Manual of Surgical Therapeutics

MANUAL OF SURGICAL THERAPEUTICS

SECOND EDITION

Departments of Surgery
University of Iowa and University of Illinois

EDITORS

Robert E. Condon, M.D., M.S.
Professor of Surgery and Head of the Department, University of Iowa College of Medicine, Iowa City

Lloyd M. Nyhus, M.D.
Professor of Surgery and Head of the Department, The Abraham Lincoln School of Medicine of the University of Illinois at the Medical Center, Chicago

PREFACE TO THE SECOND EDITION

We have been most gratified by the generally enthusiastic response to the *Manual of Surgical Therapeutics.* Our colleagues who have reviewed the *Manual* have found it to be worthwhile; this and its ready and widespread acceptance by students and house officers have been the immediate reasons for a second edition.

The *Manual of Surgical Therapeutics* continues to be for *the man on the firing line.* We have added a new section concerning normal hemostasis and the approach to the patient whose clotting mechanism is disturbed as well as one on management of facial trauma. The discussions dealing with management of trauma, shock, and cardiopulmonary arrest have been revised extensively. The chapter on surgical infections and antibiotic therapy has also been revised extensively and updated in keeping with the rapid growth of knowledge in this area.

All material has been rechecked or revised by both contributors, old and new. As well, closure of a few gaps has been accomplished by the addition of new material.

To our colleagues, we extend our continuing appreciation for their diligence and expertise, as reflected in this *Manual.*

We continue to be indebted to Mr. Fred Belliveau of Little, Brown and Company, his editorial staff, and, in particular, to Mrs. Anne N. Merian. We acknowledge the fine drawings of the hand by Mr. William R. Schwarz of the Department of Medical Illustration, University of Illinois. Special thanks are due Mrs. Ann Haddick Napoletan and Mrs. Barbara Getz, editorial assistants of the Departments of Surgery, University of Iowa and University of Illinois, respectively, without whose devotion to these pursuits the production of the *Manual* would have been much more difficult.

Robert E. Condon
Lloyd M. Nyhus

PREFACE TO THE FIRST EDITION

In the *Manual of Surgical Therapeutics* the student or house officer will find the essentials of day-to-day care of the surgical patient. It is a manual for the *man on the firing line,* and as such it is a readily available source of information on the pathophysiologic, pharmacologic, and nonoperative aspects of the care of the surgical patient. Discussions will be found on drug usage and administration, dosages and side effects, as well as on diagnostic maneuvers and the management of complications should they arise. No attempt has been made to be all-inclusive, however, since inclusiveness itself is a presumptuous goal. It is expected that when the frenetic pace of the Surgical Service slackens, the user of this *Manual* will undertake to find in standard surgical textbooks the in-depth coverage he seeks.

For a number of years, the value of another manual, the *Manual of Medical Therapeutics,* has been apparent wherever medical students and house staff are in evidence. Its edges often frayed, the medical manual is seen in the pockets of many white coats throughout the nation as well as in many dictating and nursing stations where harried medical students or house officers have temporarily misplaced it. But much of the information which the student or house officer on the Surgical Service needs is not to be found in the medical manual.

Thus the need for a surgical manual made itself apparent. This *Manual* has been prepared by the members of the Department of Surgery of the University of Illinois College of Medicine and reflects our methods and judgment in the management of selected surgical problems. We thank all the contributors for their assistance and for their understanding acceptance of the considerable exercise of editorial prerogative which the published version of their contributions represents.

Deep appreciation is due Mr. Fred Belliveau of Little, Brown and Company, his editorial staff, and, in particular, Mrs. Anne N. Merian. Special thanks are due Mrs. Ann Napoletan and Mrs. Barbara Getz, editorial assistants of the Department of Surgery, University of Illinois College of Medicine, without whose capable help and devotion it would not have been possible for us to complete this *Manual.*

Robert E. Condon
Lloyd M. Nyhus

CONTENTS

1 THE EVALUATION AND MANAGEMENT OF TRAUMA

I. GENERAL PRINCIPLES

A. Emergency evaluation The outcome after severe trauma depends on two factors: *availability of medical care* and *adequacy of early treatment.* The first objective of the physician examining an injured person is the preservation of life. Resuscitation and proper evaluation of life-endangering injuries are crucial to survival. Injudicious or inadequate emergency management can result in an unnecessary fatality or permanent disability. When dealing with acute trauma, it is impossible to separate diagnostic and therapeutic measures. Resuscitation is not dependent on an etiological diagnosis. Airway obstruction, shock, and cardiorespiratory failure often must be treated without knowledge of the precipitating cause of these disorders. Once the patient is stable, rapid and thorough evaluation of the genesis of these derangements is in order.

The **priorities of management** of an injured patient are:

1. ***Establish and maintain an adequate airway*** Patients with depressed consciousness from intoxication, cerebral injury, or shock have a high risk of aspiration of blood, food, vomitus, and dentures. Insertion of an oropharyngeal airway after aspiration and removal of any foreign bodies are the first considerations. The patient must be observed closely and suctioned frequently. He must not lie unattended flat on his back or be restrained in this position. A semiprone position is more satisfactory. Some common causes of upper airway obstruction include bleeding or edema of the mouth, tongue, posterior pharyngeal wall, and epiglottis. Penetrating wounds of these parts are particularly dangerous. Cervical spine injuries producing vertebral subluxation and retropharyngeal hematoma can compromise the upper airway. Severe maxillofacial trauma with obliteration of the nasal passage will contribute to these problems. Respiratory distress with stridor, a contusion of the neck, and a history of steering wheel injury may indicate the presence of a fractured larynx or trachea. Endotracheal intubation will be unsuccessful in such a case and emergency tracheostomy is necessary. A patient exhibiting only abdominal breathing may have a cervical vertebral dislocation and spinal cord injury. Any manipulation before stabilizing the neck may complete a partial transection of the spinal cord. Vascular injuries in the neck producing hematomas may compress the airway and require intubation. Examine for evidence of sucking chest wounds, flail chest, tension pneumo-

thorax, hemothorax, simple pneumothorax, or contused lungs. Relieve airway obstruction (suction and intubation) and respiratory insufficiency (oxygen and positive-pressure ventilation). Patients who are combative because of hypoxia can be intubated by using a rapidly acting neuromuscular depolarizing agent. Only rarely is a primary tracheostomy necessary.

2. **Assess and support cardiopulmonary function** Severe hypoxia and acidosis can result in cardiac arrest. If arrest has occurred, mouth-to-mouth resuscitation must be employed immediately, followed by endotracheal intubation. Closed chest massage is preferred (see Chap. 4) and is effective except when the chest wall is unstable, as in flail chest, or in patients with severe emphysema. In such cases, emergency thoracotomy and manual cardiac compression may be necessary. Ventilation with an "Ambu" bag and oxygen are provided while cardiac compression is carried out. Metabolic acidosis is corrected with IV sodium bicarbonate. Initial and periodic arterial blood gas and pH analyses are very helpful. ECG (electrocardiographic) monitoring is essential; cardiac defibrillation may be needed since an hypoxic and acidotic heart is prone to arrhythmias, fibrillation, and asystole (see Chap. 4).

3. **Control hemorrhage** External hemorrhage is best controlled by pressure at the source of bleeding using sterile gauze pads. Pressure is maintained until definitive proximal control is possible under optimal operative conditions. The use of tourniquets or of blind clamping is not advisable, as further injury may ensue. The possibility of loss of blood within the body cavities must be evaluated; thoracocentesis and paracentesis will help establish the diagnosis of such hemorrhage.

4. **Treat shock** Restoration of blood volume deficit is the prime therapeutic aim in management of hypovolemic shock. Major blood loss must be replaced with blood. However, blood volume losses of up to 30% (1,500 ml) may be replaced with blood substitutes. Loss of 1,000 ml may cause no significant physiological disturbance except for a fall in pulse pressure and a slight tachycardia. Loss of 1,500 ml results in tachycardia, tachypnea, and postural hypotension. Major exsanguination (over 30% of blood volume) causes the classic signs of pallor, cold clammy skin, blood pressure below 80 mm Hg (or 50% below usual value), and oliguria (urine output below 30 ml an hour). Patients with catastrophic loss of over 45% of their effective circulating blood volume are severely prostrated, hypoxic, and unresponsive. These patients, if not vigorously resuscitated, will progress to cardiopulmonary arrest. Clinical guides to assess the magnitude of blood loss are shown in Table 1-1.

The **essential steps in management of hemorrhagic shock** are:

a. **Insert intravenous catheters** Insert one or more large polyethylene intravenous catheters, one of which must be in a central (intrathoracic) vein. These may be inserted via the subclavian, external jugular or antecubital routes. Use sterile technique and dress the catheter puncture site with an antibiotic ointment and sterile gauze.

TABLE 1-1. Estimation of Blood Loss and Suggested Therapy

Magnitude of Hemorrhage		Clinical Findings	Possible Etiology	Treatment
Grade I	Minor blood loss (10–15%) 500–750 ml	Well compensated May have dizziness, tachycardia (100/min)	Blood donation Laceration Hematoma Hemothorax Extremity fracture	Control hemorrhage Infuse crystalloid if necessary
Grade II	Moderate blood loss (15–30%) 750–1,500 ml	Cool, sweaty, thirsty Tachycardia (110/min) Tachypnea (24/min) Decreased pulse pressure Slight hypotension (90–100 mm Hg)	Major laceration Subcapsular splenic rupture Fractured femur	Control hemorrhage Crystalloid and colloid infusion Oxygen administration Evaluate for obscure bleeding
Grade III	Major blood loss (30–45%) 1,500–2,250 ml	Cold, clammy, pallor Tachycardia $>$ 120/min Hypotension $<$ 80 mm Hg Oliguria $<$ 30 ml/hr Metabolic acidosis	Peripheral vascular injury Stellate liver injury Pelvic fracture	Control hemorrhage Crystalloid, colloid, and blood transfusion Oxygen administration Bicarbonate infusion Physiological monitoring
Grade IV	Catastrophic blood loss ($>$ 45%) over 2,250 ml	Cyanosis Semiconscious Respiratory distress Profound hypotension Anuria, metabolic acidosis	Traumatic amputation Visceral arterial injury Multiple injuries	Control hemorrhage Crystalloid, colloid, and blood transfusion Oxygen administration Bicarbonate infusion Antibiotics Cardiac monitoring

b. **Draw blood samples** through the venous catheters for typing and crossmatching prior to infusion of plasma expanders such as dextran, which can coat erythrocytes and interfere with type and match procedures.

c. **Administer intravenous fluids immediately** to replace blood volume deficits. The choice of fluid is not as important as the volume and rapidity of infusion. Crystalloid solutions (normal saline or Ringer's lactate) are satisfactory. More rapid volume expansion can be obtained with dextran or albumin solutions.

d. **Transfuse blood** as soon as compatible crossmatched blood is available. Maintain the hematocrit above 30%. In extreme cases, in which resuscitation is not possible with blood substitutes alone or when the hematocrit is dangerously low (below 15%), type-specific or universal donor (O-neg) blood may be given until crossmatched blood is available in order to maintain oxygen-carrying capacity.

e. **Monitor CVP (central venous pressure)** through the venous catheter placed in an intrathoracic vein. Central positioning of the catheter tip is facilitated by observing the saline column in the manometer. Smooth fluctuations concomitant with ventilation assure intrathoracic placement. Pulsation of the saline column in relation to the heart rate means the catheter tip is in the right ventricle or pulmonary artery. All catheters should be checked for position later when the chest x-rays are obtained. Serial pressure readings are essential to successful replacement of blood losses. CVP readings are not a measure of blood volume alone but reflect both the adequacy of venous return and the pumping action of the right ventricle. Absolute values are not as important as relative changes observed over a period of time. Typically, in hypovolemic shock, the CVP is well below the normal value of 4–8 cm of saline. With adequate volume replacement, a rise in CVP paralleling that of systolic blood pressure is observed. A low CVP persisting after adequate volume replacement should stimulate a search for occult bleeding. An elevated CVP in the face of a low systemic blood pressure may be a result of pericardial tamponade, myocardial infarction, or acute congestive heart failure. Changes in the quality of heart sounds, cardiac rhythm, electrocardiographic findings, and the response to volume loading will be helpful in this diagnostic dilemma.

f. **Catheterize the bladder** using a Foley catheter and measure hourly urinary output. Initially, diminished urine volume and increased specific gravity and osmolality (above 1.030 and 700 mOsm/kg, respectively) reflect the renal response in shock. A persisting low output or low concentration indices may be a result of early acute tubular failure (see Chap. 10).

g. **Obtain serial hematocrit determinations** to monitor changes in the circulating erythrocyte mass. Although the hematocrit takes several hours to equilibrate after rapid hemorrhage, measurement of the hematocrit is still the best bedside guide to the numbers of erythrocytes available for oxygen delivery. In massive rapid hem-

orrhage there may be no appreciable dilution until after initial therapy with asanguinous fluids when the deficit will become very apparent. In the post-traumatic period, blood volume estimation using radioisotopes can be of value in guiding replacement therapy.

h. **Measure blood gases and pH** After major blood loss and shock, tissue anaerobiosis results in an increased concentration of lactic acid in blood. Accumulation of hydrogen ion causes a fall in blood pH. Usually there is an attempt to compensate for this by increased ventilation permitting excretion of hydrogen ion from carbonic acid (as water and carbon dioxide). In a typically severe case, the arterial blood pH is 7.30 or below, the pCO_2 is 28 or below. Using standard nomograms (Sigaard-Anderson), the degree of metabolic acidosis is expressed as a base deficit (−10 to −20 mEq/liter). Replacement with sodium bicarbonate can be guided accurately by: *mEq of bicarbonate needed = base deficit (mEq/liter) × body wt (kg) × 0.30.* Replace approximately one-half of this calculated amount and then recheck the blood gases in 1 hr.

i. **Repeated examinations of the patient** must be made with special attention to pulse rate, pulse pressure, skin temperature and color, and the state of consciousness. A rapid pulse, narrow pulse pressure, blanched cool skin, and combativeness are signs of recurrent hypovolemia and hypoxia.

j. **Establish the etiology of shock** Blood loss from fractures, especially of long bones or the pelvis, occult rupture of the liver or spleen, and collections of blood in "silent areas" such as the pleural cavity and the retroperitoneal space must be considered. Blood loss into tissues around fracture sites may be deceptive. A fracture of the femoral shaft may cause the loss of 2–4 units of blood, but a pelvic fracture may result in loss of 10 or more pints. Hematuria, hemoptysis, bloody nasogastric suction, or rectal bleeding will suggest other sources of blood loss. Most injuries causally related to shock are either obvious on careful inspection or can be diagnosed by simple measures. Paracentesis, thoracocentesis, intravenous pyelography, and endoscopy will define most occult bleeding sites. *Inability to restore blood pressure and circulating volume in an acutely traumatized patient by adequate blood volume replacement indicates that a source of uncontrolled hemorrhage is present.* Continuing major hemorrhage is seen commonly in liver, retroperitoneal injury, and vascular injury. Further attempts to stabilize the circulation in such cases will be unsuccessful and may cause the demise of the patient. Patients with such injuries often must be taken to the operating room in shock and explored for direct access to the source of bleeding.

5. **Immobilize fractures** The old adage "splint 'em where they lie" remains the best rule. Any motion at a fracture site will cause further skeletal and soft tissue damage. Particularly important is the status of the neurovascular supply to the injured part (see Sect. VII). Pillows tied around an extremity make good temporary splints.

6. **Dress soft tissue wounds** Many times it is not possible to treat soft tissue wounds at the time of injury because other important visceral injuries require attention. In these situations it is best to elevate the wounded part, provide hemostasis by compression, and dress the area to protect it against further injury and contamination. Tetanus prophylaxis and antibiotics must be given in every case. When definitive therapy of a soft tissue wound is to be carried out, one must provide suitable anesthesia, clean the wounds and surrounding area, remove all foreign matter, debride, repair injured tissues, close the wound if indicated, apply an absorptive bulky dressing, and immobilize and elevate the injured part.

B. Clinical evaluation After successful resuscitation as outlined above, the patient enters a critical phase of continuous observation and more general evaluation. The assessment of the patient at this time must include the level of consciousness, spontaneous motion of extremities, chest excursion, and abdominal habitus, noting any obvious area of injury. All patients must be completely disrobed. A rapid assessment of the extent of injuries is performed by gentle but firm palpation of all body parts, especially those areas where injuries are suspected. Deformities or asymmetry of body parts, lacerations, and contusions demand special attention. Palpation of the scalp, facial bones, trachea, and vertebral column, as well as gentle compression of the thorax, ribs, pelvis, and extremities, will usually elicit signs of hidden fractures or dislocations prior to x-ray evaluation and permit intelligent and precise use of these studies. A careful abdominal examination, checking particularly for peritoneal irritation or distention, is mandatory. Rectal and pelvic examinations are routinely performed. Checking the pulses, temperature, and the neuromuscular tone of all extremities is necessary. The chest is auscultated for signs of pleural collapse, rub, or effusion. Changes in heart sounds or the finding of murmurs in the chest or over the extremities should raise a suspicion of vascular injury.

Obtain as thorough a history as possible. The preinjury health status, the factors causing the accident, and the conditions surrounding the accident are all important facts. Previous medical problems, allergies, medications, or impairments must be elicited and evaluated prior to any operation. This information must be sought from any available source, as it may be a crucial factor in survival. During this acute period, patients will have had an airway established, large intravenous catheters placed (one in the superior vena cava), and a Foley catheter inserted in the urinary bladder. The bladder urine specimen and subsequent specimens are sent for analysis. A nasogastric tube is inserted. Any aspirate is inspected, and the stomach emptied and kept decompressed by intermittent suction.

C. Volume replacement solutions and adjunctive therapy

1. **Crystalloid solutions** Initial asanguinous volume replacement has become an acceptable clinical practice. Normal saline (0.9% NaCl) and buffered salt solutions (Ringer's lactate) are cheap, stable in storage, readily available, free of immediate reactions, and do not require any special matching prior to use. There are theoretical advantages of the buffered salt solutions and they are preferred even

though they contain a small (28 mEq/liter) amount of lactate. This small amount of lactate is inconsequential when compared to the massive amounts of lactate produced by the body during shock. As the circulation is restored, lactate is readily converted by the liver to bicarbonate and excreted as carbon dioxide by the lungs. Noncolloid salt solutions rapidly diffuse out of the circulation of the extravascular extracellular space so that 2½ to 4 times the amount of blood lost must be infused as crystalloid solutions to restore the blood volume.

The effects of dilution on the hemoglobin and plasma protein concentrations are factors limiting use of crystalloid solutions. It is desirable to maintain the hematocrit above 30% and the total protein in excess of 6 gm/100 ml. At these values, the oxygen-carrying capacity and oncotic pressure of the blood will protect against further hypoxia and tissue edema. Although salt solutions are not ideal, early administration of these agents has been responsible for the decreased incidence of post-traumatic renal failure. There is no place for salt-free crystalloid solutions (dextrose in water) in primary resuscitation. When given in the early post-traumatic state, the water is retained because of the action of antidiuretic hormone and hypo-osmolality results.

2. **Plasma** stored for 6 months at 30°C under ultraviolet radiation still can transmit serum hepatitis. Type-specific single units of plasma may be given after simple screening crossmatch, although isosensitization may still occur. Plasma may be given without crossmatching and is an excellent blood volume expander which may be safely used when the hematocrit is above 30%. Its use also may be considered when plasma losses are anticipated, as in burns, peritonitis, and pancreatitis.

3. **Albumin** A 6% solution of human serum albumin is readily available and free of risk of hepatitis. Albumin is an effective plasma expander and has other advantages, one of which is that this protein is easily metabolized and spares body muscle proteins from catabolism.

4. **Dextrans** (clinical dextran, average molecular weight 70,000; low-viscosity dextran, mean molecular weight 40,000) Both of these dextrans are effective plasma expanders. They are inexpensive and relatively free of serious reactions. Besides the obvious intravascular expansion of the circulating blood volume, there is an added beneficial effect of these agents, since they decrease blood viscosity and improve microcirculatory flow. Decreased sludging, or the breaking up of erythrocyte aggregations, is a suggested mechanism for the observed improved general and intraorgan (liver, kidney, and gastrointestinal) blood flow. These plasma substitutes are effective in preventing renal failure and are excreted in the urine within 4–6 hr. Use of these agents may affect coagulation mechanisms both by dilution and by their viscosity-reducing properties. The use of dextrans should be limited if there are large soft tissue injuries or if an operation which will involve retroperitoneal dissection is anticipated.

5. **Blood transfusions** Major losses of whole blood must be replaced by blood to maintain a hematocrit level above 30%. A need for unmatched universal donor (O-neg) blood is uncommon, as most patients can be adequately supported initially by one of the blood

substitutes mentioned above. An increase in heart and respiratory rates, cardiac output, and tissue oxygen extraction are the major mechanisms employed to compensate for the loss of erythrocytes in hemorrhage and during the dilutional phase of volume restoration. Untoward reactions of blood transfusions are: major and minor blood group incompatibilities, isosensitization, serum hepatitis, and occasionally the transmission of bacteria or their metabolic by-products. Banked blood becomes progressively more acidotic and hyperkalemic, and there is a rapid loss of many coagulation factors. Massive rapid transfusions (over 20 pints) are associated with a high incidence of reactions.

6. **Buffer therapy** In shock, decreased tissue blood flow and oxygenation lead to anaerobic glycolysis. Excess lactic and pyruvic acids are produced and enter the circulation. The metabolism of these organic acids by the liver is depressed and metabolic acidosis supervenes.

   Sodium bicarbonate (5%) is an extracellular buffer, similar to but faster in action than sodium lactate (1/6 molar). Both agents are available in 500 ml bottles. Rapid changes in blood pH may be induced and cause cardiac arrhythmias so that patients receiving rapid infusions of buffers must be monitored by continuous ECG. Tris-hydroxymethyl-aminomethane (THAM) is an effective intracellular and extracellular buffer. This agent does not provide cation replacement and may cause central respiratory arrest. It is rarely used and is indicated only when the arterial pH is less than 7.30 and administration of sodium bicarbonate has failed to begin correction of the acidosis.

7. **Oxygen therapy and ventilatory support** All patients who display dyspnea or tachypnea should be given oxygen. Cyanosis usually will be present but may be absent if blood loss has been large and the circulating hemoglobin concentration is below 5 gm/100 ml. It must be remembered that the important mechanism of oxygen transport (over 95%) is the hemoglobin in erythrocytes. A normal hemoglobin of 15 gm/100 ml provides transport for 20 volumes percent of oxygen while a hemoglobin of 7 gm/100 ml (hematocrit 21%) will carry only 10 volumes/100 ml. This is the critical reserve level of oxygen consumption for most tissues, especially heart muscle and brain. Oxygen given by properly applied mask or by nasal catheter at 6 to 8 liters/min will increase inspired oxygen concentrations by 10%, and this will improve hemoglobin saturation and oxygen delivery to the tissues. Oxygen administered via a "T" adapter to an endotracheal or tracheostomy tube will increase the inspired oxygen concentration to 40%. Oxygen delivery in concentrations above 50% is unnecessary in managing most patients. Pulmonary oxygen toxicity may result if 100% oxygen is administered for 24 hr.

   Ventilatory support is indicated whenever voluntary respiratory volumes are inadequate and for specific conditions such as cardiopulmonary arrest, mechanical chest wall dysfunction, atelectasis, or pulmonary edema. Complete and repeated examinations for hemothorax or pneumothorax prior to starting and during ventilatory support are especially important. Previously undetected injury may be responsible for development of a tension pneumothorax if positive

pressure ventilation is employed. High inspiratory pressure, continuous positive pressure, and the use of expiratory retarding devices may all contribute to increased resistance to pulmonary blood flow. Positive pressure is transmitted to the alveolar space, pulmonary capillaries are compressed, and blood flow through the pulmonary circulation is diminished. Decreased return to the left side of the heart causes a fall in cardiac output and may prolong or increase shock, especially in a hypovolemic patient.

8. **Antibiotics** Massive trauma and shock are associated with decreased function of the reticuloendothelial system so that during the early post-traumatic period, the ability of the body to clear bacteria from the circulation is depressed. Prophylactic antibiotic therapy is needed in these circumstances. Antibiotic therapy is discussed in Chapter 5, Section IV.

9. **Diuretics** Maintenance of adequate urinary output (over 30 ml/hr) is a prime aim in the management of shock and major trauma. In shock, sympathetic compensatory mechanisms cause a decrease in renocortical blood flow and a marked elevation of circulating ADH (antidiuretic hormone) promoting increased water reabsorption from the distal tubule. Effective circulating plasma volume is thus conserved. When hypovolemia is inadequately treated, or when circulating tissue debris (hemoglobin and myoglobin) is filtered but not cleared from the tubules because of decreased urine flow, renal shutdown may result. Osmotic or chemical diuretics in addition to volume replacement should be employed early when renal function is precarious or when frank failure is suspected. Mannitol, a monosaccharide which is filtered but not reabsorbed by the kidney, induces an obligate excretion of filtered water which helps maintain renal tubular flow and patency. Up to 100 ml of 20% mannitol solution may be infused rapidly and the infusion may be repeated within the next hour if diuresis is not established. Mannitol also acts as a plasma expander; the CVP must be monitored during therapy. If no response is obtained to mannitol, give ethacrynic acid (50 mg) or furosemide (10–20 mg) IV. These agents also may be repeated within 1–2 hr. The effectiveness of diuretic agents is dependent upon an adequate circulating blood volume. Neither approach (osmotic or chemical) will be effective in hypovolemia.

   Should a urinary output be initiated, try to maintain the hourly volume at over 60 ml/hr. Urine specific gravity, which is simple to measure, or urine osmolality, determined by the freezing point depression method, is a guide to the efficacy of therapy and helps in estimating the functional status of the kidneys. A high urine specific gravity (over 1.025) or osmolality (700 mOsm/kg) indicates persistent hypovolemia; a low specific gravity (under 1.010) or osmolality (150 mOsm/kg) may mean overinfusion. An iso-osmotic value (specific gravity 1.010 or osmolality 300 mOsm/kg) may represent the effect of diuretics or be the first sign of loss of concentrating function and impending tubular failure. Further fluid or diuretic therapy may be chosen intelligently by comparing serial serum and urine osmolality measurements. Mannitol and chemical diuretics can be given intermittently each 6–8 hr as necessary to maintain adequate urine flow.