Despite advances in neurosurgical and neuroanesthesiological practice, postoperative pain continues to be undertreated. There are many modalities that may provide safe and effective postoperative analgesia. We discuss mainly systemic (e.g. opioids, nonsteroidal antiinflammatory agents) analgesic options. They still remain the most widely used method for providing pain relief in acute surgical situations. The exact choice or combination of analgesics utilized for a particular patient will depend on the riskbenefit profile and patient preferences. Especially is crucial to promptly involve the analgesics when an opioid tolerant patient requires aggressive pain treatment. But, opioid analgesia alone may not fully relieve all aspects of acute postoperative pain. Combinations of drugs acting on different mechanisms of nociceptive modulation will decrease the incidence of adverse effects and offer additive and/or synergistic effects. Analgesic concentrations of ketamine infusions remain a valuable addition to opioid administration. Complementary medicine techniques used as adjuvant therapies have the potential to improve pain management and improve postoperative distress. Neuromuscular blocking agents (NMB) in the intensive care unit (ICU) patient facilitate intubation and ventilatory support, decrease oxygen consumption, facilitate bedside procedures and diagnostics, and potentially decrease intracranial pressure. Ideally, analgesics, sedatives and/or muscle relaxants should be combined into a multimodal approach to facilitate patient recovery after surgery. Although a great deal is known about specific drugs and dosage requirements, further research is needed that clearly examines optimal scheduling regimens if we are to maximize patient care. The most important rule of pain management is that pain is what the patient says it is.

Key Words: postoperative pain; opioids; nonsteroid antiinflammatory drugs; neuromuscular blocking agents; multimodal approach.

BACKGROUND AND OBJECTIVES

Recently the medical profession faced new challenges in the effort to provide medical care that recognizes the patient as a participating entity. This evolution has gained the greatest prominence in the field of pain management. Fear of inadequate pain management rises enormous number of surveys of surgical hospital populations. Such fear reflects hesitancy by the medical professionals to treat pain aggressively in an environment like the Intensive Care Unit (ICU) really is. However, our growing knowledge of pain perception and propagation presents the opportunity to make the postoperative care better. The overall strategy for postoperative pain management must incorporate the latest systematic information for analgesic options individually adjusted to each patient’s needs. We review the most important modalities available for neurosurgical postoperative pain management, focusing on evidence obtained from prospective clinical studies and metaanalyses.

Based on the content of this article, the reader should be able to:

1. assess, plan, and implement interventions appropriate for a patient experiencing acute postoperative pain;
2. describe the safe and effective administration of loading doses and/or maintenance infusion rates of nonsteroid antiinflammatory drugs (NSAIDs), neuromuscular blocking (NMB) agents, and opioids for the treatment of acute postoperative pain in an neurological patient in the ICU; and

3. discuss the various physical and psychosocial factors that influence the patient response to pain treatment.

PATHOPHYSIOLOGY OF POSTOPERATIVE PAIN

Pain can be defined as "an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage"1. This definition acknowledges the fact that pain is a multifactorial human experience with physical, psychosocial, cultural, and religious elements. Implicit in the definition is that pain is a subjective experience and "there is no neurophysiological or chemical test that can measure pain. The clinician must accept the patient’s report of pain"2.

The studies of the neurophysiology of pain have produced a remarkable understanding of the role of nociception in the perioperative period. Surgical incision is a traumatic event triggering profound sympathetic and inflammatory responses. The inflammatory response activates peripheral nociceptors that transmit "pain" signals centrally and initiate a larger inflammatory process, amplifying that transmission and potentially altering subsequent nociceptive sensation through central sensitization. Resultant increase in catecho-lamine release and metabolic oxygen consumption amplifies metabolic activity, induces the sympathoadrenal axis, and imposes an increased influence on multiple organ systems with an overall influence on patient morbidity and mortality. Severe pain can contribute to complications such as: dysfunction of the blood clotting mechanism, immune system failure, water retention, breakdown of body tissue, and delayed return of normal gastric and bowel function3,4. One important aspect of postoperative patient care is the early rehabilitation program, which consists of coughing, deep breathing, positioning, early mobilization, and pain management. Effective management of pain is dependent upon valid and reliable assessment5. The key role of assessment has been recognized by its adoption as the fifth vital sign in the postoperative period6. For obvious reasons, patients with ineffective pain management are unable to effectively engage in this regimen. Poorly controlled postoperative pain limits patient mobilization, compromising pulmonary function and possibly increasing patient morbidity.

Postoperative patients, especially those who have undergone abdominal and thoracic surgery, have reduced pulmonary function by as much as 20% to 60% relative to their preoperative values7. For these patients, untreated pain leads to decreased ability to engage in the early rehabilitation regimen, and as a result, these patients are at a greater risk for venous stasis due to immobility and atelectasis, retained respiratory secretions, and pneumonia due to shallow respirations3,4.

<table>
<thead>
<tr>
<th>Agent</th>
<th>Use</th>
<th>Route of application</th>
<th>Dose*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Morphine</td>
<td>Postoperative analgesia</td>
<td>IM</td>
<td>0.05-0.20 mg kg⁻¹</td>
</tr>
<tr>
<td></td>
<td></td>
<td>IV</td>
<td>0.03-0.15 mg kg⁻¹</td>
</tr>
<tr>
<td>Pethidine (Meperidine)</td>
<td>Postoperative analgesia</td>
<td>IM</td>
<td>0.50-1.00 mg kg</td>
</tr>
<tr>
<td></td>
<td></td>
<td>IV</td>
<td>0.20-0.50 mg kg</td>
</tr>
<tr>
<td>Fentanyl</td>
<td>Postoperative analgesia</td>
<td>IV</td>
<td>0.50-1.50 µg kg</td>
</tr>
<tr>
<td>Sufentanil</td>
<td>Intraoperative anesthesia</td>
<td>IV(loading dose)</td>
<td>8.0-100.0 µg kg</td>
</tr>
<tr>
<td>Alfentanil</td>
<td>Intraoperative anesthesia</td>
<td>IV(maintenance infusion)</td>
<td>0.50-3.00 µg kg min⁻¹</td>
</tr>
<tr>
<td>Remifentanil</td>
<td>Postoperative analgesia/sedation</td>
<td>IV</td>
<td>0.05-0.30 µg kg min⁻¹</td>
</tr>
</tbody>
</table>

*The wide range of opioid dose reflects a large therapeutic index and depends upon which other anesthetic are simultaneously administered. For obese patients, dose should be based on ideal body weight or lean body mass, not total body weight. Tolerance can develop rapidly (i.e., within 2 hours) during IV infusion of opioids, necessitating higher infusion rates. Dose correlates with other variables besides body weight that need to be considered (e.g., age). The relative potencies of alfentanil, fentanyl, and sufentanil are estimated to be 1:7:65.
Br. 4 Strategies for postoperative pain relief in neurosurgical intensive care unit

TABLE 2
RAMSAY’S LEVEL OF SEDATION SCALE

<table>
<thead>
<tr>
<th>Level</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Anxious, agitated, or restless</td>
</tr>
<tr>
<td>2</td>
<td>Cooperative, oriented, tranquil</td>
</tr>
<tr>
<td>3</td>
<td>Awake, obeys commands</td>
</tr>
<tr>
<td>4*</td>
<td>Asleep but arousable and brisk response</td>
</tr>
<tr>
<td>5*</td>
<td>Asleep but arousable, sliggish response</td>
</tr>
<tr>
<td>6*</td>
<td>No response</td>
</tr>
</tbody>
</table>

*Based on response to loud auditory stimulus or light glabellar tap

Thereafter, poorly controlled postoperative pain may result in the development of chronic pain after surgery. Whether postoperative pain is a primary predictor of chronic pain is yet unclear. However, there is increasing recognition that long term neurophysiological changes occur much more quickly than previously anticipated, and chronic pain after surgery is common following procedures such as: limb amputation (up to 83%), thoracotomy (up to 67%), breast surgery (up to 57%), gallbladder surgery (up to 56%), and sternotomy (27%), with the severity of postoperative pain suggested as an important predictor for development of chronic pain.

Preemptive analgesia attempts to prevent the development of central sensitization and incidence of chronic pain by administration of analgesics in the perioperative period to prevent the establishment of incisional (intraoperative) and inflammatory (postoperative) injuries. Clinical trials are equivocal in the benefits of preemptive analgesia. Since both incisional and inflammatory injuries are important in initiating and maintaining central sensitization, maximum clinical benefit is observed when there is a complete blockade of noxious stimuli with extension of this blockade into the postoperative period.

Nonsurgical causative factors of pain, like urinary retention and bladder distention, can exacerbate postoperative pain. Opioids are known to inhibit the voiding reflex and can increase the tone of the detrusor muscle and the external sphincter of the bladder, resulting in bladder distention, urgency, and urinary retention. This condition is especially common among males who have hypertrophy of the prostate. The patient will need urinary catheterization if the bladder is distended and the patient is unable to void.

SYSTEMIC ANALGESIC THERAPY

Nonsteroidal antiinflammatory agents (NSAIDs). NSAIDs are a major systemic pharmacological option, providing analgesia without the potential for dependence or addiction but limited by a therapeutic ceiling. These agents inhibit central and peripheral cyclooxygenase (COX) enzymes and production of prostaglandins, resulting in attenuation of inflammation and a decrease in the mediators of nociception. NSAIDs have also spinal mechanism of action.

The discovery of two COX isoforms with different functions (COX-1: constitutive, causing platelet aggregation, haemostasis and gastric mucosal protection; COX-2: inducible, causing pain, inflammation and fever) has resulted in the development of selective COX-2 inhibitors, which differ from traditional NSAIDs that block both COX1 and COX-2. COX-2 inhibitors may give advantages of the analgesic efficacy of a traditional NSAIDs with decreased gastrointestinal haemorrhage and minimal effects on haemostasis. Independently, NSAIDs provide adequate analgesia for mild to moderate pain, although some recent data suggest that NSAIDs may be more efficacious as analgesics than previously recognized. The expression of COX-2 enzyme in the spinal neurones is believed to contribute to neuronal plasticity and central sensitization. This suggests that NSAIDs should have more active role in the management of pain after tissue injury (postoperative, trauma) and or nerve injury (neurogenic pain). Recent quantitative systematic reviews suggest that NSAIDs may be as efficacious as opioids alone. When used as an adjunct to opioids, NSAIDs may improve postoperative analgesia, reduce opioid requirements, facilitate return of gastrointestinal function, reduce nausea, decrease respiratory depression and improve patient satisfaction.

Small doses of NSAIDs are not efficient for acute pain relief. Therefore, the maximum daily dose should be administered, divided over several fixed time intervals in relation to the mean duration of effects of each agent. The average duration of analgesia is 4 – 6 hours for acetylsalicylic acid or ibuprofen, and 8 hours for diclofenac or ketorolac. There will be a need for additional opioids when postoperative pain is severe. When combined with opioids, NSAIDs may reduce postoperative opioid consumption up to 40%.

The preoperative administration of NSAIDs may significantly improve pain relief in the early postoperative period. However, one cannot use NSAIDs as preemptive analgesics, because the reduction in opioid requirements is small and limited to the first few postoperative hours. NSAIDs are associated with a number of sideeffects, including decreased haemostasis, renal dysfunction, gastrointestinal haemorrhage, and adverse effects on bone healing. NSAID-induced platelet dysfunction and inhibition of thromboxane-A2 causes decreased haemostasis, although the evidence of NSAIDs as a major source of perioperative bleeding is equivocal; a
large observational study of perioperative ketorolac did not demonstrate any significant increase in bleeding at the operative site. Patients with hypovolaemia, abnormal renal function or serum electrolytes may be at higher risk for developing NSAID-induced renal dysfunction. NSAIDs are also associated with a higher incidence of gastrointestinal bleeding and may have an adverse effect on bone healing. It is unclear whether COX-2 inhibitors will reduce the incidence of renal complications compared to traditional non-selective NSAIDs.

PARACETAMOL AND METAMIZOLE

Nevertheless they are often considered as NSAIDs, metamizole and paracetamol do not inhibit the COX enzymes at the analgesic concentrations and have no antiinflammatory activity. They have central antinociceptive effects involving serotoninergic descending inhibitory pathways. They do not show the typical sideeffects of NSAIDs on the gastric mucosa and blood platelets. The risk of agranulocytosis after administration of metamizole or paracetamol is minimal, and so is the related mortality. The efficacy of NSAIDs to paracetamol were shown to be comparable in postoperative pain from major surgery. Adding an NSAID to paracetamol may further improve postoperative pain but, on the contrary, adding paracetamol to NSAIDs had no beneficial effect on analgesia.

KETOROLAC

Ketorolac is a nonopioid analgesic that has properties similar to those of acetylsalicylic acid and ibuprofen. In contrast to opioids, which blunt the perception of pain via its action in the central nervous system, ketorolac prevents pain by acting on the site of injury. When it is administered in addition to opioids, not only will it decrease the opioid requirement by 25% to 50%, but very often pain relief will also be achieved more effectively than with the use of either of the drug classes alone.

OPIOIDS

Opioids remain a central option in the management of moderate to severe perioperative pain. They act peripherally on injured tissues to reduce inflammation, in the dorsal horn of spinal cord to diminish transmission of the nociceptive signal, and in the brain to activate inhibitory pathways of the spinal processing of "pain" signals. Opioids inhibit mreceptors, which are distributed both centrally and peripherally, and accordingly account for both the analgesic efficacy and sideeffects associated with these medications. A ceiling ef-

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**TABLE 3**

<table>
<thead>
<tr>
<th>Agent</th>
<th>Primary excretion</th>
<th>Speed of onset</th>
<th>Duration of action</th>
<th>Histamine release</th>
<th>Vagal blockade</th>
<th>Relative potency</th>
<th>Cost</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tubocurarine</td>
<td>renal</td>
<td>moderately rapid</td>
<td>long</td>
<td>marked effect</td>
<td>no effect</td>
<td>1</td>
<td>low</td>
</tr>
<tr>
<td>Metocurine</td>
<td>renal</td>
<td>moderately rapid</td>
<td>long</td>
<td>moderate effect</td>
<td>no effect</td>
<td>2</td>
<td>moderate</td>
</tr>
<tr>
<td>Atracurium</td>
<td>insignificant</td>
<td>moderately rapid</td>
<td>intermediate</td>
<td>slight effect</td>
<td>no effect</td>
<td>1</td>
<td>high</td>
</tr>
<tr>
<td>Cisatracurium</td>
<td>insignificant</td>
<td>moderately rapid</td>
<td>intermediate</td>
<td>no effect</td>
<td>no effect</td>
<td>5</td>
<td>high</td>
</tr>
<tr>
<td>Mivacurium</td>
<td>insignificant</td>
<td>moderately rapid</td>
<td>short</td>
<td>slight effect</td>
<td>no effect</td>
<td>12</td>
<td>high</td>
</tr>
<tr>
<td>Doxacurium</td>
<td>renal</td>
<td>slow</td>
<td>long</td>
<td>no effect</td>
<td>no effect</td>
<td>12</td>
<td>high</td>
</tr>
<tr>
<td>Pancuronium</td>
<td>renal</td>
<td>moderately rapid</td>
<td>long</td>
<td>no effect</td>
<td>moderate effect</td>
<td>5</td>
<td>low</td>
</tr>
<tr>
<td>Pipecuronium</td>
<td>renal</td>
<td>moderately rapid</td>
<td>long</td>
<td>no effect</td>
<td>no effect</td>
<td>6</td>
<td>high</td>
</tr>
<tr>
<td>Vecuronim</td>
<td>biliary</td>
<td>moderately rapid</td>
<td>intermediate</td>
<td>no effect</td>
<td>no effect</td>
<td>5</td>
<td>high</td>
</tr>
<tr>
<td>Rocuronium</td>
<td>biliary</td>
<td>rapid</td>
<td>intermediate</td>
<td>slight effect</td>
<td>no effect</td>
<td>1</td>
<td>high</td>
</tr>
<tr>
<td>Rapacuronium</td>
<td>renal</td>
<td>rapid</td>
<td>short</td>
<td>slight effect</td>
<td>no effect</td>
<td>0.3</td>
<td>high</td>
</tr>
</tbody>
</table>
fect is not seen, but the dose that can be administered is generally limited by side-effects, including respiratory depression, pruritus, nausea and vomiting.

Pure m-receptor agonist opioids are most often parenterally administered for the treatment of moderate to severe postoperative pain and will provide reliable onset of analgesic action, although intramuscular (IM) administration may result in a wider variability in serum drug concentrations than intravenous (IV) administration. Besides morphine, a broad spectrum of synthetic opioids (pethidine, fentanyl, alfentanil, sufentanil, remifentanil) is presently used in clinical practice. Contrary to morphine and pethidine, none phenylpiperidine analgesic (fentanyl, alfentanil, sufentanil) induces histamine release. (Table 1)

Mixed agonist-antagonist opioids (pentazocine, nalbuphine, butorphanol), compared with pure agonists, have limited flexibility in dosing. They show ceiling effects for analgesia and respiratory depression. One can obtain their maximal analgesic effect with relatively low doses, but a further increase of the dosing induces a larger incidence of side-effects20.

Partial agonist opioid – buprenorphine, has a high affinity for the m-receptor, but a low efficacy at this receptor. Therefore, buprenorphine cannot provide better analgesia than pure agonists, but may produce fewer respiratory, gastrointestinal, or urinary side-effects.

Opioid Toxicity and Respiratory Depression

Opioids depress the respiratory rate and tidal volume by decreasing the responsiveness of the respiratory centers in the pons and medulla oblongata. A respiratory rate of fewer than 8 breaths per minute is generally considered a criterion for respiratory depression13. Maximal respiratory depression usually occurs 5 to 10 minutes following IV administration of either opioid3. There is a direct correlation between the degree of patient sedation and respiratory depression. Therefore, it is important to assess and document the patient’s level of sedation based on his responsiveness each time the patient’s report of pain is evaluated.

Moderate sedation is defined by the patient’s ability to respond purposefully to verbal command and light tactile stimulation. When a patient is in mild to moderate sedation, the respiratory function is usually unaffected. A patient deeply sedated is not easily aroused but is responsive to painful stimulation. It is at this level of sedation that spontaneous ventilation may be inadequate, and the patient may need assistance to maintain a patent airway. Therefore, careful monitoring of the patient’s level of consciousness and respiratory status is especially crucial during this period. Patients’ level of sedation may be assessed by assigning them a Ramsay sedation score26. (Table 2)

Very often after pain relief is achieved, the patient can fall into a deep sleep due to exhaustion from the physiological and emotional responses to acute pain. In a monitored environment, such as the ICU, the patient should be allowed to have as much uninterrupted sleep as possible. However, it is crucial to ensure that the patient remains on continuous pulse oximetry and vital signs are continuously monitored to detect hyperventilation, airway obstruction, and abnormal changes in vital signs. The natural sleep is known to produce a decrease in sensitivity of the medullary center to carbon dioxide13. When the patient is painfree and is sleeping after receiving a high dose of opioid, the sleep state and opioids have an additive depressant effect on respiration2.

**Tramadol**

As a synthetic opioid which exhibits weak m- and k-receptor agonist activity (10% of morphine potency), tramadol exerts its analgesic effects by inhibiting the central reuptake of serotonin and norepinephrine and may also exhibit peripheral local anaesthetic properties27,28. With relatively less respiratory depression, major organ toxicity, depression of gastrointestinal motility and abuse potential - compared to traditional opioids, tramadol has been shown to be an efficacious analgesic agent for the treatment of moderate postoperative pain. It is typically administered in low-dose formulations together with either an NSAID, acetylsalicylic acid or paracetamol20. Continuous intravenous infusion provides efficient post-operative analgesia. Administered doses can go up to the 600 mg daily. In combination with metamizole may be very useful for relieving spastic visceral pain29. Dizziness, drowsiness, sweating, nausea, vomiting, dry mouth and headache are commonly reported side-effects30. They occur less frequently when the initial dose (1 – 1.5 mg kg–1) is administered as a 15 – 20 minute infusion.

**OTHER NON-OPIOID ANALGESICS.**

KETAMINE

Widely used as an anaesthetic agent, ketamine may be useful as an analgesic agent as it inhibits N-methyl-D-aspartate (NMDA) receptor activity, and therefore may prevent central sensitization31. Coordinates for perioperative dosing are unclear, although the use of low-dose ketamine infusion for postoperative analgesia has been reported and eventually may be a valuable adjunct in a multimodal analgesic regimen by enhancing analgesia and reducing opioid-related side-effects8,31. Inhibition of pain by ketamine significantly correlates with its serum concentrations. Analgesia is obtained with 10 - 20% of an anaesthetic dose20.
Ketamin could be quite useful preemptive analgesic. Pre- and peri-operative administration of low-dose ketamine may reduce postoperative morphine consumption, although the intensity of ongoing postoperative pain is less affected\textsuperscript{32}.

**NEUROMUSCULAR BLOCKING AGENTS**

Neuromuscular blocking agents (NMB) have been used postoperatively in the neurosurgical ICU for many years. However, their long-term use in the ICU gained popularity for the last few years. Approximately up to 10\% of all neurosurgical ICU patients receive NMB agents for more than 24 hours\textsuperscript{33,34}. NMB agents in the ICU patient facilitate intubation and ventilatory support, decrease agitation and restlessness, therefore decreasing oxygen demand and consumption, facilitate bedside procedures and diagnostics, and potentially decrease intracranial pressure. However, NMB agents have serious adverse effects and require close and continuous monitoring of the patient. Knowing that NMB agents have absolutely no sedative, amnestic and/or analgesic properties, they should only be used in patients who are properly sedated and receiving adequate analgesia. Presently, only nondepolarizing NMB agents are used in the neurosurgical ICU environment. Clinically, they are either: ultrashort, short, intermediate, or long acting compounds. These agents differ in their speed of onset, pharmacokinetics, adverse effects, and cost. They can be administered either by intermittent boluses or by continuous infusion. (Table 3)

Patient factors influencing the choice of the most appropriate NMB agent include: neurologic status, renal and hepatic system status and cardiovascular stability. Drug factors influencing the neuroanaesthesiologist’s decision include: speed of onset, duration of action, side effects and cost. Despite the intermittent boluses of NMB agents may be as effective as continuous infusion, some authors advocate continuous infusion only, to avoid increased intracranial pressure from unexpected stimulation such as from repositioning or tracheal suctioning\textsuperscript{35}.

**NONPHARMACOLOGIC INTERVENTIONS**

In addition to administering analgesics, a combination of various nonpharmacologic interventions should be implemented (e.g., repositioning, applying an ice package to a patient’s forehead, and repositioning the bed so that the patient can have a better visual contact with the nursing staff). Those interventions can improve patients’ responses to pain management by changing their perceptions of pain, altering pain behavior, and providing patients with a greater sense of control over pain.

Massage may be a useful adjuvant therapy for the management of acute postoperative pain. Its greatest effect appears to be on the affective component (i.e., unpleasantness) of the pain\textsuperscript{36}. Integrated with pharmacologic treatment, massage may be useful in the management of acute postoperative pain. However, nonpharmacologic therapy should only be used as an adjunct to, and not as a substitute for pain therapy.

**MULTIMODAL APPROACH TO POSTOPERATIVE CONVALESCENCE**

Despite the analgesic and physiological benefits of good analgesia, it is unclear whether pain control solely can lead to an improvement in patient outcomes. More likely, patient recovery after surgery is optimized when the benefits of quality postoperative pain control are integrated into a multimodal approach to patient convalescence, which includes aggressive control of postoperative pain to allow early patient mobilization, early enteral nutrition, education, and attenuation of the perioperative stress response through the use of regional anesthetic techniques and a combination of analgesic agents (i.e. multimodal analgesia)\textsuperscript{8,37}. Multimodal analgesia is particularly promising in controlling postoperative pain for patients undergoing neurosurgical procedures. For patients who experience severe pain, systemic opioids and NSAIDs, combined with sedatives and NMB agents, and/or with non-pharmacologic interventions may provide more effective pain control.

Multimodal approach to neurosurgical analgesia has a great potential to enhance postoperative recovery period.

Most efforts to control pain address the sensory experience. Physicians and nurses often measure the intensity, duration, and frequency of pain when evaluating the impact of treatment modalities. Yet the affective component, expressed as pain unpleasantness, is often not addressed. This is a vital, but less recognized aspect of the pain experience\textsuperscript{36}.

Compared to routine perioperative care, patients undergoing major surgery who participated in a perioperative multimodal approach to patient convalescence demonstrated a decrease in markers of the neuroendocrine stress response, including preservation of total body protein, lower pain scores, earlier return of bowel function, and earlier fulfillment of intensive care unit discharge criteria\textsuperscript{38}.

Although a multimodal approach to patient convalescence appears to hold great promise in decreasing perioperative morbidity and length of stay without compromising patient safety, ultimate success of this approach necessitates multidisciplinary collaboration, with a change in traditional principles of postoperative care\textsuperscript{8}.
SUMMARY

Management of the perioperative pain period is unique within the area of neuroanaesthesiology practice, in that these practitioners are intimately involved with ameliorating patient response to the trauma of neurosurgery. Our understanding of the neurophysiology of nociception suggests that attenuating central sensitization will result in diminished pain both in the immediate postoperative period and at distant time points, potentially preventing chronic pain development. There are many effective modalities and techniques to manage postoperative pain. The integration of these modalities and techniques into a complex model of patient recovery will facilitate postoperative convalescence.

The pain management regimen must be individualized on the basis of patient response, which is influenced by variables such as: age, body weight, preexisting pain, presence of opioid tolerance, need for ventilatory support, psychosocial factors, personal preferences, disease state and comorbidities, and the nature of the surgery.

REFERENCES:


