# PRE-OP AND POST-OP CARE

## Preoperative Assessment

### Cardiac Risk

**Ejection Fraction** under 35% (normal is 55%) poses prohibitive cardiac risk for noncardiac operations. Incidence of perioperative MI is very high, and morality for such an event is between 55 and 90%.

**Goldman's index of cardiac risk,** which dates from 1977, is no longer the preferred method of assessing cardiac risk. Functional status, based on the ability to cope with life's demands, is more commonly used now. But Goldman's remains useful for listing all the findings that predict trouble. They are (in descending order of importance) jugular venous distension, recent myocardial infarction, premature ventricular contractions, or any rhythm other than sinus, age over 70, emergency surgery, aortic valvular stenosis, poor medical condition, and surgery within the chest or abdomen. The examination will give particular attention to the high-risk situations that intervention can improve.

**Jugular venous distention**, which indicates the presence of congestive heart failure, is the worst single finding predicting high cardiac risk. If at all possible, treatment with ACE inhibitors, beta-blockers, digitalis, and diuretics should precede surgery.

**Recent transmural or subendocardial MI** is the next worst predictor of cardiac complications. Operative mortality within 3 months of the infarct is 40%, but it drops to 6% after 6 months. Thus, deferring surgery until then is the best course of action. If surgery is imperative sooner, admission to the intensive care unit (ICU) the day before is recommended to "optimize cardiac variables."

### Pulmonary Risk

**Smoking** is by far the most common cause of increased pulmonary risk, and the problem is compromised ventilation (high $PCO_2$, low forced expiratory volume in 1 second [$FEV_1$], rather than compromised oxygenation. The smoking history or the presence of chronic obstructive pulmonary disease (COPD) should lead to evaluation. Start with $FEV_1$, and if it is abnormal, follow with blood gases. Cessation of smoking for 8 weeks and intensive respiratory therapy (physical therapy, expectorants, incentive spirometry, humidified air) should precede surgery.

## Hepatic Risk

Two clinical findings and three laboratory values are used to predict **operative mortality** in patients with liver disease: encephalopathy, ascites, serum albumin, prothrombin time (INR), and bilirubin (only as it reflects hepatocyte function). The presence and severity of these factors can be combined in a variety of ways; the current favorite system is Child class, in which class A has 10% mortality, class B has 30%, and class C has 80%. But specific numbers are misleading because so many other factors influence the outcome. Suffice it to say that a patient in coma with huge ascites, albumin below 2, INR twice normal, and bilirubin above 4 could not survive a haircut, much less an operation.

## Nutritional Risk

**Severe nutritional depletion** is identified by loss of 20% of body weight over a couple of months, serum albumin below 3, anergy to skin antigens, or serum transferrin level of less than 200 mg/dL (or a combination of the above). Operative risk is multiplied manyfold in those circumstances. Surprisingly, as few as 4 or 5 days of pre-operative nutritional support (preferably via the gut) can make a big difference, and 7 to 10 days would be optimal it the surgery can be deferred that long.

## Metabolic Risk

**Diabetic coma** is an absolute contraindication to surgery. Rehydration, return of urinary output, and a least partial correction of the acidosis and hyperglycemia have to be achieved before surgery. (If the indication for surgery is a septic process, complete correction of all variables will be impossible as long as the septic process is present.)

# Postoperative Complications

## Fever

**Malignant hyperthermia** develops shortly after the onset of the anesthetic (halothane or succinylcholine). Temperature exceeds 104 F. Metabolic acidosis and hypercalcemia also occur. A family history may exist. Treat with IV dantrolene, 100% oxygen, correction of the acidosis, and cooling blankets. Watch for development of myoglobinuria.

**Bacteremia** is seen within 30-45 minutes of invasive procedures (instrumentation of the urinary tract is a classic example), and there are chills and temperature spike to or exceeding 104 F. Do blood cultures times three, and start empiric antibiotics.

Although rare, severe wound pain and very high fever within hours of surgery should alert you to the possibility of gas gangrene in the surgical wound.

**Postoperative (PO) fever in the usual range (101 – 103F)** is caused (sequentially in time) by atelectasis, pneumonia, urinary tract infection, deep venous thrombophlebitis, wound infection, or deep abscess.

**Atelectasis** is the most common source of post-op fever on the first PO day. Rule out the other causes listed above, listen to the lungs, do chest x-ray, improve ventilation (deep breathing and coughing, postural drainage, incentive spirometry). The ultimate therapy if needed is bronchoscopy.

**Pneumonia** will happen in about 3 days if atelectasis is not resolved. Fever will persist. Chest x-ray will show infiltrates. Do sputum cultures and treat with appropriate antibiotics.

**Urinary tract infection** typically produces fever starting on PO day 3. Work up with urinalysis and urinary cultures. Treat with appropriate antibiotics.

**Deep thrombophlebitis** typically produces fever starting on PO day 5 or thereabouts. Doppler studies of deep leg and pelvic veins is the best diagnostic modality (physical exam is worthless). Anticoagulate with heparin, transitioning later to warfarin.

**Wound infection** typically begins to produce fever on PO day 7. Physical exam will show erythema, warmth, and tenderness. Treat with antibiotics if there is only cellulitis; open and drain the would if an abscess is present. When these two cannot be easily distinguished clinically, sonogram is diagnostic.

**Deep abscesses** (like subphrenic, pelvic, or subhepatic) start producing fever around PO days 10-15. CT scan of the appropriate body cavity is diagnostic. Percutaneous radiologically guided drainage is therapeutic.

## Chest Pain

**Perioperative myocardial infarction** may occur during the operation (triggered most commonly by hypotension), in which case it is detected by the EKG monitor (ST depression, T-wave flattening). When it happens post-op, it is typically within the first 2 to 3 post-op days, showing up as chest pain only in one-third of the cases, and with the complications of the MI in the rest. The most reliable diagnostic test is troponin. Mortality (50-90%) greatly exceeds that of MI not associated with surgery. Treatment is directed at the complications. Clot busters cannot be used in the perioperative setting, but emergency angioplasty and coronary stenting may be used.

**Pulmonary embolus (PE)** typically happens around PO Day 7 in elderly and/or immobilized patients. The pain is pleuritic, of sudden onset, and is accompanied by shortness of breath. The patient is anxious, diaphoretic, and tachycardic, with prominent distended veins in the neck and forehead (a low CVP virtually excludes the diagnosis). Arterial blood gases show hpoxemia and hypocapnia. The standard diagnostic test is a spiral CT with intravenous dye, commonly referred to as a CT Angio. After confirming the diagnosis, start treatment with heparinization. Add an inferior vena cava filter (Greenfield) if Pes recur during anticoagulation or if anticoagulation is contraindicated.

Prevention of thromboembolism will in turn prevent PE. Sequential compression devices can be used on anyone who does not have a lower extremity fracture. In high-risk patients, anticoagulation is indicated. Risk factors include age >40, pelvic or leg fractures, venous injury, femoral venous catheter, and anticipated prolonged immobilization.

## Other Pulmonary Complications

**Aspiration** is a distinct hazard I awake intubations in combative patients with a full stomach. It can be lethal right away or lead to chemical injury of the tracheobronchial tree and subsequent pulmonary failure, or secondary pneumonia. Prevention includes NPO and antacids before induction. Therapy starts with lavage and removal of acid and particulate matter (with the help of bronchoscopy), followed by bronchodilators and respiratory support.

**Intraoperative tension pneumothorax** can develop in patients with traumatized lungs (recent blunt trauma with punctures by broken ribs) once they are subjected to positive-pressure breathing. They become progressively more difficult to “bag,” BP steadily declines, and CVP steadily rises. If the abdomen is open, quick decompression can be achieved through the diaphragm. If not, a needle can be inserted through the anterior chest wall into the pleural space (sneaking in under the drapes). Formal chest tube has to be placed later.

## Disorientation/Coma

**Hypoxia** is the first thing that has to be suspected when a post-op patient gets confused and disoriented. It may be secondary to sepsis. Check blood gases, provide respiratory support.

**Adult respiratory distress syndrome (ARDS)** is seen in patients with a stormy, complicated post-op course, often complicated by sepsis as the precipitating event. There are bilateral pulmonary infiltrates and hypoxia, with no evidence of congestive heart failure. The centerpiece of therapy is positive end-expiratory pressure (PEEP), taking care not to use excessive volume. Excessive ventilatory volumes have been shown to result in barotrauma. As source of sepsis must be sought and corrected. Extracorporeal membrane oxygenation (ECMO), done at a specialized center, may help the adult with ARDS who is not responding to PEEP. The main complication is intracranial bleeding, which can be minimized by using a venovenous connection to hook up the patient to the machine.

**Delirium tremens** (DTs) is very common in the alcoholic whose drinking is suddenly interrupted by surgery. About the second or third PO day these patients get confused, have hallucinations, and become combative. Intravenous benzodiazepines are the standard therapy, but alcohol is also effective. It can be intravenous (5% alcohol in 5% dextrose), or for those on oral intake we can actually prescribe their favorite drink.

**Hyponatremia,** if quickly induced by liberal administration of sodium-free IV fluids (like D5W) in a postoperative patient with high levels of antidiuretic hormone (ADH; triggered by the response to trauma), will produce confusion, convulsions, and eventually coma and often death ("water intoxication"). Chart review confirms large fluid intake, quick weight gain, and rapidly lowering serum sodium concentration (in a matter of hours). The problem is best prevented by including sodium in the IV fluids. Once it happens, therapy is controversial, and mortality is very high; young women are particularly vulnerable. Most clinicians use small amounts of hypertonic saline (aliquots of 100 mL of 5% or 500 mL of 3%), perhaps add osmotic diuretics.

**Hypernatremia** can also be a source of confusion, lethargy, and potentially coma – if rapidly induced by large, unreplaced water loss. Surgical damage to the posterior pituitary with unrecognized diabetes insipidus is a good example. Unrecognized osmotic diuresis can also do it. Chart review will show large, unreplaced urinary output, rapid weight loss, and rapidly rising serum sodium concentration. Rapid replacement of the fluid deficit is needed, but to "cushion" the impact on tonicity many prefer to use D5 ½ or D5 1/3 normal saline (NS), rather than D5W.

**Ammonium intoxication** is a common source of coma in the cirrhotic patient with bleeding esophageal varices who undergoes a portocaval shunt.

## Urinary Complications

**Postoperative urinary retention** is extremely common, particularly after surgery in the lower abdomen, pelvis, perineum, or groin. The patient feels the need to void but cannot do it. In-and-out bladder catheterization should be done at 6 hours post-op if no spontaneous voiding has occurred. Indwelling (Foley) catheter is indicated at the second (some say third) consecutive catheterization.

**Zero urinary output** typically is caused by a mechanical problem, rather than a biologic one. Look for plugged or kinked catheter.

**Low urinary output** (less than 0.5 mL/kg/h) in the presence of normal perfusing pressure (i.e., not because of shock) represents either fluid deficit or acute renal failure. A low-tech diagnostic test is a fluid challenge; a bolus of 500 mL of IV fluid infused over 10 to 20 minutes. Dehydrated patients will respond with a temporary increase in urinary output; those in renal failure will not. A more elegant way to decide is to measure urinary sodium: It will be less than 10 or 20 mEq/L in the dehydrated patient with good kidneys, while it will exceed 40 mEq/L in cases of renal failure. As even more sophisticated way to express the same is to determine "fractional excretion of sodium," which in renal failure exceeds 1.

## Abdominal Distention

**Paralytic ileus** is to be expected in the first few days after abdominal surgery. Bowel sounds are absent, and there is no passage of gas. There may be mild distension, but there is no pain. Paralytic ileus is prolonged by hypokalemia.

**Early mechanical bowel obstruction** because of adhesions can happen during the postoperative period. What was probably assumed to be paralytic ileus not resolving after 5, 6, or 7 days is most likely an early mechanical bowel obstruction. X-rays will show dilated loops of small bowel and air-fluid levels. Diagnosis is confirmed with an abdominal CT scan that demonstrates a transition point between proximal dilated bowel and distal collapsed bowel at the site of the obstruction. Surgical intervention is needed to correct the problem.

**Ogilvie syndrome** is poorly understood (but very common) condition that could be described as a "paralytic ileus of the colon." It does not follow abdominal surgery, but classically is seen in elderly sedentary patients (Alzheimer, nursing home) who have become further immobilized owing to surgery elsewhere (broken hip, prostatic surgery). They develop large abdominal distention (tense but not tender), and imaging studies show a massively dilated colon. After fluid and electrolyte correction, the safest thing to do is perform a colonoscopy, suck out all the air, and place a long rectal tube, IV neostigmine stimulates colonic motility, but this drug is best avoided. It has lots of side effects and is lethal if inadvertently give to someone whose colon is actually obstructed.

## Is It Small Bowel or Colon?

If you look at vertical sections of a CT scan of the abdomen that shows dilated bowel, how can you tell if you are looking at small bowel or colon? There are three clues:

1. *Location*. The colon hugs the outside boundaries of the image, while the small bowel tends to be in the center of it.
2. *Size* is very, very helpful. Make a circle between your index finger and thumb and hold it right in front of your face: That is about as big as small bowel can get. Now make a circle with both hands, with index touching index, and thumb touching thumb: The colon can attain that size.
3. *Fine details*. The edges of the colon have small indentations (haustral markings), whereas the small bowel has little lines going across ("stacked coins").

## What Are Air-Fluid Levels?

Everyone has air and fluid in the GI tract. But the churning motion of normal peristalsis makes a foam out of those. If the small bowel is obstructed, it eventually gets tired of trying to push the stuff, so that the liquid goes to the bottom and the air stays at the top. A horizontal line divides these, which can be seen in images taken with the patient in the upright position.

## Wound

**Wound dehiscence** is typically seen around the fifth post-op day after open laparotomy. The wound looks intact, but large amounts of pink, "salmon-colored" fluid are noted to be soaking the dressings (it is peritoneal fluid). The wound has to be taped securely, the abdomen bound, and mobilization and coughing done with great care, while arrangements are made for prompt reoperation to prevent evisceration now or ventral hernia later on.

**Evisceration** is a catastrophic complication of wound dehiscence, where the skin itself opens up and the abdominal contents rush out. It typically happens when the patient (who may not have been recognized as having a dehiscence) coughs, strains, or gets out of bed. The patient must be kept in bed, and the bowel must be covered with large sterile dressings soaked with warm saline. Emergency abdominal closure is required.

**Wound infections** are typically seen around the seventh post-op day. (This postoperative complication was described under the heading of fever.)

**Fistulas of the GI tract** are recognized because bowel contents leak out through a wound or drain site. They may harm the patient in a number of ways. If they do not empty directly and completely to the outside, but leak into a "cesspool" that then leaks out, the problem will be sepsis (requiring complete drainage). If they drain freely (patient is afebrile, with no signs of peritoneal irritation), there are three potential problems: fluid and electrolyte loss, nutritional depletion, and erosion and digestion of the belly wall. These problems are related to location and volume of the fistula: nonexistent in the distal colon, present but manageable in low-volume (up to 200-300 mL/day) high G"I fistulas (stomach, duodenum, upper jejunum), and daunting in high-volume (several liters per day) fistulas high in the GI tract. Fluid and electrolyte replacement, nutritional support (preferably elemental diets delivered beyond the fistula), and compulsive protection of the abdominal wall (suction tubes, "ostomy" bags) are done to keep the patient alive until nature heals the fistula. Nature will do so if there is no foreign body, radiation damage, inflammatory bowel disease, epithelialization, neoplastic tissue, distal obstruction, or steroid therapy (the "F.R.I.E.N.D.S." mnemonic) to prevent it.

## INTRAVENOUS FLUID THERAPY

If surgical patients cannot eat and drink, we have to put them on intravenous fluids. Those always include what we call "maintenance" IV therapy. Sometimes we have to do more, providing "replacement" and/or "correction." Let's consider these IV therapies one at a time.

In principle, "maintenance" is the equivalent of what a patient would have eaten and drunk if they were not sick. In practice, we include only three things for a few days: water, sodium, and potassium. Plain water cannot be infused, because it would lyse the red blood cells. To make it isotonic and safe, we typically add dextrose at 5% concentration (the famous "D5W"). A standard adult would receive an infusion of about 2-3 liters of D5W a day – which does double duty, because it also minimizes the protein breakdown of complete starvation.

For sodium and potassium, the amount to remember is around 100 mEq per day of each, but with a twist: Absent congestive heart failure or liver disease, we have enormous flexibility with sodium. It should not be zero and should not exceed a few hundred milliequivalents, but anything between 50 and 250 is fair game; precision is not required. Normal saline has 154 mEq/L of sodium, and 5% dextrose in one-half normal saline is often used as the vehicle for sodium. For potassium, the guiding principle is to link the dose to caloric intake. Total parenteral nutrition would require more than 100 mEq per day of potassium – but for just a few days of semistarvation, 40-60 mEq per day suffices. We typically use ampules of potassium chloride as the source.

By far the most common maintenance IV fluid order reads: "Dextrose 5 percent in one-half normal saline, with 20 milliequivalents of potassium chloride per liter, to run in at 125 cc per hour." But if you are too lazy to spell all that, the nurses will understand "D5 ½ NS with 20 mEq of KCl/L, to run in at 125 cc/hr."

Let's move to "replacement." Here we are talking about abnormal fluid losses that have been measured and require infusion, cc per cc, of an appropriate fluid. The most common such losses are from the GI tract; vomiting, nasogastric tube output, fistulas, massive diarrhea, malfunctioning ileostomies, and so on. If the aggregate loss is a small fraction of a patient's basic maintenance volume, we can

replace today what was recorded yesterday, then provide tomorrow what we measured today, and so on. But if the abnormal losses approach or exceed *half of daily basic needs,* more frequent replacement is prudent; every 8 hours, or even every 4.

The composition of the replacement fluid should mimic what was lost. For instance, GI fluids are isotonic with plasma and rich in potassium.

- Fluids from beyond the pylorus are alkaline. Ringer lactate with a little additional KCl (10-20 mEq/L) will do nicely.
- Pure gastric juice is very acidic. Half-normal saline with a little more potassium (20-40 mEq/L) is an appropriate replacement.

Either fluid should include 5% dextrose. A little extra sugar always helps.

If maintenance and replacement fluids are properly managed from day one, there will be no need for "correction." But if not, the patient will develop a "fluid and electrolyte disorder" that we will have to fix. The paragraphs that follow provide a few examples of these.

## Fluids and Electrolytes

**Hypernatremia** invariably means that the patient has lost water (or other hypotonic fluids) and has developed hypertonicity. Every 3mEq/L that the serum sodium concentration is above 140 represents roughly 1 L of water lost. If the problem happens slowly (several days), the brain will adapt, and the only clinical manifestations will be those of volume depletion. Therapy requires volume repletion, but it must be done so that thew volume is corrected rapidly (in a matter of hours), while the tonicity is only gently "nudged" in the right direction (and goes back to normal in a matter of days). This is achieved by using D5 ½ NS rather than D5W. Hypernatremia of rapid development (such as in osmotic diuresis or diabetes insipidus) will produce CNS symptoms (the brain has not had time to adapt), and correction can be safely done with more diluted fluid (D5 1/3 NS, or even D5W).

**Hyponatremia** means that water has been retained and hypotonicity has developed, but there are two different scenarios (easily distinguishable by the clinical circumstances). In one, a patient who starts with normal fluid volume adds to it by retaining water because of the presence of inappropriate amounts of ADH (for instance, post-op water intoxication, or inappropriate ADH secreted by tumors). In the other, a patient who is losing large amounts of isotonic fluids (typically from the GI tract) is forced to retain water if he has not received appropriate replacement with isotonic fluids. Rapidly developing hyponatremia (water intoxication) produces CNS symptoms (the brain has not had time to adapt) and requires careful use of hypertonic saline (3% or 5%). In slowly developing hyponatremia from inappropriate ADH, the brain has time to adapt, and therapy should be water restriction. In the case of the hypovolemic, dehydrated patient losing GI fluids and forced to retain water, volume restoration with isotonic fluids (NS or Ringer lactate) will provide prompt correction of the hypovolemia and allow the body to unload the retained water and return the tonicity to normal slowly and safely. The choice between the two is determined by acid-base status. Use normal saline only if there is alkalosis. Ringer lactate is better for acidotic patients and those whose pH is normal.

**Hypokalemia** develops slowly (days) when potassium is lost from the GI tract (all GI fluids have lots of K) or in the urine (because of loop diuretics or too much aldosterone), and it is not replaced. Hypokalemia develops very rapidly (hours) when potassium moves into the cells, most notably when diabetic ketoacidosis is corrected. Therapy is obviously potassium replacement. Remember that the safe "speed limit" of IV potassium administration is 10 mEq/h (which can be exceeded only if you know what you are doing).

**Hyperkalemia** will occur slowly if the kidney cannot excrete potassium (renal failure, aldosterone antagonists), and it will occur rapidly if potassium is being dumped from the cells int the blood (crushing injuries, dead tissue, acidosis), The ultimate therapy for hyperkalemia is hemodialysis, but while waiting for it we can help by "pushing potassium into the cells" (50% dextrose and insulin), sucking it out of the GI tract (NG suction, exchange resins), or neutralizing its effect on the cellular membrane (IV calcium). The latter provides the quickest protection.

## pH or Nanoequivalents?

The pH scale was invented in 1909 by a Danish biochemist by the name of Sorensen. He was not a physician. He dealt with the enormous scale of hydrogen ion concentrations in nature, which range from 1 to 0.000 000 000 000 01. Converting that to a scale from 1 to 14 was no mean feat.

But we medical practitioners do not have to contend with such a vast spectrum in the blood of patients. We should never have adopted the pH scale. It is counterintuitive. First, it is upside-down: More hydrogen ions = lower pH. Second, it is not linear: It's a logarithm. Decimal points in the pH do not convey the true magnitude of $H^+$ changes in the blood, which can go from 20 to 120.

We are comfortable measuring blood electrolytes in milliequivalents. Hydrogen ion is just another electrolyte, albeit one we have in tiny amounts. Moving the decimal point to the right give us a suitable unit: nanoequivalents. The normal is 40. The minimum concentration compatible with life is 20 (severe alkalosis), and the maximum is 120 (horrible acidosis). Easy to grasp. Intuitive. Linear. Perfect.

But there is more. As you know, acid-base balance has two components, metabolic and respiratory. We measure the latter with $pCO_2$, and by a happy coincidence the normal happens to be 40. Normal amount of hydrogen ion in the blood: 40 nanoeqivalents. Normal partial pressure of $CO_2$: 40. Those two values are reported by the lab with the blood gases. When you see 40 and 40, you know everything is okay. The other report that comes with the electrolytes will confirm the bicarbonate is also in good shape.

When a patient has a pure respiratory problem, those two numbers we get with the blood gases move pretty much in parallel. If they do not, you are instantly alerted to the fact that there is a metabolic problem as well. Either both systems are deranged, or one is compensating for the other. Your clinical skills – history and physical – then take over and solve the puzzle. Neat.

If this topic bores you, just turn the page and keep using pH. No big deal. If it intrigues you, I suggest you read a little book published by Lippincott, Williams & Wilkins called *Fluids and Electrolytes in the Surgical Patient.* It had five editions in the U.S. and was translated and published in Spanish, Italian, and Greek. I highly recommend it. But I am biased. I wrote that thing.

**Metabolic acidosis** can occur from excessive production of fixed acids (diabetic ketoacidosis, lactic acidosis, low-flow states), from loss of buffers (loss of bicarbonate-rich fluids from the GI tract), or from inability of the kidney to eliminate fixed acids (renal failure). In all three cases the blood pH is low (<7.4), the serum bicarbonate is low (<25), and there is a base deficit. When abnormal acids are piling up in the blood, there is also an "anion gap" (serum sodium exceeds by more than 10 or 15 the sum of chloride and bicarbonate), which does not exist when the problem is loss of buffers. The treatment in all cases must be directed at the underlying cause. If it is loss of bicarbonate, we should replace bicarbonate or use bicarbonate precursors, such as lactate or acetate.

In all cases of long-standing acidosis, of whichever etiology, renal loss of $K^+$ leads to a deficit that does not become obvious until the acidosis is corrected. Thus, we must be prepared to replace potassium as part of our therapy.

**Metabolic alkalosis** occurs from loss of acid gastric juice or from excessive administration of bicarbonate (or precursors). There is a high blood pH (>7.4), high serum bicarbonate (>25), and a base excess. In most cases, an abundant intake of KCI (between 5 to 10 mEq/h) will allow the kidney to correct the problem. Only rarely is ammonium chloride or 0.1 N HCI needed.

Respiratory acidosis or alkalosis results from impaired ventilation (acidosis) or abnormal hyperventilation (alkalosis). They are recognized by abnormal $PCO_2$ (low in alkalosis, high in acidosis) in conjunction with the abnormal pH of the blood. The therapy must be directed at improving ventilation (in acidosis) or reducing it (in alkalosis).

## Nutritional Support of the Surgical Patient

As noted earlier in this chapter, intravenous fluid therapy is required in the patient who cannot eat and drink. There is medical agreement that a patient's inability to eat and drink for just a few days would not justify the complexity of complete nutritional support. But if 10 or more days of starvation are anticipated or have taken place, the time comes to provide it.

Before we go any further, let me point out that nutrition, while beneficial, is never an emergency that commands priority over other therapeutic needs. For instance, patients who are in and out of shock, or have electrolyte imbalances, or are septic, are not candidates for nutritional support. We deliver calories and nutrients only when everything else is on an even keel and the only remaining problem is the inability to eat.

How we give nutritional support depends on the condition of the GI tract. If it cannot work at all, we choose **total parenteral nutrition (TPN)**. This uses fat emulsions as the main caloric source, along with modest amounts of amino acids, sugar, and all the other nutrients. Peripheral TPN takes advantage of the fact that regular veins can tolerate a slightly higher-than-normal osmolar load, not to exceed 1,000 mOsm/L.

Much better than relying on fat would be to identify the patients who might be able to tolerate **enteral feedings of elemental nutrients.**

As a by-product of programs that send humans into space, the medical profession has acquired very useful products – the first one was Vivonex – for feeding patients who cannot digest food but can absorb predigested nutrients. We now have a collection of those elemental diets. For a couple of reasons, they are designed to be dripped through tubes that have been positioned beyond the stomach. The first reason is trivial: their taste. Amino acids have the flavor of rotten fish. Adding other flavors did not help, and astronauts rejected them – which is why NASA gave those solutions to us. Delivery by tube eliminates that problem. The second reason is much more important: Vomiting and aspiration can kill you in a great big hurry. You cannot put anything in the stomach if the GI tract is not 100% normal; delivering amino acids through a tube that goes well beyond the stomach avoids this risk. The tubes are either fed in until they reach their destination or placed at the time of surgery (a feeding catheter jejunostomy). But one more warning: Verify radiologically that *tubes are where they are supposed to be*. Tubes can be regurgitated, tubes can migrate. Never trust a tube.

Elemental diets have two very important advantages over parenteral nutrition: a beneficial trophic effect for the GI mucosa, and a cost of about $10 a day – much cheaper than TPN.

**Radiology for the Surgical Consumer**

I am not a radiologist, but as a clinician I use their products. This brief overview of what is available, the indications for each study, and their relative cost will help you determine which way to go in many examination questions.

**Plain x-ray**. About $20. X-rays are widely available but handicapped by several limitations: They cannot penetrate the skull; they use ionizing radiation; and they superimpose the densities of all the tissues they go through. They can only see black, white, and a few shades of gray. In many applications x-rays have already been superseded by CT scans, but they are still useful where black and white suffice: broken or dislocated bones, and chest x-rays.

**Sonogram.** About $150. A transmitter aims sound beams at the target, and echoes are read back. The readings are used to create images (sonogram, ultrasound, or echo – different names for the same thing) or to measure flow by sing the Doppler principle. If you get both, we call the "duplex" scanning. Sonograms are best at the interface of solid and liquid (perfect for gallstones or urinary tract obstruction, and an echocardiogram is our standard way to look at morphological abnormalities of the heart). The enemy of sonograms is air, which makes the echoes harder to read. Sonograms have many limitations – they cannot go through bones, and they are very much operator dependent, both to conduct and to read. (To nonspecialists, the images look like "black-and-white TV on the blink.") But there is no safer way to look inside a human being: Nothing needs to be injected. There is no radiation. The only way to kill a patient with an ultrasound is to hit him over the head with the sonogram machine.

**CT scan.** About $350. Computed tomography (CT) scans are glorified x-rays. They use the same basic ionizing radiation, but now aimed from many different angles and put together by a computer. They are capable of penetrating the skull and showing black, white and hundreds of shades of gray. CT scans have taken over for head trauma, the cervical spine, the abdomen, kidney stones, and many other applications where x-rays were used in the past.

**MRI**. About $1,500. Much more expensive and not as widely available as the CT, magnetic resonance imaging (MRI) gives much more detail. MRI was first known as "magnetic nuclear resonance," which patients rejected outright. (When patients heard the word "nuclear," they thought about atomic bombs. The technology was renamed.) From a consumer perspective, MRI is just a "glorified CT" – although in reality it is a completely different technology. Magnetic pulses line up elements within the nuclei of the cells, producing exquisitely detailed images. MRI is the first choice for looking at soft, mushy targets in the body: spinal cord, brain tumors, the structures inside the knee, herniated disks, soft tissue sarcomas. MRI is not useful to guide interventional studies, however, because any ferrous metal in the vicinity will go flying across the room when the machine is turned on.

**PET scan.** About $6,500. A positron emission tomography (PET) scan gives a picture of metabolic activity. It was first used by neuroscientists to see what parts of the brain were active when volunteers were happy, or sad, or angry (or horny). Now PET is used primarily in the workup of lung cancer, to determine whether enlarged mediastinal nodes are old scars from back when the patient dug tunnels for a living or metastases rapidly growing.