



Major Hemorrhage

Massive transfusion · damage-control resuscitation · stop the bleeding, do not just replace it

TXA < 3 h
and blood, not saline

ACTIVATE EARLY, AND ON CLINICAL GROUNDS

Loss of one blood volume in 24 h, 50% in 3 h, bleeding faster than 150 mL/min, or transfusion needed to stay alive. Do not wait for a hemoglobin: the initial value is normal in brisk bleeding because the patient has not yet hemodiluted, so a reassuring number early means nothing. **Activating the protocol is what gets balanced products to the bedside** instead of sequential single units, and under-activation is far more damaging than over-activation.

FIRST MOVES *control the source while you resuscitate; these run in parallel*

STOP THE BLEEDING

Direct compression, tourniquet, hemostatic packing, pelvic binder. Definitive control by surgery, interventional radiology or endoscopy is the actual treatment: transfusion only buys the time to get there. Call surgery, IR and hematology at activation, not once products are running.

TXA WITHIN 3 HOURS

Tranexamic acid 1 g IV over 10 min, then 1 g over 8 h (CRASH-2, Lancet 2010; CRASH-3, Lancet 2019 for head injury). Earlier is better and the benefit decays fast. Given beyond 3 h it is associated with harm, so the time of injury, not the time of arrival, is what counts.

RESTRICT CRYSTALLOID

Give blood, not liters of saline. Crystalloid dilutes clotting factors, worsens acidosis and drops the temperature, feeding the lethal triad. Permissive hypotension to a systolic of about 80 to 90 until bleeding is controlled, so you do not pop a fresh clot. The exception is traumatic brain injury, where the injured brain needs perfusion: target a MAP of at least 80.

TRANSFUSE *empiric ratio first, then goal-directed once numbers are back*

Target	Aim	If not met
Empiric ratio	RBC : FFP : platelets = 1 : 1 : 1 in fixed packs until labs return	PROPPR, JAMA 2015 compared 1:1:1 with 1:1:2. Its primary endpoints, 24-h and 30-day mortality, were negative. The 1:1:1 group achieved more hemostasis and fewer deaths from exsanguination at 24 h, which were secondary findings, and that is the honest basis for the balanced ratio.
Hemoglobin	7 to 9 g/dL during active bleeding	Transfuse red cells. Do not chase a normal hemoglobin while still bleeding.
Platelets	> 50, and > 100 for CNS or eye injury, or ongoing bleeding	One adult pool.
Fibrinogen	> 1.5 to 2.0 g/L	Cryoprecipitate or fibrinogen concentrate. Fibrinogen falls first and fastest of all the clotting factors, so it is the one most often left uncorrected.
PT / aPTT	< 1.5 × normal	FFP 15 to 20 mL/kg, or PCC if on warfarin.
Ionized calcium	> 1.1 mmol/L	IV calcium chloride or gluconate. Stored blood is citrated and citrate chelates calcium, so massive transfusion reliably causes hypocalcemia. Calcium is required for the clotting cascade and for myocardial contractility, making this a cause of both refractory coagulopathy and refractory hypotension. Check it early and repeatedly.

THE LETHAL TRIAD *each element worsens the other two, so all three are treated at once*

HYPOTHERMIA

Clotting enzymes are temperature-dependent and slow markedly below 34 °C, so a cold patient is coagulopathic whatever the numbers say. Warm the patient, the fluids and the blood, use forced-air warming, and stop exposing them once the survey is done.

ACIDOSIS

Acidosis impairs the clotting cascade and blunts the response to vasopressors. The fix is restoring perfusion and stopping the bleeding, not bicarbonate. Follow lactate and base deficit as resuscitation markers.

COAGULOPATHY

Trauma-induced coagulopathy is present on arrival in a substantial minority, before any dilution. Balanced ratio plus TXA plus calcium, then viscoelastic testing (ROTEM or TEG) to target the specific defect once available.

BY SETTING, AND WHAT TO REVERSE

Setting	Additional measures
Trauma	Damage-control resuscitation and damage-control surgery: control contamination and bleeding, then return once resuscitated rather than completing a long operation on a cold, acidotic patient. Pelvic binder, early CT or straight to the operating room, TXA early.
Obstetric	Uterotonics, bimanual compression, balloon tamponade, and the operating room. TXA early. Obstetric hemorrhage is frequently underestimated because young patients compensate until they crash.
GI	Endoscopy or interventional radiology; in variceal bleeding add octreotide and ceftriaxone. Surgery if uncontrolled.
Reverse anticoagulants	Warfarin: 4-factor PCC plus vitamin K. DOACs: idarucizumab for dabigatran, andexanet alfa for factor Xa inhibitors, or PCC if unavailable. Ask what they take at activation, since this changes the products you need.

MONITOR, AND KNOW WHEN TO STOP *over-transfusion has its own harms*

WHAT TO REPEAT, AND HOW OFTEN

Full blood count, coagulation screen, fibrinogen, blood gas (lactate and base deficit) and ionized calcium every 30 to 60 min during active bleeding. Numbers drawn an hour ago describe a patient who no longer exists, which is why viscoelastic testing at the bedside is preferred once available.

DE-ESCALATE ONCE CONTROLLED

Stand the protocol down deliberately and tell the blood bank, or products keep arriving. Over-transfusion causes TACO, TRALI, hyperkalemia and thrombosis, and the balanced ratio that was right during exsanguination is wrong once bleeding has stopped. Then switch to VTE prophylaxis, which these patients need sooner than instinct suggests.

PEARLS & PITFALLS

- Blood in a balanced ratio, and TXA within 3 hours. Crystalloid dilutes clotting factors and cools the patient.
- A normal initial hemoglobin means nothing in brisk bleeding. Activate on clinical grounds.
- Check ionized calcium early and repeatedly. Citrate chelates it, and low calcium causes both coagulopathy and hypotension.
- Definitive control is the treatment. Transfusion only buys time to reach the operating room, IR suite or endoscope.

