Pharmacologic Management of Upper Extremity Chronic Nerve Pain

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KEYWORDS

• Chronic pain • Neuropathic • Nerve • Tricyclic • Gabapentin • Postsurgical

KEY POINTS

- Chronic pain complicates all types of surgery, including upper extremity surgery done with excellent technique.
- Chronic postsurgical pain is usually neuropathic even though it is not described as burning or electrical but is more commonly described as deep and throbbing.
- Cutaneous neuromas (scar neuromas) are common and can only be excluded by injecting the scar.
- Medication management tailored to neuropathic pain is often effective and the principles of such management are outlined in the article.

Chronic pain following surgery is a major complication affecting between 10% and 30% of patients following a wide variety of surgeries.[1] The hand and upper limb are especially susceptible to postsurgical pain because of the rich innervation and unique demands of the upper extremity. Neuropathic pain after hand/upper limb surgery is likely produced through at least 3 potential mechanisms: (1) transection of cutaneous branches during skin incision with neuroma formation in the skin scar[2–5]; (2) adherence of postsurgical scar tissue to nerves either in the skin or deeper without transection of these nerves (exacerbated by the postoperative immobilization often required for bone healing)[6]; and (3) entrapment of nerve branches remote from the skin incision. This entrapment can be proximal to the surgical insult caused by edema tracking along the nerve course and resulting in compression at sites where surrounding tissue is noncompliant.[7] Another factor that puts the upper limb at risk for neuropathic pain is that few parts of the body are as mobile, or have the degree of excursion, as the nerves traveling from the cervical spine to the fingertips. These nerves cross multiple joint lines but, after trauma, nerves can become tethered in immobile cutaneous scars surrounded by keratinocytes secreting chemokines and growth factors that promote painful neuromas.[8–13] Beyond the physical characteristics of the upper limb, many central and peripheral mechanisms also likely contribute to the development of chronic pain and undermine the results of a technically perfect surgery.[14]

Many physicians think that neuropathic pain is tingling or electrical and therefore complaints of sore, heavy, dull, or throbbing pain are considered not neuropathic in nature. However, surveys of patients with neuropathic pain, such as spinal cord injury pain, acute herpes zoster, postherpetic neuralgia, and diabetic neuropathy, found that this pain was often described as a dull throbbing or heavy pain.[15–20] Therefore the most important point in better treating the chronic pain after surgery is to recognize that this pain is more frequently than expected neuropathic in origin. There are some data suggesting that some of the medications that are frequently used to treat neuropathic pain work best for those people whose pain is not described in classic neuropathic

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terms but who describe their pain as sore, dull, or heavy.[17–19]

Similarly, it is my own personal experience that cutaneous scar neuromas are invariably described by patients as a diffuse and deep pain. Therefore, no patient with chronic postsurgical pain can dissuade me from injecting their cutaneous scar with local anesthetic. For me, the only convincing evidence that a scar neuroma is not present and a major source of the patient's chronic pain is failure to have temporary profound analgesia in response to a scar injection with local anesthetic. Scar neuroma is an underexplored and underreported area of investigation in the literature on chronic postsurgical pain.

This article describes the basics of pharmacologic treatment of neuropathic pain (the most common type of chronic pain after surgery). This article is intended for upper extremity surgeons and allied practitioners who are not pain specialist but for whom a better understanding of how to treat nerve pain will be of great benefit.

GENERAL TENETS

1. Do not be in a hurry. The medications that most successfully reduce neuropathic pain and have the most durable analgesic effects all require some patience to be maximally successful. An expectation that they will work as quickly as commonly used pain relievers (opioid medications: eg, Vicodin, codeine) results in profound disappointment for both provider and patient. More importantly, impatience may cause you and your patient to prematurely put aside one of the few medications that can produce long-term profound pain relief. An expectation that neuropathic pain medicines will work on the time scales patients have come to expect from their previous exposure to opioids will cause a drug that might have been successful to be rejected. In addition to differing in their time to effectiveness, medications used for neuropathic chronic postsurgical pain also seem to have a threshold effect not commonly seen with the opioids. A patient may take a small quantity of an opioid and experience minor relief and know from experience that if they take more it is likely to provide greater relief. In contrast, many of the antidepressants and anticonvulsants used to manage neuropathic pain (henceforth referred to as antineuropathics) seem to have a threshold effect; a patient might feel no relief at 25 mg or 50 mg of nortriptyline, and then at 75 mg start to feel relief, and then at 150 mg feel profound relief. Patients' initial experience of the drug may therefore not reflect their subsequent experience with a higher dose.
2. Antineuropathics should be started at ineffective doses. When started at an effective doses antineuropathic medications often create side effects. These side effects can largely be avoided by slow titration from a low dose, which allows patients to accommodate to the medication effects. For example, duloxetine creates nausea in as many as 25% of patients when started an effective dose of 60 mg. However, if started at the ineffective dose of 20 mg a day, and then increasing the dose by 20 mg weekly up to 60 mg, then nausea becomes an infrequent side effect.[21–23] It is generally difficult to talk someone into retrying a drug that they are convinced makes them vomit (or be dopey and so forth), so it is better to start at a low, explicitly ineffective dose and gradually titrate up to an effective dose. I tell patients that I am going to give them a certain drug at a dose that does not work. We use the first few weeks not to help with the pain but just to get the patient's body used to this medication, then after a few weeks we start to explore doses that might help. Failure to have this kind of conversation with the patient results in loss of confidence in the physician and the medication when the medication fails to improve pain in the first few days.
3. There is some evidence that even though these drugs may exert some effect immediately at any given dose, the degree of pain relief may build substantially over the first few weeks of treatment.[22,23]
4. Unlike penicillin, for which there is a clear correct dose, with all of these antineuropathic medications doses have to be tailored to the individual. Given current technology it is impossible to know what dose may be too much for one person and not enough for another. Thus you have to test a given dose in a given person and then slowly change the dose to see the degree of side effects and the degree of pain relief. So the policy is:
   a. Start low (start at a dose that you are confident will not create side effects; usually a dose too low to be effective)
   b. Go slow (increase the dose by small increments every few days or each week so that you plan to reach the target dose in 1 to 2 months)
   c. But go! (when you start one of these medications do not just pick a low dose and leave it there, or decide that it does not work just because it is not having an effect at that low dose)