Postprocedure Pain Management of Interventional Radiology Patients

Olga Hatsiopoulou, MD, Robert I. Cohen, MD, and Elvira V. Lang, MD

Postprocedure pain management of patients after interventional procedures has to take into account residual drug actions from pre- and intraprocedure medications. A variety of sedatives, narcotics, local anesthetics, nonopioid analgesics, and nonsteroidal antiinflammatory agents can be adjusted to the patient's needs and risk factors. The article addresses the safe use of these agents in addition to reflections on assessment and the cognitive elements of the pain experience.

J Vasc Interv Radiol 2003; 14:1373–1385

Abbreviations: NSAID = nonsteroidal antiinflammatory drug, PCA = patient-controlled analgesia

INTERVENTIONAL radiologists have assumed increasingly more encompassing roles in patient management. As with all health care services, public or private, consumers are most concerned with receiving the highest quality of service (1). As radiographic procedures become more invasive, complex, readily available, and widely offered to high-risk patients, issues of analgesia and anxiolysis in radiology are more critical than ever. This article reviews methods and techniques that the interventional radiologist should be aware of and effective in when dealing with postoperative patient pain issues. The radiologist should be in tune with the needs of the patient and his/her family and should work closely with the nursing staff and the other hospital disciplines. Advice should be sought from anesthesiology and pain specialist colleagues in the case of the high-risk, frail, or elderly patients, those with anticipated ongoing pain, and when polypharmacy may be an issue.

GENERAL CONSIDERATIONS

Patients' postprocedure comfort depends largely on the intraprocedure experience. During interventional procedures under standard care conditions, pain increases with the length of the procedure, regardless of the amount of drugs given (2). Intraprocedure stress can override the anxiolytic and analgesic effects of drugs, resulting in large amounts of medication given. However, after the procedure, the full drug effect may become unimpeded resulting in deeper postprocedure sedation than desirable. Because premedications and intraprocedure drugs can carry over their therapeutic and adverse effects into the recovery period, it is important both to become familiar with extended effects and interactions of drugs used in a specific patient and to be aware of the fundamentals of pain perception and assessment.

Carryover from Premedication and Procedural Pain Management

Some practices use premedication as the sole method of analgesia and sedation or as an adjunct to intraprocedure analgesia and sedation. Commonly used agents include opiates, sedatives, anticholinergics, and occasionally steroids (as prophylaxis to contrast medium reactions) (3). Premedication agents may be used for their analgesic, sedative, and amnesic effects. They may also reduce gastric secretions and nausea and vomiting and may prevent unwanted contrast medium reactions. However, premedications may alter the patient's mental status, making direct assessment of mental status during and after the procedure challenging or impossible. For this reason, some physicians avoid their use.

Some opiates, such as morphine, have a slower onset compared with others, such as fentanyl (4). The differences in onset of drug action, combined with unpredictable drug absorption (whether given orally or subcutaneously) and possible delays in patient transfer may result in unreliable/suboptimal effects by the time that the patient reaches or leaves the procedure room. Drug interactions with baseline and intraprocedure medication, liver and renal function, and patient age and weight may affect drug levels postoperatively. These factors should be taken into consideration when devising the analgesia and sedation plan and postoperative care.

Pain management during interventional procedures often relies on local

From the Departments of Radiology (O.H., E.V.L.) and Anesthesia (R.I.C.), Beth Israel Deaconess Medical Center, Harvard Medical School, 330 Brookline Avenue (WCC 308), Boston, Massachusetts 02215. Received February 3, 2003; revision requested March 12; final revision received April 29; accepted May 2. Supported by the U.S. Army Medical Research and Material Command under DAMD 17 to 01 to 1-0153 and the National Center for Complementary and Alternative Medicine (RO1 AT 0002-05 and K24 AT 01074-01). **Address correspondence to** E.V.L.; E-mail: elang@bidmc.harvard.edu

None of the authors have identified a potential conflict of interest.

DOI: 10.1097/01.RVI.0000085769.63355.24

anesthetics and intravenous conscious sedation by combining morphine (derivatives) and benzodiazepines (4–12). For tumor embolizations, intraarterial lidocaine may be used (13). Some interventionalists may use spinal or epidural anesthesia or a regional celiac ganglionic blockade in procedures with greater anticipation of increased pain perception (14).

Most procedure personnel administer intravenous conscious sedation as they perceive the need, which may be at variance with the patient's pain perception (15). Pain management varies widely, even for equivalent procedures (15), and practice philosophy more than the patients' needs may govern medication administration (15,16). Some providers medicate only when patients become restless (17), some titrate until the patient becomes drowsy (16) and develops slurred speech or ptosis (7,18), and a few use deep sedation or general anesthesia in anticipation that patients may move or otherwise fail to cooperate.

When pain perception increases during lengthy procedures (2), physicians often resort to administering excessive amounts of sedatives and opiates with adverse outcomes. Most interventionalists can attest to the fact that highly aroused patients can override significant amounts of sedatives and opiates during their procedures but slide into a poorly arousable state afterward. Recovery of such patients then poses a considerable problem.

Postprocedure Assessment and Management of Distress

Immediately after the procedure, patients are typically observed intensely in a recovery area and then transported to their respective wards or observation beds when discharge criteria have been met. Electrocardiography, blood pressure, heart rate, and oxygen saturation monitoring is often used in immediate recovery settings (19).

During and after the procedure, vital signs and respiratory status are monitored at frequent intervals (eg, every 5 minutes) for procedure complications and any adverse drug effect such as respiratory or cardiovascular depression. Use of end-tidal CO_2 monitoring to detect early apnea is very helpful, especially for patients who remain sedated after the procedure. Pain ratings are considered the "fifth" vital sign and are best obtained with the same instruments used before and during the procedure and later on the ward in the interest of continuity of care.

In general, health professionals are fairly accurate in assessing patients' pain-related distress (20). However, this accuracy decreases as procedures become more challenging and stressful for the operator (20). In addition, nursing personnel may underestimate postoperative distress. The patient's self-rating is still considered the single most reliable descriptor of the pain experience (5,21). There are several graphic pain scales that score the pain and help to assess the efficacy of the pain management (22,23). They include visual numeric scales in horizontal or vertical arrangement (Thermometer scales) with or without verbal description, Likert-type scales with expressions of severity, and face scales. Self-reporting scales (23–33) generally have similar statistical power, efficacy, and response problems (29). Of the self-reported scales available to measure pain intensity, the visual numeric scale is particularly easy to administer, correlates well with other intensity scales, is sensitive to change, and has demonstrated success also with elderly adults (23). It typically elicits responses on a scale of 0 (no pain at all) and 10 (worst pain possible), and patients either identify a number while viewing the scale or by placing an X. In the case of radiologic procedures, immobilization of patients on the procedure table or in bed afterward can make use of visual scales cumbersome. Fortunately, a purely verbal 0–10 linear numerical scale, with 0 = no pain at all and 10 = worst pain imaginable, has been validated and can be used as an alternative (33,34).

Because pain and anxiety are interrelated (35,36), measurements of anxiety can be helpful but are not yet considered routine. Ratings for anxiety can be obtained by relatively extensive instruments such as the Spielberger State and Trait Anxiety Scale, a multiquestion assessment (37). However, in practice, a simple verbal analog scale can be used successfully in the interventional setting and for patient follow-up. This scale is a verbal analog rating scale with the extremes (0 = no anxiety at all and 10 = worst anxiety possible) (38). This scale correlates substantially with the Spielberger State Anxiety Inventory, and both tests have similar relationships to neuroticism, extroversion, conscientiousness, agreeableness, and openness to experience as measured by the "Big Five Inventory" (39,40).

DRUGS USED FOR PAIN MANAGEMENT IN THE PERI- AND POSTPROCEDURE PERIOD

Overview

Postprocedure management includes nonsteroidal antiinflammatory drugs (NSAIDs), narcotics, sedatives, antiemetics, and local anesthetic field and nerve blocks (41). Routes include oral, mucosal, rectal, subcutaneous, intramuscular, transcutaneous, and intravenous. Medication can be administered by providers as needed, by the clock, or by the patient with patient-controlled analgesia (PCA). In a PCA approach, the patient self-delivers intravenous drugs within preset limits.

Because there is an overlap between drugs used during and after procedures, we focus on potential aftereffects and drug interactions of these medications.

Opioids

Mode of Administration.—Opioids are the most potent analgesics available for treatment of postprocedure pain. The commonly used opioids are summarized in **Table 1** (42). Opioids are administered postoperatively via the intravenous, intramuscular, subcutaneous, oral, or rectal route. On the hospital ward, opioids may be administered by nursing personnel as needed, on a regular schedule, or self-administered with a PCA device (see below). Given as needed by medical personnel, patients are at risk of receiving inadequate analgesia because providers may underestimate the pain level, overestimate the opioid effect and duration, or fear opioid abuse and side effects (43). However, under- and overmedication can also occur with PCA.

The use of opiate transdermal patches has not yet found entry into

Table 1
Approximate Equianalgesic Doses of Commonly Used Opiate Analgesics

Drug	Equianalgesic Parenteral Dose	Equivalent Enteral Dose	Typical Clinic Dose
Morphine*	10 mg	30 mg (10–30% oral bioavailability)	15–30 mg p.o.
Hydromorphone (Dilaudid)	1.5 mg	7.5 mg	2–6 mg p.o.
Fentanyl*	100 μg	200–800 U OTFC‡	25–50 μg/h patch
Meperidine (Demerol)	100 mg	300 mg	50–100 mg p.o.
Methadone (Dolophine)	10 mg†	20 mg (50% oral bioavailability)	5–10 mg p.o.
Codeine	75 mg	150 mg	30–60 mg p.o.
Hydrocodone (in Lorcet, Lortab, Vicodin, others)	NA	30 mg	5–10 mg p.o.
Oxycodone* (Roxicodone, also in Percocet, Percodan, Tylox, others)	14 mg (parenteral formulation NA in U.S.)	15–30 mg (50% oral bioavailability)	5–10 mg p.o.

When changing drugs, start with a dose 50% less than the calculated equianalgesic dose, then titrate upward in subsequent doses based on patient response. Safe dosing may be affected by patient weight, general health status, vital signs, and comfort levels. Patient variability is very high. In a typical clinic setting, prescribed individual doses would likely be lower than those used in the postprocedure setting for patients with high pain intensity.
* For patients taking sustained-release formulations before the procedure, it may be wise to continue doses before the procedure.
† Response to methadone may vary, especially in patients taking high doses of other opioids before the procedure. Use caution because these opiate-tolerant patients may be very sensitive to methadone.
‡ Oral transmucosal fentanyl citrate buccal lozenge, 800 μg = 10 mg intravenous morphine in one study (88).
Note.—p.o. = orally; NA = not available.

the routine management of postprocedure discomfort but may be an attractive alternative in select patients when postprocedure pain is expected to continue for a long time (weeks or months). For patients on transdermal fentanyl patch before the procedure, this treatment may be considered without interruption to treat baseline pain and supplement with additional short-acting opiates. Discontinuing the patch should be considered when the procedure is expected to significantly lessen the pain, as with, for example, vertebroplasty.

Epidural or intrathecal administration of opiate is available for patients expected to have very high postprocedure pain levels or for patients who cannot tolerate enteral or parenteral opiate administration. This modality may be particularly helpful in treating intense postembolization pain, such as in preoperative renal embolization. Placement and removal of epidural catheters in anticoagulated patients are discouraged because of an increased risk of epidural hematoma. In situations in which this cannot be avoided, ongoing neurologic monitoring is suggested.

The intravenous route has the most rapid onset of action and the shortest duration and thus is the safest for treating acute postprocedure pain when compared with intramuscularly or subcutaneous administration. The intramuscularly or subcutaneous route can result in drug accumulation, especially when multiple doses are given in a short time in an attempt to treat pain of high intensity. Systemic absorption may be reduced and unreliable when vascular drug uptake is reduced, as in cases of hypothermia, hypovolemia, and peripheral vasoconstriction. Of the commonly used sites, the deltoid muscle gives the most consistently rapid intramuscular absorption. Avoid the intramuscular/subcutaneous route in cases of shock or sepsis.

The oral route can be used in patients who can tolerate oral intake and have no contraindication to oral administration (eg, depressed gag reflexes). Because the enteral route involves drug elimination by the gut and liver, the analgesic effect may again be unreliable, the onset of action slow, and establishment of an effective serum level may be delayed. The oral route is unsuitable for patients with intestinal malabsorption and anatomic or functional short gut syndrome. Sublingual, intranasal, and rectal administration may bypass the enterohepatic circulation and may achieve a more rapid increase in circulating analgesic concentration levels.

Opioid Delivery via PCA.—In the recovery area, postprocedure pain of high and moderate intensity can be treated with an intravenous opiate titrated until pain levels become tolerable. Then PCA can be started so that the patient can maintain the analgesic state. During this titration, valuable information is gained about the amount of opiate necessary to treat the pain. The dose may be large in patients with high tolerance and/or with intense pain levels. For moderate pain in an opiate-naive patient, begin with a low dose, such as 1–2 mg morphine. The distribution half-life to the brain for morphine is approximately 5 minutes. If there has been no change in pain intensity after 5–10 minutes, repeat with a higher dose, such as 2–4 mg. After waiting another 5–10 minutes, if there is no decrease in pain, check that the intravenous line is running properly and in a patient on long-term opioid therapy, an even higher dose may be tried. If after one or two initial doses, the pain is still at moderate intensity but has decreased some or even a little, the previous dose should be repeated rather than an increased dose. If pain intensity has significantly decreased, wait and administer a lower dose next time if the pain is not controlled. If the patient starts with very intense pain,

Table 2
Opioids Commonly Used in PCA with Examples of Doses and Lockout Times

Drug	Loading Dose to Treat Intense Pain before Starting PCA	Usual PCA dose	Opioid-Tolerant Dose	Lockout Time (min)
Morphine	2–4 mg, repeat up to 30 mg/70 kg	1 mg	5 mg	5–10
Fentanyl	25–50 μg, repeat up to 300 μg/70 kg	10 μg	50 μg	3–6
Hydromorphone (Dilaudid)	0.4–0.8 mg, repeat up to 6 mg/70 kg	0.2 mg	1 mg	5–10
Meperidine (Demerol)	Not recommended for PCA use due to possibility of seizure due to normeperidine accumulation			

*Always follow local PCA guidelines regarding doses and lockout intervals.

start with higher doses and escalate if there is no response to the selected dose, or repeat a dose if a partial response is elicited, and so on.

PCA dosing can be individualized for each patient. Suggestions are given in **Table 2**. If the PCA dose is too high for a given patient, a side effect may result in negative feedback, resulting in failure to achieve analgesia. For each patient, there is a balance among an inadequate dose, a sufficient dose, and an overdose eliciting adverse effects.

It is important to know how to dose the PCA correctly for each patient and to recognize common failures so that adjustments can be made and analgesia achieved. For example, when a patient reports a high pain level, the correct response is to bolus with additional intravenous opiate and usually to increase the PCA dose. However, if questioning reveals that 5 minutes after a delivered dose, the patient becomes sedated, falls asleep, and then awakens in pain after 15 to 30 minutes, the correct response may be to decrease the PCA dose. Although counterintuitive, decreasing the dose prevents toxicity (sedation), and the patient remains awake to call for additional doses that, during 1 hour, result in more medication delivered and effective analgesia. A similar negative feedback results with other side effects. A patient who becomes nauseated or dizzy after each dose may fail to self-administer sufficient analgesic. A lower dose and shorter lockout interval on the PCA may be helpful. Other problems occur when patients believe opiate medication to be bad or evil or for cultural reasons are reluctant to self-medicate.

A dose too low also provides negative feedback when the patient fails to get sufficient relief to justify pushing the button again. This is negatively reinforced when each PCA dose fails to produce significant relief. Even a correct PCA dose may fail when initial pain levels are not treated by the initial intravenous administration of sufficient doses of the opiate to be used in the PCA because analgesic serum levels are never obtained.

Factors Affecting Opioid Response.— Patient characteristics that may affect the response to an opiate include age, weight (especially obesity and malnutrition), hypoalbuminemia, and systemic diseases, particularly hepatic and renal impairment.

Age

Sensitivity to opioids may increase with age, owing in part to pharmacokinetics (44,45). For example, a decrease in clearance increases the duration of effect, and high or repeat doses may cause opioid concentration in the systemic circulation to be higher and remain longer than expected (44).

Weight and Nutrition

The free unbound fraction of the drug reaches the target receptors and produces the analgesic effect. The rest is bound to proteins, mainly albumin and α_1-glycoprotein. In hypoalbuminemic patients, the bound fraction is reduced, leaving more free fraction to cause the opioid effect, hence increasing the patient's sensitivity to the drug. It is important to titrate the doses accordingly for patients who may have low protein levels, for example, the malnourished, the chronically ill, and those with inflammatory bowel disease and protein deficiencies.

Systemic Disease

Opioids undergo hepatic metabolism, such as conjugation of morphine to morphine-3-glucuronide, which is then excreted by the kidney. Decreased hepatic function results in reduced hepatic clearance and prolonged drug effects. Patients with renal insufficiency may experience metabolite accumulation, which, in the case of meperidine, can result in neurotoxicity, central nervous system excitation, and seizures (46). For this reason, meperidine has fallen out of favor. Accumulation of the normeperidine metabolite may become problematic, even in adults without renal insufficiency when the meperidine dose is significantly greater than 500 mg/d for more than several days.

Adverse Effects with Opioid Use.— Adverse effects are generally dose related and resolve quickly when opioid serum levels fall.

Respiratory Depression

Opioids acting on μ-receptors in the brain stem interfere with respiratory rhythmicity, often resulting in decreased respiratory rate, respiratory pauses, irregular and periodic breathing, desaturation, and hypercarbia. The normal sympathetic response to an increase in serum carbon dioxide leads to increased ventilation. Ventilatory drive in response to increasing levels of carbon dioxide is inhibited by opiates. High serum carbon dioxide levels augment opioid sedation in a dangerous positive feedback loop leading to apnea.

Naloxone, naltrexone, and nalbuphine all reverse the μ-opioid respiratory depressant effect. However, caution is advised when administering a μ-antagonist. Although it may be possible to give a low dose that significantly improves ventilatory response

and respiratory rate, high doses may be dangerous. If they unmask μ-opioid receptor analgesia, there may be a sudden surge of high levels of pain leading to a massive sympathetic discharge resulting in myocardial ischemia.

When the duration of action of a μ-antagonist (such as naloxone) is less than that of a μ-agonist (such as morphine or fentanyl), the patient should be monitored for a longer period until it is unlikely that respiratory depression will recur. The circulating concentration of the μ-antagonist decreases faster than the circulating concentration of the μ-agonist. Some institutions suggest increased monitoring for return of respiratory depression for more than an hour and as long as 2 hours after a dose of a short-duration μ-antagonist such as naloxone.

Nausea

Narcotics have a direct stimulatory effect on the central chemoreceptor trigger zone in the medulla and may lead to nausea and vomiting, particularly when the dose exceeds that required for analgesia in a given patient. For a given patient, one opiate may be more likely to trigger nausea and vomiting than another opiate. This emetic effect is reported to be more severe with morphine than fentanyl. However, it should also be kept in mind that untreated pain can also stimulate the chemoreceptor trigger zone and result in nausea (47).

Increased Muscle Tone

Narcotics increase the smooth muscle tone of the pylorus, ileocecal valve, and the sphincter of Oddi. This may result in decreased gastric emptying and bouts of biliary colic in susceptible patients. Stimulation of the detrusor muscle tone can result in urinary retention. These are μ-receptor effects and can be reversed by μ-antagonists such as naloxone.

Increased skeletal muscle tone can sometimes become manifest, particularly when a high dose of opiate is administered rapidly and can result in increased muscle rigidity affecting the muscles of the chest and abdominal wall (48,49). The exact mechanism of this effect is not clear. Because of the chest wall stiffness, ventilation may be uncomfortable or possibly difficult to maintain. The effect is often transient, and reassurance may be all that is necessary; however, in rare but life-threatening cases, high-flow oxygen with assisted mask ventilation should be initiated, and an anesthesiologist should be called immediately. In these cases, administration of a muscle relaxant, such as succinylcholine, and intubation of the trachea may become necessary.

Narcotic Antagonists

Naloxone hydrochloride (Narcan) is a pure opioid antagonist competing for opioid receptors with agonist. It is used to reverse narcotic overdose effects, particularly respiratory depression (50). Naloxone hydrochloride is administered intravenously as a bolus injection; onset of action is rapid, within 1–2 minutes. It is supplied in vials of 0.02, 0.4, and 1.0 mg/mL. These should be readily available for emergency use. For postprocedure opioid-induced sedation and respiratory depression, the manufacturer recommends that the dose be titrated in 0.05–0.1-mg increments and waiting 2–3 minutes between doses until the desired effect is achieved by monitoring the patient's respiratory effort and rate. The authors recommend a slightly lower dose 0.04–0.08 mg/70 kg for use in interventional radiology. Higher doses, such as 0.2–1 mg, are appropriate for drug overdose in the emergency department or to reverse μ-opioid effects when reemergence of pain is not an issue.

By carefully titrating the dose, it is possible to reverse respiratory depression without eliminating all the analgesic effect and without producing unwanted side effects such as nausea and vomiting. A catecholamine response to sudden severe pain due to analgesia reversal with naloxone may result in cardiac dysrhythmias and can often be prevented by careful titration of low doses.

It is important to stress that naloxone is a short-acting antagonist. The patient should be monitored and not left unattended after the respiratory depression is reversed because the narcotic effect may well reappear and further reversal may be required. Current practice guidelines suggest observing a patient for at least 1–2 hours after the last dose of reversal agent. Extended recovery is suggested for patients who received reversal after a large or long-acting opioid dose, especially if the dose was administered intramuscularly, subcutaneously, as needed, or orally.

Benzodiazepines

Benzodiazepines provide sedation, amnesia, and anxiolysis but no direct analgesia. However, they decrease the component of unpleasantness in pain. The short duration of action of a single, low dose of midazolam is owing to rapid redistribution out of the central nervous system. However, repeated or high doses may have a longer clinical effect. The half-life of midazolam is approximately 3 hours and 6 hours for the active metabolite 1-hydroxymidazolam. Diazepam has a long elimination half-life, as long as 48 hours, and its active metabolites may have an even longer elimination half-life.

Benzodiazepines undergo hepatic metabolism. Diazepam and midazolam have active metabolites that can accumulate in patients with renal dysfunction. For this reason, dose reduction should be considered in patients with hepatic or renal disease as well as careful titration during the procedure. Midazolam may have a half-life of as long as 20 hours in patients with renal failure.

Benzodiazepines cause a dose-related central respiratory system depression that can lead to complete apnea. Apnea is more likely with rapid intravenous administration of midazolam in elderly or debilitated patients and in the presence of hepatic impairment. Respiratory depressive effects tend to be synergistic when benzodiazepines are administered with opioids.

Benzodiazepines can lead to hypotension and tachycardia, especially in the elderly, severely ill patients, or in patients with unstable cardiovascular status. Parenteral preparations of benzodiazepines (apart from midazolam) contain organic solvents such as propylene glycol that produce pain on injection. This can cause venous irritation, swelling, and consequently thrombophlebitis. Although rarely recommended for intravenous use, diazepam should be administered slowly via a rapidly flowing intrave-

Table 3
Commonly Used Nonopioid Medications Including NSAIDs and Cyclooxygenase-2 Agents

Drug	Class	Route	Onset (h)	Duration (h)	Maximal Dose (mg/24 h)
Acetaminophen	Para-aminophenol	Oral	0.5	2–4	3,000–4,000
Aspirin	Salicylate	Oral	0.5–1.0	2–4	3,600
Naproxen	NSAID	Oral	1	4–7	1,500
Fenoprofen	NSAID	Oral	1	4–6	3,200
Ibuprofen	NSAID	Oral	0.5	4–6	3,200
Ketorolac	NSAID	IV/IM/Oral	0.5–1.0	4–6	120
Rofecoxib (Vioxx)	Cox-2	Oral	0.5	24	50
Celecoxib (Celebrex)	Cox-2	Oral	1–3	12–24	200
Valdecoxib (Bextra)*	Cox-2	Oral	1–3	24	20

* U.S. Food and Drug Administration approval for IV formulation pending.
Note.—IV = intravenous; IM, intramuscular; Cox-2, cyclooxygenase-2. Cox-2 agents may offer significant potentiation of opiate analgesia and may have less effect on platelet function than other NSAIDs and should be considered when treating postprocedure pain in a setting in which hemorrhage is a concern.

nous line to reduce venous irritation and discomfort.

Benzodiazepine Antagonists.—The effects of benzodiazepines can be reversed by the antagonist flumazenil. Flumazenil is a competitive inhibitor of benzodiazepine activity at the γ-aminobutyric acid/benzodiazepine receptor complex, and it reverses the sedative effects of the benzodiazepines. Flumazenil [0.2 mg (2 mL)] is administered intravenously for over 15 seconds. As many as four additional doses of 0.2 mg can be administered at 60-second intervals (for a total dose of 1 mg) to restore the desired level of consciousness. Because the duration of action of flumazenil is shorter than that of the benzodiazepine it reverses, resedation is common. If resedation occurs, repeated doses (of ≤1 mg) may be administered at 20-minute intervals. In patients with hepatic disease, the clearance of flumazenil is reduced and the dose and frequency of administration may need to be reduced. Flumazenil has been associated with the precipitation of seizures in high-risk populations. Therefore, flumazenil is not recommended when signs or symptoms of tricyclic antidepressant overdose are present. Inpatients on long-term benzodiazepine treatment, flumazenil can precipitate benzodiazepine withdrawal with hot flashes, agitation, dizziness, mild confusion, emotional liability, and mild sensory distortions.

It is important to be aware that flumazenil may not reverse respiratory depression in patients who have received benzodiazepines in combination with opioids. In these patients, concurrent administration of low-dose (0.02–0.04 mg/70 kg) naloxone can be considered. After reversal of the benzodiazepine effect, patients must be monitored for resedation, respiratory depression, or other residual benzodiazepine effects for an appropriate period based on the dose and duration of effect of the benzodiazepine used. Extension of recovery time by 1–2 hours for patients who have received benzodiazepine reversal is a common practice. Resedation is least likely when a low-dose of a short-acting benzodiazepine (such as midazolam) has been used. Resedation is most likely when long-acting benzodiazepines are administered in high doses or multiple lower doses.

Nonopioids for Postoperative Pain Management

Nonopioid analgesics may be used alone or in combination with opioids and local anesthetic agents. Commonly used nonopioids include acetaminophen and NSAIDs. An overview is given in **Table 3**. The World Health Organization recommends that analgesics be used in a stepwise pattern beginning with NSAIDs, progressing to weak opiates, then strong opiates. The addition of NSAIDs can significantly improve the efficacy of opiate analgesia, reduce opiate dose, and lessen side effects.

Acetaminophen (Tylenol) decreases fever by an effect on the hypothalamus leading to sweating and vasodilation. It also inhibits the effect of pyrogens on the hypothalamic heat-regulating centers. It may cause analgesia by inhibiting central nervous system prostaglandin synthesis; however, because of minimal effects on peripheral prostaglandin synthesis, acetaminophen has no antiinflammatory effects. It has antipyretic and analgesic effects similar to those of aspirin, although specific differences in mechanism of action have been suggested (51). A dose of acetaminophen may be administered before the procedure to reduce the dose of opiate that may be required.

In some respects, acetaminophen is safer than NSAIDs because it has fewer side effects. For example, acetaminophen does not cause an anticoagulant effect or ulceration of the gastrointestinal tract and is safe to use in patients with a history of peptic ulceration and bleeding diathesis and after discharge for procedures such as biopsies. Although acetaminophen has few adverse effects when taken in usual therapeutic doses, chronic and even acute toxicity can develop after long-term, symptom-free use. Acetaminophen overdose can lead to hepatotoxicity with liver necrosis and death. Acetaminophen overdose is a medical emergency.

Acetylsalicylic acid (aspirin) has antipyretic, antiinflammatory, and analgesic effects. The antipyretic effect is owing to an action on the hypothala-

Table 4
Characteristics of Commonly Used Local Anesthetic Agents

Agent	Most Dilute Commerical Formulation	Onset of Action (min) (Infiltration)	Duration of Action (min) (Infiltration)	Maximal Dose (mg/70 kg) (with Vasoconstrictor)	Maximal Dose (mL/70 kg) (of Dilute Formulation with Vasoconstrictor)
Lidocaine (Xylocaine)	0.5% (5 mg/mL)	4–10	60–120	300 mg (500 mg)	60 mL (100 mL)
Bupivacaine (Marcaine, Sensorcaine)	0.25% (2.5 mg/mL)	8–12	120–240	175 mg (225 mg)	70 mL (90 mL)
Mepivacaine (Carbocaine, Polocaine)	1% (10 mg/mL)	6–10	90–180	400 mg (500 mg)	40 mL (50 mL)
Levobupivacaine (Chirocaine)	0.25% (2.5 mg/mL)	8–12	120–240	150 mg (NR)	60 mL
Ropivacaine (Naropin)	0.2% (2 mg/mL)	8–12	120–240	175 mg (NR)	87.5 mL

Maximal doses are given for use without a vasoconstrictor. Always calculate the maximal single dose taking into consideration the patient's weight and the actual concentration of the local anesthetic preparation used; hence, for a 70-kg patient, the maximal dose of 1% lidocaine without epinephrine is 30 mL. Always note whether a vasoconstrictor is added in the preparation because this alters the maximal recommended dose. For example, the maximal dose of lidocaine with epinephrine would be 500 mg/70 kg (50 mL of 1% solution of lidocaine with epinephrine).

mus, resulting in heat loss by vasodilation of peripheral blood vessels and by promoting sweating. The antiinflammatory effects are probably mediated through inhibition of cyclooxygenase, which results in a decrease in prostaglandin synthesis and other mediators of the pain response. Aspirin also produces inhibition of platelet aggregation by decreasing the synthesis of endoperoxides and thromboxanes. For this reason, aspirin should not be used in patients with severe anemia, bleeding diathesis, or a history of peptic ulcer disease; in conjunction with anticoagulant therapy; or immediately pre- and postoperatively. The platelet aggregation inhibition tends to be dose related, and the effect on a given platelet tends to last the life of the platelet.

When low-dose aspirin is administered to prevent platelet adhesion and reduce the risk of stroke, heart attack, or postangioplasty platelet aggregation, caution should be used when prescribing other NSAIDs. Nonaspirin NSAIDs reversibly inhibit cyclooxygenase, which regains activity as the NSAID level falls. When a dose of aspirin is taken after a dose of ibuprofen, these platelet sites are "protected" from irreversible aspirin acetylation of cyclooxygenase. When the ibuprofen concentration decreases, these platelet sites become active and the patient may be at increased risk of a thrombotic event (51).

Other NSAIDs

NSAIDs have analgesic, antiinflammatory, and antipyretic properties. NSAIDs are available in oral preparations as well as parenterally. Ketorolac (Toradol) and soon valdecoxib (Bextra) are intravenous formulations that can be given during or after the procedure to treat mild and moderate pain. Valdecoxib, available now in an oral formulation, is believed to inhibit cyclooxygenase-2 without inhibiting the form of cyclooxygenase responsible for platelet aggregation and can be used in patients in whom the risk of hemorrhage is too high to receive ketorolac. The manufacturer recommends that intravenous ketorolac be used for only a very limited duration and then should be switched to an oral formulation. We found ketorolac particularly useful during uterine fibroid embolization; one dose during the procedure and one dose in the evening after the procedure seem to provide reasonable pain relief and reduce the need for opiates.

NSAIDs are advantageous in the perioperative/procedure period in that they do not provoke the common opioid adverse effects of nausea and vomiting, sedation, urinary retention, gastric stasis, and constipation. However, the risk/benefit ratio should be reconsidered when NSAIDs is used in patients with asthma who have a history of sensitivity, in patients with a history of peptic ulcer disease, and in patients on anticoagulant therapy. NSAIDs may produce or exacerbate renal insufficiency and should be used with caution in patients with this condition. It may also produce or exacerbate hypertension. It is best to limit the use of ketorolac to a few days postoperatively.

Local and Regional Anesthesia

Overview and General Considerations.—Local anesthesia is used mostly during procedures but can also be effective in postprocedure care. It can be used as local infiltration around catheter and puncture sites and for regional nerve blocks. Topical preparations such as Hurricane spray (Benzocaine) and EMLA creme (eutectic mixture of local anesthetic: lidocaine and prilocaine) may sometimes be all that is needed to provide local anesthesia to the skin.

Table 4 summarizes the properties and maximal recommended doses of the injectable local anesthetics (52). The more widely used local anesthetics are lidocaine, mepivacaine, and bupivacaine (53,54). One of the most

common preparations is a mixture of 1% lidocaine without or with epinephrine at 1:100,000 concentration.

The duration of local anesthetic effect in a tissue is reduced in tissues with greater perfusion. The faster a local anesthetic is taken up into the systemic circulation, the faster its effect will be terminated. Therefore, the onset of action will be greater and the duration of the effect shorter for more vascular sites of injection where the uptake rate into the systemic circulation is greater. Following the same principle, the addition of a local vasoconstrictor into the local anesthetic preparation will decrease its systemic uptake and enhance its potency in the local tissue. Epinephrine is the vasoconstrictor most commonly used with lidocaine. Other vasoconstrictors such as norepinephrine and phenylephrine have also been added. Preparations with vasoconstrictors should be used with caution in areas of limited blood supply (ears, noses) or in distal appendages (fingers, toes, penis) because of a potential risk of local necrosis.

Although most local anesthetics produce sensory and motor block at nearly the same concentration, three of the amide local anesthetics have a differential effect favoring a block of sensory fibers, with higher concentrations required to produce motor block. The most commonly used of this group is bupivacaine (Marcaine/Sensorcaine). Touting reduced cardiac toxicity, levobupivacaine (Chirocaine), and ropivacaine (Naropin) also produce differential sensory block similar to bupivacaine.

Differential Block: Fiber Type.—Local anesthetics work by blocking sodium channels in membranes, thereby inhibiting nerve action potentials and pain transmission. Sodium channel sensitivity and distribution in nerve fibers may influence pain transmission (55). Thus, different fiber types would be expected to respond with greater or lesser sensitivity to a given local anesthetic type and/or a given concentration of that anesthetic. Most sensitive are the small, slowly conducting unmyelinated nerves called C fibers. Carrying many of the nociceptive signals into the central nervous system resulting in the perception of pain, these nerves seem to be sensitive to lower concentrations of local anesthetic than larger, faster nerves responsible for proprioception and motor activation. Thus, local anesthetic concentrations that do not fully block sensation or motor function can lead to analgesia. Interestingly, in several settings, including inflammation, the number of sodium channels increases significantly and the nerve becomes more sensitive. The frequency of its firing may increase until the nerve fires repetitively even without a stimulus, a phenomenon known as wind-up that leads to perceived pain in the absence of a painful stimulus (allodynia). Very low concentrations of local anesthetic may be effective in treating this pain when given by infusion in a pain clinic setting.

Local Anesthetic Toxicity.—Beyond nociceptive inhibition, increasing doses of local anesthetic deepen the sensory block, and finally produce a profound motor block. As the local anesthetic is absorbed into the systemic circulation, distant sodium channels in distant tissues can become blocked. If the concentration of local anesthetic rises high enough, it can block sodium channels in inhibitory interneurons in the brain leading to loss of consciousness and, at even higher concentration, seizures.

Sodium channels are also present in the cardiac conduction system. High circulating concentrations of local anesthetic can lead to heart block and malignant re-entrant rhythms with ventricular fibrillation that may be resistant to electrical defibrillation until the level of local anesthetic falls. Patients with cardiac toxicity may require prolonged cardiopulmonary resuscitation or even cardiopulmonary bypass until local anesthetic levels fall sufficiently for a normal rhythm to be restored.

Interestingly, like chemotherapeutic agents, local anesthetics are drugs for which maximal dose guidelines are offered. The most commonly used local anesthetic, lidocaine, has a suggested maximal limit of 4–5 mg/kg. If a provider would prefer to think in terms of the volume administered, then for 0.5% lidocaine, this is 1 mL/kg. For bupivacaine at 0.25%, it is easy to remember 1 mL per kg (= 2.5 mg/kg = 175 mg/70 kg).

If a high dose of local anesthetic is injected into a vessel, central nervous system and cardiac levels may rise quickly to toxic levels. The recommended doses are designed to reduce the occurrence of systemic toxicity (high serum levels) as the local anesthetic is gradually absorbed from body tissues. Staying within the recommended dose will not prevent toxic levels from occurring when a dose meant for soft tissue injection is directly injected into a vessel. Thus, before injection through a nonmoving needle, aspiration for possible blood return should be done routinely with every injection. An exception can be made when the needle is moved continuously during the injection. It is still possible for a needle to be aligned with a vessel resulting in toxicity if a high dose is injected rapidly.

When maximal or greater than maximal recommended doses are administered, the risk increases for seizures or cardiac arrest as the drug level rises with tissue absorption. With lidocaine in a well-perfused tissue (intercostal block), a peak serum level may occur in less than 30 minutes. With bupivacaine, a local anesthetic that is both more lipid soluble and more tightly bound to receptor protein, the serum peak may be delayed by more than 40 minutes and reinjection within this time can lead to toxicity. When the dose approaches the recommended maximum, it is safer to wait before administering additional doses with a guideline of at least 20 minutes for lidocaine and 40 minutes for bupivacaine.

Treating Local Anesthetic Toxicity

Patients experiencing visual disturbances, tinnitus, tongue numbness, muscle twitching, and lightheadedness should be suspected of having local anesthetic toxicity. They need urgent attention and may require resuscitation within seconds or minutes. When signs and symptoms of local anesthetic toxicity are apparent, the local anesthetic injection should be stopped immediately and the patient's vital signs continuously monitored.

In cases of mild toxicity from intravascular injection in which the patient is experiencing perioral paresthesia and dysphoria, it may suffice to reassure the patient that the symptoms

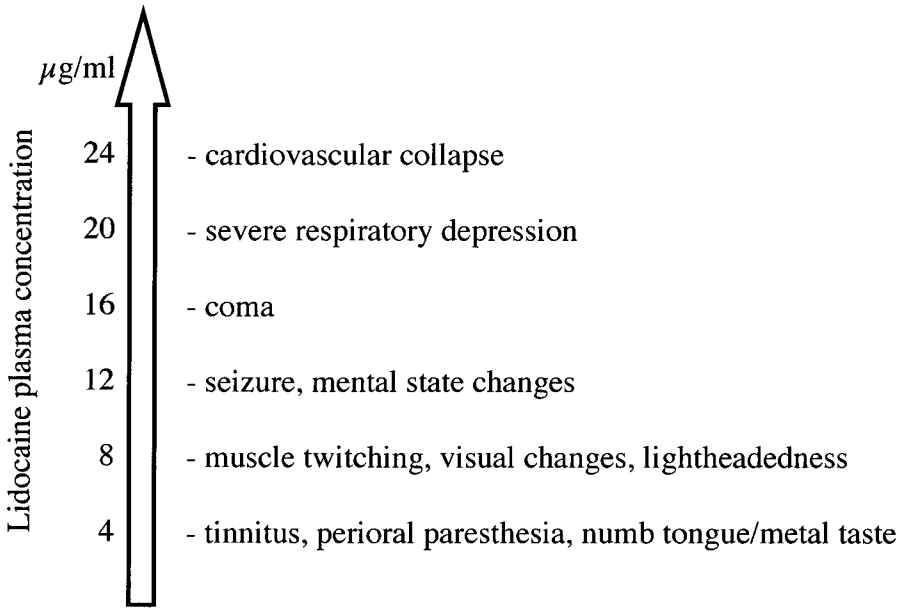

Figure. Lidocaine toxicity as a function of its plasma concentration. (Adapted and reprinted with permission, from reference 56.)

will subside shortly. The patient should be informed that taking deep breaths will help the symptoms pass more quickly. These deep breaths not only trigger a relaxation response but cause an alkalotic shift in brain tissue pH, facilitating passage of somewhat acidic local anesthetic molecules out of nerve cell membranes. This may not work in situations in which tissue injection of a toxic dose is producing an increasing serum concentration of local anesthetic, although it is still reasonable to give oxygen to a patient experiencing toxicity and to use a bag valve mask to deliver positive pressure ventilation if the patient loses mental alertness. Drugs that raise the seizure threshold, such as a benzodiazepine, may be necessary to treat severe central nervous system toxicity (seizures). Intubation may be required and, if cardiac arrest occurs, resuscitation until the patient recovers or until sufficient time has elapsed for the serum level to fall below toxic levels (several hours). Rapid access to cardiopulmonary bypass may effectively maintain perfusion and should be considered part of the treatment of severe toxicity, especially for the more cardiotoxic local anesthetics such as bupivacaine.

Lidocaine.—With lidocaine, there is a spectrum of toxicities as the serum level increases (**Figure**) (56). At low serum levels (1–4 μg/mL, the therapeutic level for treatment of ventricular ectopy), patients may complain of perioral paresthesia or tinnitus. As the concentration increases toward 8 μg/mL, patients may have significant changes in mental status. Occasionally, a patient who is not cooperative during a painful procedure may have lost the ability to volitionally control his/her behavior due to central nervous system toxicity of the local anesthetic. Thus, if a maximal dose of local anesthetic has been approached, adding a low dose of opioid or stopping the procedure and waiting for the local anesthetic level to fall would produce a better result than attempting to give additional local anesthetic at this point. If the serum level of lidocaine increases to 12 μg/mL, a seizure may result. Cardiac arrest becomes likely at twice this concentration. In this respect, lidocaine has a high therapeutic index. The serum concentration leading to significant change in mental status is twice the concentration leading to benign reportable side effects. Doubling the serum level again leads to seizures, but cardiac arrest is not likely until the concentration is doubled yet again. Safe use of local anesthetic mandates that the provider look for early signs of toxicity, such as perioral paresthesia, and solicit this information from the patient repeatedly before giving additional doses of local anesthetic. Injection of intraarterial lidocaine during tumor embolizations can be an effective intraoperative measure in postoperative pain management (13). When a local anesthetic is intentionally administered intravascularly, it should be kept in mind that, at least for lidocaine, this is an off-label use for a drug that has a long safety record for treatment of ventricular arrhythmia, in which a loading dose of 1–1.5 mg/kg (approximately 100 mg/70 kg) is the current American Heart Association guideline. This would represent 10 mL 1% lidocaine. After 10–15 minutes, it may be possible to give additional lidocaine safely in patients with normal cardiac output and hepatic function. For patients taking medication such as β-adrenergic antagonists (to reduce hepatic blood flow) or cimetidine (to inhibit hepatic metabolism), consider reducing the dose. During transcatheter tumor therapy, the safety margin of lidocaine theoretically increases when lidocaine is injected after the embolization material starts slowing flow, reduces perfusion, and thus potentially reduces systemic uptake (see below). One review advocates 30-mg boluses of intraarterial lidocaine to as much as 200 mg (= 20 mL 1% solution) total during liver embolization (57).

Bupivacaine.—Bupivacaine has several advantages over lidocaine, provided toxicity from intravascular injection and/or high levels from tissue absorption of high doses do not occur. Because it more avidly binds to the sodium channel, its duration of action is considerably longer, making it more effective during the postprocedure period. Also, it has a differential effect at a lower concentration that is even more significant than lidocaine for blocking sensory nerves. Solutions of dilute bupivacaine of ≤0.25% inhibit sensory fibers to a much greater degree than motor fibers.

With a delayed onset of action compared with that of lidocaine due to its lower pH, a greater potency due to its greater lipid solubility, and a longer duration of action due to its

greater receptor-protein binding, it is not surprising that patients report perioral paresthesia, tinnitus, and mild sedation at very low serum levels (<1 μg/mL). Compared with lidocaine, bupivacaine has a much lower therapeutic index because cardiac arrest may occur at the same serum concentration that produces loss of consciousness or seizures. Additional caution regarding total dose and avoidance of intravascular injection should be exercised when bupivacaine is used. Patients should not receive more than 175 mg/70 kg (225 mg/70 kg if mixed with a vasoconstrictor such as epinephrine at 1:200,000). For the same reasons, bupivacaine should therefore not be used for intravenous regional anesthesia or for intraarterial injection.

Levobupivacaine.—Bupivacaine is a racemic mixture of two isomerically active forms. Most of the nerve conduction blockade is due to the L or levorotatory structure, whereas most of the cardiotoxicity is due to the D or dextrorotatory molecule. Levobupivacaine (Chirocaine) shares the desirable characteristics of differential block (stronger sensory than motor block) and long duration of action with its racemic cousin. If higher doses are required during a procedure, this drug may be a safer alternative.

Ropivacaine.—Similar in structure but less lipid soluble than bupivacaine, ropivacaine (Naropin) is less potent and may be less active at sodium channels within the myocardium. Ropivacaine is not optically active so that it does not have a D form and may be less cardiotoxic for this reason. Like the L form of bupivacaine, ropivacaine appears to spend less time bound to cardiac sodium channels. Like its analogues, it exhibits a differential block. In a recent study comparing levobupivacaine with ropivacaine in the setting of interscalene block for surgical anesthesia, both agents had equal efficacy when injected at an equal concentration and volume. In the postoperative setting, when a more dilute solution was infused, levobupivacaine was more potent than ropivacaine.

Pain during Injection.—Pain during injection of the local anesthetic is caused by direct tissue trauma produced by the needle, mechanical distention of the tissues by the local anesthetic injected, and a physiologic response to an injectate of low pH. Pain can be lessened if a buffer is added to increase the pH of the injectate (58–61). It is best to use 1 mL sodium bicarbonate (8.4% = 1 mEq/mL) to buffer 10–20 mL lidocaine but only 0.1 mL to buffer 10 mL bupivacaine. Local anesthetic injection pain can also be reduced by the use of empathic language and avoiding the use of negative suggestions such as "this will sting/burn/hurt" (2,62,63). Alternative wording such as a feeling or sensation of "fullness," "tingling," "coolness," or a similar neutral descriptor suffice to prevent a startle response and reduce the experience of discomfort.

Hypersensitivity.—Hypersensitivity reactions have been reported with local anesthetics and are more common with ester formulations such as procaine (Novocaine) or benzocaine (Lanacane) and less common with amide formulations such as lidocaine or bupivacaine. Ester hydrolysis yields paraaminobenzoic acid, which triggers a reaction in sensitive individuals. Hypersensitivity reactions with amide local anesthetics are more commonly due to the vehicle or preservatives, such as methylparaben (a natural constituent of blueberries), which is added to multidose vials as a preservative and antifungal. The most common "allergic" reactions are actually caused by intravascular injection of the local anesthetic with epinephrine, which can produce profound tachycardia, hypertension, and dysphoria. When local anesthetic with epinephrine is injected accidentally intravascularly, the heart rate increases. An increase in heart rate of more than 10 beats per minute corresponds typically to an intravenous dose of 15 μg/70 kg epinephrine. Careful questioning may reveal that a hypersensitivity reaction has not occurred and it is safe to use that local anesthetic in a subsequent procedure. A vasovagal episode associated with the injection can also generate a report of perceived sensitivity to local anesthetic.

Patient-Controlled Analgesia

PCA is a process that permits the patient to administer small boluses of an analgesic at regular intervals. PCA consists of an electronically controlled infusion pump with a timing device. When the patient pushes a button, it triggers the device to administer intravenously a predetermined dose of the drug. The timer prevents the administration of further boluses until a predetermined time interval has lapsed (the lockout time). In addition to the demand doses, loading doses and continuous infusion rates of drug can be programmed into the device. The PCA pump contains a computer that records the machine's "history" (eg, the number of requests and the actual deliveries of drugs). Thus, it can provide an indirect assessment of postprocedure comfort and the patient's self-medicating behavior.

PCA is an effective pain management technique when used appropriately. PCA is thought to enhance comfort while providing patients with a means of control (64,65), and patients have reported that PCA improves their recovery (66). This sense of control and reduced anxiety by not having to rely on another person for administration of pain relief have been shown to have psychological benefits on patients (67). However, careful patient selection and patient and staff education regarding PCA use are paramount in the success of this technique. Patients unable to understand the use of PCA, such as the mentally impaired or demented and those who cannot physically operate the system, are obviously excluded. Age is no exclusion criterion per se: Duggleby and Lander (64) showed that elderly subjects were equally adept as users of PCA as middle-aged subjects.

Patients should be educated with verbal instructions and visual aids such as leaflets and videos in the use of the device. They should also be supervised in use of the PCA pump as previously discussed in the section "Opioid Delivery via PCA" and feel confident in its operation and success. However, despite all educational efforts we have seen on occasion patients who are reluctant to press the delivery button and therefore suffer and others who become "trigger happy" to their own peril.

The ideal opioid for PCA use would be one with a reliable and rapid onset of action, intermediate duration, and minimal narcotic side effects. The most commonly used opioids are mor-

phine, hydromorphone, and fentanyl (42,68) (**Table 1**). As such, there is a risk with all opioids, as discussed previously. For example, serious respiratory depression has been reported at an incidence of 0.5% (69).

Factors associated with respiratory depression in a PCA setting include staff errors during set up, patient factors, and device malfunctions. Patients should therefore be assessed at regular intervals and vital signs, sedation, and analgesia levels monitored and documented. For some patient populations, the use of two PCA devices may be considered: one for analgesia and one for anxiolysis. However, this creates greater complexity.

Cognitive Factors of the Pain Experience and Nonpharmacologic Adjuncts

A patient's pain does not only depend on the severity of the physical stimulus. For example, pain after tumor embolization is common and can be considerable but varies highly among individuals (70–73). The volume of embolized tissue and the degree of infarction did not correlate with the pain intensity in a recent study (73). A similar relative independence of stimulus severity and response in a given interventional radiology setting were seen in another study in which patient pain reports were nearly identical despite differences in the invasiveness of the procedure (74,75).

In the postoperative course, a patient's previous medical experiences can greatly influence levels of distress during a current admission. Memory of pain and expectation have been found to affect subsequent pain intensity (76–79). Patients who have had repeat interventional radiologic procedures tend to experience greater pain and receive more sedatives and narcotics (75). The adverse effect of conditioning and pretreatment expectations is also well described in cancer patients who develop anticipatory nausea on repeat chemotherapy and in burn patients who become increasingly apprehensive of upcoming dressing changes (80,81). Pain perception after procedures therefore cannot be seen in isolation and will be influenced by previous medical encounters and will depend on the experience of the preceding interventional procedure. In a previous study, pain perception increased linearly with procedure time under standard care conditions despite liberal access to intravenous conscious sedation (2). This identifies time as a critical parameter in the pain experience. A study of abdominal interventional procedures confirmed this assumption by reporting greater pain when procedures were technically more difficult (and thus likely more time intensive) (12). In addition, Schutz et al (82) identified length of procedure as a negative predictor of satisfaction with pain management in a group of patients undergoing colonoscopy with intravenous conscious sedation. Laboratory research indicates that exposure to acute pain makes individuals more attentive to external cues, such that they report increasing pain over time even in the absence of a painful stimulus (83). Previous work also showed that this time-dependent increase in pain can be averted when patients receive nonpharmacologic analgesia in the form of self-hypnotic relaxation (2). Such methods can be easily learned by interventional radiology personnel (74) and the appropriate techniques have been summarized in the literature (84).

Additional Options

For patients with underlying chronic illnesses or expected long duration of painful symptoms, early inclusion of hospital-based "pain services," if available, is advisable. These services provide the entire spectrum of conventional and integrative modes of discomfort management including enteral and parenteral medications, transdermal drug delivery (eg, skin patches of fentanyl or clonidine) (85), acupuncture, and psychological support, to name a few. The goal is to have a solid plan for pain management before the patient is discharged.

CONCLUSION

Postoperative pain management is a complex and fascinating area. Both the physical and psychological needs of patients must be met before, during, and after procedures. This can only be achieved by a comprehensive knowledge of the methods available to the interventionalist to provide safe and reliable pain relief, tailored for each individual patient.

References

1. Mott K. The consumer perspective. Med J Aust 2001; 175:75–76.
2. Lang EV, Benotsch EG, Fick LJ, et al. Adjunctive non-pharmacological analgesia for invasive medical procedures: a randomised trial. Lancet 2000; 355: 1486–1490.
3. Lasser EC, Berry CC, Talner LB, et al. Pretreatment with corticosteroids to alleviate reactions to intravenous contrast material. N Engl J Med 1987; 317: 845–849.
4. Miller DL, Wall RT. Fentanyl and diazepam for analgesia and sedation during radiologic special procedures. Radiology 1987; 162:195–198.
5. Acute Pain Management Guideline Panel. Acute pain management: operative or medical procedures and trauma. Clinical practice guideline (AHCPR pub. no. 92–0032). Rockville, MD: Agency for Health Care Policy and Research, Public Health Service, U.S. Department of Health and Human Services, 1992.
6. Skehan SJ, Malone DE, Buckley N, et al. Sedation and analgesia in adult patients: evaluation of a staged-dose system based on body weight for use in abdominal interventional radiology. Radiology 2000; 216:653–659.
7. Cragg AH, Smith TP, Berbaum KS, Nakagawa N. Randomized double-blind trial of midazolam/placebo and midazolam/fentanyl for sedation and analgesia in lower extremity angiography. AJR Am J Roentgenol 1991; 157: 173–176.
8. Miller DL, Wall R. Fentanyl and diazepam for analgesia and sedation during radiologic special procedures. Radiology 1987; 162:195–198.
9. Keeffe EB. Technical monitoring during radiologic interventions—what can be done and what must be done. In: Steinbrich W, Gross-Fengels W, eds. Interventional radiology. Adjunctive medication and monitoring. New York: Springer, 1993;71–78.
10. Mueller PR, Wittenberg KH, Kaufman JA, Lee MJ. Patterns of anesthesia and nursing care for interventional radiology procedures: a national survey of physician practices and preferences. Radiology 1997; 202:339–343.
11. Magni VC, Frost RA, Leung JWC, Cotton PB. A randomized comparison of midazolam and diazepam for sedation in upper gastrointestinal endoscopy. Br J Anaesth 1983; 55:1095–1101.
12. Kennedy PT, Kelly IM, Loan WC, Boyd CS. Conscious sedation and analgesia for routine aortofemoral arteriography.

A prospective evaluation. Radiology 2000; 216:660–664.
13. Hartnell GG, Gates J, Brophy DP, Stuart K, Underhill J, McEniff NJ. Reduction of pain and other complications of hepatic chemoembolization by adjunctive intra-arterial lidocaine. Radiology 1997; 205(S):156.
14. Coldwell DM, Loper KA. Regional anesthesia for hepatic arterial embolization. Radiology 1989; 172:1039–1040.
15. Lang EV, Chen F, Fick LJ, Berbaum KS. Determinants of intravenous conscious sedation for arteriography. J Vasc Interv Radiol 1998; 9:407–412.
16. McDermott VGM, Chapman ME, Gillespie I. Sedation and patient monitoring in vascular and interventional radiology. Br J Radiol 1993; 66: 667–671.
17. Zollikofer CL, Antonucci F, Stuckman G, Mattias P. Drug therapy during venous interventions and vena cava filter procedures. In: Steinbrich W, Gross-Fengels W, eds. Interventional radiology. Adjunctive medication and monitoring. New York: Springer, 1993; 128–132.
18. Kandarpa K. Commonly used medications. In: Kandarpa K, ed. Handbook of cardiovascular and interventional radiologic procedures. Boston: Little, Brown, 1989;194–217.
19. American Society of Anesthesiologists. Practice Guidelines for Sedation and Analgesia by Non-Anesthesiologists. An Updated Report by the American Society of Anesthesiologists Task Force on Sedation and Analgesia by Non-Anesthesiologists. Anesthesiology 2002; 96:1004–1017.
20. Baron RS, Logan H, Kao CF. Some variables affecting dentists' assessment of patients' distress. Health Psychol 1990; 9:143–153.
21. National Institutes of Health. The integrated approach to the management of pain. J Pain Symptom Management 1987; 2:35–44.
22. U.S. Department of Health and Human Services. Acute Pain management Guideline Panel. Acute pain management: operative or medical procedures and trauma. Clinical practice guideline (AHCRPR pub. no. 92-0032). Rockville, MD: Agency of Health Care Policy and Research, Public Health Service, U.S. Department of Health and Human Services, 1992.
23. Herr K, Mobily P. Comparison of selected pain assessment tools for use with the elderly. Appl Nurs Res 1993; 6:39–46.
24. Egbert LD, Battit GE, Turndorf H, Beecher HK. The value of the pre-operative visit by an anaesthetist. JAMA 1963; 185:553–555.
25. Melzack R. The McGill pain questionnaire: Major properties and scoring methods. Pain 1975; 1:277–299.
26. Price DD, McGrath PA, Rafii A, Buckingham B. The validation of visual analogue scales as ratio scale measures for chronic and experimental pain. Pain 1983; 17:45–56.
27. Guilford JP. Rating scales. In: Guilford JP, ed. Psychometric methods. New York: McGraw-Hill, 1954; 263–301.
28. Jensen MP, Karoly P, Riordan EF Jr, Bland F, Burns RS. The subjective experience of acute pain—an assessment of the utility of 10 indices. Clin J Pain 1989; 5:153–159.
29. Jensen MP, Karoly P, Braver S. The measurement of clinical pain intensity: a comparison of six methods. Pain 1986; 27:117–126.
30. Herr K, Mobily P. Comparison of selected pain assessment tools for use with the elderly. Appl Nurs Res 1993; 6:39–46.
31. Gracely RH, Wolskee PJ. Semantic functional measurement of pain: integrating perception and language. Pain 1983; 15:389–398.
32. Gescheider G. Psychophysical scaling. Annu Rev Psychol 1988; 39:169–200.
33. Murphy D, McDonald A, Power A, Unwin A, MacSullivan R. Measurement of pain: a comparison of the visual analogue with a nonvisual analogue scale. J Clin Pain 1988; 3:197–199.
34. Paice JA, Cohen FL. Validity of a verbally administered numeric rating scale to measure cancer pain intensity. Cancer Nurs 1997; 20:88–93.
35. Cornwall A, Donderi DC. The effect of experimentally induced anxiety on the experience of pressure pain. Pain 1988; 35:105–113.
36. Graham L, Conley J. Evaluation of anxiety and fear in adult surgical patients. Nursing Research 1971; 20:113–122.
37. Spielberger CD, Gorsrech RL, Lushene RE. Test manual for the state: trait anxiety inventory. Palo Alto, CA: Consulting Psychologists Press, 1970.
38. Benotsch E, Lutgendorf SK, Watson DW, Fick LJ, Lang EV. Rapid anxiety assessment in medical patients: evidence for the validity of verbal anxiety ratings. Ann Behav Med 2000; 22:199–203.
39. John OP, Donahue EM, Kentle RL. The "Big Five Inventory"—versions 4a and 54. Technical Report. Berkeley, CA: Institute for Personality and Social Psychology, University of California, 1991.
40. Spielberger CD. State-trait anxiety inventory. Palo Alto, CA: Consulting Psychologists Press, 1983.
41. Lang EV, Porter DH. Analgesia and sedation for interventional radiological procedures. In: Society of Cardiovascular and Interventional Radiology. Patient care in interventional radiology. SCVIR syllabus. Fairfax, VA: Society of Cardiovascular and Interventional Radiology 1999;65–103.
42. Austrup ML, Korean G. Analgesic agents for the postoperative period. Opioids. Surg Clin North Am 1999; 79: 253–273.
43. Marks RM, Sachar EJ. Undertreatment of medical inpatients with narcotic analgesics. Ann Intern Med 1973; 78:173–181.
44. Inturrisi CE. Clinical pharmacology of opioid analgesics. Anesthesiol Clin North America 1989; 7:33–49.
45. Scott JC, Stanski DR. Decreased fentanyl and alfentanil dose requirements with age. A simultaneous pharmacokinetic and pharmacodynamic evaluation. J Pharmacol Exp Ther 1987; 240: 159–166.
46. Szeto HH, Inturrisi CE, Houde R, Saal S, Cheigh J, Reidenberg MM. Accumulation of normeperidine, an active metabolite of meperidine, in patients with renal failure or cancer. Ann Intern Med 1977; 86:738–741.
47. Chia YY, Kuo MC, Liu K, et al. Does postoperative pain induce emesis? Clin J Pain 2002; 18:317–323.
48. Bailey PL, Stanley TH. Pharmacology of intravenous narcotic anesthetics. New York: Churchill Livingstone, 1986;764.
49. Sokoll MD, Hoyt JL, Gergis S. Studies in muscle rigidity, nitrous oxide, and narcotic analgesic agents. Anesth Analg 1972; 51:16–20.
50. Longnecker DE, Grazis PA, Eggers GW Jr. Naloxone for antagonism of morphine-induced respiratory depression. Anesth Analg 1973; 52:447–453.
51. Catella-Lawson F, Reilly MP, Kapoor SC, Cucchiara AJ, DeMarco S, Tournier B, et al. Cyclooxygenase inhibitors and the antiplatelet effects of aspirin. N Engl J Med 2001; 345:1809–1817.
52. Lind LJ, Mushlin PS. Sedation, analgesia, and anesthesia for radiologic procedures. Cardiovasc Intervent Radiol 1987; 10:247–253.
53. Smith DW, Peterson MR, DeBerard SC. Local anesthesia. Topical application, local infiltration, and field block. Postgrad Med 1999; 106:57–60, 64–66.
54. Chan SK, Karmakar MK Chui PT. Local anaesthesia outside the operating room. Hong Kong Med J 2002; 8:106–113.
55. Li HL, Galue A, Meadows L, Ragsdale DS. A molecular basis for the different local anesthetic affinities of resting versus open and inactivated states of the sodium channel. Mol Pharmacol 1999; 55:134–141.

56. Covino BG. Clinical pharmacology of local anesthetic agents. In: Cousin MJ, Bridenbaugh PO, eds. Neural blockade in clinical anesthesia and management of pain, 2nd ed. Philadelphia: Lippincott, 1988;111–144.
57. Ramsey DE, Kernagis LY, Soulen MC, Geschwind JFH. Chemoembolizaton of hepatocellular carcinoma. J Vasc Interv Radiol 2002; 13:S211–S221.
58. Christopher R, Buchanan L, Begalia K. Pain reduction in local anesthetic administration through pH buffering. Ann Emerg Med 1988; 17:117–120.
59. Davies, Robert John. Buffering the pain of local anaesthetics: a systematic review. Emerg Med 2003; 15:81–88.
60. Erramouspe J. Buffering local anesthetic solutions with sodium bicarbonate: literature review and commentary. Hosp Pharm 1996; 31:1275–1281.
61. McKay W, Morris R, Mushlin P. Sodium bicarbonate attenuates pain on skin infiltration with lidocaine, with or without epinephrine. Anesth Analg 1987; 66:572–574.
62. Spanos NP, Horton C, Chaves JF. The effects of two cognitive strategies on pain threshold. J Abnorm Psychol 1975; 84:677–681.
63. Lang EV, Joyce JS, Spiegel D, Hamilton D, Lee KK. Self-hypnotic relaxation during interventional radiological procedures: effects on pain perception and intravenous drug use. Int J Clin Exp Hypn 1996; 44:106–119.
64. Duggleby W, Lander J. Patient-controlled analgesia for older adults. Clin Nurs Res 1992; 1:107–113.
65. Schelling G, Weber W, Mendl G, Braun H, Cullman H. Patient controlled analgesia for shock wave lithotripsy: the effect of self-administered alfentanil on pain intensity and drug requirement. J Urol 1996; 155:43–47.
66. Bucknell S, Sikorski K. Putting patient-controlled analgesia to the test. MCN Am J Matern Child Nurs 1989; 14:37–40.
67. DiNobile C. Patient-controlled analgesia: a new trend in pain control. J Post Anesth Nurs 1988; 3:154–161.
68. Lutz LJ, Lamer TJ. Management of postoperative pain: review of current techniques and methods. Mayo Clin Proc 1990; 65:584–596.
69. Etches RC. Respiratory depression associated with patient-controlled analgesia: a review of eight cases. Can J Anaesth 1994; 41:125–132.
70. Ravina JH, Herbreteau D, Ciraru-Vigneron N, et al. Arterial embolization to treat uterine myomata. Lancet 1995; 346:671–672.
71. Worthington-Kirsch RL, Popky GL, Hutchins FL. Uterine arterial embolization for the management of leiomyomas: quality-of-life assessment and clinical outcome. Radiology 1998; 208: 625–629.
72. Goodwin SC, McLucas B, Lee M, et al. Uterine fibroid embolization for treatment of uterine leiomyomata midterm results. J Vasc Interv Radiol 1999; 10: 1159–1165.
73. Roth AR, Spies JA, Walsh SM, Wood BJ, Gomez-Jorge J, Levy EB. Pain after uterine artery embolization for leiomyomata: Can its severity be predicted and does severity predict outcome? J Vasc Interv Radiol 2000; 11: 1047–1052.
74. Lang EV, Laser E. Training interventional radiology personnel in nonpharmacologic analgesia. Radiology 1996; 201:214.
75. Lang EV, Chen F, Fick LJ, Berbaum KS. Determinants of intravenous conscious sedation for arteriography. J Vasc Interv Radiol 1998; 9:407–412.
76. Bachiocco V, Morselli A, Carli G. Self-control expectancy and postsurgical pain: relationships to previous pain, behavior in past pain, familial pain tolerance models, and personality. Pain Symptom Manage 1993; 8:205–214.
77. Bachiocco V, Scesi M, Morselli AM, Carli G. Individual pain history and familial pain tolerance models: relationships to post-surgical pain. Clin J Pain 1993; 9:266–271.
78. Eich E, Reeves JL, Jaeger B, Graff-Radford SB. Memory for pain: relation between past and present intensity. Pain 1985; 23:375–379.
79. Walmsley PNH, Brockopp DY, Brockopp GW. The role of prior pain experience an expectations on postoperative pain. J Pain Symptom Manage 1992; 7:34–37.
80. Montgomery GH, Tomoyasu N, Bovberg DH, et al. Patients' pretreatment expectations of chemotherapy-related nausea are an independent predictor of anticipatory nausea. Ann Behav Med 1998; 20:104–109.
81. Patterson DR, Everett JJ, Burns GL, Marvin JA. Hypnosis for the treatment of burn pain. J Consult Clin Psychol 1992; 5:713–717.
82. Schutz SM, Lee JG, Schmitt CM, Almon M, Baille J. Clues to patient dissatisfaction with conscious sedation for colonoscopy. Am J Gastroenterol 1994; 89:1476–1479.
83. Bayer TL, Coverdale JH, Chiang E, Bangs M. The role of prior pain experience and expectancy in psychologically and physically induced pain. Pain 1998; 74:327–331.
84. Lang EV, Lutgendorf S, Logan H, Benotsch EG, Laser E, Spiegel D. Nonpharmacologic analgesia and anxiolysis for interventional radiological procedures. Semin Interv Radiol 1999; 16:113–123.
85. Ashburn MA, Lind GH, Gillie MH, de Boer AJF, Pace NL, Stanley TH. Oral transmucosal fentanyl citrate (OTFC) for the treatment of postoperative pain. Anesth Analg 1993; 76:377–381.