OPTIMIZING CIRCULATION/EVALUATING VOLEMIA AND FLUID RESPONSIVENESS

From the 3rd Goal-Directed Therapy Symposium, presented by the Departments of Anesthesiology, David Geffen School of Medicine at the University of California, Los Angeles and the University of California, Irvine, School of Medicine

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Optimizing Circulation

Etiology of circulatory shock: although commonly attributed to cardiac pump function, hypovolemia, obstruction, and vasomotor tone (artificial construct); every patient in cardiogenic shock shows inflammatory response with elevation of cytokines and decreased vasomotor tone, every trauma patient shows septic picture independent of hypovolemia, and patients with massive pulmonary embolism (PE) experience cardiogenic shock and sepsis (ie, shock states categorize patients, but large components of each characteristic found in all patients with full-blown shock [fundamental etiologies differ])

Physical findings: increased sympathetic tone causes tachycardia, hyperpnea, diaphoresis, and decreased organ perfusion with decreased urine output (UO), ileus, altered sensorium, and lactic acidosis; in all cases, hypotension occurs late (indicates treatment has been delayed too long and constitutes medical emergency); cardiac output (CO) important only for maintaining flow

Physiologic perspective: hypotension caused by hypovolemia (hemorrhage), cardiac pump function (myocardial infarct), obstruction (PE), or vasomotor paralysis (sepsis); management—administer fluids and vasopressors (maintain mean arterial pressure [MAP]), then review evidence of organ perfusion; give additional fluids, if necessary, to maintain flow; rise in central venous pressure (CVP) indicates heart failure and requires dobutamine; target MAP 65 mm Hg (fundamental starting point); titrate care to clinical markers

Cardiac output: varies with metabolic demand (no “normal” level of CO); threshold levels important (extreme low cause for concern); cardiac index (CI) of 1 L/min/m² possibly adequate for hypothermic paralyzed patient, while 6 L/min/m² inadequate for seizing and hyperthermic patient; following volume relative to treatment (rather than targeting absolute number) important

Study by Bland et al: evaluated survival based on spontaneous oxygen delivery (CO multiplied by arterial O₂ constant [Do₂]); and found good outcomes with Do₂ >450 mL/min (>600 mL/min best) without intervention; alternative interpretation—sick patients show low spontaneous O₂ delivery, while healthy patients show high O₂ delivery (and “sick people die more often than nonsick people”)

Perioperative optimization: preoperative goal-directed therapy reduces mortality in patients presenting with critical illness, but studies show no benefit for those with preexisting injury (eg, renal failure, coma); early goal-directed therapy with maintenance of Do₂ >600 mL/min/m² throughout surgery (ie, preoptimization) associated with improved outcomes in these patients; postoptimization effective for patients who have had “surgical misadventure”

Target values: oxygen saturation—transfusion of blood improves oxygen saturation without increasing blood flow; mixed venous O₂ saturation (SvO₂) useful only as negative predictor, with low values indicating trouble; direct therapy to target oxygen delivery (oxygen transport)—data show decreases in infection rate, cardiovascular dysfunction, and mortality; beneficial even if target not reached; act of resuscitation most important (overresuscitation must be avoided); postoperative wound infection result of ischemia, which may stem from poor resuscitation; elective surgery—increased CO results in reduced postoperative nausea (gut ischemia), shorter length of stay, increased patient satisfaction (with lower cost), and increased patient flow

Organ injury: aggressive resuscitation—not beneficial in patient with organ injury, but improves outcomes before its development (ie, “you cannot bring back the dead”)

Cost of health care: government decides acceptable level of expenditures on health care; cost-effectiveness analysis favors preoptimization; study by Rhodes and Bennett—showed permanent benefit of 2 hr of goal-directed therapy, with mean increase in survival >3 yr; all patients with no complications had good outcomes, but protocol group better able to weather complications because of resuscitation; other studies show both pre- and postoptimization beneficial in patients with complications

Protocolized care: ensures consistency of care across shifts and between doctors; improves processes and decreases errors; overall benefit seen in rates of gastrointestinal complications in high- and low-risk patients; rate of liver complications minimized (liver profoundly resistant to ischemia)

Tissue wellness: susceptibility to ischemia varies among different types of tissue; study of macrocirculatory parameters and organ injury found dobutamine increases CO but impairs microcirculatory flow and decreases hepatic function (ie, no relationship exists between macro- and microcirculation)

Recommendations: monitor “real things” (eg, UO, MAP, capillary refill, sensorium); measure adequacies of flow; measure delivered functional parameters (eg, vascular occlusion test, stroke volume variation, pulse pressure variation); trends more important than absolute values; manage patient with fluids and vasopressors (eg, norepinephrine); consider increasing

Educational Objectives

The goal of this program is to improve outcomes of patients in circulatory shock. After hearing and assimilating this program, the clinician will be better able to:

1. Determine the etiology of circulatory shock.
2. List the end-organ effects of improving oxygen delivery.
3. Use the stop-flow test to estimate mean systemic pressure.
4. Recognize the effects of alteration in unstressed blood volume on cardiac output.
5. Explain the relationship between stroke volume variation and volume responsiveness.

Faculty Disclosure

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Evaluating Volemia and Fluid Responsiveness

Primary clinical assessment: identify hemodynamic instability early (before patients become too ill for intervention to be effective); determine appropriate therapy, then apply therapy in consistent fashion; bedside cardiovascular physiology — determine CO from circulation

Cardiac function: one-third of equation, with tone and volume completing circuit; right atrial pressure fulcrum for CO; role of heart to keep right atrial pressure low; CO plotted against right atrial pressure forms Starling curve; passive increase in right atrial pressure causes pressure gradient for venous return to fall and flow to cease; when pressure in right atrium equal to pressure in periphery, flow becomes 0 (mean systemic pressure); venous return equals CO; ventricular function curve simultaneously represents venous return curve; only one point (equilibrium point) represents intersection of CO and right atrial pressure curves (function of ventricular pump function, blood volume, blood flow distribution, and peripheral vasomotor tone [altered during disease]); impaired contractility decreases ventricular function, increases right atrial pressure, and decreases CO; patients in chronic heart failure retain fluid (only means of maintaining CO without drugs) and develop congestive heart failure; fluid retention causes mean systemic pressure to rise

Mean systemic pressure: determined by static blood volume, vasomotor tone, and dynamic processes of blood flow distribution; lowered by hypovolemia (hypervolemia skews curve in opposite direction); vasodilation or vasovagal syncope causes pressure to vasodilate, and right atrial pressure to fall with no change in blood volume (cause of hypotension during spinal or general anesthesia); norepinephrine causes mean systemic pressure to rise with no change in blood volume; therefore, hypotensive patients should not be given norepinephrine to restore CO unless blood volume also corrected (hypovolemic patients receiving norepinephrine increase CO to point of death)

Measurement of mean systemic pressure: evaluate dynamic swings in right atrial pressure to stroke volume and right ventricle instantaneously; apply end-inspiratory pause maneuver (track fall in CO and rise of right atrial pressure for different levels of continuous positive airway pressure); use systemic to mean right atrial pressure, CVP, and CO; peripheral vascular occlusion test — rise in pleural pressure during positive-pressure breathing causes increase in CVP and decrease in right-ventricular filling pressure, with decrease in right-ventricular stroke volume; plot right atrial pressure against right-ventricular stroke volume on beat-to-beat basis to obtain estimate of mean systemic pressure; identical results obtained with end-inspiratory pause (easy to perform at bedside); using inspiratory pauses at various points and measurements of CO, speaker demonstrated that mean systemic pressure rises or falls with volume loading or head-up tilt; rapid vascular occlusion shows decay in venous and arterial pressures; curve slows and stop-flow value equals mean systemic pressure; any patient can be tested using arterial line; results of stop-flow test available on beat-to-beat basis and equal to values calculated with formulas; study of septic animal model confirmed accuracy of stop-flow test under all conditions

Effective blood volume: mean systemic pressure function of blood volume in periphery and indicates effective circulating blood volume of patient; most blood in unstressed, volume, and only stressed volume determines mean systemic pressure; capacitance — change in total volume; compliance most important; mean systemic pressure (and CO) rise with increase in volume; shifting blood from gut to legs (which have low unstressed volume) results in decrease in unstressed volume and markedly increased mean systemic pressure with constant blood volume; increased sympathetic tone distributes blood volume away from gut into areas with less unstressed volume (primary mechanism for increasing CO during stress); vasodilation causes venous return to fall by increasing unstressed volume (resulting in fainting); altering unstressed volume primary mechanism for altering driving pressure for venous return (explains why stressed pressure of CVP and total blood volume not related); CVP should never be used to assess volume status but can signal cor pulmonale (ie, stop fluids if rapid rise in CVP occurs)

Hypotension in sepsis: due to increased unstressed volume, rather than change in compliance; initial fluid challenge for patients with sepsis results in increased blood pressure (BP) due to increased unstressed volume; comparison of volume-responsive patients with nonresponsive patients showed higher mean systemic pressures in those no longer volume-responsive (ie, additional fluid in full system elicits no response); stressed volume of volume-responsive patients small compared with other patients; increased vasomotor tone causes rise in mean systemic pressure; slope of line reciprocal of resistance to venous return (slope decreases with increase in vasomotor tone because resistance increased); vasodilation decreases resistance and increases slope; French study — demonstrated preservation of CO when norepinephrine discontinued; slope of line increases in presence of decreased mean systemic pressure (resistance to venous return compensates for fall in mean systemic pressure)

Dobutamine: speaker found that it decreases resistance overall, with resistance to venous return decreased by one-third; acts as vasodilator and causes mean systemic pressure to fall (causes hypovolemic patients to become hypotensive); increasing contractility alone increases CO and decreases right atrial pressure; with addition of dobutamine, mean systemic pressure and resistance decrease (slope increases, and CO further increases; explains increased CO seen in volume-resuscitated patients with heart failure)

Cardiac performance: ability of heart to maintain CO with increased pressure; calculated by dividing driving pressure for venous return (ie, mean systemic pressure minus right atrial pressure) by mean systemic pressure; volume loading alters cardiac power; plot of mean systemic pressure against ratio of driving pressure has slope of 1 if heart “perfect”; increasing mean systemic pressure causes no change in CVP and increases CO; slope decreases proportionally with decreasing performance of heart; volume loading flattens curve for “good” heart (volume response decreases); “bad” heart starts low and rapidly enters poor range; patients with heart failure, hypovolemia, or sepsis may all have arterial pressure of 80 mm Hg (ie, BP value offers no information about cardiovascular status of patient); patients with, eg, heart failure, sepsis, can have high filling pressure with low contractility; single value for CO reveals nothing about cardiovascular status of patient; if mean systemic pressure known, combining these parameters can narrow possibilities to heart failure or hypovolemia; responses to volume loading — differ among patients with hemorrhage vs sepsis vs cardiogenic shock; mean systemic pressure increased without improvement in CO in patients with cardiogenic shock; those with sepsis or hemorrhage showed no change in mean systemic resistance, but had increased CO

Critical closing pressure: right arterial pressure can be measured independently; arterial resistance nonlinear (linked to arterioles and precapillary sphincters); when stroke volume plotted against arterial pressure, intercept point significantly higher than CVP (critical closing pressure of body); in pressure-flow relationship with CO vs arterial pressure, slope represents resistance, but zero intercept much higher than right atrial pressure; zero flow pressure critical closing pressure of arterioles; in acute endotoxic shock, critical closing pressure lost
with dilation of arterioles, without change in resistance vessels (clinical presentation of sepsis); arterial resistance found to be much lower than systemic vascular resistance.

**Volume responsiveness:** determination can incorporate pulse pressure and stroke volume variation (dynamic values); positive-pressure breathing — end-diastolic volume decreases with inspiration, and left ventricular stroke volume falls (stroke volume variation); stroke volume variation lost in non-volume responsive patients; responsiveness indicated with 2 different variances; arterial elastance important because BP does not appreciably rise in response to volume in vasodilated patients; pulse pressure to stroke volume variation falls with vasodilation.

**Patient management:** when MAP increased by 20 mm Hg with norepinephrine, mean systemic pressure, right atrial pressure, and venous resistance increase, with decrease in stroke volume variation due to increased afterload (response in CO variable); low stroke volume variation functions as marker of poor cardiac performance; patients with increased CO show greater stroke volume variation.

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**Estimated time to complete the educational process:**

- Review Educational Objectives on page 1: 5 minutes
- Take pretest: 10 minutes
- Listen to audio program: 60 minutes
- Review written summary and suggested readings: 35 minutes
- Take posttest: 10 minutes
1. All patients in full-blown circulatory shock show components of cardiac pump function, hypovolemia, obstruction, and vasomotor tone, regardless of the primary etiology.
   (A) True  (B) False

2. Which of the following is a late finding in circulatory shock and constitutes a medical emergency?
   (A) Decreased urine output  (C) Hypotension
   (B) Hyperpnea  (D) Lactic acidosis

3. Select the true statement about oxygen delivery.
   (A) In addition to improving oxygen saturation, transfusion of blood increases blood flow
   (B) Improved oxygen delivery beneficial only if target flow reached
   (C) Postoperative wound infections may result from ischemia due to poor resuscitation
   (D) Mixed venous O₂ saturation (SVO₂) can be used as a positive predictor

4. All the following statements about protocolized care are true, except:
   (A) Ensures consistency of care across shifts and between doctors
   (B) Improves processes and decreases errors
   (C) Significantly reduces the rate of liver complications
   (D) Reduces rates of gastrointestinal complications in high- and low-risk patients

5. Choose the correct statement about tissue effects in the patient in circulatory shock.
   (A) All types of tissue equally susceptible to ischemia
   (B) Dobutamine improves hepatic function
   (C) Dobutamine has similar effects on macro- and microcirculation
   (D) None of the above

6. All the following statements about the stop-flow test are true, except:
   (A) Results available on beat-to-beat basis
   (B) Accurate under all conditions
   (C) Stop-flow value equals mean systemic pressure
   (D) Requires a central venous pressure monitor

7. Most blood is in unstressed volume and has no effect on mean systemic pressure.
   (A) True  (B) False

8. Which of the following accurately describes the effect of dobutamine on circulatory shock?
   (A) Increases chronotropy
   (B) Decreases mean systemic pressure and resistance
   (C) Increases right atrial pressure
   (D) Increases mean arterial pressure in hypovolemic patients

9. Select the true statement about stroke volume variation.
   (A) Caused by decreased end-diastolic volume during the expiratory phase of positive-pressure ventilation
   (B) Increases in non-volume responsive patients
   (C) Dynamic value that can be used in determination of volume responsiveness
   (D) Pulse pressure to stroke volume variation increases with vasodilation

10. Low stroke volume variation is a marker for which of the following?
    (A) Poor cardiac performance  (C) Decreased unstressed blood volume
    (B) Increased fluid responsiveness  (D) Decreased afterload

Answers to Audio-Digest Anesthesiology Volume 56, Issue 17: 1-D, 2-B, 3-C, 4-C, 5-A, 6-C, 7-A, 8-C, 9-A, 10-D