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Postoperative Care

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INTRODUCTION

The care of the surgical patient requires an accurate understanding of the pathophysiological changes that occur perioperatively. During this period, the body attempts to maintain systemic homeostasis despite multiple iatrogenically induced alterations. Given the proper environment and appropriate interventions, the body should eventually correct for these derangements. The surgeon's goal during the postoperative period is two fold. First, he or she must provide appropriate support that allows for maintenance of homeostasis and prevention of potential complications. Second, he or she must recognize unfavorable trends in the course of recovery and respond expeditiously to prevent further compromise. With diligent care, the surgical patient should eventually return to his or her preoperative level of function.

SUPPORTIVE POSTOPERATIVE CARE

Vital Signs

Much information can be obtained by close monitoring of the vital signs, including blood pressure, pulse, and respiratory rate. More importantly, the trend and changes of these measurements more accurately reflect the patient's ongoing condition. In the immediate

postoperative period, frequent measurements are usually obtained by the recovery room staff. Selected parameters are more important during various stages of the recovery period. Initially, respiratory rate and blood pressure are of greater significance during recovery from anesthesia, as it reflects hemodynamic stability and level of anesthetic reversal. Later, after adequate analgesia and pulmonary function has been obtained, pulse rate correlates better with intravascular volume status. After discharge from the recovery room, vitals should be monitored every four hours until stable and then every eight hours depending on the patient's progress.

Postoperative Activity

Early ambulation is extremely important after surgery. In addition to improving diaphragmatic excursion with its subsequent decrease in pulmonary atelectasis, it also prevents the development of deep venous thrombosis. We normally require that our patients ambulate within 12 hours of surgery. Thereafter, they are assisted to walk three to four times a day.

To further decrease pulmonary ventilating detects and improve mobilization of mucous secretions, patients are encouraged to cough and breath deeply. To assist with this respiratory exercise, an incentive spirometer is used at least every hour while awake.

Nutrition

The energy requirements due to the stress of surgery are often increased with a concurrent rise in the basal metabolic rate. Where possible, advancement to a regular diet should be obtained as soon as the patient's condition permits. Nutritional considerations are usually unnecessary after uncomplicated surgery. If allowed to eat within one week, these patients will generally do well. However, for the chronically ill individual undergoing extensive surgery, early postoperative nutritional supplementation is essential for timely wound healing and immunological defense.¹

Normal recovery from surgery may include transient loss of appetite and mild nausea. This usually is secondary to the anesthetic agents and other perioperative medications used. Symptoms can easily be managed by antiemetics such as Compazine 10 mg IM every four hours as needed. However, after a major transperitoneal procedure, adynamic ileus may be responsible for continued nausea, abdominal distention, and absence of flatus. The return of small bowel function occurs within six hours of surgery and is highlighted by the return of bowel sounds. Gastric emptying and pyloric sphincter function requires two to three days and coincides with improved nausea and decreased nasogastric tube output. Finally, the return of colonic peristalsis that occurs in three to five days is signaled by the passage of flatus.

Patients with gastrointestinal dysmotility should be initially kept NPO for 48 hours or until symptoms of nausea resolve. For those who are: severely symptomatic with continued vomiting and distention, a nasogastric tube should be inserted and placed on suction. As gastric motility returns, noted by decreased NG output and resolution of nausea, the tube can be removed and

the patient advanced to a clear liquid diet. Prior to oral advancement, the patient should receive at least 100 grams of glucose in the form of 2 liters of 5% dextrose per day; this will minimize protein catabolism during this starvation period.

Progression to a regular diet should be withheld until full return of gastrointestinal motility is confirmed. This is noted with the production of flatus and findings of a nondistended abdomen. If prolonged dysfunction is noted or anticipated, early nutritional support is recommended. Two major forms of delivery are available: enteral feeding either via a nasal feeding tube or surgically placed feeding jejunostomy is the preferred route of administration, provided that the gastrointestinal tract is functional. Hyperalimentation through a central venous catheter is utilized if the enteral route cannot be used to maintain adequate nitrogen balance.²

Fluids and Electrolyte Management

Postoperative fluid management is dependent on:

Current deficits

Maintenance requirements

Abnormal losses

The status of the patient's current conditions should first be determined. Utilizing information from the patient's history such as preoperative vomiting, bowel distention, oral intake, intraoperative hemorrhage, extravascular fluid accumulation (third space), and previous fluid replacement, clues to the patient's fluid deficits can be determined. Physical examination, vital signs, recent weight change, and record of fluid balance can also help to determine the status of the intravascular volume and total body water. If uncertainty exists regarding the patient's actual fluid status, invasive monitoring can be used to measure central venous pressure or left ventricular filling pressure, (preload), using the Swan-Ganz catheter.

Daily maintenance requirement for water is calculated to replace insensible losses and to provide adequate volume to excrete the solute load derived from daily metabolism. In the healthy individual with normal renal function, the daily requirement is approximately one liter per day. However, in the surgical patient with a higher insensible loss and less than optimal renal concentrating ability, a daily maintenance requirement between 35–40 ml/kg/day is necessary.

Abnormal losses can be estimated by the addition of NG tube output, fistula tract drainage, and sump suction discharge. Quantitation of the drainage tubes is easily performed. However, internal third space fluid loss is extremely variable and difficult to predict. Adequacy of fluid replacement is strictly based on clinical response to estimated volume replacement.

Electrolyte replacement after uncomplicated surgery rarely requires more than sodium chloride and potassium supplementation, both at 1 mEq/ Kg/day. This requirement is easily met by giving 0.25% normal saline with 20 mEq KCl/liter at the volume predicted above for daily fluid needs. In patients who have continued isotonic losses from the NG tube, fistulas, or peritoneal surfaces,

isotonic fluid is required to prevent electrolyte aberrations. In individuals with complicated large volume drainage, the electrolytes of the drainage should be analyzed and replacement adjusted accordingly.³

In consideration of the volume, electrolytes, and glucose requirements discussed above, we generally begin with a maintenance of D5 1/4 NS with 20 mEq KCl/liter at 100 ml/hr for the 60 kg patient. Additional fluid requirements for low blood pressure, inadequate urine output, or decreased CVP/left ventricular filling volume is supplemented with normal saline.

Pain Control

Liberal use of postoperative analgesics is essential for recovery. Adequate pain control allows for early ambulation, improved pulmonary toilet, and decreased overall stress. The most effective regimen for pain control requires small frequent dosing, preferably via the intravenous route. An extension of this rationale has led to the recent uses of patient controlled analgesia (PCA). This system consists of a preprogrammed infusion pump that is controlled by a hand-held button. An intravenous dose of premeasured narcotic is delivered when the button is depressed, providing a timely bolus of analgesia. Patients on a PCA have a decreased overall narcotic use and have improved pain control.⁴

With the normal progression of recovery, patients can usually be switched to an oral narcotic within two to four days. For those who are difficult to wean from IV narcotics intramuscular injection can be used, thus prolonging the half-life of the effective dose.

An alternative form of pain control is gaining in popularity. This involves placement of an epidural catheter by an experienced anesthesiologist. The catheter is subsequently infused with a preservative-free narcotic agent that bathes the epidural space and provides quality pain control.⁵

Wound Care

The management of the incisional wound is based on the normal biology of the healing process. Conceptually, healing can be divided into three phases. Initially, after tissue injury, inflammatory changes occur as local chemical mediators are released causing an influx of leukocytes. Epidermal migration results in epithelialization within 24 to 48 hours, thus separating the wound from the external environment. Endothelial budding is also noted with the production of granulation tissue within the subdermal surface. By the fifth postoperative day, the effect of epithelialization and neovascularization allows the skin edges to be held in approximation, provided that the edges are spared from undue tension.

The second phase of healing is marked by fibroblast proliferation. Rapid collagen deposition is noted from the fifth postoperative day and reaches its maximal volume by the seventeenth day. Ultrastructurally, the collagen fibers seen during this phase are disoriented, with suboptimal

interfibril cross-linking. The tensile strength of the wound steadily increases from the fifth day onward due to the production of this collagen lattice.

The third phase involves the maturational process of the collagen network that is already in place. Complex cross-linking occurs with replacement of the previous fibers with a thicker, more organized, polarized collagen. Tensile strength continues to increase with this remodeling for up to two years without any additional increase in collagen content.⁶

Wound care is based on understanding of the biological principles of healing. With the attainment of epithelial continuity, the operative dressing can be removed after 24–48 hours. However, if wound drainage is noted upon inspection, a sterile dressing must be replaced until the drainage ceases and epithelialization is assured.

The timing of suture removal is based on two opposing guidelines. For purposes of providing added strength to skin approximation, suture should be left in place until adequate tensile strength is obtained by collagen deposition and maturation. On the other hand, allowing sutures to stay in place for a lengthy period will increase scar formation at the sites of skin penetration. In healthy individuals with an abdominal incision, sutures or staples can generally be removed on the fifth postoperative day followed by placement of adhesive strips. This allows for maximal cosmetic benefit while providing adequate support for wound stability. The timing of the removal can be adjusted in either direction depending on the importance of each opposing factor. Thus, individuals who are nutritionally compromised, in whom cosmesis is of less importance, sutures may be allowed to stay for a longer period of time.

POSTOPERATIVE COMPLICATIONS

Shock

Shock is a state of inadequate tissue perfusion and, as such, is directly proportional to blood pressure. Causes of postoperative hypotension include:

- Hypovolemia from decreased intravascular volume
- Decreased peripheral resistance from sepsis or neurogenic collapse
- Cardiogenic failure

Clinical presentation can be very helpful in differentiating these types of shock and may direct subsequent management. Because the central problem is lack of perfusion to vital organs, oliguria and decreased mental status are objective signs of inadequate tissue perfusion. Due to its life-threatening potential, immediate therapy should be initiated prior to diagnostic evaluation. Intravenous fluid bolus should be given to those suspected of vascular collapse and the patient placed in the Trendelenberg position (head down and feet up).

When the patient stabilizes and if the cause of the hypotension is still in doubt, invasive monitoring may be required. A central line placed in the superior vena cava via either the internal jugular or subclavian vein can be used to measure the intravascular volume. Using the midaxillary line as a reference point, the central venous pressure (CVP) normally ranges from 8 cm to 12 cm of water. Low volume is reflected by low CVP. In patients with possible right heart failure, other cardiac dysfunction,⁷ sepsis,⁸ respiratory failure, or severe preeclampsia,⁹ CVP is inadequate to evaluate actual left heart filling pressure. Utilization of a Swan-Ganz catheter allows for a more accurate measurement of left ventricular end-diastolic pressure (left atrial pressure).¹⁰ Pulmonary capillary wedge pressure (PCWP), normally between 10 cm and 18 cm of Hg, is a measurement of left heart filling volume, thus correlating with intravascular fluid status.¹¹

Low CVP or PCWP indicates decreased effective volume. Immediate replacement with normal saline should be undertaken until normal parameters are reached or the blood pressure stabilizes. Serial blood counts are obtained, and if continued drop is noted and the patient continues to be symptomatic, blood is transfused. With massive blood transfusion coagulation parameters such as the prothrombin time, partial thromboplastin time, platelets, and calcium are monitored. Derangements will require correction with fresh frozen plasma, platelets, or calcium gluconate, respectively. Persistent blood loss may require surgical reexploration or possibly intraarterial embolization of the responsible artery.

Hypotension with normal intravascular volume requires measurement of cardiac output (CO) and systemic vascular resistance (SVR) using the Swan-Ganz catheter by the thermal dilution technique. In septic shock, CO is high while SVR is low; in addition, associated fever, rigors, and leukocytosis are present. Treatment includes normalization of PCWP with fluid, followed by the initiation of vasopressors. Dopamine may be started at 2 µg/kg/min and titrated up to maintain a mean arterial pressure of 70–80 mm of Hg.¹² The ultimate management of septic shock is oriented toward elimination of the source of sepsis while maintaining appropriate antibiotic coverage. Broad-spectrum antibiotics are initially used and subsequently narrowed according to culture results. Drainage of infected focus are discussed under “Postoperative Fever” below. Cardiogenic shock is suggestive when PCWP is high and CO is low. SVR is usually either high or normal. Standardization of myocardial function is obtained by dividing CO by body surface area, giving the cardiac index (CI). Administration of dobutamine starting at 3 µg/kg/min for inotropic support should be initiated to maintain a CI above 2.2 liters/min/m².¹³ Identifying other treatable causes of cardiogenic dysfunction such as arrhythmias, hypoxia, acidosis, pericardial tamponade, or massive pulmonary embolism is the key for cardiogenic stabilization. Continued myocardial decompensation may require placement of an intra-arterial balloon pump and subsequent evaluation for mechanical cardiac support.

Respiratory Compromise

Respiratory care plays an important role in the care of the postoperative patient. Anesthesia, splinting, and immobilization leads to retention of pulmonary secretions and atelectasis.¹⁴ Turning, ambulation., coughing, percussion, postural drainage, and use of an incentive spirometer are all integral parts of an aggressive pulmonary toilet program. Use of bronchodilators, especially in patients who smoke or have underlying pulmonary disease, is extremely beneficial in the immediate postoperative period. We suggest the use of a selective B2 agonist such as terbutaline or albuterol every six hours in an aerosol form.¹⁵

Initial manifestation of pulmonary dysfunction can present clinically as dyspnea, tachypnea, increasing anxiety, or diminished mental alertness. Documentation of impaired pulmonary function requires evaluation of the arterial blood gas. Subsequent therapy is based on an accurate interpretation of these results. To understand any abnormalities noted, pulmonary function can first be discussed in terms of two relatively separate processes: respiration and ventilation.

Respiration pertains to gas exchange, with tissue oxygenation considered the main objective. Partial pressure of arterial oxygen (PaO_2) is a direct reflection of respiratory success. *Ventilation*, on the other hand, describes only the mechanical process of air movement. Determination of its success requires measurement of the arterial CO_2 content (PaCO_2). Factors that affect respiration include the concentration of the inspired air (FIO_2), the diffusion capability of oxygen across the alveolar-capillary membrane, the hemoglobin concentration, and the adequacy of peripheral perfusion.¹⁶ Ventilation relies largely on respiratory rate and tidal volume.

In the surgical patient, acute hypoxia is most likely due to the development of a ventilation perfusion mismatch. Such inequality may arise if alveoli are perfused but not ventilated, resulting in an "intrapulmonary shunt,"¹⁷ as commonly seen with postoperative atelectasis. On the other hand, if alveoli are adequately ventilated but poorly perfused, as with pulmonary emboli or pulmonary edema, an "alveolar dead space" (increased functional residual capacity) will be present. Ventilation failure, in this setting, is often due to excessive sedation from anesthetics or narcotics, and residual paralysis is due to neuromuscular agents used during surgery.

Treatment of respiratory dysfunction requires urgent oxygen therapy when the PaO_2 falls below 60 mm Hg. Oxygen can be administered by nasal cannula or face mask. A maximum of 4 liters can be given by cannula, corresponding to a FIO_2 of 35%. Higher flow should not be used, due to its drying and irritating effect on the nasal mucosa. A face mask can deliver up to 40%–60% FIO_2 . Ventilation failure is corrected by placement of an oral airway with assisted ventilation until the effects of the drug are reversed.

Patients who cannot maintain a PO_2 of 60 mm Hg, a PCO_2 less than 50 mm Hg, or a respiratory rate of less than 45 will require intubation and mechanical support. FIO_2 should be kept below 50% to decrease the risk of oxygen toxicity from the production of free oxygen radicals. Use of positive end-expiratory pressure (PEEP) can support oxygenation by reducing FRC, thus reducing

the requirements of FIO₂. However, PEEP should not be administered above 10 mm of H₂O without close hemodynamic monitoring and diligent surveillance for barotrauma with its resultant pneumothorax.

Postoperative Fever

Temperature elevation above 99.9° F (37.5° C) in the surgical patient should alert the physician of potential complications. Evaluation and subsequent therapy is dependent on how soon after surgery the fever develops. Within the first two days after surgery fever is commonly caused by released pyrogens or pulmonary atelectasis.¹⁸ Pyrogens arise from hematogenous seeding of either leukocytes or bacteria, an example of which occurs during the manipulation of a pelvic abscess.¹⁸ Atelectasis commonly develops from hypoventilation due to mechanical splinting from incisional pain. Less commonly, a necrotizing wound infection should be ruled out by inspection and palpation of the incision. Signs of crepitus, pain, and edematous discoloration may be indications for aggressive intraoperative debridement and drainage. Provided that the patient does not show signs of toxicity (*i.e.*, hypotension, persistent tachycardia, mental confusion, or respiratory distress) treatment is largely symptomatic using antipyretics, incentive spirometer, and increased ambulation.

Fever occurring between postoperative days 2 and 4 potentially could be due to an upper urinary tract infection, an infected intravenous line, or pulmonary pneumonia. Work-up for adequate diagnosis may include a urinalysis, chest x-ray, sputum culture, and a thorough physical exam with attention to all IV sites. UTI or pneumonia, if found, should be treated as a nosocomial infection with appropriate broad-spectrum antibiotics. Suspicious IV lines should be removed, followed by local heat and elevation. If daily inspection shows progression of the phlebitis, the involved vein should be surgically excised.¹⁹ Deep venous thrombosis should be confirmed if clinical signs are suspicious and intravenous heparin started (see "Thromboembolic Disease" below).

After the fifth surgical day the differential for new or persistent fever expands further. While wound infections from less virulent organisms continue to be a threat, intraabdominal or intrapelvic abscess should be considered. For wound infection, the skin should be opened to the fascia and the drainage gram stained and cultured. If concurrent cellulitis is noted or infection is found to have systemic effects, broad-spectrum antibiotics are used and adjusted when culture results are obtained. Thereafter, local wound care with adequate drainage is usually curative. Without evidence of a wound infection, a CT scan should be obtained.²⁰ Localization of an abdominal abscess will require either surgical or percutaneous drainage with concurrent use of intravenous antibiotics. Failure of adequate drainage will necessitate aggressive operative intervention to prevent progression into multiple organ failure due to repeated bouts of bacteremia and its subsequent activation of the complement, leukocytic cascade.²¹

Oliguria

Urine output is important because it is a direct reflection of tissue perfusion. The patient who voids less than 17 ml of urine/hour is by definition oliguric. However, for most patients, a urine flow of less than 30 ml/hour should demand clinical attention. Oliguria can be either prerenal, renal, or postrenal. To determine the cause of the low output a variety of laboratory data, clinical measurements, and physical findings should be gathered to assist in the diagnosis. Heart rate, orthostatic changes, and daily weights are easily measurable and usually correlate with the intravascular volume status. More accurately, CVP or a Swan-Ganz catheter measuring PCWP can be used in diagnostically difficult situations. Laboratory data, including serum sodium, BUN/creatinine ratio, fractional excretion of sodium, and serum osmolality, are suggestive but often nondiagnostic in the acute situation.²² Physical examination noting jugular venous distention, mucous membrane, pulmonary rales, S3 heart sounds, or pitting edema adds valuable data to the clinical evaluation. Once a working diagnosis is made, therapy should be provided. The patient's response is followed and his or her condition re-evaluated, thus confirming or disproving the accuracy of the initial diagnosis. When corrected, potential complications such as volume overload or acute tubular necrosis can be avoided.

In the early postoperative period, a prerenal etiology is most often the cause of decreased urine output. Decreased effective intravascular volume is often due to perioperative blood lost and third space sequestration of extracellular fluid. Isotonic fluid enters the traumatized tissue or the nonfunctioning gut and decreases the effective volume. Initial treatment with isotonic solution may be given in 300–500 ml bolus and repeated up to a liter depending on the patient's underlying cardiovascular capability. Failure to respond may require invasive monitoring to determine the actual volume status. Other prerenal causes of oliguria include cardiogenic failure and will require either diuretics and/or inotropic agents such as dobutamine, depending on PCWP and CO as measured by the Swan-Ganz catheter.

Postrenal oliguria may be due to ureteral obstruction from hyperangulation or inadvertent ligation of the ureters at the time of surgery. This of course would have to be bilateral and would produce anuria. When such a condition is suspected, renal ultrasound should be obtained to identify the presence of hydronephrosis. If further intervention is required, ureteral catheters are both diagnostic and therapeutic.

Renal causes of oliguria are suspected when pre and postrenal causes are excluded. While many causes of parenchymal damage exist, the most common pathology in the immediate postoperative period is acute tubular necrosis. More specifically, this results from (1) ischemia due to hypoperfusion, or (2) nephrotoxicity secondary to aminoglycoside or radiocontrast dye use. Supporting laboratory data such as those previously mentioned, in addition to microscopic examination of the urine, should be obtained. Initial management requires close attention to fluid

balance and electrolyte abnormalities. If possible, fluid diuresis should be initiated by the use of intravenous Lasix. Dosages; should be doubled with each successive administration until an adequate response (approximately 100 ml/hr) is achieved or the maximal dosage of Lasix 600 mg/day, is reached. Maintenance of a nonoliguric state will greatly facilitate subsequent fluid management.²³ If good urinary output cannot be achieved, maintenance fluid replacement should be adjusted to include only insensible losses and ongoing losses from the previous 24 hours. Potassium administration should be stopped immediately until current levels are obtained. If conservative management fails to maintain homeostasis, use of hemodialysis should be undertaken to correct azotemia, uncontrolled hyperkalemia, or fluid overload.²²

Electrolyte and Acid-Base Abnormalities

Hypernatremia is a relative deficit of water volume compared to sodium concentration. The major causes of hypernatremia include:

Decreased water intake as compared to solute intake, such as in concentrated enteral feeding or hypertonic parenteral fluid administration

Excessive water loss compared to sodium loss such as in diabetes insipidus, glycosuria, and diuretic use

Intrinsic renal disease with decrease renal tubule response to ADH

Endocrine disturbance such as Cushing's syndrome or hyperaldosteronism

Clinically, patients present with confusion, seizures, stupor, or coma. Urine electrolyte evaluation may indicate the cause of increased sodium concentration. Treatment depends on correction of the primary cause, as well as slow replacement with D5W or hypotonic fluid.

Low serum sodium concentrations (hyponatremia) are a far more common clinical problem.

Hyponatremia may be due to an actual decrease in extracellular sodium (depletional hyponatremia), but is far more often secondary to an increase in extracellular water (a dilutional hyponatremia). In the latter instance, patients are usually edematous or have significant third-space filling of extracellular fluid, such as in the gastrointestinal tract. This is in contrast to the patient with depletional hyponatremia, who shows clear evidence of decreased extracellular fluid volume with low plasma volume, poor tissue turgor, and signs of hypotension. The presence of dilutional hyponatremia can be easily verified with a urine sodium measurement that is in the normal range.

The patient with a low serum sodium concentration may on occasion have the syndrome of inappropriate antidiuretic hormone (SIADH). SIADH may accompany intracranial lesions, pulmonary disease, malignant disease, and administration of certain drugs such as diuretics and vinca alkaloids. The treatment is to restrict the patient's intake of water to approximately one

liter/day. However, should the serum fall below 110, there is a significant risk of seizures. If symptomatic, patients require careful administration of hypertonic solutions. Fluid overload and congestive heart failure can result from aggressive hypertonic infusions.

In assessing the problems of potassium balance in the postoperative patient, internal mechanisms for potassium distribution related to serum pH must be taken into consideration. Patients who are acidotic have an increase serum concentration of hydrogen ions. This results in an increased movement of these ions into the cells. To maintain neutrality, potassium ions are subsequently pumped out of the cells, thus increasing the potassium concentration in the serum. Conversely, in alkalosis, the serum potassium decreases as the potassium moves into the cells. In addition, cellular destruction at the time of surgery can lead to leakage of intracellular potassium into the serum, with a subsequent rise in serum potassium.

When the serum potassium exceeds 7 mEq/liter, there is a significant risk of a fatal cardiac arrhythmia. Early warning signs can be seen on the electrocardiogram (EKG), with peaking of T waves followed by prolongation of the PR interval and widening of the QRS complex. The treatment of hyperkalemia first consists of stopping all potassium administration to the patient. In life-threatening hyperkalemia, immediate administration of NaHCO₃ should be considered, which causes the transient hydrogen and potassium flux just described. The serum potassium can then be lowered by the simultaneous infusion of glucose and insulin (1 unit of insulin for every 5 g of glucose). Administration of ion-exchange resins such as kayexalate will bind potassium into the gastrointestinal tract and thus lower the serum potassium concentration. However, its onset of action is relatively slow and may require several hours for clinical response.

Hypokalemia in the postoperative patient is commonly due to obligatory losses of potassium in the urine, but can easily be avoided by potassium maintenance solutions as suggested above. In general, hypokalemia can be corrected with 20 mEq to 40 mEq KCl/liter of IV fluids. However, in severe cases of hypokalemia (serum potassium less than 3 mEq/liter), 10 mEq KCl can be administered in a Buretrol (drip) of 50 ml solution over 1 hour.

In significant long-standing hypokalemia, there usually coexists a magnesium deficiency, which can be evaluated by serum magnesium level. If present, it can be corrected with 50% MgSO₄, 10–20 ml intravenously over 24 hours.²⁴

Management of fluid and electrolyte imbalance in the postoperative patient also requires attention to acid-base abnormalities. The acid-base status of the patient is best assessed by the arterial blood gas and serum CO₂. Values outside of their normal parameters can usually be categorized into specific acid-based abnormalities.

Acidosis with a corresponding high pCO₂, indicating respiratory acidosis, is usually caused by hypoventilation. This can be a common problem in the recovery room and can easily be confirmed by noting a decreased vital capacity as measured with a portable spirometer. The vital

capacity should be greater than 30 ml/kg, which usually correlates with a tidal volume of approximately 500 ml. Such values would indicate sufficient ventilation in the average adult. Correction of hypoventilation in such a patient will lead to correction of the respiratory acidosis. The patient with a high pH and low PCO₂ has respiratory alkalosis, which is usually secondary to hyperventilation. The most common causes of hyperventilation are hypoxia, anxiety, compensation for metabolic acidosis, excess mechanical ventilation, shock, and septicemia. Paresthesia and tetany resembling hypokalemia can be seen with severe respiratory alkalosis. The treatment of respiratory alkalosis is simply that of correcting the underlying cause. Metabolic acidosis is characterized by a low pH and a low HCO₃ and can be seen with lactate acidosis from hypoxia, ketoacidosis from diabetes, and renal acidosis from uremia. An additional cause is loss of alkali from intestinal fistulas. Once again, the treatment of metabolic acidosis is to correct the underlying cause. If the pH is below 7.25, the administration of NaHCO₃ is indicated. For patients on respirators, the respirator can be adjusted to make the patient hyperventilate and lower the PCO₂, thus helping correct the acidosis. In severe cases of metabolic acidosis with significant amount of HCO₃ replacement, calcium should be given, because the serum calcium will drop with the correction of the acidosis.

Patients with a high pH and high bicarbonate have metabolic alkalosis, the predominant cause of which in the postoperative patient is loss of acids through gastric secretions or iatrogenic over-correction. Both these conditions are best corrected by the administration of normal saline along with symptomatic support.

Thromboembolic Disease

Thrombosis of veins in the lower extremities or the pelvis is a common complication of gynecological surgery. Patients classically present with a warm leg that is edematous, painful, and tender. Stasis is a major cause of venous clotting. This usually occurs as a result of either venous outflow obstruction or from conditions that render the legs hypotonic. Outflow obstruction may be caused by obesity, the gravid uterus, or congestive heart failure, whereas the hypotonic limb occurs during anesthesia or from prolonged bed rest.

The other major cause of venous thrombosis is related to patients who are hypercoagulable. Individuals who have an underlying malignancy or who sustained previous endothelial damage are at increased risk for clot formation. The cancer patient undergoing lymphadenectomy is therefore much more prone to thrombosis.

Deep vein thrombosis may be complicated by pulmonary embolism, which occurs when a proximal thrombus breaks off and travels to the pulmonary artery. Frequently these small clots reach the pulmonary circulation, where they occlude some small vessels, then retract, undergoing fibrinolysis, and disappear without any clinical symptoms.²⁵ However, significant obstruction of pulmonary arteries can cause a whole spectrum of clinical problems from massive obstruction

leading to sudden death, to recurrent occlusion of the pulmonary vasculature leading to chronic pulmonary hypertension. However, pulmonary infarction is, in fact, a rare event that occurs in fewer than 10% of patients with pulmonary emboli.²⁶

The diagnosis of venous thrombosis frequently requires objective radiological techniques, because signs and symptoms alone are frequently inaccurate.²⁷ The diagnostic standard used to in the evaluation includes contrast venography and pulmonary arteriography. However, these tests are invasive, expensive, and expose the patient to additional risks. As a result, noninvasive techniques have been developed over the past 20 years that decrease patient morbidity while improving the accuracy ,of the clinical diagnosis. Among the various ultrasonic and nuclear medicine tests that are available, we have consistently found that the combination of a venous doppler study and venous plethysmography to be most helpful during the evaluation of potential thrombosis. Although the doppler is dependent on the experience of the technologist, its use is inexpensive and versatile. For the evaluation of pulmonary emboli, the radioisotope V/Q lung scan remains useful to detect ventilation/perfusion defect.²⁸ Its sensitivity and specificity is enhanced when correlated with the chest x-ray and clinical suspicion.

The treatment of deep vein thrombosis or pulmonary embolism consists primarily of anticoagulation therapy. Heparin is the drug of choice but only prevents further progression of the existing thrombus; clots that are already present are not affected by its use. Intravenous heparin, 1000 units/kg, is given as an initial bolus as soon as the diagnosis is suspected. This is then followed by a continuous infusion at 1000 units/hr. Subsequent rate is adjusted to maintain a prothrombin time at two times control. Noninvasive screening, previously discussed, should be obtained to confirm the diagnosis. If evaluation proves negative, the heparin is discontinued. However, if these initial tests are equivocal, more invasive diagnostic studies are employed and the results treated accordingly.²⁷ Fibrinolysis has been used successfully but is rarely indicated in the postoperative patient.

Massive, life-threatening pulmonary emboli may require embolectomy via transvenous suction or open pulmonary embolectomy. After adequate control of the; acute process, prolonged anticoagulation is attained by the use of Coumadin to maintain a prothrombin time at 1.3 times control. Continued emboli despite adequate anticoagulation or in patients in whom its use is contraindicated, a vena caval (Greenfield) filter is used to trap the venous clots.²⁹

Prophylaxis of thromboembolic disease in the gynecological surgical patient is important owing to the high incidence of postoperative deep venous thrombosis and pulmonary embolism. Patients should be encouraged to move their legs frequently while in bed. Early ambulation is of paramount importance, and a footboard should be provided for those patients who are not likely to achieve early ambulation. Elastic stockings can compress the legs and divert the blood from the superficial to deep venous veins, improving flow and reducing stasis.

Patients with cancer, a previous history of deep venous thrombosis and pulmonary embolism, obesity, congestive heart failure, and those using oral contraceptives should, in addition, receive prophylactic heparin. Perioperative prophylactic heparin has been shown to decrease the incidence of deep venous thrombosis from approximately 30% to below 10% without any significant complications.²⁶ The most popular regimen is low-dose heparin given at a dose of 5000 units every 12 hours by subcutaneous injection. The initial dose must be given at least two hours prior to surgery, because deep venous thrombosis commonly occurs during the surgical procedure. Patients at extremely high risk of thromboembolic disease, such as those with a previous history of deep venous thrombosis or pulmonary embolism, should receive 5000 units of heparin subcutaneously every eight hours. Heparin is continued until the patient is completely ambulatory, which is generally one to two days prior to discharge from the hospital. A normal coagulation profile should be seen before initiating any of the prophylactic heparin regimen just described. Platelet count and partial thromboplastin time should be monitored every two to three days while the patient is on heparin treatment, because thrombocytopenia and full anti-coagulation can be seen with these treatments.

In patients in whom heparin is contraindicated or in whom additional preventive measures are desired, the use of sequential pneumatic compression stockings are equally as effective as heparin therapy. For maximal benefit these stockings are placed prior to anesthetic induction and compression initiated prior to muscular relaxation.³⁰

Urinary Retention

The morbidity of urinary retention can lead to patient discomfort and prolonged hospital stay. Bladder overdistention can be easily avoided by awareness of its possible existence and attention to the urinary output. In the acute postoperative patient, retention is due to a minimal number of etiological factors. During prolonged operative procedures or in patients with significant postoperative pain, bladder capacity may be surpassed. The musculature becomes overdistended and its ability to contract is compromised. This dysfunction may persist despite subsequent decompression. Preventive measures includes intraoperative Foley placement or early intermittent catheterizations every 6–8 hours. Treatment of patients with overdistention dysfunction requires placement of an indwelling Foley catheter for 24–48 hours, thus allowing recovery of the injured musculature.³¹

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