Case Studies in Critical Care of the Surgical Patient

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Table of Contents

Introduction ................................................. 2
Case 1 Presentation ................................. 2
Case 2 Presentation ................................. 5
Case 3 Presentation ................................. 7
Summary Points .............................................. 10
References ................................................. 10
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INTRODUCTION

In the United States, 20% of all hospital stays require an intensive care unit admission (ICU). There are nearly 6000 ICUs in the United States with nearly 60,000 ICU beds. Dedicated surgical ICUs represent 12% of all total ICUs, while 65% of U.S. ICUs are mixed medical-surgical units. The most common reason patients require ICU care is organ failure. For surgical patients not suffering organ failure, ICU admission for close monitoring commonly follows operations that are high risk or are performed in high-risk patients. Slightly less than 5% of all ICU admissions are trauma patients.

ICU care is expensive and complicated, and strategies are being developed to reduce the morbidity and mortality of patients requiring intensive care. For surgical patients, goal-directed care and rapid recognition and treatment of potential complications have resulted in increased long-term survival benefits. ICU care in surgical patients has also led to increases in survival by avoidance of postoperative complications. This manual uses case presentations to highlight and review major concepts in intensive care of the surgical patient, including acute kidney injury, acute respiratory failure, and nutrition.

CASE 1 PRESENTATION

PRESENTATION

A 42-year-old man was involved in a high-speed motor vehicle collision 48 hours prior. He underwent an emergent exploratory laparotomy with splenectomy and colon resection at the time of initial presentation. He remains critically ill, requiring mechanical ventilation. He initially received several units of blood products and has received maintenance intravenous fluids in the last 24 hours. His urine output has been 0.2 mL/kg/hour for the last 12 hours.

What are the potential causes for low urine output?

ACUTE KIDNEY INJURY

Renal dysfunction is common in ICU patients, occurring in about 5% to 20% of all patients, and is termed acute kidney injury (AKI). AKI is defined as an abrupt decline in the ability of the kidney to filtrate waste products (glomerular filtration rate, GFR) in the blood, resulting in acute azotemia. The etiology of AKI in the critically ill patient includes prerenal azotemia, intrinsic renal parenchymal disease, or postrenal azotemia. The common pathway of prerenal azotemia is decreased renal perfusion resulting from a decrease in the extracellular volume from excessive volume losses (hemorrhage), decreased effective circulating volume (heart failure), or excessive third-spacing in the interstitial spaces from conditions such as pancreatitis or intra-abdominal sepsis. Prerenal azotemia is generally considered reversible if the causative etiology is addressed in a timely manner. If not addressed, it can progress to a type of intrinsic renal disease that affects the tubules known as acute tubular necrosis (ATN), which is the most common type of AKI occurring in hospital settings. Intrinsic renal parenchymal disease can be preexisting in surgical ICU patients or develop as a result of profound volume depletion leading to ATN. The etiology of postrenal azotemia is generally an obstructive process, and in patients who have recently undergone intra-abdominal operation this should be excluded as a cause of postoperative renal dysfunction. Obstructive causes can include ureteral injury or urethral/prostate obstruction. Hemorrhage in the bladder can lead to clot formation in a trauma patient, causing secondary obstruction of the urethra.

What is the workup for suspected AKI in a critically ill surgical patient?

All patients should have a Foley catheter placed to monitor hourly urine output and relieve any urethral or prostate obstruction. If concern exists for postrenal azotemia causes, a renal ultrasound should be performed to rule out hydronephrosis. If a Foley catheter is placed, it should be connected to a bladder pressure monitoring system to assess bladder pressure and compliance. Urodynamic studies should be performed to rule out intrinsic bladder disease.
catheter is in place already, it should be flushed to ensure proper functioning. If the patient suffers from a condition that potentially could lead to abdominal compartment syndrome, bladder pressures should be measured. Abdominal compartment syndrome leads to AKI by causing a significant decrease in blood flow to the kidney (prerenal cause) due to the elevated pressure in the abdomen, resulting in the development of renal ischemia and subsequent ATN.

Serum electrolytes, plasma osmolality, urine electrolytes, urine osmolality, blood urea nitrogen (BUN), serum creatinine, and urinalysis should be obtained. The plasma and urine electrolyte values are utilized to calculate the fractional excretion of sodium (FENa) to determine if the cause of AKI is likely to be prerenal (FENa <1%) or intrinsic from ATN (FENa >1%). The FENa is calculated as the ratio of sodium excreted (urinary sodium × volume) to the sodium filtered (serum sodium × GFR), or (Urine sodium/serum sodium) divided by (Urine creatinine/serum creatinine) × 100 (Figure 1). Prerenal azotemia will result in a high urine osmolality (>350 mOsm/L), low urine sodium (<20 mEq/L), and an elevated BUN-to-serum-creatinine ratio (>20). Urine from patients with intrinsic renal disease, including ATN, will have casts with cellular elements, high urine sodium (>30 mEq/L), normal urine osmolality (300 mOsm/L), and a BUN-to-serum-creatinine ratio around 10. It is important to be aware that several conditions causing ATN can make the FENa difficult to interpret, including contrast nephropathy, hepatic cirrhosis, rhabdomyolysis, sepsis, and acute glomerulonephritis. Each of these conditions results in a low FENa. Diuretic administration will falsely elevate the FENa. Under these conditions, measurement of fractional excretion of urea is more accurate.

CASE 1 CONTINUED

The patient is found to have a serum potassium of 5.9 mEq/L, serum creatinine of 4 mg/dL, BUN of 80 mg/dL, and urine sodium of 40 mEq/L. The calculated FENa is suggestive of ATN. A Foley catheter is in place and is functioning appropriately. A renal ultrasound is unremarkable and an electrocardiogram is normal without peaked T waves. The patient’s urine output has decreased further and is now less than 0.2 mL/kg/min. His central venous pressure (CVP) is 22 mm Hg and he has had increasing oxygen needs on the ventilator over the last 6 hours, now requiring a fraction of inspired oxygen (FiO₂) of 80%.

$$FENa = \frac{\text{Urine}_{Na} \times \text{Serum}_{Cr}}{\text{Serum}_{Na} \times \text{Urine}_{Cr}} \times 100$$

Figure 1. Calculation of fractional excretion of sodium (FENa). Cr = creatinine; Na = sodium.

- What are the indications for renal replacement therapy and what are the types of therapy available?

RENAI REPLACEMENT THERAPY

Most patients with AKI in the ICU will recover with supportive treatment and will not require renal replacement therapy (RRT). For patients in which the etiology of AKI is postobstructive, relief of the obstruction, such as with a Foley catheter or nephrostomy tube, results in brisk and dramatic improvement in renal function. For prerenal causes, volume resuscitation to restore renal perfusion is the first line of therapy. If the patient has not progressed to ATN, adequate resuscitation generally improves renal function if the patient is volume deficient. For patients who have ATN, most will not require RRT unless they develop complications of AKI, including volume overload compromising respiratory status, hyperkalemia, significant refractory acidosis, or uremia (Table 1).

Hyperkalemia is a common complication of AKI that must be addressed in a timely manner. Treatment is generally not required until the serum potassium level rises above 6 mEq/L or peaked T waves are seen on electrocardiogram. First-line therapy is to promote urinary excretion of potassium via use of volume expansion or loop diuretics (such as furosemide) while driving potassium intracellularly using glucose (25–50 g) and insulin (10 units regular insulin intravenously). The increased excretion takes hours for onset of action, while the insulin and glucose work within minutes. Driving the potassium intracellularly, however, is only a temporizing measure that generally lasts a few hours. Inhaled albuterol can also be used to drive the potassium intracellularly and has a slightly longer onset of action than insulin/glucose, but the therapeutic effect lasts a bit longer as well. Intravenous calcium should be reserved for patients with hyperkalemia-induced heart block. It is also a temporizing measure with a rapid onset of action but a short duration of effect (< 30 minutes).

For patients meeting the indications for RRT, the goal is to restore an appropriate intravascular volume status and remove excess solutes for control of azote-
Critical Care of the Surgical Patient

Continuous RRT is available in many different forms, with the most common options being slow continuous ultrafiltration (SCUF), continuous venovenous hemofiltration (CVVH), or continuous venovenous hemodiafiltration (CVVHDF). Isolated ultrafiltration (SCUF) involves the removal of primarily volume alone, and solute removal is dependent only upon convection, resulting in relatively poor control of azotemia. SCUF does not have dialysate flow. Conversely, CVVH utilizes a highly permeable hemodialysis membrane to allow high ultrafiltrate rates, and solute is removed via a convective transport. Because the ultrafiltrate rate is high, volume has to be replaced continuously via the circuit to avoid hypotension. With CVVH, large volumes can be removed and titrated hourly while still achieving good solute removal. CVVHDF uses dialysate flow to improve the clearance of solute.

- What is the prognosis for AKI?

Mortality is fairly high for critically ill patients with AKI, approaching 50% to 80%.5,7,8 This high mortality reflects that the patients often have multisystem failure, and the presence of AKI often serves as a marker of overall illness.5,9 Nonoliguric renal failure has a better prognosis, with survival rates as high as 75%, while patients with oliguric renal failure fare worse, with mortal-
ity approaching 70%. Converting an oliguric patient to a nonoliguric patient through the use of diuretics has not been shown to lead to improvement in survival. If a patient recovers from AKI, return of renal function occurs fairly rapidly, with most survivors reaching dialysis independence by 30 days.\(^8\) Patients with preexisting renal insufficiency, extremes of age, and certain precipitating events (burns, sepsis, severe ischemia) tend to do worse overall and are the least likely to recover function if they survive.\(^8,9\) Patients with AKI secondary to medications, contrast dye, and prerenal causes addressed quickly tend to be the most likely to survive and recover function.

**CASE 1 CONCLUSION**

The physician places a temporary dialysis catheter in the patient’s right internal jugular vein. CVVH is initiated, with removal of volume and correction of his metabolic acidosis and electrolytes. Over the next 6 days, his overall condition improves. His need for dialysis decreases and his renal function improves. He is discharged to a rehabilitation facility on postinjury day 15.

**CASE 2 PRESENTATION**

**INITIAL PRESENTATION**

A 21-year-old helmeted man is involved in a motorcycle crash and sustains bilateral femur fractures and a grade III liver laceration. He arrives to the hospital hemodynamically stable and requiring 2 L of oxygen via nasal cannula. He is admitted to the surgical ICU for additional care. Over the course of the next several hours, he begins to have a rising oxygen requirement. He is currently receiving 6 L of oxygen via nasal cannula to maintain an oxygen saturation of 92%.

- **What further workup should the patient have?**
- **What is the differential diagnosis?**

The patient should have a chest x-ray performed and an arterial blood gas measurement. The clinical scenario suggests that the patient will likely have a large alveolar-arterial oxygen gradient and is developing progressive respiratory failure. Young, otherwise healthy patients who develop respiratory failure in the first 24 hours following their traumatic injury are usually suffering from acute lung injury (ALI), acute respiratory distress syndrome (ARDS), or direct pulmonary injury due to significant pulmonary contusions. Less common causes include fat emboli syndrome (seen in patients with long-bone fractures), pulmonary edema from massive volume resuscitation, and massive transfusion.\(^11\) By far, ALI and ARDS are the most common cause of respiratory failure and affect more than 200,000 people per year, with a mortality rate approaching 30% to 40%.\(^11\) ALI is a less severe form of lung injury than ARDS. The 2 syndromes share a common pathophysiology and represent a continuum of a single disease process.

**CASE 2 CONTINUED**

Over the next 12 hours of care, the patient has increasing oxygen requirements, ultimately requiring intubation. A chest x-ray is performed after intubation and shows the development of diffuse bilateral pulmonary infiltrates.

- **What is the definition of ALI and ARDS?**

**ACUTE LUNG INJURY AND ACUTE RESPIRATORY DISTRESS SYNDROME**

The American-European Consensus Conference (AECC) has defined ARDS as an acute process of noncardiogenic pulmonary edema leading to dyspnea and hypoxemia that occurs in patients without a chronic pulmonary disease. Diagnosis of both ALI and ARDS requires the presence of (1) acute onset, (2) unilateral or bilateral infiltrates, and (3) pulmonary capillary wedge pressure ≤ 18 mm Hg or no clinical sign of right atrial hypertension. ARDS is the more severe condition, as it is defined by the presence of a Pa\(\text{O}_2/Fi\(\text{O}_2\) ratio of ≤ 200, and ALI is the less severe form with a Pa\(\text{O}_2/Fi\(\text{O}_2\) ratio ≤ 300. Patients can suffer from ALI or ARDS without progressing to requiring mechanical ventilation, although most ultimately do. There are many causes of ALI and ARDS, including trauma, burns, sepsis, drug reactions, cardiopulmonary bypass, transfusion, and aspiration.

- **What is the pathophysiology of ARDS and ALI?**

The common pathway of the development of ARDS is increased pulmonary microvascular permeability resulting in an influx of protein-rich fluid into the lungs. It occurs in 2 phases, with the early phase being termed the “exudative” phase, or the peak of the pulmonary edema seen in the lungs. This leads to hyaline membrane formation in the alveolar space and an increase in inflammation within the lung parenchyma. This second phase is known as the “proliferative” phase of ARDS. It is common for the second phase to result in an increase in ventilatory dead space leading to worsened perfusion-to-ventilation mismatch.
Thus, the patient's hypoxemia becomes increasingly refractory to increased oxygen supplementation, and the minute ventilation requirements to achieve adequate gas exchange rise substantially.

**CASE 2 CONTINUED**

The patient is placed onto a mechanical ventilator receiving 6 mL/kg of tidal volume, with a respiratory rate of 26 breaths/min, positive end-expiratory pressure (PEEP) of 14 cm H2O, and FiO2 of 80%. His last arterial blood gas analysis results are significant for a pH of 7.24, Pco2 of 60 mm Hg, Paco2 of 56 mm Hg, and bicarbonate level of 25 mEq/L.

- **How is ARDS treated and what is lung protective ventilation?**

The treatment for ARDS is largely supportive. If an identified insult is present, early treatment of the inciting condition is important. Unfortunately, ARDS commonly still progresses. The mainstay of treatment is lung protective ventilation where patients are ventilated at lower tidal volumes and higher respiratory rates to avoid the secondary lung injury that occurs from overdistention of the alveoli (with resultant volutrauma). ARDS is often a heterogeneous lung disease, with areas of the lung that are relatively normal, areas with mild to moderate atelectasis, and areas with dense consolidation in the most dependent zones of the lung. Thus, as larger tidal volumes are delivered to the lung, the alveoli in the more normal lung are subjected to volutrauma without achieving the intended benefit of improving aeration of the densely consolidated portions of the lung. In 2000, a landmark randomized trial was published by the ARDS Network (ARDSnet) comparing patients ventilated with traditional tidal volumes (12 mL/kg of predicted body weight [PBW]) to patients ventilated with lower tidal volumes of 6 mL/kg/PBW. The study included 861 patients and demonstrated a 22% lower in-hospital mortality in patients treated with lower tidal volumes. This strategy became known as lung protective ventilation.

Lung protective ventilation includes the concept of permissive hypercarpnea, where patients are allowed to develop a respiratory acidosis in exchange for allowing mechanical ventilation at lower tidal volumes. This prevents the alveolar overdistention that has historically been seen in the treatment of ARDS. The tidal volume utilized is generally 6 mL/kg of PBW. PBW is determined based upon height measured in inches (males: 50 + 2.3[height in inches – 60]; females: 45.5 + 2.3[height in inches – 60]). Minute ventilation (tidal volume in liters × respiratory rate) is maintained by increasing the patient’s respiratory rate. PEEP and FiO2 are adjusted by a protocol to maintain oxygen saturation between 88% and 95% and achieve an FiO2 less than 60% as soon as possible. The FiO2 is lowered to avoid toxicity from delivering oxygen above FiO2 60%. Patients with cardiac ischemia or elevated intracranial pressure are not generally candidates for lung protective ventilation as hypercarbia increases heart rate, with subsequently increased myocardial oxygen demand. Hypercarbia also worsens intracranial hypertension. Maintaining euvolemia and avoiding intravascular volume overload may also play a role in the treatment of ARDS. The pathophysiology of ARDS involves the development of significant pulmonary edema at even relatively normal volume status. Theoretically, restricted volume may reduce the hydrostatic pressure in the lungs, and therefore lessen pulmonary edema formation if instituted early in the course of ARDS. A randomized clinical trial demonstrated that those patients kept at a targeted CVP below 4 mm Hg (compared with CVP 10–14 mm Hg) had a shorter interval of mechanical ventilation, but survival was not improved. Despite no survival benefit, most clinicians believe that a restrictive volume strategy may be helpful in the care of these patients. Further investigation is ongoing through an ARDSnet trial.

- **What are the rescue therapies for severe refractory ARDS?**

**ARDS RESCUE THERAPIES**

For patients with severe ARDS, lung protective ventilation alone may not be enough to overcome the profound hypoxemia associated with ARDS. For those patients, additional strategies have been studied with varying supportive evidence for their utility. Prone positioning can improve oxygenation, but whether it improves survival remains controversial. Prone positioning appears to improve oxygenation by shifting segments of lung with atelectasis from dependent maximal blood flow areas to nondependent areas. This decreases ventilation-to-perfusion mismatch and allows more effective gas exchange. The largest survival benefit for prone positioning has been shown in studies where it is instituted early.

The use of inhaled nitric oxide has theoretical benefits in the care of patients with ARDS, but results in clinical trials have not been as promising as anticipated. Nitric oxide selectively vasodilates pulmonary arterioles and capillaries where gas ex-
change occurs. Thus, blood flow is increased in areas of ventilated lung and away from the nonventilated areas. In this fashion, inhaled nitric oxide reduces the amount of shunting and improves the gas exchange. Nitric oxide is expensive and, given the paucity of clinical effectiveness data, it should be reserved for patients with isolated respiratory failure with severe hypoxemia.17

Inverse ratio ventilation allows more time in the inspiratory phase and less time in the expiratory phase. Typical mechanical ventilation involves a 2 to 3 times longer interval in the expiratory phase than the inspiratory phase. From a theoretical standpoint, equalizing the inspiratory and expiratory intervals allows a longer time period of the respiratory cycle to be spent in inspiration, recruiting areas of the lung that are relatively resistant to other recruitment strategies. Any improvement in oxygenation seen with this strategy may take place over many hours. Because this is not the typical way natural respiration occurs, patients often require significant sedation to tolerate this method. Also, as the expiratory phase is shortened, patients may not have time to adequately expel the tidal volume delivered and will be at risk of developing auto-PEEP. Auto-PEEP occurs secondary to inspired gas that remains at the level of alveoli at end-expiration. If a patient has inadequate exhalation time, this gas remains, leading to increased positive pressure in the lungs, making it more difficult to create a pressure gradient that is needed to exchange gas effectively. Thus, the patient must generate a higher negative inspiratory pressure to overcome this increased positive pressure, and the work of breathing rises.

High frequency ventilation (HFOV) and extracorporeal membrane oxygenation (ECMO) both have theoretical benefits for hypoxic respiratory failure, but definitive evidence of survival differences has not been demonstrated in clinical trials of these techniques.18 HFOV involves ventilating patients with a special ventilator that achieves very small tidal volumes at high frequencies. The tidal volumes are as small as 1 mL/kg and the frequencies range from 60 to 300 breaths per minute. Patients must be deeply sedated and often pharmacologically paralyzed in order to tolerate this method. It is primarily reserved as a last effort salvage therapy. ECMO involves removing blood, passing it through a membrane oxygenator, and returning it to the circulation (arterial or venous). Older studies did not support its use in refractory ARDS, but there has been renewed interest in recent years. To date, there are no modern day randomized clinical trials.

Given that the pathophysiology of ARDS appears to involve significant inflammation, especially in the proliferative phase of the illness, the use of corticosteroids to blunt the inflammatory response has theoretical benefits. Despite extensive study, there is no definitive conclusion as to the benefit versus harm of routine steroid administration in the treatment of ARDS. Several recent meta-analyses have yielded conflicting results, and most clinicians do not routinely utilize steroids.19–21 A randomized, controlled trial failed to show a benefit for patients when steroids were given during the proliferative phase of the illness.22

**CASE 2 CONCLUSION**

The patient requires mechanical ventilation and support of oxygenation. Despite PEEP and positive pressure ventilation, his PaO2/FiO2 ratio decreases to less than 100, then gradually improves. He undergoes tracheostomy on post-injury day 10. He is discharged to a rehabilitation facility on post-injury day 22.

**CASE 3 PRESENTATION**

**INITIAL PRESENTATION**

A 55-year-old woman presents with succus draining from a recent midline laparotomy wound. She underwent an exploratory laparotomy with proximal small bowel anastomosis for a closed loop bowel obstruction approximately 2 weeks ago. Her immediate postoperative hospital course was uneventful except for a wound infection. Upon radiologic evaluation, a computed tomography scan demonstrates a fistula from the proximal jejunum (Figure 2). There is no associated abscess or uncontrolled spill of contrast. When she presents, she is hypotensive and tachycardic, with an elevated BUN of 46 mg/dL and a serum creatinine of 3 mg/dL. She is admitted to the ICU for further care.

- **What is a fistula?**

A fistula can be defined as an abnormal passage between 2 epithelialized surfaces. The fistula present in this patient is likely enterocutaneous or colocutaneous in nature. About 75% to 85% of all enterocutaneous fistulas are associated with previous operations, with additional causes including malignancy, radiation, and inflammatory bowel disease.23

Defining and classifying a fistula is an important step in understanding the natural course of the fistula as well as potentially predicting whether or not a fistula
will close spontaneously. A fistula can be classified physiologically (based on output), anatomically, or by etiology (Table 2). In general, high output (>500 mL/day) fistulas are more likely to be small bowel in origin and less likely to close spontaneously.23,24

**What are the FRIENDS of a fistula?**

The FRIENDS of a fistula alludes to a commonly taught mnemonic that is used to help recall attributes of a fistula that make spontaneous closure much less likely: F is for foreign body, R for radiation, I for infection or inflammation, E for epithelialized track, N for neoplasia, D for distal obstruction, and S for short, wide tract.

**After stabilization and resuscitation, what are the important factors to consider for the patient’s care?**

Patients with proximal small bowel fistulas often suffer electrolyte abnormalities (most commonly hypokalemia) and dehydration from the loss of proximal gastrointestinal contents from the fistula.10 If the gastrointestinal losses are substantial, patients can develop metabolic acidosis from bicarbonate losses. If a fistula is not contained, patients may present with sepsis. If sepsis is present, control of fistula effluent and abscess drainage must be obtained in order to convert an intra-abdominal leak into a controlled fistula.25 Steps to decrease the fistula output and achieve local control should also be undertaken. For proximal fistulas, patients are given nothing by mouth, nasogastric tubes are placed, and patients are given nutrition intravenously to decrease the fistula outputs. The skin around the cutaneous opening of a fistula must also be protected to prevent excoriation and breakdown from exposure to intestinal contents. A sump-type catheter system may be useful in order to control fistula effluent.

**CASE 3 CONTINUED**

The patient is resuscitated, her electrolytes are corrected, and nutrition therapy is initiated with total parenteral nutrition (TPN) on hospital day 2.

**What role does nutrition play in healing of an enterocutaneous fistula?**

Nutrition is crucial for the treatment and supportive care of an enterocutaneous fistula. Enteral nutrition has several advantages over TPN, including immunologic, and is the preferred method of nutrition. Unfortunately, it is often not possible for most patients suffering from a proximal small bowel fistula to receive enteral nutrition due to resultant increased fistula output or lack of access to the gastrointestinal tract distal to the fistula for feeding.25 Critically ill patients who receive enteral nutrition have a reduced incidence of infectious complications compared with those who receive parenteral nutrition.27 Regardless of the method of nutrition, patients with proximal fistulas have high metabolic needs. Most patients should initially receive 20 kcal/kg per day of carbohydrates and fat and 0.8 g/kg per day of protein.26 Ultimately, patients may require as many as 30 kcal/kg of carbohydrates and fat and 1.5 to 2.5 g/kg per day of protein.28

**CASE 3 CONTINUED**

After several days of parenteral nutrition, the patient’s nutritional parameters remain suboptimal despite being fed an appropriate number of kcal/kg based upon predictive calculations.

**What are additional methods to determine a patient’s metabolic needs more accurately?**

An estimation of nutritional needs can be determined by analyzing nitrogen balance by collecting a 24-hour urine sample and measuring the total urine urea nitrogen. Each gram of urea contains 467 mg of nitrogen. The amount of nitrogen consumed can be calculated by estimating 1 gram of nitrogen absorbed for each 6.25 g of protein intake. The goal is to achieve a positive nitrogen balance of 2 grams per day.

A more accurate method of estimating a patient’s caloric needs is the metabolic cart study, also known as indirect calorimetry. With this method, exhaled gas...
C r i t i c a l  C a r e  o f  t h e  S u r g i c a l  P a t i e n t

is analyzed to determine a respiratory quotient (RQ) which reflects the composition of the diet. If a patient is being primarily fed carbohydrates, the RQ is 1. If a patient has a diet consisting of mostly lipids, the RQ is 0.7. If the patient has a diet rich in protein, the RQ is near 0.8. For patients who are appropriately fed, the RQ should be around 0.9. Patients who are overfed will have a RQ above 1.

What are the major considerations for the use of TPN?

Because of the high osmotic load of the solution, parental nutrition requires a dedicated port of a central venous catheter. Caloric needs are generally estimated as being between 25 to 30 kcal/kg/day for critically ill patients. The components of TPN include dextrose, amino acids, electrolytes, trace elements, and minerals. Lipids are also commonly given either added to the TPN or infused separately. The majority of calories are given as dextrose and lipids, with dextrose representing the bulk of the non-protein calories. Dextrose is commonly given in 15-47% concentrations. Dextrose should not exceed more than 7.2 g/kg per day, as the body's maximum rate of dextrose oxidation is 5 mg/kg per minute. Lipids generally come in 10% or 20% emulsions, yielding 1.1 kcal/mL or 2.2 kcal/mL, respectively. At least 3% of nonprotein calories must be given as lipids in order to avoid essential fatty acid deficiency. A maximum of 60% of nonprotein calories can be given as lipid. Typically, less than 30% of the nonprotein calories are given as lipids as higher concentrations of lipids have an immunosuppressive effect. Amino acids include all the essential and nonessential amino acids except arginine and glutamine; they are given in concentrations between 5% and 15% and represent the nitrogen intake of the patient. The typical protein intake per day should be between 1 and 2 g/kg.

Trace elements and vitamins have been shown in clinical trials to confer a survival advantage.27

- What laboratory values should be routinely monitored for patients receiving TPN?

When TPN is first initiated, glucose should be monitored every 6 hours, and serum electrolytes, calcium, magnesium, and phosphate should be measured daily. Once the serum parameters reach a steady state, the values are typically measured twice weekly along with nutritional parameters including C-reactive protein, pre-albumin, retinol-binding protein, serum beta-2 transferrin, albumin, and liver function tests. For patients receiving lipids, triglycerides should also be measured weekly. Serum transferrin levels have been shown to correlate with spontaneous fistula closure.30

CASE 3 CONCLUSION

The patient is given increased protein in her TPN and her nutritional parameters improve. Her wound is controlled with local care. Her output remains in excess of 1000 mL/day. Given the continued high output, the physician determines that it is unlikely that the fistula will close spontaneously. TPN is continued for an additional 12 weeks, after which the patient is prepared for operation. At operation, extensive intra-abdominal adhesions are found, which are lysed. A small bowel resection encompassing the fistulized section of bowel is performed, and an anastomosis is completed. The abdomen is closed with the aid of a biological mesh. The patient regains bowel function, TPN is discontinued, and she is started on enteral nutrition. She is discharged home in good condition on postoperative day 12.

Table 2. Classification of Enterocutaneous Fistulas

<table>
<thead>
<tr>
<th>Category</th>
<th>Type of Fistula</th>
<th>Uses of Information</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anatomy</td>
<td>Internal versus external</td>
<td>May suggest cause of fistula</td>
</tr>
<tr>
<td></td>
<td>Anatomic course</td>
<td>Assists in planning operative closure</td>
</tr>
<tr>
<td></td>
<td>Output (mL/day)</td>
<td>May predict spontaneous closure</td>
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<tr>
<td></td>
<td>Low (&lt;200)</td>
<td>Predicts mortality</td>
</tr>
<tr>
<td></td>
<td>Moderate (200–500)</td>
<td>Predicts metabolic derangements</td>
</tr>
<tr>
<td></td>
<td>High (&gt;500)</td>
<td></td>
</tr>
<tr>
<td>Etiology</td>
<td>By underlying disease process</td>
<td>Predicts closure rate</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Predicts mortality</td>
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**SUMMARY POINTS**

- Causes of acute kidney injury (AKI) in the critically ill patient include prerenal azotemia (reduced renal perfusion due to decreased extracellular volume from excessive volume losses, decreased effective circulating volume, or excessive third-spacing in the interstitial spaces), intrinsic renal parenchymal disease (preexisting or caused by profound volume depletion), or postrenal azotemia (usually an obstructive process).

- The fractional excretion of sodium (FENa), based on urine and plasma electrolyte values, can be used to determine if AKI is due to prerenal causes (FENa, <1%) or intrinsic from acute tubular necrosis (FENa >1%).

- Indications for renal replacement therapy in AKI include metabolic abnormalities, acidosis, anuria/oliguria, and fluid overload.

- Diagnosis of acute lung injury (ALI) and acute respiratory distress syndrome (ARDS) requires the presence of (1) acute onset, (2) unilateral or bilateral infiltrates, and (3) pulmonary capillary wedge pressure ≤18 mm Hg or no clinical sign of right atrial hypertension. ARDS, the more severe condition, is defined by the presence of a PaO$_2$/FiO$_2$ ratio of ≤200, while ALI is the less severe form with a PaO$_2$/FiO$_2$ ratio of ≤300.

- Lung protective ventilation in treatment of ARDS allows a respiratory acidosis to develop in exchange for allowing mechanical ventilation at lower tidal volumes. The tidal volume utilized is generally 6 mL/kg of predicted body weight. Positive end-expiratory pressure and FiO$_2$ are adjusted to maintain oxygen saturation between 88% and 95% and achieve an FiO$_2$ less than 60% as quickly as possible.

- Enteral nutrition is preferable to total parenteral nutrition in the treatment of patients with enterocutaneous fistula but is not possible in most patients with a proximal small bowel fistula due to the resultant increased fistula output or lack of access to the gastrointestinal tract.

- Caloric needs for critically ill patients are generally estimated as being between 25 and 30 kcal/kg/day. The majority of calories are given as dextrose and lipids, with dextrose representing the bulk of the non-protein calories.

- Dextrose should not exceed more than 7.2 g/kg per day as the body’s maximum rate of dextrose oxidation is 5 mg/kg per minute. In most cases, less than 30% of the nonprotein calories are given as lipids. Typical daily protein intake is between 1 and 2 g/kg.

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12. Ventilation with lower tidal volumes as compared with tradi-


