesia. These patients with established preoperative central sensitization have less favorable outcome after surgery and are more prone to develop CPSP [46,47]. A recent clinical trial concerning limb amputation highlighted the impact of an effective preoperative pain control on CPSP development. Optimized preoperative analgesic treatment not only may modulate underlying central sensitization but also has an effect on patient's psychologic mood [7].

Psychosocial vulnerability

Mental health, that is, optimism and positive attitude, has an important impact on the patient's willingness to recover. Emotional and attentional mechanisms of pain processing already known to play a role in chronic pain conditions have recently attracted interest in trauma and perioperative conditions [2,21,48]. Obviously, there is a vulnerable population who present with reduced ability to cope with pain, to anticipate pain and to control pain when confronted with it. Anxiety trait and psychological distress, for example, depression, are predictors of severe postoperative pain which itself carries an increased risk for persistent pain [49]. Attentional avoidance of negative experiences in general does not protect but makes people more vulnerable to a variety of stressors. Pain hypervigilance, a strong attentional bias toward pain defined as an automatic prioritization of pain, conscious or not, aimed to avoid physical threat can be maladaptive like in fibromyalgia and other chronic pain conditions. This preoperative mental state is predictive of both severe postoperative pain and CPSP [21]. Pain catastrophizing is associated with emotional distress states (depression) and risk of severe postoperative pain. Further, its impact may extend beyond the trauma period as it compromises social functioning after delivery [50] and may predict CPSP [21]. The notion of psychological flexibility–inflexibility has recently prompted attention. Individual's inability to act effectively in presence of unpleasant thoughts, emotions or symptoms like pain is referred as psychological inflexibility [51]. Although it correlates with chronic pain adjustment and functioning, to date, no study has assessed its role which might be a central mediating process in the progression from acute to CPSP [51].

The subacute pain period

Whether a distinct transition period exists between acute and chronic pain or whether chronic pain is a continuum of acute postoperative pain remains unclear. Nevertheless, subacute pain which can last for several weeks after surgery (Table 1) represents a neglected area of clinical investigation [52•], although it can be severe with a negative impact on patient's rehabilitation [16]. A few prospective studies have demonstrated the predictive value of 30-day or 6-week postoperative pain intensity as a risk factor for CPSP after inguinal hernia repair [15] and cosmetic breast surgery [20]. Pain intensity and incidence (14% at day 30 vs. 7% at day 7) may even increase during the subacute pain period as shown after herniotomy, perhaps revealing a neuropathic pain process [15]. Similarly after thoracotomy, neuropathic pain may develop (from 8% in the immediate postoperative period increasing up to 22% at 3 months) [9]. Poorly relieved long-lasting postoperative pain not only has a negative psychological impact but might also contribute to maintain an underlying central sensitization process, thereby interfering with rehabilitation. Most changes in health-related quality of life occur during the first weeks and months after surgery, after which patients' condition appear to remain stable [53]. Consequently, we should pay particular attention to patients' evolution during the early recovery period.

Conclusion

The major risk factors involved in chronic pain development post-trauma and postsurgery are well known. The phenomenon is complex and multifaceted. However, recent studies have underlined some important points on which to focus, mainly during the acute and subacute postinjury periods. A more dynamic approach of both acute pain resolution, individual's endogenous pain-modulatory processes and psychological functioning seems mandatory to improve our understanding of the progression from acute to chronic pain.

Acknowledgements

Conflicts of interest

There are no conflicts of interest.

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Papers of particular interest, published within the annual period of review, have been highlighted as:

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