The transition from acute to chronic pain

Introduction

Why some patients develop chronic pain after an acutely painful event remains an enigma. For example, over 90% of the population will experience acute back pain at some time in their lives. In most cases this resolves but, in a few, it does not, even though these patients have radiologic pathology similar to those in whom the pain does improve.

Similarly, there are few people who escape the hands of the surgeon. What is becoming increasingly apparent is that those who do experience the sharp end of the scalpel frequently experience persistent pain as a result of the damage caused by this instrument. The reasons for this chronic post-surgical pain are discussed in this presentation, as are possible preventative strategies.

Definition of chronic post-surgical pain (CPSP)

From Macrae and Davies:

- Pain developed after a surgical procedure;
- Pain is of at least 2 months duration;
- Other causes for the pain should have been excluded (e.g. continuing malignancy or chronic infection);
- The possibility that the pain is continuing from a pre-existing problem must be explored and exclusion attempted.

Incidence

There are frequently wide variations in the incidence of chronic pain after specific operations. This is because studies that have been performed, which directly investigate the incidence of chronic pain after an operation, find a far higher incidence than those studies in which chronic pain incidence was only part of the study. Despite this, it is often startling to see how high the incidence of chronic post-surgical pain is, and there are suggestions that patients should be informed of this before consenting to surgery (Table I).

Aetiology

Pain is subjective, but may be caused by various factors. Exactly what causes CPSP remains unclear but it appears that it is related to both operative nerve damage, resulting in neuropathic pain, and that it is modified by psychosocial factors which may have been pre-existing or developed post-operatively as a consequence of the chronic pain experience.

Following surgery, neuropathic pain can be caused by damage to peripheral nerves or central nervous system (CNS) sensory transmission. Inflammatory pain due to persistence of the inflammatory response or chronic infection may also be a factor. It is important to differentiate neuropathic from non-neuropathic pain, in order to effectively design strategies to prevent and to treat these entities.

| Type of surgery                | Incidence of chronic pain (%) |
|-------------------------------|-------------------------------|
| Amputation                    | 30 - 85                       |
| Thoracotomy                   | 5 - 67                        |
| Mastectomy                    | 11 - 57                       |
| Inguinal hernia repair        | 0 - 63                        |
| Sternotomy                    | 28 - 56                       |
| Cholecystectomy               | 3 - 56                        |
| Knee arthroplasty             | 19 - 43                       |
| Breast augmentation           | 13 - 38                       |
| Vasectomy                     | 0 - 37                        |
| Radical prostatectomy         | 35                             |
| Gynaecological laparotomy     | 32                             |
| Iliac crest bone harvest site | 30                             |
| Hip arthroplasty              | 28                             |
| Saphenectomy                  | 27                             |
| Hysterectomy                  | 25                             |
| Craniotomy                    | 6 - 23                        |
| Rectal amputation             | 12 - 18                       |
| Caesarean section             | 12                             |
| Dental surgery                | 5 - 13                        |
Neuropathic pain is diagnosed clinically, and there are no specific diagnostic tests for this condition. Rasmussen et al have proposed the following criteria for the diagnosis of neuropathic pain:3

- Pain in a neuroanatomically defined area, i.e. corresponding to a peripheral or central innervation territory;
- History of relevant disease or lesion in the nervous system, which is temporally related to development of pain;
- Partial or complete sensory loss in all or part of the painful area;
- Confirmation of a lesion or disease by a specific test, e.g. surgical evidence, imaging, clinical neurophysiology, biopsy.

Whilst studies based on self-reporting of symptoms by patients may not identify neuropathic pain, other studies which have used more refined investigative tools such as sensory threshold changes are more sensitive and more likely to elucidate neuropathic pain.4,5

The link between neuropathic pain and CPSP is complicated. Many patients in whom there are clear signs of peri-operative nerve damage (e.g. a numb area) do not experience CPSP.4 When comparing cases where a sensory nerve was deliberately “sacrificed” during surgery with cases where efforts were made to preserve it, the incidence of CPSP appears to be similar.7,8

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Prevention of CPSP

It is become increasingly apparent that, in order to prevent CPSP, one must prohibit noxious inputs from reaching the CNS for the entire peri-operative period. Failing that, one has to prevent the CNS changes that result from painful stimuli starting. If, at any time during the peri-operative period, there is an episode of “breakthrough” pain, then irreversible CNS changes may be triggered and CPSP may result.

This explains why pre-emptive regional anaesthesia does not consistently reduce CPSP, whereas preventative regional anaesthesia may do. Here the difference is that, in the former, the block is given before surgical incision but not continued whereas, in the latter, the block is given before incision but continued well into the postoperative period.

Pre-emptive antineuropathic pain medication similarly shows no effect on CPSP, whereas preventative antineuropathic pain medication may be beneficial.20,21

Multimodal analgesic techniques are being increasingly used to provide maximal analgesia with minimal side-effects with the aim of reducing CPSP.22 Whilst, in theory, these can be highly effective,
more often than not the problem remains of ensuring that the patient actually receives the analgesic medication prescribed and at the correct time. All too frequently the patient only receives analgesia when pain has returned. Nursing staff must be educated to administer analgesia regularly in order to prevent pain. Surgeons need to understand the importance of maintaining epidural infusions well into the postoperative period and not to discontinue them prematurely.

Conclusion

Over the last ten years, there has been a substantial increase in the understanding of the problem of CPSP amongst anaesthesiologists. This information needs to be more widely disseminated to other healthcare professionals. Patients should be given information regarding the likelihood of CPSP so that they can make more informed decisions regarding whether to undergo certain operations and, if they do proceed with surgery and develop CPSP, understand why it has occurred and not attribute undeserved blame to the surgeon. The patient would also then be more likely to have his or her pain acknowledged and receive appropriate treatment.

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