Selective Review of Key Perioperative Renal-Electrolyte Disturbances in Chronic Renal Failure Patients*

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The medical care of chronic renal failure patients is often complicated by the comorbid conditions of hypertension and coronary artery disease in the perioperative period. The limitations on solute and water excretion imposed by renal dysfunction increase the susceptibility of this population to both salt deficit and surfeit, as well as hyponatremia and hypernatremia perioperatively. Accurate assessment and successful treatment of these complications in renal failure patients require understanding of the concept of electrolyte-free water, proper utilization of diuretics, and calculated prescription of fluid therapy. The presence of hyperkalemia in the adapted renal failure patient generally indicates a severe reduction in glomerular filtration, such that nonrenal hypokalemic treatments are imperative. IV calcium-based therapy and infusion of insulin with glucose represent the mainstays of immediate therapy, and sodium bicarbonate therapy should be given only when severe acidemia is present. Perioperative aggravation of preexistent hypertension is common. Rebound hypertension attributable to injudicious adjustment of the medical regimen should be diligently searched for first, before any new therapies are recommended. Relief of pain or anxiety may be all that is necessary. Briefly acting calcium channel blocker therapy should not be employed in these cases, and smooth IV control by a variety of agents is preferable, the choice of the agent contingent on the clinical scenario.

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Herein follows a selective review of certain key renal-electrolyte disorders encountered during the perioperative period in patients with chronic renal insufficiency (Table 1). The selection of topics reflects the authors’ biases as to the more common and more important fluid-electrolyte issues seen during the perioperative period in patients with variable degrees of renal failure.

DISORDERED VOLUME REGULATION

Adaptation

The product of the serum sodium concentration and the glomerular filtration rate (GFR) defines how many milliequivalents of sodium enter the nephrons in a given period. Under normal conditions, with a serum sodium concentration of 140 mEq/L and a GFR of 120 mL/min, the filtration rate of sodium is 16.8 mEq/min or 24,192 mEq/d. The amount of sodium that is eventually excreted is tightly linked to the daily intake. Thus, someone ingesting and excreting 200 mEq of sodium each day excretes a mere 0.8% (200 ÷ 24,192) of his or her filtered load, or put another way, normally, 99.2% of the filtered sodium undergoes tubular reabsorption. Patients with advanced but stable chronic renal failure (CRF) may also ingest and therefore, excrete 200 mEq of sodium daily, despite a GFR that might be 12 mL/min. With a serum sodium level of 140 mEq/L but a GFR of 12 mL/min, only 1.68 mEq/min or 2,419 mEq/d are filtered. Thus, the fraction of filtered sodium excreted rises to 8% (200 ÷ 2,419), or the fractional reabsorption decreases from 99.2 to 92%. By inhibiting the tubular reclamation of filtered sodium, the patient with advancing renal failure remains in salt balance, despite a large and variable intake.

Although many factors underlie the damaged kidneys’ adaptation to maintain salt balance, most agree that the key influential forces include the increased load of solute per remaining nephron, increased extracellular fluid (ECF) volume and a variety of paracrine, humoral, and hormonal compounds that modify tubular sodium reabsorption. With the loss of nephrons, total GFR decreases, but hypertrophy of remaining glomeruli and adaptive intrarenal hemodynamic changes cause the filtration rate of remaining nephrons to become supranormal. The ensuing increased filtration of solute by residual
Table 1—Selective Review of Key Perioperative Renal-Electrolyte Disturbances in CRF Patients

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nephrons permits the resulting osmotic diuresis, primarily through urea excretion, to limit sodium reabsorption. While an increasing number of local and systemic compounds have been shown to alter renal salt reabsorption, their integrated physiologic roles remain unsettled.

Clinical Implications

The presence of two normal kidneys affords great latitude in the amount of salt one may ingest. In the face of excess salt exposure, normal kidneys can excrete hundreds of milliequivalents of sodium, whereas in the absence of dietary sodium, salt-free urine may be elaborated. Increasing degrees of renal damage impose limitations on this vital regulatory process.

Salt Overload

Patients with CRF, especially those with primary glomerular disease, are usually hypertensive, and their BP is usually salt sensitive. Depending on age and the duration of renal damage, coronary artery disease is often present. The combination of sustained hypertension and coronary artery disease assures that many CRF patients have abnormal left ventricular anatomy and function. The injudicious use of salt solutions in the perioperative period will conspire with the aforementioned pathophysiologic forces to sensitize patients with advanced renal failure to pulmonary edema.

Salt Loss

The above-described adaptive forces that limit renal salt reabsorption predispose patients to salt loss and hypovolemia when challenged by salt restriction. Within 3 to 4 days of initiating severe salt restriction, subjects with normal renal function can reduce sodium excretion to almost zero. When salt restric-

Perioperative Management of Salt Balance

In patients with established CRF, most clinicians believe that a “well-filled” intravascular space will minimize any decrease in the GFR following general anesthesia and major surgery. Although studies to corroborate this notion are hard to find, Tasker et al., in 1974, prophylactically administered normal saline solution (1 to 4.5 L) to five CRF patients before surgery. Their lack of any postoperative decline in GFR was attributed to their salt exposure (Fig 1). In CRF patients with stable cardiac function and BP, we discontinue treatment with diuretics and liberalize salt intake for 2 or 3 days prior to elective major surgery. An IV infusion of 0.45% saline solution with 5% dextrose is administered at 75 mL/h, beginning the night prior to the day of the operation. BP and cardiac status are closely monitored and the infusion is changed to accommodate their needs. Although open to debate, we urge that pulmonary capillary wedge pressure monitoring be performed intraoperatively and postoperatively in CRF patients whose myocardial function is known to be compromised. The various humoral forces operative during anesthesia and surgery render such patients sensitive to fluid overload. Our unproven assumption is that the protection afforded to the kidney and heart from careful stabilization of intravascular filling outweighs the complications of the procedure.

Patients with end-stage renal disease who receive long-term hemodialysis, in general, should receive dialytic therapy 12 to 24 h prior to elective surgery. Fluid-electrolyte balance can be achieved with this procedure. Serum electrolytes should be reanalyzed 2 or 3 h before surgery. Complications notwithstanding, routine hemodialysis may be resumed within 24 to 36 h postoperatively.

Disordered Water Balance

Pathophysiology

Hypernatremia and hyponatremia are the chemical footprints of disordered water balance. These
changes in sodium concentration, of course, bear no relationship to the state of body sodium content because hyponatremia or hypernatremia may occur in either edematous (sodium overloaded) hypovolemic (sodium deficient) or euvolemic patients.

**Water Metabolism**

Synthesis and secretion of arginine vasopressin (AVP) are unaltered in renal failure. Although residual nephrons usually respond to AVP and generate, excrete, or retain electrolyte-free water (EFW) normally to supranormally, the absolute decrease in GFR and number of residual nephrons limits the maximal capacity to excrete EFW (see below). The solute diuresis per remaining nephron tends to limit the urinary concentrating mechanism (see above); however, the normal thirst mechanism generally prevents the development of hypernatremia. Although most patients with advancing CRF remain in water balance until the serum creatinine concentration approaches 10 mg/dL, it becomes easier for water excess or prolonged thirsting to override the EFW excretion or retention as renal failure progresses. Furosemide and other “loop diuretics” exaggerate disordered water balance by inhibiting both urinary concentrating and diluting processes.

**Electrolyte-Free Water**

One must understand the concept of EFW excretion before a rational fluid prescription can be crafted for any patient, including those with chronic renal insufficiency, receiving IV therapy. Intracellular water (ICW) overload with hyponatremia and ICW deficits with hypernatremia are the only clinically recognizable consequences of disordered water balance. The presence or absence of urea does not chronically influence the distribution of water between intracellular and extracellular spaces. Thus, the level of urea in the plasma and urine should be ignored when assessing the state of water balance. Because urea does contribute to serum and urinary osmolality, the latter measurement is of little or no value in clinical assessment of fluid balance. Only
those factors that influence the transmembranous movement of water are of physiologic importance to the assessment of water balance. *Tonicity* defines the concentration of those solutes that do influence the transcellular distribution of water. Therefore, tonicity is accounted for by sodium and its accompanying anions and glucose in serum and by sodium, potassium, and their anions in urine. In the absence of glucosuria, the urinary glucose content can be ignored. For all intents and purposes, plasma tonicity is defined as twice the serum sodium concentration plus the plasma glucose (mg/L) divided by its molecular weight (180) (if glucose is expressed in mg/dL, its value should be divided by 18). The tonicity of urine is defined as twice the sum of the sodium and potassium concentrations. Unlike plasma, urinary potassium concentration is usually quite high and therefore makes a major contribution to urinary tonicity. Because potassium is confined to the ICW where its concentration approximates 140 mEq/L, it accounts for most of the intracellular tonicity. Thus, large losses of potassium from the body ultimately derive from the intracellular space. This loss causes transient intracellular hypotonicity that is quickly corrected as water moves outwardly into the relatively more hypertonic extracellular space. This translocation of cellular water simultaneously returns intracellular tonicity toward normal while reducing extracellular tonicity. Once the tonicity of both fluid compartments is equal, net water movement ceases. Thus, body potassium balance, in general, and urinary potassium content, specifically, play key roles in assessing fluid balance.

When the serum concentration of sodium and glucose are 140 mEq/L and 90 mg/dL, respectively, plasma tonicity is 285 mOsm/L (ie, $[2 \times 140] + [90 \div 18]$). Excretion of *isotonic urine* requires that twice the sum of the urinary concentrations of sodium and potassium equals 285 mOsm/L. Stated another way, when the sum of the urinary concentrations of sodium and potassium equals 140 mEq/L, the urine is isotonic to normal plasma. When the total urinary cation concentration is $< 140$ mEq/L, the urine is *hypotonic*. If 1 L of urine contains a total of 70 mEq of cation, it would be equivalent to half-normal saline solution. This single liter of urine may be divided into two virtual volumes, 500 mL of water and 500 mL of an isotonic solution of cation. In other terms, mixing 500 mL of water with 500 mL of normal saline solution recreates 1 L of half-normal saline solution. The urinary loss of 500 mL of normal saline solution will shrink the remaining ECF space, without altering the remaining serum sodium concentration. The urinary loss of 500 mL of EFW will raise the concentration of serum sodium by a predictable amount.

**Clinical Implications**

**End-Stage Renal Disease**

Patients receiving regular hemodialysis treatments have an obvious and ready means for adjusting surfeits or deficits in water. It is unusual to find major changes in serum sodium concentration in this setting.

**Antidiuretic Hormone**

Anesthesia, pain, narcotics, hypoxemia, and hypovolemia all may individually, or in combination, stimulate the excessive release of AVP. These stimuli, indigenous to the perioperative period, override the ability of hypotonic hyponatremia to normally suppress AVP release, predisposing patients with functioning kidneys to hyponatremia. So long as fluid administration is carefully crafted to the patient’s needs, the presence of excessive AVP is unimportant.

**Fluid Replacement in Special Settings**

**Hyponatremia**: Patients with hypotonic hyponatremia, who clinically appear to have a normal ECF volume, have an excess of total body water. The absence of edema is, of course, related to two issues: two thirds of the retained water resides within cells, and the mild clinically inapparent volume expansion reestablishes a new steady-state salt balance. Thus, these patients excrete the salt they ingest, albeit, in a relative small volume of fluid. Key issues guiding the repair of hyponatremia include *not raising* the serum sodium concentration by more than 10 to 12 mEq/L/d and enhancing EFW excretion.

When the rate of repair of chronic hyponatremia exceeds 10 to 12 mEq/L/d, demyelinating CNS disorders may occur.$^{10–12}$ The cellular adaptation to chronic hyponatremia entails the slow export of solute from cells. The extracellular transport of solute and water from cells of the CNS diminishes their jeopardy from cerebral edema, but sensitizes them to the crenating effect of rapidly rising serum tonicity. Central pontine myelinolysis is the prototypical disorder engendered by the overly rapid repair of hypotonicity.$^{10–12}$

The nondialytic management of normovolemic, hypotonic hyponatremia generally entails returning the isotonic portion of the increase in urine volume provoked by diuretic therapy. The increased urine volume created by loop diuretics is generally hypo-
tomic to plasma. To initiate therapy, it is reasonable to assume that the urine excreted will be approximately half-normal saline solution in character, ie, the sum of the urinary concentrations of sodium and potassium will equal 70 mEq/L. Thus, each milliliter of urine will be composed of 0.5 mL of normal saline solution and 0.5 mL of EFW. In nonemergent circumstances, one could simply replace each milliliter of such urine with 0.5 mL of normal saline solution. In this way, total body sodium level will remain unchanged while water loss proceeds. If 1 L of urine were excreted in 3 h, this replacement regimen will have induced the net loss of 500 mL of EFW. This protocol may be appropriate for some patients. However, if more rapid water loss is required, the 70 mEq of cation excreted in 3 h could have been replaced with only 140 mL of 3% saline solution (ie, 0.5 mEq/mL). In this fashion, a net of 860 mL of EFW would have been shed, compared with 500 mL. Increases in the dose of loop diuretics can increase urine flow and thereby increase the rate of EFW loss.

Clinical trials are proceeding with agents that block renal AVP receptors. These “aquaretics” should simplify the therapeutic attainment of appropriately rapid rates of EFW loss. Because patients with excessive AVP levels can actively excrete sodium, but not water, they can become hyponatremic during administration of normal saline solution. For example, a patient with AVP hypersecretion who receives 154 mEq of sodium in 1 L of normal saline solution as therapy for hyponatremia may produce only 700 mL of urine. This scenario produces 300 mL of negative EFW, thereby lowering the serum sodium concentration from water retention. Thus, the kidney extracts and excretes the salt, but retains some of the infused water.

Diuretic Therapy of Congestive Heart Failure

Failure to appreciate the need to replace EFW excreted during diuretic therapy for congestive heart failure (CHF) often results in “some good news” and “some bad news.” The “good news” is that the excreted isotonic urine shrinks the ECF, consequently providing symptomatic therapy; the “bad news” is that failure to replace the excreted EFW causes progressive hypernatremia. Many fail to recognize that returning the EFW deficit to a patient with symptomatic, but improving, CHF does little to change intravascular volume. One liter of administered water distributes in the body fluid compartments such that only 83 mL is retained in the intravascular space. In general, until the urinary cations are measured, replacing each milliliter of diuretic-induced urine with 0.5 mL of 5% dextrose in water will permit salutary contraction of the ECF while preventing the development of hypernatremia.

Edematous Patients With Either Hyponatremia or Hypernatremia

Regardless of the serum sodium concentration, the desired effect of diuretic therapy in edematous patients is to rid the body of sodium. The EFW excreted along with the isotonic portion of urine need not be replaced in the hyponatremic patient, whereas it must be replaced in diuresing hypernatremic patients. Hyponatremic patients, of course, require further water restriction whereas hypernatremic patients require additional water administration.

Hyperkalemia

The serum potassium concentration reflects the delicate balance struck between dietary intake, GI absorption, the cation’s transcellular distribution, and finally, its renal excretion. Under steady-state conditions, the urinary excretion of potassium matches milliequivalent for milliequivalent the net GI absorption. The delicacy of this balance is put into perspective when one weighs the fatal consequence of an abrupt 3- to 4-mEq/L increment in serum potassium concentration (ie, the addition of approximately 50 to 60 mEq of potassium ion to the entire ECF) against the reality that 50 to 100 mEq/d of the cation traffic from the GI tract through the ECF and into the urine. One must always remember that 140 mEq/L of intracellular potassium (ie, > 4,000 mEq) continuously diffuses into the ECF only to be recaptured by the ubiquitous membrane-associated sodium-potassium-adenosine triphosphatase (Na-K-ATPase).

The threat of hyperkalemia increases as renal failure progresses. Indeed, 1 to 1.5% of hospitalized patients with CRF develop life-threatening degrees of hyperkalemia. The GI, renal, and hormonal adaptations to advancing renal failure maintain stable normokalemia, despite advanced CRF. Diabetes and certain tubulointerstitial diseases notwithstanding, hyperkalemia generally is not seen until the GFR is < 10 mL/min.

Adaptations

Normally 90% of the 50 to 100 mEq of potassium ingested daily is absorbed without the exertion of any regulatory force. Because potassium is secreted continuously into the intestinal lumen and subsequently reabsorbed, the difference between the oral intake and that in the stool reflects only the minimum amount of potassium absorbed. As renal failure
progresses, the net fractional reabsorption of the cation decreases from 90% to as low as 60 to 70%.

The increase in net colonic potassium secretion is only partially accounted for by aldosterone but may be further stimulated by acidosis, cholinergic agents, and bisacodyl (Dulcolax).

Accordingly, patients with advancing renal failure are generally placed on regimens of low potassium diets, being instructed to avoid salt substitutes, fruits, vegetables, etc. Constipation is also potentially dangerous because it countervails the GI adaptation, acting to enhance the net absorption of potassium. These two therapeutic issues must, of course, be attended to in the perioperative period.

Although the detailed understanding of how the damaged kidney adapts to maintain potassium balance is not fully defined, certain facts are clear. The adaptation occurs in the distal nephron, entails an enhancement of potassium secretion and is only partially dependent on aldosterone. Because these adaptive changes enhance the excretion of potassium, it is unusual to see serious hyperkalemia until oliguria supervenes. Disorders causing diminished renin, angiotensin, aldosterone synthesis or secretion, or renal responsiveness to the mineralocorticoid cause hyperkalemia at lesser degrees of renal failure. Various drugs that limit potassium secretion (trimethoprim/sulfamethoxazole, amiloride, triamterene, spironolactone) predispose to hyperkalemia. Extrarenal volume depletion, by limiting the distal delivery of sodium and fluid, also limits renal potassium excretion and accounts for much of the hyperkalemia seen perioperatively.

Insulin exerts a regulatory effect on the cellular uptake of potassium. Thus, fasting patients with CRF suppress insulin and tend to become hyperkalemic. Allon and colleagues have amply demonstrated this clinically important point in which they showed a time-dependent increase in plasma potassium level in fasted individuals with CRF that was reparable with a dextrose-insulin infusion (Fig 2). Glucose-containing solutions, therefore, should be provided to such patients who are not taking food by mouth.

Therapy of Hyperkalemia

Cardioprotection: Patients with ECG signs of hyperkalemia are at risk for life-threatening arrhythmias and should receive IV calcium. Ten milliliters of 10% calcium gluconate infused during ECG monitoring over 10 min should rapidly stabilize myocardial conduction by raising the threshold potential. This therapy "buys the time" needed for other treatments that are aimed at lowering the serum potassium level to become effective. Repeated doses of calcium may be required. Rapid infusion of calcium may precipitate digoxin-related arrhythmias in those patients taking the glycoside.

Redistribution of Potassium

Sodium Bicarbonate: The evidence now seems clear that alkali administration does not lower increased serum potassium levels in CRF patients. Whether alkali lowers serum potassium level in other conditions is unclear. Because of the negative inotropic and venoconstrictive effects of acidemia, we believe that alkali ought to be administered to those with severe acidemia (pH < 7.20).

Insulin/Glucose: Although peripheral resistance to
insulin’s action on glucose uptake develops in CRF, the hormone’s action on potassium uptake remains intact.19 Insulin stimulates the activity of membrane-bound Na-K-ATPase, thereby enhancing the net movement of extracellular potassium into the intracellular fluid. Rapid increase in plasma glucose concentration increases tonicity and causes intracellular fluid water to move extracellularly, which in turn increases the intracellular potassium concentration. By rendering the electrochemical gradient more favorable for potassium’s egress from the cell, the net uptake of the cation that insulin would have otherwise effected may be reduced. Indeed, some diabetics become frankly hyperkalemic from hyperglycemia.22 Adding 6 to 10 U of regular insulin while administering 25 to 50 g of glucose should be effective.

β2-Adrenergic Stimulation: Enhanced activation of cellular Na-K-ATPase by augmenting intracellular cyclic adenosine monophosphate levels can be utilized to lower the serum potassium concentration therapeutically. Selective β2-agonist therapy increases cellular cyclic adenosine monophosphate levels and drives potassium into cells against their electrochemical gradient. This treatment modality is rapidly and easily delivered. Albuterol as a nebulized aerosol (10 to 20 mg) can reduce elevated serum potassium levels effectively for up to 2 h.19,20 Caution is warranted though, since tachycardia and arrhythmias deriving from this therapy may compromise the cardiovascular status of patients with coronary artery disease. We maintain that β2-agonist therapy should be rendered only after all other measures have proven themselves unsuccessful, especially given the often heightened adrenergic state of the perioperative patient.

HYPERTENSION

Pathophysiology

Hypertension is frequently encountered in CRF patients undergoing surgical procedures. Numerous factors may promote the development or worsening of hypertension in the perioperative venue in either previously normotensive or hypertensive subjects. Most renal failure patients become hypertensive and many have had primary hypertension prior to their renal disease.23 Hypervolemia and augmented sympathetic nervous system discharge likely represent the two primary factors underlying the secondary hypertension of CRF.

Anxiety and Medications

Preoperative anxiety, pain, and discontinuation of antihypertensive therapy may lead to severe hypertension, thereby worsening angina and/or diminishing left ventricular output such that pulmonary edema may ensue.24 However, diuretic medications represent the sole exception to the rule of continuing treatment with the BP medications throughout the perioperative period, including on the day of surgery. Diuretic therapy should be discontinued 2 or 3 days before a scheduled operation. However, in some patients with the nephrotic syndrome or CHF, diuretics may be necessary for clinical stabilization of their cardiopulmonary status. Medications that may cause or aggravate hypertension should always be sought in the medical record and treatment with them should be discontinued prior to surgery, so long as “rebound hypertension” is not a complication of the offending agent(s). Such agents include non-steroidal anti-inflammatory drugs, antihistamines, and decongestants.25

Operative Factors

Within the operative suite, the BP may rise during endotracheal intubation or manipulation of the oropharynx.26 Similarly, manipulations of the urethra or rectum may also worsen hypertension. During lower-limb surgery, hypertension may develop after the application of tourniquets.27

Cardiac Surgery

In cardiac surgery that involves cardiopulmonary bypass, hypertension is a common occurrence. Hypertension may occur before, during, or after bypass. BP elevations may also occur in the absence of any precipitating factor following coronary artery bypass surgery, valvular replacement, and resection of aortic coarctation, presumably because of exaggerated sympathetic activity.28

Other Factors

Within the recovery room, anxiety, pain, hypoxemia, and hypercarbia may singly, or in combination, engender BP elevation. Hypoglycemia may also elevate the BP in patients with more advanced renal failure who are particularly susceptible to this complication, especially those who require insulin and are routinely fasted prior to surgery.29

Perioperative Management of Hypertension

Relief of anxiety and pain is paramount in prevention of perioperative hypertension. Short-acting analesics and anxiolytics may reap great rewards in this regard, preventing the addition of more drugs to the already complex pharmacopoeia taken by many renal failure patients. All patients should continue...
taking their BP medications until the time of surgery, even when they are allowed nothing by mouth. In this regard, treatment with medications should be resumed at the earliest opportunity in the postoperative period.30 Following surgery, some medications may be unable to be given, having no oral analog. In such cases, an IV or transdermal preparation of the same class should be used. For example, abrupt discontinuance of centrally acting agents such as α-methyldopa and clonidine and the β-blocker propranolol, because of an inability to deliver them orally, might precipitate hypertension in an individual with a previously well-controlled condition.31 This complication could be sidestepped by simply applying the transdermal delivery system for clonidine, with several days overlap between the “patch” and the oral compound.30 For patients with preexistent or highly suspected cardiovascular disease and hypertension undergoing noncardiac surgery, appropriate risk stratification and clinical assessment must be made.32,33 In these patients, treatment with perioperative β-blocker, atenolol, has proved beneficial, reducing the 6-month mortality with minimal side effects.34 However, specific recommendations regarding digitalis, nitrates, and calcium channel blockers (CCBs) cannot be made, except that short-acting CCB therapy should not be employed to rapidly lower the BP. The risk of perioperative bleeding may be increased by CCBs.35 When hypertension occurs, despite the best efforts to prevent it, and oral medications cannot be used, IV treatment with labetalol, esmolol, enalaprilat, nicardipine, or nitroprusside is recommended.36–40 For patients who experience tachycardia or myocardial ischemia with hypertension, β-blockade is the preferred choice. However, in the setting of CHF and hypertension, angiotensin-converting enzyme inhibitors are preferred. Nitroprusside is usually reserved for patients with medical-surgical crises.39 Renal and hepatic failure patients are more susceptible to thiocyanate and cyanide toxic reactions. Such cases, an IV or transdermal preparation of the same class should be used. For example, abrupt discontinuance of centrally acting agents such as α-methyldopa and clonidine and the β-blocker propranolol, because of an inability to deliver them orally, might precipitate hypertension in an individual with a previously well-controlled condition.31 This complication could be sidestepped by simply applying the transdermal delivery system for clonidine, with several days overlap between the “patch” and the oral compound.30 For patients with preexistent or highly suspected cardiovascular disease and hypertension undergoing noncardiac surgery, appropriate risk stratification and clinical assessment must be made.32,33 In these patients, treatment with perioperative β-blocker, atenolol, has proved beneficial, reducing the 6-month mortality with minimal side effects.34 However, specific recommendations regarding digitalis, nitrates, and calcium channel blockers (CCBs) cannot be made, except that short-acting CCB therapy should not be employed to rapidly lower the BP. The risk of perioperative bleeding may be increased by CCBs.35 When hypertension occurs, despite the best efforts to prevent it, and oral medications cannot be used, IV treatment with labetalol, esmolol, enalaprilat, nicardipine, or nitroprusside is recommended.36–40 For patients who experience tachycardia or myocardial ischemia with hypertension, β-blockade is the preferred choice. However, in the setting of CHF and hypertension, angiotensin-converting enzyme inhibitors are preferred. Nitroprusside is usually reserved for patients with medical-surgical crises.39 Renal and hepatic failure patients are more susceptible to thiocyanate and cyanide toxic reactions. Such patients require even greater vigilance when receiving this agent and prophylactic therapy may be in order. In addition, rarely, but dosestrously, the compound may induce a “coronary steal” syndrome. Lastly, optimization of the ECF volume with calculated diuresis in the hypervolemic patient should be accomplished prior to surgery.

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