Hemodynamics

Preload Responsiveness

Preload responsiveness means that a fluid bolus of 500 mL will increase the cardiac output by 15%. Of course, being preload-responsive doesn't necessarily mean you need more preload—chances are that you are preload-responsive right now, but you don't need an infusion of fluids. So, a good rule of thumb to follow is:

- Preload-responsive + signs of hypoperfusion = isotonic fluid bolus
- Preload-unresponsive + signs of hypoperfusion = vasopressors
- Preload-responsive but no signs of hypoperfusion = do nothing

Things that indicate preload-responsiveness:

- Arterial line stroke volume variation ≥ 10% or pulse pressure variation ≥ 13%[1,2]. The best way to measure this is with the LidCO device. It will display cardiac index, SVV, and PPV. A femoral arterial line is probably better, but a radial line will also work.
- Ultrasound IVC diameter variation ≥ 13%[2]. Find the IVC about 2 cm below the right atrium. Measure the diameter and variation using M-mode. With ventilator breaths, the IVC will get bigger during inspiration and smaller during expiration. Take the maximum diameter and minimum diameter. Divide the *difference (Dmax – Dmin)* by the *mean diameter ([Dmax + Dmin]/2)*. Multiply the result by 100 to express it as a percentage.
- Increase in cardiac output with passive leg raising to 30 degrees. The patient should be supine. Elevate the legs to 30 degrees for 30-60 seconds. See what the cardiac output does—this is best done with the LidCO. The passive leg raise is the equivalent of a 300 mL blood bolus, so if the cardiac output increases it indicates the patient is preload responsive. This can be done in spontaneously breathing, or even non-intubated, patients.

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1 In a patient breathing passively on assist-control ventilation. These are not reliable in patients who are not on a ventilator or who are breathing on pressure-support ventilation.

2 Unreliable if the cardiac rhythm is irregular

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Things that don't correlate with preload-responsiveness:

- Central venous pressure—no better than a coin flip.
- Pulmonary artery occlusion pressure—a coin flip plus about 5%. Still lousy.
- Right ventricular end diastolic volume index—this depends on multiple calculations, each with a significant margin of error. Not that helpful.
- Static measurement of the IVC diameter—the dynamic measurement in a ventilated patient has much better positive predictive value. Static measurement of the IVC correlates with the CVP, but we already know that the CVP is worthless.

Don't forget the low-tech but high-yield technique of repeated bedside examinations. Feel the toes, look at the neck veins, and gauge the urine output. Serial lactate measurements or SvO_2 monitoring can be helpful.

Fluid Therapy

Remember that isotonic fluids are designed as resuscitation fluids, and as such should be given as boluses to restore the intravascular volume (and no more). While hypovolemia is bad, salt-water drowning is not much better! **There is little need for salt-containing "maintenance fluids" in the ICU.** The best maintenance fluids are given in the form of enteral nutrition. The continuous infusion of salt-containing fluids over days in the ICU leads to pulmonary and interstitial edema, and should be avoided except in certain cases (rhabdomyolysis, correction of electrolyte disturbances).

0.9% saline, also known as (ab)normal saline, is the most commonly used fluid in the MICU. That may not be a good thing. An increasing body of evidence shows that the resulting hyperchloremia and metabolic acidosis (if chloride rises, bicarbonate must go down) can have a harmful effect. The pH in a bag of normal saline is 5.0, after all. There's no increase in mortality, but a chloride-liberal strategy is associated with more renal failure and more need for dialysis and CRRT.

Lactated Ringer's solution is a better option for routine use. There is potassium in it, but only 4 mEq/L. Unless you're giving large amounts to someone who has dialysis-dependent renal failure, it shouldn't be a problem. Using LR helps avoid the metabolic acidosis and hyperchloremia with large-volume resuscitation. The lactate is converted to bicarbonate in the liver, and the chloride content is more physiologic (109 mEq/L, vs. 154 mEq/L in 0.9% saline). If anyone says that normal saline is a "medical" fluid and LR is a "surgical" fluid, don't listen to that person anymore. Patients are patients, physiology is physiology, and chemistry is chemistry.

Colloids (Hextend, Dextran, albumin) haven't been proven to be any better than crystalloid

(NS and LR), but they are significantly more expensive. 25% albumin can be useful when used in conjunction with diuretics in patients with volume overload. Albumin also can be used as a rapid volume expander when you need to get a lot of fluid in quickly. Patients with spontaneous bacterial peritonitis do improve when given 1.0 g/kg of albumin on day 1 and 1.5 g/kg on day 3 (divided doses), likely by preventing acute renal failure from sepsis. For the most part, though, albumin and other colloids don't have much if any advantage over crystalloid and shouldn't be used routinely.

Vasopressors

Vasopressors are used to raise the blood pressure via peripheral vasoconstriction. Inotropes increase cardiac output by either increasing the heart rate or the stroke volume (more squeeze). Some agents have both vasopressor and inotropic properties; others have one or the other, but not both.

- Epinephrine is God's own pressor, with both α and β activity. It gets all sympathetic receptors when given in pharmacologic doses and will increase peripheral vascular tone and cardiac output. The problems with epinephrine are related to its potency—a small increase in dose can really increase the heart rate, blood pressure, or cause significant peripheral vasoconstriction. Think of it as hitting the gas on a Ferrari. It works, but it can be difficult to titrate without running into problems. Nevertheless, it's a good drug for really sick patients. The dose is 1-20 mcg/min.

- Norepinephrine: primarily a vasopressor (α receptors), but it does have some β-1 inotropy to increase cardiac output. This is the drug of choice in septic shock, where there is both a distributive and cardiogenic component. It's also useful for profound cardiogenic shock and shock from a massive pulmonary embolism. The dose is 1-30 mcg/min.

- Dopamine: primarily an inotrope at lower doses (β-1, β-2), with more vasopressor activity (α) at higher doses. It can be useful for cardiogenic shock but tachydysrhythmias can be a problem. The dose is 5-20 mcg/kg/min.

- Dobutamine: an inotrope, not a vasopressor. Action is on the β-1 and β-2 receptors. This causes increased heart rate, contractility, and vasodilation. This is good for CHF with mild hypotension, but if the SBP is < 90, dopamine is probably a better choice. The dose is 5-40 mcg/kg/min.

- Milrinone: another inotrope that works via phosphodiesterase inhibition. It's useful for decompensated CHF and sometimes causes less tachycardia than dobutamine. The dose is 0.375-0.750 mcg/kg/min.

- Phenylephrine: a pure α vasoconstrictor. It works best in situations of pure vasoplegia with preserved cardiac function, like spinal shock. It's also used when tachydysrhythmias occur with norepinephrine. Most patients with septic shock have

some cardiac dysfunction, so phenylephrine isn't the best choice in that setting. The dose is 40-180 mcg/min.

- Vasopressin: a pure vasoconstrictor via the peripheral vasopressin receptors. In septic shock, endogenous vasopressin gets depleted quickly. The resulting vasopressin deficiency makes the patient relatively insensitive to the effects of catecholamines, both endogenous and exogenous. Replacing at a constant rate (0.03-0.04 units/minute) will help the catecholamines be more effective.

The most important thing to remember is that all of these drugs have side effects and should be titrated to get the minimal acceptable result—in most cases, there's no need to get a mean arterial pressure more than 65, and so increasing norepinephrine to get a "normal" mean pressure of 93 would result in a lot of necrotic fingers and toes. Our bodies can vasodilate some vascular beds and vasoconstrict others. Not so with pressors—it's all or nothing.

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