# Acute Pain Management

This textbook is written as a comprehensive overview of acute pain management. It is designed to guide clinicians through the impressive array of different options available to them and to patients. Since the late 1990s, there has been a flurry of interest in the extent to which acute pain can become chronic pain and how we might reduce the incidence of such chronicity. This overview covers topics related to a wide range of treatments for pain management, including the anatomy of pain pathways, the pathophysiology of severe pain, pain assessment, therapeutic guidelines, analgesic options, organization of pain services, and the role of anesthesiologists, surgeons, pharmacists, and nurses in providing optimal care. It also discusses the use of patient-controlled analgesia and how this may or may not be effective and useful.

Dr. Raymond S. Sinatra currently serves as Professor of Anesthesiology at Yale University School of Medicine. He received his MD as well as a PhD in neuroscience at SUNY Downstate School of Medicine and completed his anesthesiology residency at the Brigham & Women's Hospital, Harvard Medical School. Dr. Sinatra joined the faculty at Yale in 1985 and organized one of the first anesthesiology-based pain management services in the United States. In addition to directing the service, he has served as principal investigator for dozens of clinical protocols evaluating novel analgesics and analgesic delivery systems. Dr. Sinatra has authored more than 130 scientific papers, review articles, and textbook chapters on pain management and obstetrical anaesthesiology and was senior editor of an earlier textbook titled *Acute Pain: Mechanisms and Management*. Dr. Sinatra annually presents papers and lectures at both national and international meetings and serves as a reviewer for several anaesthesiology and pain management journals.

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Eugene R. Viscusi

# Foreword: Historical Perspective, Unmet Needs, and Incidence

*Henry McQuay*

It is a delight and an honor to be asked to write the foreword for this text on acute pain management. We have an impressive array of different options for acute pain management (Figure F.1), and not all of them were available in the late 1970s.

As a simple example of the improvement in knowledge, compare the analgesic efficacy work of Moertel and colleagues[1] with that available to us now (Figure F.2). We can use these league tables of relative efficacy to say with some authority how well on average the different analgesics compare. This leaves us, of course, with the real-world issues of, for example, how the individual patient will react, prior experience, and drug-drug interactions.

Yet, we have the continued embarrassment of surveys that show that a substantial number of patients still endure severe pain after their surgery or trauma. This "unmet need" is a mixture of our failure to implement effective analgesic strategies and the inadequacy of those strategies. Acute pain teams date back to the early 1980s, and their policies and education of both patients and caregivers have made a difference. There is little excuse now for the failure to provide adequate analgesia for straightforward cases, but we need to acknowledge that there are also difficult cases. Many of the patients whose care causes problems for the teams seem, locally for us at least, to be the patients with chronic pain problems who are already on substantial analgesic therapy (e.g., chronic gastrointestinal disease) or substance abusers. Things the teams can do well include the education and patient advocacy roles within the institution. Things they may struggle with include changing behavior and provision of seamless care across nights and weekends.

Since the late 1990s there has been a flurry of interest in the extent to which acute pain can become chronic pain and how we might reduce the incidence of such chronicity.

Perhaps the most important thing this foreword points out is the sheer scale of the problem. From the chronic pain perspective, it appears now that surgery may be the most common cause of nerve damage pain and should perhaps be something that patients are warned about as a possibility in the consenting process. Mechanistically, one can ask what happens to cause this surgical pain to become chronic. I have always been skeptical that there is some psychological factor, pejoratively some weakness, that causes some patients to have the problem and others not. As an example, take a patient who had an inguinal herniorrhaphy 3 years ago: the procedure was performed perfectly and result was perfect. This year he had the other side done, and the same procedure was performed by the same surgeon. The patient described very severe postoperative pain, qualitatively and quantitatively quite different from the first operation, and this severe pain persisted. Something happened to cause the pain, and one cannot invoke a psychological explanation because of the perfect result the first time. What can we do about this? We still have no strong evidence that analgesia delivered before the pain does anything radically different from the same analgesia given after the pain, let alone that it preempts the development of this type of chronicity. It may be that unexpected severe pain is a red flag, but that is not easy to spot given the huge variations in pain intensity experienced after a given procedure. But it might be something we could pursue. Teasing apart precisely what happens during surgery would be another approach.

The measurement of the analgesic efficacy of preemptive strategies is another of the outstanding methodological issues in acute pain management. Our current methods allow us to measure the relative change in pain intensity. If the patient has no pain initially, then the method is invalid. This is the conundrum in measurement of the analgesic efficacy of preemptive strategies, because we have no idea whether the patient would have had no pain without the intervention. We claim that the patient had no pain because of the intervention, but they may not have had any pain without it.

A second cause of methodological angst is the use of patient-controlled analgesia (PCA) as an outcome measure. Many of the current crop of studies – for instance, those studying prophylactic antiepileptic drugs – use PCA in this way and report reduced PCA opioid consumption compared with controls. Unfortunately, this difference in consumption is not reported at valid equivalence in pain scores in the two groups. The control groups

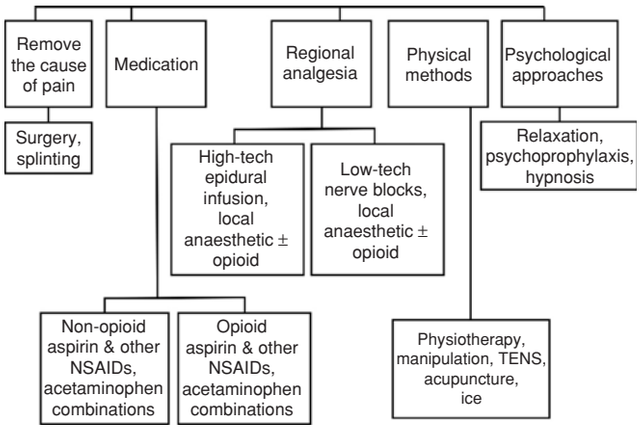


Figure F.1: The different options for acute pain management.

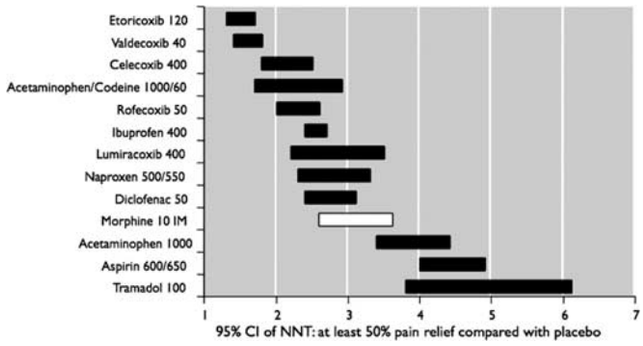


Figure F.2: Relative analgesic efficacy of analgesics in postoperative pain: number-needed-to-treat (NNT) for at least 50% pain relief over 6 hours compared with placebo in single-dose trials of acute pain.

commonly fail to use the PCA to lower their pain scores to the same level as is seen in the "active" group. Unless the pain scores are equivalent, it is very difficult to interpret the difference in PCA consumption. We need urgently to establish the validity of PCA as an outcome measure.

The editors and the authors of this book are to be congratulated on keeping academic and practical attention focused on acute pain, because there is room to both improve our current practice by learning from the best and try to answer some of the important outstanding issues.

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