# 9
# Postoperative Management: Pain and Anesthetic, Fluids and Diet

Tracey D. Arnell and Robert W. Beart, Jr.

Many of the major advancements and changes in the care and survival of the surgical patient have occurred in the postoperative period. This is frequently recognized in regard to critical care, but has been just as remarkable in the non–intensive care unit patient. Significant changes in reimbursement and patient population patterns have either driven, or allowed for, better survival, less morbidity, earlier discharge, and more ambulatory procedures. The major changes have been in the areas of postoperative feeding, activity, pain control, and ulcer and deep venous thrombosis (DVT) prophylaxis. In an attempt to incorporate this knowledge and in conjunction with physician extenders such as nurse practitioners and physician assistants, patient care pathways are being increasingly instituted and validated. The focus of this chapter will be on the non–intensive care unit inpatient.

## Pain Control

The trends toward decreased length of hospital stay and more ambulatory procedures necessitate a good understanding of the mechanisms of pain and its relief. It has been clearly demonstrated that adequate pain control is necessary to maximize cardiac and respiratory function and decrease the risk of complications.[1–3] On a more practical note, the Joint Commission on Accreditation of Healthcare Organizations (JCAHO) now requires specific assessment and documentation of treatment of pain. Despite this, the management of acute pain is still less than ideal. In a survey of 250 patients who had undergone surgery (38% outpatient), 82% of respondents reported experiencing pain. Of these, 39% described severe to extreme pain and 47% moderate pain. One might think this is a result of the trend toward ambulatory procedures, but it was more common in the inpatient setting.[4] Although of course not all inclusive, what follows is a review of the components of pain and options for treatment.

### Physiology

The subjective sensation of pain is made up of many components, both physical and psychological. One definition is that acute pain is "the initiation phase of an extensive, persistent nociceptive and behavioral cascade triggered by tissue injury." The cascade begins with tissue injury that causes nociceptive neurons to begin firing and the local release of inflammatory mediators in the periphery. Once nociceptors become sensitized, the threshold necessary for further activation is lowered and their discharge rate increases. Put simply, less painful stimuli ultimately result in more pain perception. This effect is amplified by the environment of inflammation and its mediators. The nociceptive signals are carried by A delta and C fibers to the spinal cord dorsal horn and the ascending pathways to the central nervous system. Integration of signals occurs at all levels in this pathway. Different analgesic choices will target different parts of this cascade. As a result, analgesic types can be combined to more effectively manage multiple components of pain.[5]

### Techniques

In the inpatient setting of abdominopelvic surgery, the major modalities of postoperative pain control are patient-controlled anesthesia (PCA), opioids, nonsteroidal antiinflammatory drugs (NSAIDs), and epidural anesthesia. Preemptive analgesia is another tactic and includes preincisional infiltration of local anesthetics and administration of NSAIDs and intraoperative epidural anesthesia. Along less traditional lines, massage, acupuncture, and biofeedback therapy are being used in some institutions.

### Opioids

Opioids are the most frequently used medication in perioperative pain management. Their mechanism is via specific opioid receptors as well as nonspecific antiinflammatory actions. They block transmission of nociceptive afferent signals in the spinal dorsal horn and involve efferent messaging by activating inhibitory pathways supraspinally. Additionally, they act locally in the areas of tissue injury to inhibit inflammation.[5]

Opioid side effects include respiratory depression, pruritus, nausea, vomiting, and constipation. Titration of morphine to pain is extremely important in avoiding respiratory depression because the respiratory center receives nociceptive input that counterbalances the depression. When pain is reduced by other means such as adjunct medications and nerve blocks, the amount of morphine must be reduced. Partial agonists have been developed (buprenorphine, tramadol) that may reduce these complications, but presently they are infrequently used and clinical experience is lacking.[6,7]

The most frequently used opioid is morphine, and it is against which all other choices are compared. The second most frequently used opioid is meperidine and it will be discussed specifically. Initially, it was developed as an anticholinergic agent but was found to have analgesic effects. The anticholinergic effect and the potential for less smooth muscle spasm in areas such as the colon, biliary tract, and renal system is one reason it continues to be used in acute pain management. In fact, when used in equianalgesic doses with morphine, meperidine has the same spasmodic effect on smooth muscle.[8] The analgesic effects of meperidine are inferior to those of morphine, and its duration of effectiveness is significantly less than 4 hours. Compounding this ineffectiveness is the use of the intramuscular (IM) route. The absorption is highly variable with variable blood levels resulting in poor pain control. In one series, only 30% of postoperative patients achieved 50% pain relief after injection of 100 mg of meperidine.[9,10] Meperidine causes central nervous system excitation, seizures, increased respiratory depression, has a propensity for addiction, and produces metabolites with little analgesic but significant neurotoxic potential.[8] All of these factors have led the JCAHO to discourage the use of meperidine in its pain guidelines. At best, meperidine, given its short duration of action and significant risk of serious side effects with repeated use, should have an extremely limited role in pain management of postoperative patients.

The route of administration of opioids is more important than the specific opioid used in terms of onset of action. For the intravenous (IV) route and the oral route, there is little difference among various opioids. The IV route is effective within minutes, whereas the oral route varies between 1 hour for standard release and 2–4 hours for sustained relief. The greatest variability occurs with IM administration based on the lipophilic nature of the drug. The more lipophilic, the quicker the onset of pain relief.[11]

For IV delivery, PCA has been used successfully for more than 30 years and is one of the recommended modes of pain control by the American Society of Anesthesiologists in their practice guidelines. Improved pain control, patients' satisfaction, and decreased pulmonary complications have been found in two large reviews comparing PCA with conventional opioid analgesia in postoperative patients.[12,13] Although more expensive, PCA opioid use is a safe and effective mode of delivery. Making the transition from IV pain control to oral pain control should be made with knowledge of the pain requirements based on the most current IV dosages. Table 9-1 lists equianalgesic doses of the IV and oral forms of several frequently used medications.

## Nonopioid

### *Nonsteroidal Antiinflammatory Drugs*

As previously described, the mechanism of pain production and perception is altered by the inflammatory cascade. By decreasing the production of mediators such as prostanoids, the perception of painful stimuli may also decrease. Nonsteroidal medications inhibit cyclooxygenase (COX) in the periphery and spinal cord and this may be the mechanism by which they are effective in diminishing hyperalgesia.[14] Their action is mediated by their effect on COX-2 receptors and result in analgesic and antiinflammatory effects. The side effects are largely a result of inhibition of COX-1 receptors which occur most frequently in the gastrointestinal (GI) tract, renal tissue, and platelets. The effectiveness of NSAIDs in the management of acute pain has been demonstrated in multiple disciplines of surgery including but not limited to orthopedic, oral, abdominal, and spinal surgery. There remain concerns regarding their safety in the surgical patient because of the risk of GI bleeding and, especially, surgical site bleeding. Overall, the use of these agents in postoperative surgical patients has been found to be safe, but there are risks of GI bleeding, renal injury, and surgical bleeding. In the largest review of the use of ketorolac in 1996, 10,272 patients receiving ketorolac were compared with 10,247 receiving opiates. The rate of complications for ketorolac compared with opiates was GI bleeding 2.1% versus 1.9%, serious operative site bleeding 1.5% versus 1.8%. In subanalysis, it was found that the major risk factors that significantly increased these

TABLE 9-1. Equianalgesic dosages of frequently prescribed IV and oral medications

| | Approximate equianalgesic | | Starting dosage, adults >50 kg | |
|---|---|---|---|---|
| | IV/SC/IM | PO | IV/SC/IM | PO |
| Morphine | 10 mg q 3–4 h | 30 mg q 3–4 h | 10 mg q 3–4 h | 30 mg q 3–4 h |
| Codeine | 75 mg q 3–4 h | 130 mg q 3–4 h | 60 mg q 2 h | 60 mg q 3–4 h |
| Hydromorphone | 1.5 mg q 3–4 h | 7.5 mg q 3-4 h | 1.5 mg q 3–4 h | 6 mg q 3–4 h |
| Hydrocodone | | 30 mg q 3–4 h | | 10 mg q 3–4 h |
| Meperidine | 100 mg q 3 h | 300 mg q 2–3 h | 100 mg q 3–4 h | |
| Oxycodone | | 30 mg q 3–4 h | | 10 mg q 3–4 h |

*Source:* Tarascon Pocket Pharmacopeia, 2002 classic shirt pocket edition. Loma Linda, CA: Tarascon Publishing. PO, per os.

risks were patient age greater than 75, daily dosage exceeding 105 mg/day, and courses longer than 5 days.[15] Used within these parameters, NSAIDs are safe and effective as an adjunct or by themselves for the postoperative patient.

### *Antihistamines*

Histamine is known to activate nociceptive fibers and may participate in mediating pain. For this reason, antihistamines have been proposed as adjuncts to pain management. The mechanism of antihistamines in analgesia is unclear but may involve opioid receptors or presynaptic inhibition of histamine receptors. Despite positive findings in animal models, clinical studies have been conflicting. The confounding factors of sedation and poor methodology do not allow for recommendations for their use as single agents. As adjuncts, they may have benefit although the same confounding factors exist in these data. As more selective antihistamines with less sedation become available, these questions may be answered.[16]

## Epidural Anesthesia

Epidural anesthesia functions at the dorsal horn preventing afferent conduction of nociceptive stimuli. For patients undergoing laparotomy and lower abdominal and pelvic surgery, epidural anesthesia may have better pain control, patient satisfaction, and potentially return of bowel function with fewer side effects.[17,18] In a randomized study of colorectal patients undergoing thoracic epidural placement for colorectal resections, resolution of ileus and control of postoperative pain was significantly improved compared with those receiving a PCA.[19] These findings were supported in a series of patients undergoing proctocolectomy.[20] In a series of patients undergoing laparoscopic colon resection randomized to epidural versus PCA, the differences were not significant. The type of medication infused may also have a significant influence on the outcomes postoperatively as discussed in a Cochrane review in which those patients receiving local epidural anesthetics had reduced GI paralysis with comparable pain control.[21] The additional time and cost involved with an epidural has been the primary reason it has not been adopted in a more widespread manner.

## Preemptive Analgesia

The debate over the effectiveness of preemptive analgesia continues. Initial animal studies demonstrated that the doses of analgesia necessary to prevent central hyperexcitability in rats was significantly less than that necessary to reverse it.[22] The concept is that by preventing the initial stimulation of central pain pathways, there will be decreased sensitization to noxious stimuli. When increased sensitization occurs, it is referred to as hyperalgesia and suggests that the same stimuli will produce different degrees of effects based on the state of the target. Hyperalgesia may result from upregulation of afferent pathways and the inflammatory mediators involved in the perception of pain.[23] This led to the evaluation of preemptive analgesia in human studies.

A review of the 80 randomized controlled trials regarding the comparison of preemptive and postoperative pain relief attempted to reach a consensus regarding preemptive analgesia trials in humans. The only end point examined was level of pain. The trials were divided into NSAIDs, IV opioids, epidural analgesia, caudal analgesia, and peripheral local anesthetics. Although there were a few studies that demonstrated improved pain control at various time points postoperatively, this was not consistent and not overall. The findings were that "timing of analgesia did not influence the quality of postoperative pain control, whatever the type of preemptive analgesia."[24] A comparison of preincisional versus postincisional epidural anesthesia with a combination of lidocaine and fentanyl including a control with a sham epidural showed very minimal difference between the former two groups in terms of postoperative morphine consumption. There was a 20% decrease in morphine use compared with the sham epidural as might be expected.[25] It has been suggested that the focus should shift from comparing preoperative and postoperative analgesia, to developing more comprehensive, multimodality paradigms of surgical pain control.

## "Nontraditional" Adjuncts

Acupuncture and acupressure have been used for thousands of years and are now being increasingly used in Western medicine. There are many reports of their use in control of surgical pain, but few of these are randomized. The studies that have been randomized have been mixed in their findings as well as the type of acupuncture or acupressure. The methods include needles, pressure, and electrical stimulation and the number and location of sites is variable. The purported benefits are decreased need for opioids, decreased nausea, and lower plasma cortisol and epinephrine release.[26,27]

Modalities that address the psychological perception of pain, rather than only the physiologic, are being examined. It has been suggested that techniques such as massage may better address the psychological aspect. In the one randomized study of this in patients with acute surgical pain, there was no difference in the consumption of opioids in 202 patients.[28] For similar reasons, relaxation techniques and the use of music have been suggested.

Overall, the data are very limited for these therapies. Most have few risks associated with them (acupressure, psychological methods) and are becoming available in some hospitals. Their role in the management of acute postoperative pain remains to be seen in larger, randomized trials.

# Perioperative Fluid Management

Basic fluid requirements under normal circumstances are approximately 2500 cc/day in a 70-kg adult. This allows for the 1500 cc of urine necessary to excrete waste products including urea, potassium, and sodium. A very simple formula

for calculating basic fluid needs is 1500 cc for the first 20 kg with 20 cc/kg for the remaining weight. As a result of surgical stress, there is an increase in renin, aldosterone, and antidiuretic hormone release and activation of the sympathetic system resulting in sequestration of fluid (third spacing) and increased volume requirements. Additional losses may occur from blood loss, diarrhea, nasogastric tubes, and abdominal drains and these should be accounted for. Assuming a return to homeostasis, this fluid retention begins to resolve with a return to normal of the hormones and sympathetic nervous system. Table 9-2 lists the composition of the frequently administered colloids and should serve as a guide for replacement based on calculated fluid losses.

The management of perioperative fluid has not received much attention in terms of postoperative recovery and complications until recently. It may be that the routine administration of maintenance IV fluids is deleterious. In two randomized controlled trials of colorectal patients, a relatively restricted perioperative fluid administration schedule was used. The groups randomized to the restricted fluid had fewer complications in terms of cardiopulmonary events and tissue healing complications as well as quicker resolution of intestinal ileus.[29,30] The difference in cardiopulmonary complications was also found in a Cochrane review of patients undergoing orthopedic surgery.[31] There is still little information about perioperative fluid management of patients, but changes in standard regimens may be on the horizon.

## Ulcer Prophylaxis

In many institutions, ulcer prophylaxis is a routine part of the postoperative orders. In patients without risk factors, or personal history, this is unnecessary. The incidence of clinically significant GI bleeding in hospitalized patients in this age of ulcer prophylaxis has been well characterized for the critically ill and is less than 0.2%.[32] In this population, mechanical ventilation, coagulopathy, prolonged hypotension, and organ failure have been the most consistently identified risk factors for the development of stress ulcer bleeding.[33,34] Despite this information, inappropriate use of these agents continues as demonstrated in a review of 226 patients admitted to the medical unit. In this population, prescribed ulcer prophylaxis was not indicated in 65% of patients yet a significant number of these patients were discharged on these medications.[35]

The choice of agents for prophylaxis has greatly increased. Table 9-3 lists the most common agents, mechanisms, and effectiveness. In a review of the studies comparing therapies for stress ulcer prophylaxis, Hiramoto et al.[36] concluded that H2 antagonists, sucralfate, and proton pump inhibitors are effective in decreasing the risk of clinically significant bleeding. Proton pump inhibitors, however, are the most potent gastric acid suppressant and, theoretically, may be more effective.

TABLE 9-3. Mechanism of frequently used ulcer prophylaxis medications

| | Mechanism |
|---|---|
| Antacids | • Neutralizes acid |
| Sucralfate | • Mucosal production<br>• Stimulates mucous, HCO3, prostaglandin secretion (inhibits acid secretion)<br>• Coat ulcer base |
| H2 Antagonists | • Blocks stimulation of histamine receptor and production of H+ |
| Proton pump inhibitors | • Blocks H+/K+ ATPase pump (final step of acid production) |

## DVT Prophylaxis

Although the occurrence of a fatal pulmonary embolism (PE) is rare, venous thromboembolism (VTE), both symptomatic and asymptomatic, is relatively common in the surgical patient. In one study, 0.8% of patients admitted after surgical procedures developed symptomatic VTE. Of note, 66% of these occurred in the 3 months after discharge.[37] PE is the most preventable cause of death in hospitalized patients in the United States and was listed as the cause of death in 0.45% of deaths.[38] In light of the many available, low-risk forms of prophylaxis, this should be a part of the care of the postoperative patient.

Of the different therapies available, the costs and potential risks are variable. The potential risk factors are many, and are listed in Table 9-4.[39] Stratification of patients based on their risk for occurrence of VTE/PE should guide the choice of prophylaxis (Table 9-5). Each of the proposed therapies will be discussed in regard to institution, dosage, and effects.

### Elastic Stockings

The literature available on the use of elastic stockings is based on the use of graduated compression stockings. They function by compressing the lower extremity in a gradual manner, with the greatest pressure at the ankle, encouraging venous return. If not fitted properly, they may actually be constrictive and increase the venous pressure below the knees, decreasing

TABLE 9-2. Composition of extracellular fluid and common crystalloid solutions

| Type | Na+ | Cl– | K+ | Ca++ | Mg++ | HCO3– | Lactate |
|---|---|---|---|---|---|---|---|
| Extracellular fluid | 142 | 103 | 4 | 5 | 3 | 27 | |
| NaCl 0.9% (normal saline) | 154 | 154 | | | | | |
| Lactated Ringers | 131 | 111 | 5 | 2 | | | 29 |
| D5/0.45% saline | 77 | 77 | | | | | |
| Plasmalyte 148 + glucose (plasmalyte) | 148 | 97 | 5 | | 1 | 40 | |
| Sodium bicarbonate 8.4% | 1000 | | | | | 1000 | |

TABLE 9-4. Common causes of hypercoagulability

| Risk factor |
|---|
| Age |
| Type of Surgery |
| Orthopedic lower extremity |
| Major surgery |
| Previous VTE |
| Malignancy |
| Pregnancy |
| Estrogen use |
| Obesity |
| Heart failure |
| Thrombophilic disorders |
| Factor V Leiden |
| Essential thrombocytosis |
| Prothrombin G 20210 A mutation |
| Immobilization |
| Hospitalization |

venous return.[40] In a Cochrane review, they did reduce the risk of VTE in moderate-risk patients.[41] As a solo prophylaxis, they should be reserved for the low-risk patient. Otherwise, they should be used in conjunction with other measures.

## Sequential Compression Devices

These devices offer a very effective, low-risk prophylaxis for DVT. The mechanism is both direct and systemic. Locally, they compress the deep venous system decreasing stasis and encouraging venous return. On a systemic level, they increase the fibrinolytic activity by reducing plasminogen activator.[42] There are several types available including a foot pump, calf- and thigh-high devices. There are experimental and clinical data that suggest the devices may be equivalent, although the original studies were based on the thigh-high devices. Killewich et al.[43] studied the hemodynamics of the foot pump system and their conclusions were that there are measurable increases in the venous outflow with these devices. In patients undergoing hip replacement, the foot pump was equally effective as compared with low-molecular-weight heparin (LMWH).[44] No direct comparisons of the different devices are available.

An additional consideration is compliance. For maximal benefit in patients undergoing surgery, they should be placed before the induction of anesthesia and functioning throughout an operation. Postoperatively, their effectiveness can be compromised because of patient, physician, and nursing compliance. Cornwell et al.[45] observed the compliance of trauma patients with the use of sequential compression devices (SCDs). They defined full compliance as the SCDs being on the patient and functioning upon six observations. Based on this, 19% of patients were fully compliant and SCDs were on in 53% of observations.[45] When used properly, SCDs are a safe and effective prophylactic measure in the low- and moderate-risk patient.

## Low-dose Unfractionated Heparin

Unfractionated heparin has been evaluated since the 1970s as a form of prophylaxis and has been shown to be safe in the majority of surgical patients. It consists of molecules that range in size from 3000 to 33,000 Da and binds to antithrombin (ATIII) and accelerates the inhibition of thrombin and other coagulation factors, particularly factor X. In a large randomized trial from 1970, low-dose unfractionated heparin (LDUH) decreased the risk of fatal PEs in the postoperative population from 0.7% to 0.1% in 4000 patients.[46] This was supported in a large metaanalysis of 70 randomized trials. The risk of DVT, PE, and fatal PE was decreased by more than 50%.[47] Although effective, one concern has been the risk of bleeding in the postoperative patient. There has been a small increase in postoperative bleeding in most studies, but the majority of these events are wound hematomas.

A more frequent side effect of heparin is heparin-induced thrombocytopenia (HIT). It is less common with prophylactic than therapeutic heparin, but may occur in 5%–15% of patients. HIT may cause a paradoxical hypercoagulable state with arterial and venous thrombosis. The platelet count should be followed in patients receiving routine heparin and discontinued immediately if diminishing.

It is recommended that subcutaneous (SC) heparin be started within 2 hours of an operation and continued until the patient is fully ambulatory. The dosage is generally 5000 U every (q) 12 hours. This may be increased in those patients in the high-risk category to 7500 U q 12 hours or 5000 U q 8 hours.

## Low-Molecular-Weight Heparin

LMWH consists of heparin molecules in a smaller range and size than LDUH (3500–6000 Da). The mechanism is the same

TABLE 9-5. DVT prophylaxis guidelines

| | Age (y) | Surgery | Risk factors | DVT | PE | Recommendation |
|---|---|---|---|---|---|---|
| Low | <40 | Minor | None | 0.4% | <0.5% | Early ambulation *or* Elastic stockings *or* IPC |
| Moderate | | | | 2%–4% | 1%–2% | Early ambulation *and* Elastic stockings *or* IPC *or* LDUH *or* LMWH |
| A | Any | Minor | Present | 4% | | |
| B | <40 | Major | None | | | |
| C | 40–60 | Minor | None | | | |
| High | | | | 4%–8% | 2%–4% | Early ambulation *and* Elastic stockings *and* IPC *or* LDUH *or* LMWH |
| | >60 | Minor | ±Other | | | |
| | >40 | Major | None | | | |
| | <40 | Major | Present | | | |

as LDUH regarding the acceleration of ATIII inactivation of Xa, but it does not inactivate thrombin. It also does not bind as strongly to plasma moieties so has greater bioavailability, longer half-life, and more predictable plasma levels. Because of this, partial thromboplastin time does not need to be monitored.[48] The incidence of HIT is also lower than LDUH (2.7% versus 0%).[49]

LMWH is at least as effective as LDUH in preventing DVT in postoperative general surgery and colorectal surgery patients without an increase in bleeding complications. A large European trial randomized 1351 patients undergoing abdominal surgery to LDUH or LMWH. The incidence of thromboembolic complications was equal (4.3% versus 4.7%), but patients in the LMWH group experienced fewer bleeding complications, primarily wound hematomas (8.3% versus 11.8%).[50] A metaanalysis of only prospective randomized trials of 5520 patients, including one trial with 1300 colorectal patients, confirmed these results.[51]

With this type of evidence, the question may be why LMWH is not the standard prophylaxis for surgical patients rather than LDUH. Primarily, it is the issue of cost-effectiveness. Based on their findings in a randomized prospective trial of 936 colorectal surgery patients, the authors of the Canadian Multicentre Colorectal Deep Vein Thrombosis Prophylaxis Trial attempted a cost analysis in both Canadian and US dollars for the use of LDUH and LMWH (enoxaparin). Based on their findings of equal effectiveness and a trend toward more bleeding in the LMWH group, they concluded that LDUH was more cost effective. Even with the assumption of greater effectiveness and equal bleeding, LMWH was twice as expensive as LDUH therapy. Their conclusion was: "Although heparin and enoxaparin are equally effective, low-dose heparin is a more economically attractive choice for thromboembolism prophylaxis after colorectal surgery."[52,53]

## Duration

The risk of DVT and PE does not end with the discharge of the patient from the hospital. This is especially true given the decreasing lengths of stay and, therefore, the decreasing time available for prophylaxis while patients are hospitalized. In addition to the previous study cited by White et al. in which 66% of events occurred following discharge. Agnelli et al. found that 40% of DVT/PE events in patients operated on for cancer happened more than 21 days following surgery.[37,59a] It has been postulated that screening patients prior to discharge for DVT using ultrasound or venography and continuing anticoagulation in the population with positive findings would identify a population requiring continued anticoagulation. Pelligrini et al. reported in a prospective series of orthopedic patients that this was not successful in decreasing outpatient events, as 2.2% of patients with negative venograms developed a DVT requiring readmission with three deaths (0.15%).[60a] In a randomized prospective trial of 332 patients deemed high risk having undergone curative pelvic or abdominal cancer resection, those patients who received 21 additional days of enoxaparin had a 4.8% rate of DVT versus 12.0% in the group receiving only in hospital prophylaxis.[61a] Given these findings, cosideration for extended prophylaxis in patients who are at moderate and high risk for thrombotic events must be given.

The American Society of Colon and Rectal Surgeons Practice Parameters for prevention of venous thromboembolism are presented in Appendix A, Chapter 8.

## Anticoagulation

Although there are fairly well-defined recommendations available for the preoperative management of patients on chronic anticoagulation therapy, there is little regarding the postoperative resumption of therapy. The urgency and timing of postoperative anticoagulation can be inferred from the data regarding risk of adverse thromboembolism overall, tempered by an understanding of the risk of bleeding. Clearly, postoperative bleeding risks are influenced by the surgical procedure performed.

Overall, the risk of thrombotic and embolic events may be increased in the surgical patient and those in whom warfarin therapy has been abruptly stopped. In surgical patients not anticoagulated, changes in levels of fibrin D-dimer and other hemostatic markers associated with thrombosis have been found to be increased.[54,55] In those patients taking oral anticoagulation, there is biochemical evidence that there may be a rebound hypercoagulable state after the withdrawal of oral anticoagulation, perhaps increasing the risk even more.[56] Most studies have not borne this out in clinical practice, however.

An estimation of risk will help in guiding the need and timing for beginning anticoagulation postoperatively. A summary of the risk categories based on diagnosis and the general recommendation for anticoagulation is shown in Table 9-6.[57,58]

## Diet

The resumption of a diet is critical to the recovery of the patient undergoing intestinal surgery. Before discharge, it is accepted that patients should tolerate oral analgesia, not require IV hydration, and demonstrate return of intestinal tract function. The order in which these occur varies by practitioner, however. The most traditional approach to these patients is postoperative nasogastric tube decompression, followed by advancement of oral intake based on demonstration of GI function by flatus and bowel movements. On the other extreme is the institution of a regular diet immediately after surgery with changes based on the clinical status. Much literature has accumulated in reference to the viability of these approaches.

Since the 1980s, many groups have evaluated the need for nasogastric tube decompression in the elective abdominal surgery patient.[58–61] The trials failed to show a benefit in

TABLE 9-6. Risk factors for adverse events based on diagnosis and anticoagulation recommendations

| | Atrial fibrillation | Prosthetic valves | Thromboembolism |
|---|---|---|---|
| Adverse event risk | • 1%–8.5% strokes per year | • 8%/y without anticoagulation<br>• 2%/y with anticoagulation | • 40%/y recurrence <1 mo<br>• 10%–15%/y 1–3 mo<br>• 5%/y >3 mo |
| **Risk** | | | |
| High | • Event <30 d<br>• Mitral valve disease | • Event <30 d<br>• Mural thrombus<br>• Placement <90 d<br>• Multiple valves<br>• Caged-ball valve<br>• Mitral position<br>• Previous event<br>• Atrial fibrillation<br>• ↓LV function<br>• Pregnancy | • Recent event <30 d |
| Intermediate | • Previous events<br>• Age >75 y<br>• ↓LV function<br>• Left atrial enlargement<br>• Ischemic disease<br>• Hypertension<br>• Diabetes | • Bi-leaflet or tilting-disc >90 d<br>• Bioprosthetic valves 31–90 d | • Event 1–3 mo<br>• Obesity<br>• Malignancy<br>• Familial prothrombotic state<br>• Preoperative immobility |
| Low | • All others | • None | • Event >3 mo<br>• No event |

reduction of complications including anastomotic, hospital stay, or return to normal GI function. Combined with the patient discomfort and the loss of the lower esophageal sphincter as a protective mechanism, this has prompted most surgeons to abandon their routine use.

The advent of laparoscopic colon resection has facilitated a more aggressive approach to postoperative feeding regimens.[62,63] Several trials demonstrated that the majority of patients tolerated oral intake in the immediate postoperative period, regardless of the presence or absence of traditional markers of return of GI function. This approach has been used in the open colectomy patients as well. In a nonrandomized study of elderly patients (mean age, 77 years) undergoing open colon resection, 90% tolerated early feeding (clear liquids on day 2, regular diet day 3). There were no anastomotic leaks or abscesses in this group.[64] In a recent metaanalysis, 11 studies with 837 patients were identified which compared liberalized diet immediately postoperative to nothing by mouth until evidence of GI function. These included patients undergoing all types of GI surgery, not specifically colon surgery. Overall, there was a reduction in postoperative infections, both directly related to the surgical procedure and other infections such as pneumonia. There were actually fewer anastomotic complications and a shorter length of stay. The only negative finding was a small increase in the number of patients experiencing vomiting. This did not translate to more wound complications.[65]

In addition to having been shown to be safe and well tolerated, there are several theoretical advantages to early feeding. The potential benefits are related to maintenance of intestinal integrity from a biochemical and immunologic perspective. It has been clearly shown that malnutrition in the surgical patient is associated with increased morbidity and mortality.[66–68]

This has increased the interest in achieving adequate postoperative nutrition. In animal models, survival from peritonitis is significantly improved with enteral nutrition, and almost universally fatal with administration of TPN.[69] In one of the largest prospective, randomized clinical trials, Bozzetti et al.[70] randomized 159 malnourished postoperative cancer patients each to enteral versus parenteral nutrition. Seventy-nine of these patients underwent colon surgery, approximately equally divided between the two groups. Overall, there were fewer complications in the enterally fed group (34% versus 49%). Anastomotic leaks and intraabdominal abscesses were not significantly different between the groups. Of note, 21% of enterally fed patients required reduction in caloric intake or switch to parenteral nutrition (8%).[70]

In summation, early feeding after elective abdominal surgery and specifically colon surgery, has been shown to be safe and generally well tolerated. This may improve patients' comfort, and there is a growing body of evidence that early nutrition may improve outcome and reduce complications. These data are most convincing for the malnourished patient, and for the use of enteral nutrition.

## Steroids

It is not infrequent that patients undergoing colorectal surgical procedures are taking exogenous steroids. Usually this is in the inflammatory bowel disease population, but there are many other clinical situations that may be encountered. Important considerations include identifying those patients at risk for adrenal insufficiency, equivalent oral and parenteral dosages, the effect of surgical stress on dosage requirements,

and the timing of tapering to presurgical dosages or cessation of treatment. A brief review of the physiology of steroid homeostasis will help in understanding the recommendations.

Glucocorticoids are essential for protein, carbohydrate, and fat metabolism. Their overall effect is to increase gluconeogenesis by allowing for the production of amino acids by proteolysis and lipolysis. They also stimulate metabolism by their inotropic effects and enhancement of norepinephrine and epinephrine. Glucocorticoid production in the adrenal cortex is stimulated by the anterior pituitary gland via adrenocorticotropic hormone. The hypothalamus stimulates the pituitary by secreting corticotrophin-releasing hormone. Both of these regulatory hormones are inhibited by the end product cortisol by negative feedback. The usual production of cortisol in an unstressed individual is approximately 20 mg/day. During periods of maximal stress, this production may increase up to 150 mg/day.[71–74] The degree of stress is directly related to the magnitude of the procedure, and the anesthetic, with general anesthesia producing the greatest increases.[75]

It has been clearly demonstrated that adrenal atrophy and suppression occurs with exogenous steroid administration. This is a result of the negative feedback effect on adrenocorticotropic hormone by exogenous cortisol and the lack of stimulation of the adrenal cortex. This can take up to 1 year to recover and patients are frequently asymptomatic during this time if not exposed to stress. During this period, the potential for acute adrenal insufficiency exists.

In actuality, the occurrence of adrenal insufficiency in the surgical population is quite rare. The majority of reports are anecdotal. In fact, in one of the only randomized studies recently, those patients randomized to receiving only their usual daily dosage of steroid perioperatively did not experience symptoms of adrenal insufficiency. The numbers were too small (N = 18, N = 12 in the placebo group) to detect a small difference and the dosages of steroids in most patients were quite low, but this supports the rarity of the occurrence.[76] Salem et al.[77] reviewed the body of evidence regarding the perioperative use of steroid coverage. As they describe, the current usage is based on two anecdotal reports in 1952 which led to recommendations that became the standard of care. From their review, they conclude that the vast majority of patients are over-treated and recommendations should be tailored to identifiable populations at greatest risk. These populations are stratified based on the dosage and duration of treatment an individual patient has received. The risk of suppression can be predicted and this is shown in Table 9-7. As to the question of testing of the adrenal axis, it has not been clearly demonstrated that identified suppression leads to clinical insufficiency.[78,79]

The duration of the taper postoperatively is most impacted by the surgical procedure. For most outpatient procedures, the degree of postoperative stress is considered minor and patients can be returned to their preoperative dose immediately. For major surgery, stress dosages should be continued until signs of surgical stress have resolved. This varies from patient to patient as postoperative ileus, cardiac and pulmonary complications and infections pose additional stress. For patients with an uncomplicated postoperative course, this generally begins on the third day. Once the taper begins, it can be carried out rapidly over a period of a few days to the preoperative dosage. Table 9-8 shows the equivalent steroid dosages for the parenteral and enteral steroids.[73,77,80]

TABLE 9-7. Risk of adrenal suppression from exogenous steroids and recommendations for replacement

| | Dose | Duration | Recommendation |
|---|---|---|---|
| High risk* | >20 mg/d | >3 wk | • 100 mg at induction<br>• 100 mg q 8 hrs throughout period of "stress" |
| Intermediate | >5 mg/d<br><20 mg/d | >3 wk | • Prophylaxis *or*<br>• Testing of the axis |
| Low | Any dose<br><5 mg/d | >3 wk<br>Any time | • No prophylaxis |

*Patients with Cushing's syndrome are considered high risk regardless of dosage or duration of steroid administration.

It is important to recognize the signs of adrenal insufficiency because they may occur both in the immediate postoperative period and beyond in the event of a complication. These include bowel obstruction, anastomotic leak, surgical and nonsurgical infections. Symptoms may include hypoglycemia, cardiovascular collapse, fatigue, abdominal pain, nausea, and vomiting. In the postoperative patient presenting with a change in intestinal function, steroid withdrawal should be considered in the at-risk population. Stelzer et al.[81] reviewed their 60 steroid-dependent patients who underwent pouch surgery and developed signs and symptoms of a bowel obstruction. They found that 43 had no objective signs of mechanical obstruction and promptly resolved their symptoms within 4 hours of steroid administration. At the other extreme of intestinal function, Rai and Hemingway[82] reported on a patient presenting with high ileostomy output which was responsive to steroids.

## Clinical Pathways

With an awareness of the benefits of practicing evidence-based medicine, the development of standardized postoperative protocols is a reasonable next step. Potential benefits include decreased length of stay with more efficient utilization of hospital beds and personnel, and potentially fewer mistakes because of standardization of care. Many groups have reported their successful application of such clinical pathways specifically in regard to colorectal surgery. The protocols are variable with respect to pain management and

TABLE 9-8. Equivalent steroid dosages

| Glucocorticoid | Equivalent dose (mg) | Half-life (h) |
|---|---|---|
| Prednisone | 5 | 18–36 |
| Dexamethasone | 0.5 | 36–54 |
| Hydrocortisone | 20 | 8–12 |
| Methylprednisolone | 4 | 18–36 |

the use of cathartics, but individually show a reduction in length of stay with acceptable outcomes. The major end points have been length of stay, readmission rate, complication rate, and patient satisfaction.

The trend toward earlier discharge began first with the laparoscopic colon resection patients. In 1995, Bardram et al.[83] prospectively followed eight patients over the age of 70 undergoing laparoscopic-assisted colectomy (extracorporeal anastomosis). The regimen involved thoracic epidural catheters intra- and postoperatively for pain control with the avoidance of opioids. A protein-enriched diet and ambulation were begun immediately according to a predetermined protocol. Patients were discharged after they had a normal bowel movement. Six of eight patients went home on the second day; two patients waited until day 3 because of "social" reasons. There were no readmissions and all patients were satisfied.[83] These results were reproduced in a group of 16 patients with a median age of 71 years undergoing open sigmoid colectomy. The protocol consisted of an epidural catheter during and after surgery for pain control. Immediately after surgery, a regimen of mobilization, cisapride and magnesium, and liberal diet including protein drinks was begun. The median length of stay was 2 days (range, 2–6 days) and readmissions were 3, not related to intestinal complications (headaches, social secondary to blindness) and there were no complications.[84]

Recently, a randomized trial compared patients undergoing open intestinal resection who followed a "fast-track" versus the "traditional" pathway. The traditional patients had a nasogastric tube placed that was removed when the drainage was low, and had sips of liquids until the occurrence of flatus and/or stool. The fast-tract patients began a regular diet if they tolerated liquids the evening of surgery and were encouraged to ambulate. No epidural catheters were used. The length of stay was significantly shorter (5.4 versus 7.1 days) and there was no difference in readmissions, complications, or patient satisfaction.[85] The same group has demonstrated that this approach is feasible and safe in the patient with significant comorbidity undergoing "complex" operations as well.[86]

Factors that are not necessarily emphasized in these studies, but are clearly present, include the involvement of ancillary staff and patient education. Preoperatively, patients should be educated regarding the expectations of the pathways in terms of their activity and diet. Additionally, an attempt to explain realistic expectations of what patients may expect in terms of pain and discomfort will help in compliance with the protocol. The caregivers, both family and hospital staff, must also be involved and aware of the pathway. Preoperative printed instructions and wall charts may help in achieving this understanding.

## Conclusion

Clearly, the many facets to the postoperative care of the individual patient are complex and as varied as the population treated. The goal of this summary is to provide general recommendations and a framework on which to guide medical decision making. Consistency in postoperative care helps ancillary staff and patients in regard to expectations and understanding their course, but as the clinical situation evolves, changes may be necessary. A basic knowledge of the principles involved and the options available is crucial in delivering the appropriate care.

## References

1. Gust R, Pecher S, Gust A, Hoffmann V, Bohrer H, Martin E. Effect of patient-controlled analgesia on pulmonary complications after coronary artery bypass grafting. Crit Care Med 1999;26:2218–2223.
2. Major CP, Greer MS, Russell WL, Roe SM. Postoperative pulmonary complications and morbidity after abdominal aneurysmectomy: a comparison of postoperative epidural versus parenteral opioid analgesia. Am Surg 1996;62:45–51.
3. Kouraklis G, Glinavou A, Raftopoulos L, Alevisou V, Lagos G, Karatzas G. Epidural analgesia attenuates the systemic stress response to upper abdominal surgery: a randomized trial. Int Surg 2000;85:353–357.
4. Apfelbaum JL, Chen C, Mehta SS, Gan TJ. Postoperative pain experience: results from a national survey suggest postoperative pain continues to be unmanaged. Anesth Analg 2003;97:534–540.
5. Carr DB, Goudas LC. Acute pain. Lancet 1999;353:2051–2058.
6. McQuay H. Opioids in pain management. Lancet 1999;353: 2229–2232.
7. McQuay HJ. Potential problems of using both opioids and local anaesthetic. Br J Anaesth 1988;61:121.
8. Latta KS, Ginsberg B, Barkin RL. Meperidine: a critical review. Am J Ther 2002;9:53–68.
9. Austin KL, Stapleton JV, Mather LE. Multiple intramuscular injections: a major source of variability in analgesic response to meperidine. Pain 1980;8:47–62.
10. Erstad BL, Meeks ML, Chow H, et al. Site-specific pharmacokinetics and pharmacodynamics of intramuscular meperidine in elderly postoperative patients. Ann Pharmacother 1997;331:23–28.
11. Collins SL, Faura CC, Moore RA, Mcquay HJ. Peak plasma concentrations after oral morphine: a systematic review. J Pain Symptom Manage 1998;16:388–402.
12. Walder B, Schafer M, Henzi I, Tramer MR. Efficacy and safety of patient-controlled opioid analgesia for acute postoperative pain. A quantitative systematic review. Acta Anaesthesiol Scand 2001;45:795–804.
13. Ballantyne JC, Carr DB, Chalmers TC, Dear KB, Angelillo IF, Mosteller F. Postoperative patient-controlled analgesia: meta-analysis of initial randomized control trials. J Clin Anesth 1993;5:182–193.
14. McCormack K. Non-steroidal anti-inflammatory drugs and spinal nociceptive processing. Pain 1994;59:9–44.
15. Strom BL, Berlin J, Kinman JL, et al. Parenteral ketorolac and risk of gastrointestinal and operative site bleeding. A postmarketing surveillance study. JAMA 1996;275:376–382.
16. Raffa RB. Antihistamines as analgesics. J Clin Pharm Ther 2001; 26:81–85.
17. Mann C, Pouzeratte Y, Boccara G, et al. Comparison of intravenous or epidural patient-controlled analgesia in the elderly after major abdominal surgery. Anesthesiology 2000;92:433–441.
18. Eriksson-Mjoberg M, Svensson JO, Almkvist O, Olund A, Gustafsson LL. Extradural morphine gives better pain relief than

patient-controlled i.v. morphine after hysterectomy. Br J Anaesth 1997;78:10–16.
19. Carli F, Trudel JL, Belliveau P. The effect of intraoperative thoracic epidural anesthesia and postoperative analgesia on bowel function after colorectal surgery: a prospective randomized trial. Dis Colon Rectum 2001;44:1083–1089.
20. Scott AM, Starling JR, Ruscher AE, DeLessio ST, Harms BA. Thoracic versus lumbar epidural anesthesia's effect of pain control and ileus resolution after restorative proctocolectomy. Surgery 1996;120:688–695.
21. Jorgensen H, Wetterslev J, Moiniche S, Dahl JB. Epidural local anaesthetics versus opioid based analgesic regimens on postoperative gastrointestinal paralysis, PONV, and pain after abdominal surgery. Cochrane Database Syst Rev 2000;4:CD001893.
22. Woolf CJ. Evidence for a central component of postinjury pain hypersensitivity. Nature 1983;308:386–388.
23. Coderre TJ, Katz J, Vaccarino AL, Melzack R. Contribution of central neuroplasticity to pathological pain. Review of clinical and experimental evidence. Pain 1993;52:259–285.
24. Moiniche S, Kehlet H, Dahl JB. A qualitative and quantitative systematic review of preemptive analgesia for postoperative pain relief. Anesthesiology 2002;96:725–741.
25. Katz J, Cohen L, Schmid R, Chan VWS, Wowk A. Postoperative morphine use and hyperalgesia are reduced by preoperative but not intraoperative epidural analgesia. Implications for preemptive analgesia and the prevention of central sensitization. Anesthesiology 2003;98:1449–1460.
26. Sakurai M, Suleman MI, Morioka N, Akca O, Sessler DI. Minute sphere acupressure does not reduce postoperative pain or morphine consumption. Anesth Analg 2003;96:493–497.
27. Kotani N, Hashimoto H, Sata Y, et al. Preoperative intradermal acupuncture reduces postoperative pain, nausea and vomiting, analgesic requirement and sympathoadrenal responses. Anesthesiology 2001;95:349–356.
28. Piotrowski MM, Paterson C, Mitchinson A, Kim HM, Kirsh M, Hinshaw DB. Massage as adjuvant therapy in the management of acute postoperative pain: a preliminary study in men. J Am Coll Surg 2003;197:1037–1046.
29. Lobo DN, Bostock KA, Neal KR, et al. Effect of salt and water balance on recovery of gastrointestinal function after elective colonic resection: a randomised controlled trial. Lancet 2002; 359:1812–1818.
30. Brandstrup B, Tonnesen H, Beier-Holgersen R, et al. Effects of intravenous fluid restriction on postoperative complications: comparison of two perioperative fluid regimens. Ann Surg 2003;238:641–648.
31. Price J, Sear J, Venn R. Perioperative fluid volume optimization following proximal femoral fracture. Cochrane Database Syst Rev 2002;(1):CD003004.
32. Pimental M, Roberts DE, Bernstein CN, Hoppensack M, Duerksen DR. Clinically significant gastrointestinal bleeding in critically ill patients. Am J Gastroenterol 2000;95:2801–2806.
33. Terdiman JP, Ostroff JW. Gastrointestinal bleeding in the hospitalized patient: a case-control study to assess risk factors, causes, and outcome. Am J Med 1998;104:349–354.
34. Cook DJ, Fuller HD, Guyatt GH, et al. Risk factors for gastrointestinal bleeding in critically ill patients. N Engl J Med 1994; 330:377–381.
35. Nardino RJ, Vender RJ, Herbert PN. Overuse of acid-suppressive therapy in hospitalized patients. Am J Gastroenterol 2000;95: 3118–3122.
36. Hiramoto JS, Terdiman JP, Norton JA. Evidence based analysis: postoperative gastric bleeding: etiology and prevention. Surg Oncol 2003;12:9–19.
37. White RH, Romano PS, Zhou H. A population-based comparison of the 3 month incidence of thromboembolism after major elective/urgent surgery. Thromb Haemost 2001;86:2255.
38. Horlander KT, Mannino DM, Leeper KV. Pulmonary embolism mortality in the United States, 1979–1998: an analysis using multiple-cause mortality data. Arch Intern Med 2003;163: 1711–1717.
39. Kaboli P, Henderson MC, White RH. DVT prophylaxis and anticoagulation in the surgical patient. Med Clin North Am 2004;87: 77–110.
40. Best AJ, Williams S, Crozier A, Bhatt R, Gregg PJ, Hui AC. Graded compression stockings in elective orthopedic surgery. An assessment of the in vivo performance of commercially available stockings in patients having hip and knee arthroplasty. J Bone Joint Surg Br 200;82:116–118.
41. Amarigiri SV, Lees TA. Elastic compression stockings for prevention of deep vein thrombosis. Cochrane Database Syst Rev 2000:CD001484.
42. Camerota AJ, Chouhan V, Harada RN, et al. The fibrinolytic effects of intermittent pneumatic compression: mechanism of enhanced fibrinolysis. Ann Surg 1997;226:306–313.
43. Killewich LA, Sandager GP, Nguyen AH, Lilly MP, Flinn WR. Venous hemodynamics during impulse foot pumping. J Vasc Surg 1995;22:598–605.
44. Warwick D, Harrison J, Glew D, Mitchelmore A, Peters TJ, Donovan J. Comparison of the use of a foot-pump with the use of low molecular weight heparin for the prevention of deep-vein thrombosis after total hip replacement. J Bone Joint Surg Am 1998;80:1158–1166.
45. Cornwell EE, Chang D, VelmahosG, et al. Compliance with sequential compression device prophylaxis in at-risk trauma patients: a prospective analysis. Am Surg 2002;68:470–473.
46. Prevention of fatal postoperative pulmonary embolism by low doses of heparin. An international multicentre trial. Lancet 1975;2:45–51.
47. Collins R, Scrimgeour A, Yusuf S, Peto R. Reduction in fatal pulmonary embolism and venous thrombosis by perioperative administration of subcutaneous heparin. Overview of results of randomized trials in general, orthopedic, and urologic surgery. N Engl J Med 1988;318:1162–1173.
48. Simmons ED. In: Bongard FS, Sue DY, eds. Antithrombotic Therapy. In Current Critical Care Diagnosis and Treatment. 2nd ed. New York: McGraw Hill; 2002;905–924.
49. Warkentin TE, Levine MN, Hirsh J, et al. Heparin-induced thrombocytopenia in patients treated with low-molecular weight heparin or unfractionated heparin. N Engl J Med 1995;332:1330–1335.
50. Kakkar VV, Boeckl O, Boneu B, et al. Efficacy and safety of a low-molecular-weight heparin and standard unfractionated heparin for prophylaxis of postoperative venous thromboembolism. European multicenter trial. World J Surg 1997;2:2–8.
51. Mismetti P, Laporte S, Darmon JY, Buchmuller A, Decousus H. Meta-analysis of low molecular weight heparin in the prevention of venous thromboembolism in general surgery. Br J Surg 2001; 88:913–930.
52. Etchells E, McCleod RS, Geerts W, Barton P, Detsky AS. Economic analysis of low-dose heparin vs the low-molecular weight heparin enoxaparin for prevention of venous thromboembolism after colorectal surgery. Arch Intern Med 1999;159:1221–1228.
53. McLeod RS, Geerts WH, Sniderman KW, et al. Subcutaneous heparin versus low-molecular-weight heparin as thromboprophylaxis

in patients undergoing colorectal surgery: results of the Canadian colorectal DVT prophylaxis trial: a randomized, double-blind trial. Ann Surg 2001;233:438–444.
54. Palareti G, Legnani C, Guazzaloca G, et al. Activation of blood coagulation after abrupt or stepwise withdrawal of oral anticoagulants: a prospective study. Thromb Haemost 1994;72:222.
55. Genewein U, Haeberli A, Straub PW, Beer JH. Rebound after cessation of oral anticoagulant therapy: the biochemical evidence. Br J Haematol 1996;92:479.
56. Watts SA, Gibbs NM. Outpatient management of the chronically anticoagulated patient for elective surgery. Anaesth Intensive Care 2003;31:145–154.
57. Kearon C, Hirsh J. Current concepts: management of anticoagulation before and after elective surgery. N Engl J Med 1997;336: 1506–1511.
58. Bauer JJ, Gelernt IM, Salky BA, Kreel I. Is routine postoperative nasogastric decompression really necessary? Ann Surg 1985;201: 233–236.
59. Argov S, Goldstein I, Barzilai A. Is routine use of the nasogastric tube justified in upper abdominal surgery? Am J Surg 1980; 130:849–850.
59a. Agnelli G, Boli G, Capussotti L. et al. A clinical outcome based prospective study on venous thromboembolism after cancer surgery. Ann Surg 2006;243:89–95.
60. Meltvedt R, Knecht B, Gibbons G, et al. Is nasogastric suction necessary after elective colon resection? Am J Surg 1985;149: 620–622.
60a. Pelligrini VD, Donaldson CT, Farber DC, Lehman EB, Evarts CM. Prevention of readmission for venous thromboembolic disease after total hip arthroplasty. Clin Ortho and Related Research 2005;441:56–62.
61. Wolff BG, Pemberton JH, Van Heerden JA, et al. Elective colon and rectal surgery without nasogastric decompression. Ann Surg 1987;154:640–642.
61a. Bergqvist D, Agnelli G, Cohen AT, et al. ENOXACAN II Investigators. Duration of prophylaxis against venous thromboembolism with enoxaparin after surgery for cancer. N Engl J Med 2002;346:971–980.
62. Wexner SD, Cohen SM, Johansen OB, et al. Laparoscopic colorectal surgery: a prospective assessment and current perspective. Br J Surg 1993;80:1602–1605.
63. Jacobs M, Verdeja JC, Goldstein HS. Minimally invasive colon resection (laparoscopic colectomy). Surg Laparosc Endosc 1992;1:144–150.
64. DiFronzo LA, Yamin N, Patel K, O'Connell TX. Benefits of early feeding and early hospital discharge in elderly patients undergoing open colon resection. J Am Coll Surg 2003;197:747–752.
65. Lewis SJ, Egger M, Sylvester PA, Thomas S. Early enteral feeding versus "nil by mouth" after gastrointestinal surgery: systemic review and meta-analysis of controlled trials. BMJ 2001;323: 773–776.
66. Apelgren KN, Rombeau JL, Twomey PL, Miller RA. Comparison of nutritional indices and outcomes in critically ill patients. Crit Care Med 1982;10:305–307.
67. Klonoff-Cohen H, Barrett-Connor EL, Edelstein SL. Albumin levels as a predictor of mortality in the healthy elderly. J Clin Epidemiol 1992;45:207–212.
68. Dervenis C, Augerinos C, Lytres D, Dells S. Benefits and limitations of enterol nutrition in the early postoperative period. Arch Surg 2003;387:441–449.
69. Petersen SR, Kudsk KA, Carpenter G, Sheldon G. Malnutrition and immunocompetence: increased mortality following an infectious challenge during hyperalimentation. J Trauma 1981;21:528–533.
70. Bozzetti F, Braga M, Gianotti L, Gavazzi, Mariani L. Postoperative enteral versus parenteral nutrition in malnourished patients with gastrointestinal cancer: a randomized mulitcentre trial. Lancet 2001;358:1487–1492.
71. Chernow B, Alexander HR, Smallridge RC, et al. Hormonal responses to graded surgical stress. Arch Intern Med 1987;145: 1273–1278.
72. Kehlet H. A rational approach to dosage and preparation of parenteral glucocorticoid substitution therapy during surgical procedure. Acta Anaesthesiol Scand 1975;19:260–264.
73. Orth DN, KovacsWJ. The adrenal cortex. In: Wilson JD, Foster DW, Kronenberg HM, et al., eds. Williams Textbook of Endocrinology. 9th ed. Philadelphia: WB Saunders; 1988:517–664.
74. White PC, Pescovitz OH, Cutler GB. Synthesis and metabolism of corticosteroids. In: Becker KL, ed. Principles and Practice of Endocrinology and Metabolism. 2nd ed. Philadelphia: Lippincott; 1995:647–662.
75. Udelsman R, Norton JA, Jelenich SE, et al. Responses of the hypothalamic-pituitary adrenal and rennin-angiotensin axis and the sympathetic system during controlled surgical and anesthetic stress. J Clin Endocrinol Metab 1987;64:986–994.
76. Glowniak JV, Loriaux DL. A double-blind study of perioperative steroid requirements in secondary adrenal insufficiency. Surgery 1997;121:123–129.
77. Salem M, Tainsh RE, Brombert J, Loriaux DL, Chernow B. Perioperative glucocorticoid coverage. A reassessment 42 years after emergence of a problem. Ann Surg 1994;219:416–425.
78. Bromberg JS, Baliga P, Cofer JB, et al. Stress steroids are not required for patients receiving a renal allograft and undergoing operation. J Am Coll Surg 1995;180:532–536.
79. Boots JMM, van den Ham ECH, Christiaans MHL, van Hooff JP. Risk of adrenal insufficiency with steroid maintenance therapy in renal transplantation. Transplant Proc 2002;34:1696–1697.
80. Rolih C, Ober K. The endocrine response to critical illness. Med Clin North Am 1995;79:211–224.
81. Stelzer M, Phillips JD, Fonkalsrud EW. Acute ileus from steroid withdrawal simulating intestinal obstruction after surgery for ulcerative colitis. Arch Surg 1990;125:914–917.
82. Rai S, Hemingway D. Acute adrenal insufficiency presenting as high output ileostomy. Ann R Coll Surg 2003;85:105–106.
83. Bardram L, Funch-Jensen P, Jensen P, Crawford ME, Kehlet H. Recovery after laparoscopic colonic surgery with epidural analgesia, and early oral nutrition and mobilization. Lancet 1995;345:763–764.
84. Kehlet H, Mogensen T. Hospital stay of 2 days after open sigmoidectomy with a multimodal rehabilitation programme. Br J Surg 1999;86:227–230.
85. Delaney CP, Fazio VW, Senagore AJ, Robinson B, Halverson AL, Remzi FH. "Fast track" post-operative management protocol for patients with high co-morbidity undergoing complex abdominal and pelvic colorectal surgery. Br J Surg 2001;88:1533–1538.
86. Delaney CP, Zutshi M, Senagore AJ, Remzi FH, Hammel J, Fazio VW. Prospective, randomized, controlled trial between a pathway of controlled rehabilitation with early ambulation and diet and traditional postoperative care after laparotomy and intestinal resection. Dis Colon Rectum 2003;46:851–859.

Request PDF | Postoperative Management: Pain and Anesthetic, Fluids and Diet | Many of the major advancements and changes in the care and survival of the surgical patient have occurred in the postoperative period. This is | Find, read and cite all the research you need on ResearchGate.Â  The major changes have been in the areas of postoperative feeding, activity, pain control, and ulcer and deep venous thrombosis (DVT) prophylaxis. In an attempt to incorporate this knowledge and in conjunction with physician extenders such as nurse practitioners and physician assistants, patient care pathways are being increasingly instituted and validated. @inproceedings{Beck2009PostoperativeMP, title={Postoperative Management: Pain and Anesthetic, Fluids and Diet}, author={David E. Beck and Patricia L. Roberts and John L. Rombeau and Michael J Stamos and S D Wexner}, year={2009} }. David E. Beck, Patricia L. Roberts, +2 authors S D Wexner. Adequate pain control is necessary to maximize cardiac and respiratory function and decrease the risk of complications. The Joint Commission on Accreditation of Healthcare Organizations (JCAHO) now requires specific assessment and documentation of treatment of pain. View via Publisher. Save to Library. Create