TRAUMA

FLUID MANAGEMENT

Early resuscitation • Hemostatic strategy • Post-resuscitation care

for Anesthesiology & Critical Care

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Goals of intraoperative fluid management

The main objectives are:

1. Maintain adequate intravascular volume and cardiac output.
2. Ensure sufficient tissue and organ perfusion.
3. Maintain electrolyte and acid–base balance.
4. Prevent hypovolemia and fluid overload.
5. Minimize postoperative complications, including pulmonary edema, acute kidney injury, and delayed gastrointestinal recovery.

Strategy	***Main principle***
Liberal fluid therapy	Relatively generous fluid administration
Restrictive fluid therapy	Avoid unnecessary fluid administration
Zero-balance therapy	Minimize unnecessary fluid retention
Goal-directed fluid therapy	Individualize fluids according to hemodynamic response
Damage control resuscitation	Balanced hemostasis

Damage control resuscitation (DCR)

1. **Hemostatic resuscitation** — Minimize crystalloids; use warmed blood products; correct acidemia
2. **Permissive hypotension** — Selected patients only, until bleeding is controlled
3. **Damage control surgery** — Operative or angiographic hemorrhage control
4. **Goal-directed hemostasis** — Identify and correct coagulopathy
5. **Adjuncts** — such as transamine

These strategies are intended to proceed simultaneously.

How perioperative fluid practice evolved

The review describes a shift in practice over roughly 25 years:

Early 2000s

Liberal fluid use

Concern: under-resuscitation

→

Second decade

Restrictive approach

Concern: fluid overload

→

Recent years

More balanced approach

Avoid both excess and hypovolemia

GDFT uses hemodynamic parameters to guide fluid administration and cardiovascular support.

Parameters that may be monitored include:

- Stroke volume (SV)
- Cardiac output (CO)
- Stroke volume variation (SVV)
- Pulse pressure variation (PPV)
- Mean arterial pressure (MAP)
- Lactate concentration
- Urine output
- Peripheral perfusion

The resuscitation problem

Hemorrhage

↓ circulating volume → ↓ venous return → ↓ cardiac output → impaired tissue perfusion

Endothelial injury

↑ capillary permeability → intravascular-to-interstitial fluid shift → tissue edema

Treatment tension

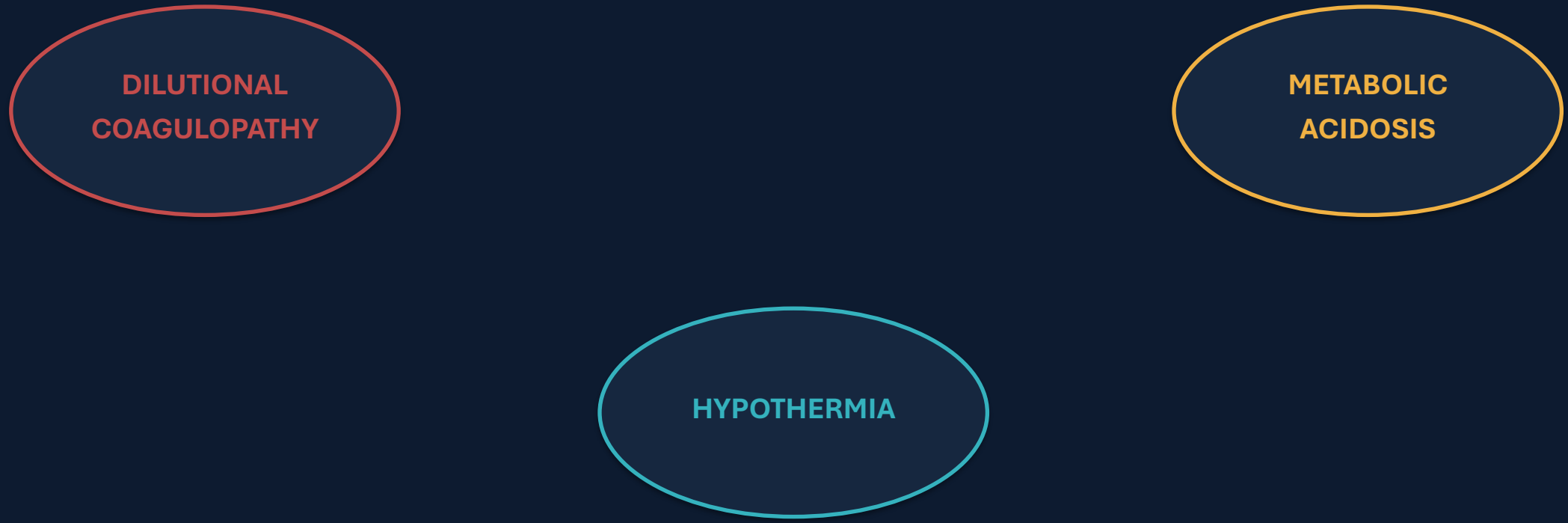
Support organ perfusion while limiting dilution, hypothermia, and further bleeding

The target is not a normal blood pressure at any cost.

It emphasizes balancing organ perfusion with hemostasis until definitive hemorrhage control.

The trauma “lethal triad”

Aggressive crystalloid loading can amplify all three.



Potential downstream effects

Intra-abdominal hypertension / ACS • Extremity compartment syndrome • Ileus • Pulmonary and tissue edema

Hemostatic resuscitation

Limit crystalloid

Avoid large-volume crystalloid administration during active hemorrhage.

Warm the patient

Use warmed fluids and blood products; prevent hypothermia.

Blood component strategy

PRBC : FFP : platelets in 1–2 : 1 : 1 pattern.

The objective is to approximate whole-blood resuscitation and reduce dilutional coagulopathy.

Static measures: useful, but imperfect

Examples discussed in the review

- Central venous and
- Pulmonary artery pressures
- Systemic arterial pressure and
- Heart rate
- Urine output
- Lactate,
- Base deficit, and
- other laboratory measures

Why caution is needed

Static parameters can represent volume status poorly. Oliguria may reflect anesthetic effects or surgical stress; blood pressure and heart rate do not consistently track cardiac output.

Rising lactate and falling mixed-venous oxygen saturation can help identify global tissue hypoperfusion.

Clinical classes of hemorrhage

Parameter	Class I	Class II	Class III	Class IV
Blood loss	<15%	15–30%	31–40%	>40%
Heart rate	↔	↔ / ↑	↑	↑ / ↑↑
Blood pressure	↔	↔	↔ / ↓	↓
Pulse pressure	↔	↓	↓	↓
Urine output	↔	↔	↓	↓↓
GCS	↔	↔	↓	↓
Base deficit (mEq/L)	0 to −2	−2 to −6	−6 to −10	≤ −10

Clinical signs are estimates; interpret together with physiology and ongoing blood loss.

Dynamic monitoring and fluid responsiveness

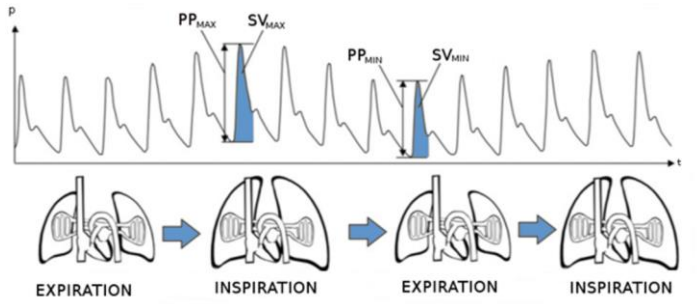
Waveform variation

Pulse pressure variation (PPV),

Stroke volume variation (SVV),

Systolic blood pressure variation(SBPV), and

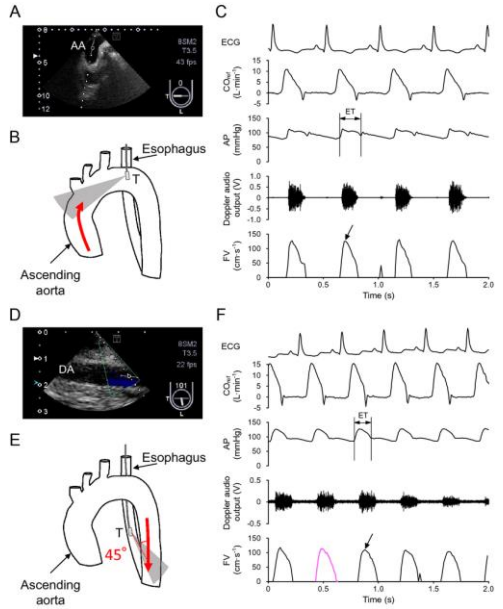
IVC diameter changes.



Ultrasound

Transesophageal Doppler can estimate aortic flow;

Transthoracic or transesophageal echocardiography can assess cardiac filling and function.

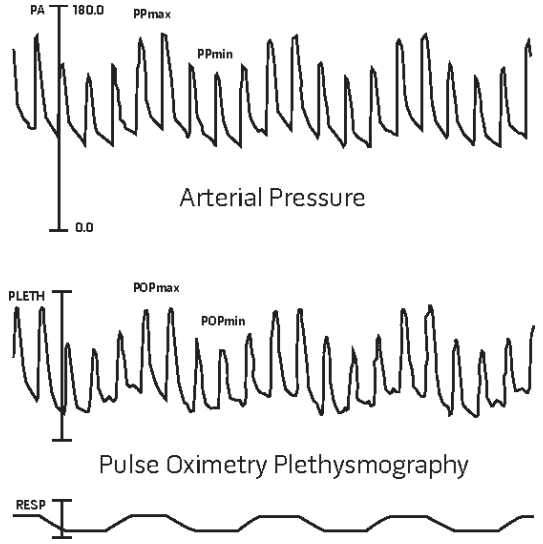


Other technologies

Pleth variability index,

thoracic bioimpedance, and

pulse-wave analysis may be used, but accuracy limitations remain.



Dynamic indices generally assess fluid responsiveness better than static measures—but they are not universally reliable.

Machine learning and dynamic indices

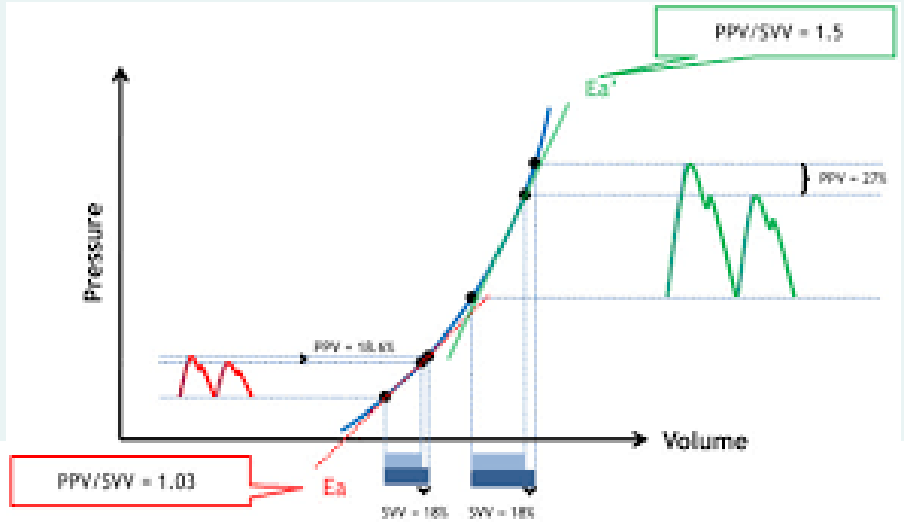
Hypotension Prediction Index (HPI)

- Edwards Acumen; arterial waveform features **give a 0–100 score, alert typically ≥85**
- **Warns up to ~15 min before MAP <65 (Hatib 2018)**
- **HYPE trial: less time-weighted hypotension; later** trials smaller or inconsistent; outcome benefit unproven
- Needs arterial line (finger-cuff version exists); alert needs a treatment protocol; alert fatigue



Dynamic indices

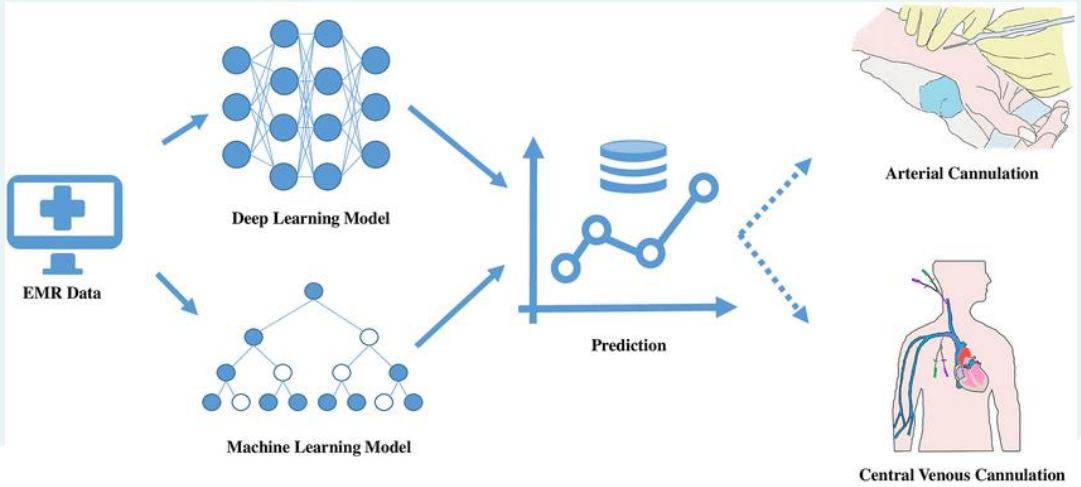
- PPV/SVV: need controlled ventilation, adequate tidal volume, sinus rhythm
- Pleth variability index: noninvasive but less reliable
- Dynamic arterial elastance (Eadyn)
- Stroke volume, CO and pulse pressure trends often fall before MAP



Preoperative predictors and bedside ultrasound

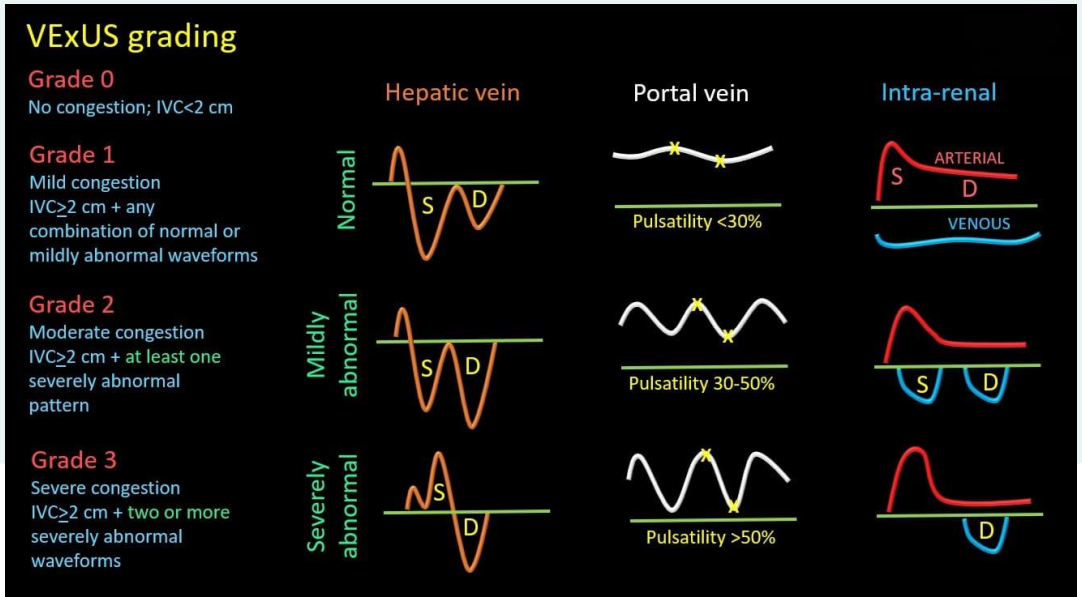
Preoperative and induction risk

- Older age, higher ASA class, low baseline MAP
- ACEi/ARB on day of surgery, hypovolemia
- High propofol or opioid doses
- Shock index (HR/SBP); orthostatic or ambulatory BP findings
- ML models on preoperative data in development



POCUS

- IVC collapsibility/distensibility
- LV size and function (hyperdynamic, underfilled)
- Carotid corrected flow time, passive leg raise with SV measurement, VExUS
- Operator-dependent and intermittent: complements continuous monitoring

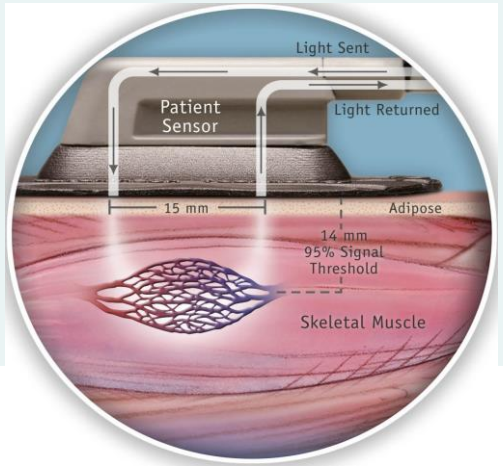


Other signals and practical points

Other physiologic signals

- Sudden end-tidal CO_2 drop: falling CO or embolism
- Cerebral NIRS may fall before systemic pressure
- Analgesia monitors and HRV: limited evidence
- Tachycardia plus narrowing pulse pressure: classic early hypovolemia





Practical points

- Arterial line detects hypotension earlier than intermittent cuffs
- Even short MAP <65 episodes linked to AKI and myocardial injury; risk rises with depth and duration
- Best results: predictive alert plus a response protocol

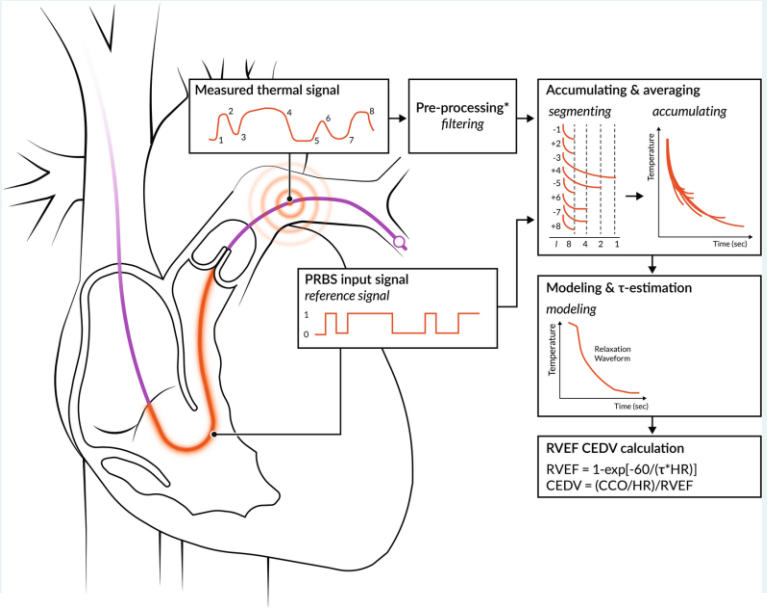
Cardiac Output Monitoring

Classification by invasiveness and measurement principle

Reference methods and transpulmonary thermodilution

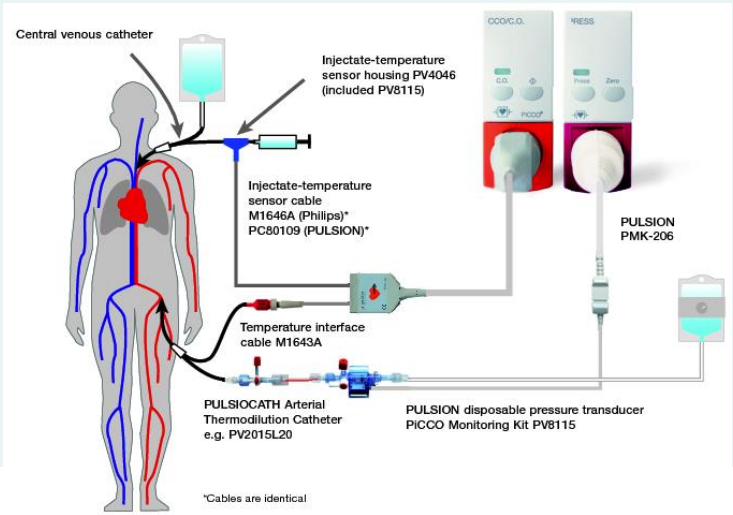
Classic reference

- Pulmonary artery catheter: bolus or continuous thermodilution; invasive, limited outcome benefit
- Fick principle (O_2 consumption, AV O_2 difference): theoretical gold standard, rarely practical



Transpulmonary thermodilution

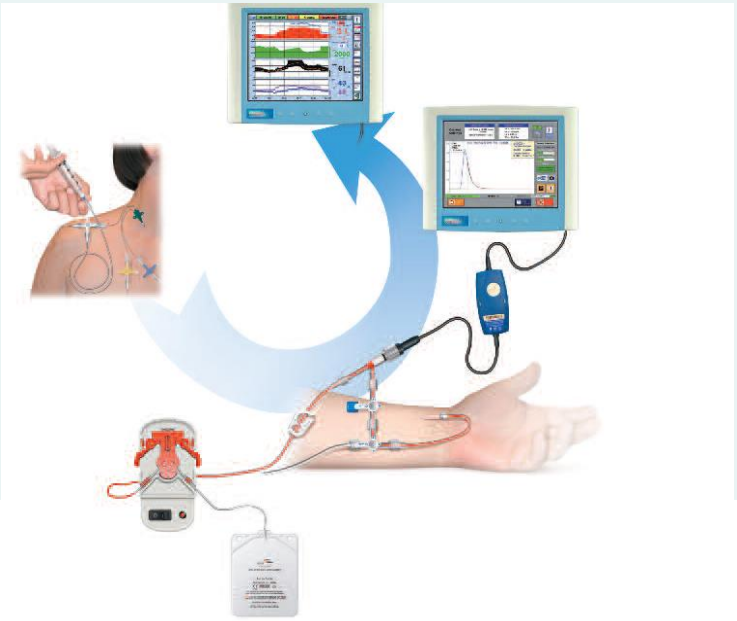
- PiCCO (Getinge/Pulsion): central venous injection plus femoral/axillary arterial thermistor line
- Gives CO, global end-diastolic volume, extravascular lung water, pulse contour between calibrations
- EV1000/VolumeView (Edwards) works similarly



Pulse contour analysis

Calibrated

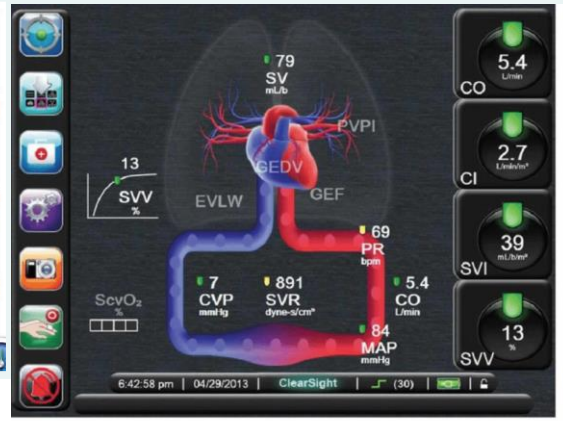
- LiDCOplus: lithium dilution calibrates a pulse power algorithm
- Lithium caveats: non-depolarizing relaxants can interfere with the sensor; avoid in early pregnancy and in patients on lithium
- PiCCO (see previous slide)



Uncalibrated

- LiDCOrapid (pulse power)
- FloTrac/Vigileo (Edwards); platform for HPI
- MostCare (PRAM)
- Finger cuff: ClearSight/Nexfin (volume clamp), CNAP
- Less accurate in vasoplegia, rapid tone changes, arrhythmias

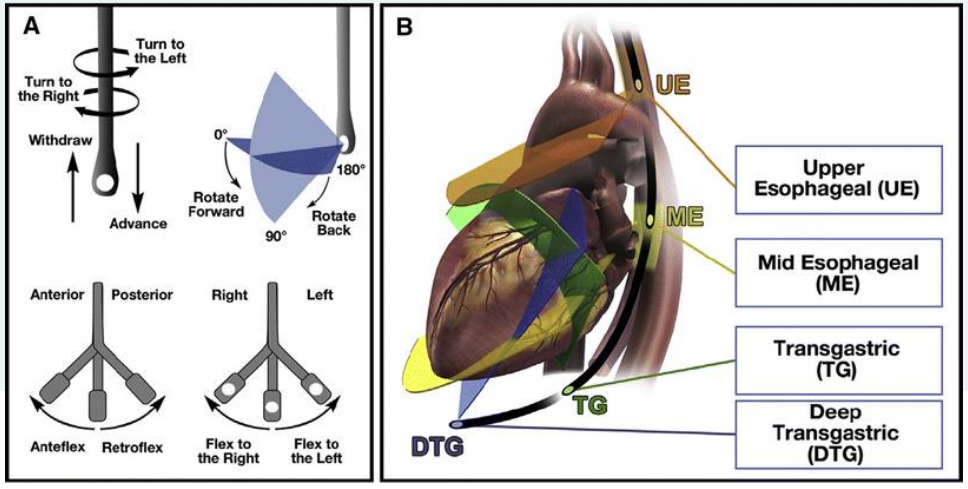




Doppler, bioimpedance and other methods

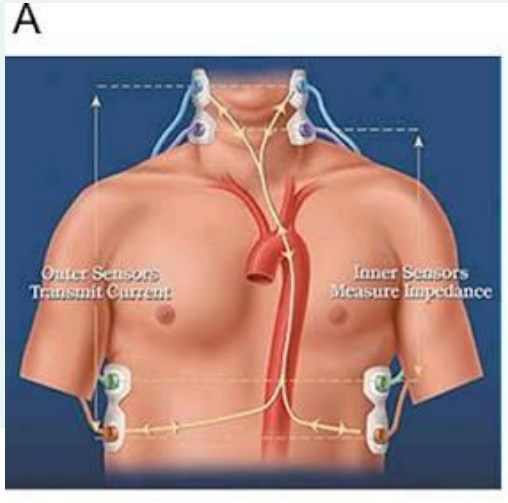
Doppler / ultrasound

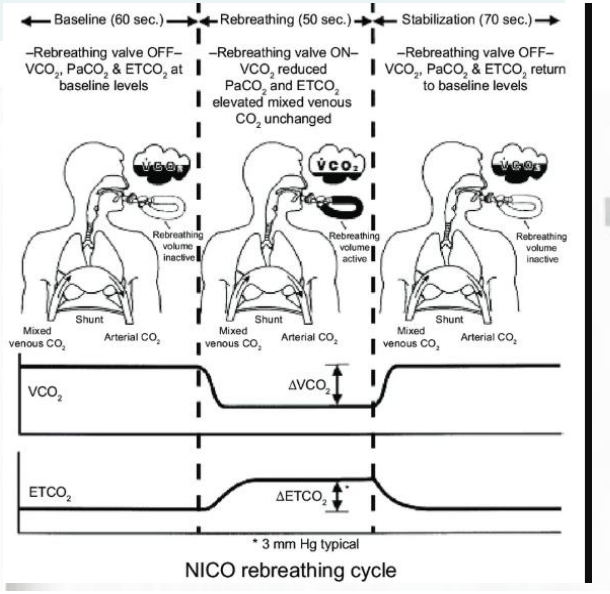
- Esophageal Doppler (CardioQ-ODM): descending aortic flow; strong GDFT outcome evidence; anesthetized patient
- USCOM 1A: noninvasive continuous-wave Doppler at aortic/pulmonary valve; nomogram-estimated valve area; operator-dependent, intermittent; useful in ED and pediatrics
- TTE/TEE: VTI × LVOT area gives stroke volume



Bioimpedance / other

- Thoracic bioimpedance and bioreactance (NICOM/Cheetah Starling): convenient, accuracy mixed in critical illness and surgery
- Partial CO_2 rebreathing (NICO): Fick with CO_2; needs ventilation, less used now
- Applied echo and carotid Doppler for trend estimation





Comparison of CO monitors

Category	Examples	Invasiveness	Calibration	Strength
Thermodilution	PAC, PiCCO	High	Yes	Reference; extra volumetrics
Pulse contour (calibrated)	LiDCOplus	Moderate	Lithium	Accurate trends
Pulse contour (uncalibrated)	FloTrac, LiDCOrapid	Arterial line	No	Easy, continuous
Finger cuff	ClearSight	Noninvasive	No	Low risk
Doppler	CardioQ, USCOM	Semi/non	No	Strong GDFT evidence
Bioreactance	NICOM	Noninvasive	No	Simple; mixed accuracy

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Initial crystalloid choice

Suspected TBI / spinal injury

0.9% sodium chloride is the chapter's preferred initial crystalloid because balanced solutions may be slightly hypotonic and could aggravate cerebral edema.

Other hemorrhaging trauma

A balanced crystalloid is preferred (e.g., lactated Ringer's / Hartmann's, Ringer's acetate, Plasma-Lyte, Sterofundin).

Crystalloid choice is only one part of the strategy: volume and rate matter.

Do not let crystalloid administration delay blood products or definitive hemorrhage control.

Colloids and albumin

- Colloids have not shown a major clinically meaningful volume-expansion advantage over crystalloids in the cited evidence.
- Synthetic colloids in severely injured patients were associated with increased hemodialysis requirement and multiple-organ failure in registry data.
- Hydroxyethyl starch may impair coagulation: weaker clot formation and impaired platelet aggregation are described.
- In one study, 4% albumin was associated with higher 28-day mortality than saline; the difference persisted at 2 years.
- Conclusion: colloids / albumin do not offer a major advantage in early trauma resuscitation.

Hypertonic saline: theory vs outcomes

Proposed advantages

Small-volume plasma expansion • Immunomodulatory / anti-inflammatory effects • Reduction of ICP

Evidence

No improvement in patient-oriented outcomes in hemorrhagic shock or TBI; one cohort showed higher mortality among patients not transfused in the first 24 h.

Hypertonic saline is not recommended as a routine trauma resuscitation fluid

Permissive hypotension: injury-specific targets

Injury pattern (active bleeding)	Target SBP
Penetrating trauma	60–70 mmHg
Blunt trauma without head injury	80–90 mmHg
Blunt trauma with TBI / spinal injury	100–110 mmHg

Permissive hypotension is not for TBI or spinal injury.

After hemorrhage control, transition toward restoring tissue perfusion and organ function.

Initial bolus: small, warm, reassess

Adults

1 L isotonic crystalloid (chapter also describes ~15 mL/kg)

Children <40 kg

20 mL/kg initial bolus

If blood unavailable

Small aliquots: 250 mL or 4 mL/kg in selected critically hypoperfused patients

After the initial bolus, reassess vital signs, end-organ perfusion, and tissue oxygenation.

Avoid reflexively repeating large crystalloid boluses.

Response to the initial bolus

Pattern	Clinical response	Likely implication	Next step
Rapid responder	Normalizes rapidly	Usually <15% blood loss	Continue evaluation; limited further resuscitation
Transient responder	Improves, then deteriorates	Ongoing bleeding / 15–40% loss	Blood products; urgent hemorrhage control
Minimal / nonresponder	Continues to deteriorate	Severe loss / >40% or other shock	Immediate definitive intervention

A transient response is a warning sign—not reassurance.

Practical initial resuscitation workflow

A — Airway & breathing assessment

B — Two large-bore peripheral IVs (≥18G)

C — Crossmatch, laboratory tests, and blood gas

D — Warm isotonic crystalloid: 1 L adult / 20 mL/kg child

E — **Classify response: rapid / transient / minimal**

F — **Prioritize blood products and hemorrhage control when indicated**

After hemostasis: assess perfusion, not BP alone

Lactate

Serial measurement and clearance help assess adequacy of tissue perfusion.

Base deficit

A marker of metabolic derangement and resuscitation adequacy.

$ScvO_2$

Listed among microcirculatory oxygen-delivery indices.

Normalization of vital signs does not guarantee adequate resuscitation.

Maintain normothermia and provide adequate analgesia to help reverse vasoconstriction.

DYNAMIC TREATMENT

When should fluid resuscitation stop?

1 RESUSCITATION

Treat shock and restore perfusion; repeated boluses may be needed.

2 OPTIMIZATION

Refine perfusion and oxygenation; reassess frequently and use fluid more conservatively.

3 STABILIZATION

Maintain water/electrolyte balance with minimal maintenance infusion; aim for neutral or negative balance.

4 DE-ESCALATION

Support recovery by mobilizing accumulated fluid; aim for negative balance.

Frequent reassessment is essential; reduce IV fluids during optimization and consider stopping during stabilization.

Post-resuscitation fluid management

- Estimate ongoing losses: wounds, drains, persistent capillary leak, fever, and hypercatabolism.
- Individualize fluid type and dose according to the estimated deficit and clinical context.
- Uncorrected hypovolemia may worsen tissue hypoperfusion and organ dysfunction.
- Excess fluid may impair oxygen delivery, wound healing, and homeostasis.
- Before giving additional fluid, assess fluid responsiveness; use small aliquots and monitor benefit and harm.

Principle: maintain perfusion while preventing unnecessary fluid accumulation.

Deresuscitation: the recovery phase

When to consider

After stabilization, especially with tissue edema, compartment syndromes, pulmonary edema, ARDS, or contusions.

How

Active fluid removal with diuretics and/or ultrafiltration, as clinically appropriate.

Why it matters

Persistent positive cumulative balance is associated with prolonged ICU stay and ventilatory requirements.

A restrictive recovery-phase strategy and active fluid removal may facilitate earlier extubation.

Take-home messages & source

- Limit crystalloids; prioritize early blood products and definitive hemorrhage control.
- Use 0.9% saline when TBI is suspected; balanced crystalloids for other hemorrhaging patients.
- After 1 L in adults, classify the response—especially identify transient responders.
- After hemostasis, use perfusion markers; during recovery, prevent and actively manage fluid overload.