ICM on Open Injuries - Consensus Recommendation

RQ: Should debridement be deferred until serum lactate has normalised in polytrauma or isolated long-bone open fractures?

Section 1: Background (<300 words)

Timely debridement remains a central principle in open fracture care because devitalised tissue and contamination increase the biological risk of deep infection. Historically, operative irrigation and debridement were treated as emergencies, often within a 6- to 8-hour window, although the evidence supporting a rigid threshold has been debated [1]. Modern management combines early antibiotics, resuscitation, wound excision, lavage, skeletal stabilisation and staged reconstruction.

Serum lactate is a marker of tissue hypoperfusion, anaerobic metabolism and response to resuscitation. In orthopaedic polytrauma, persistent hyperlactataemia and metabolic acidosis are associated with pulmonary complications, organ failure, prolonged ventilation and increased resource use [2-7]. Early Appropriate Care protocols use improvement thresholds such as lactate <4.0 mmol/L, pH >=7.25 or base excess >=-5.5 mmol/L to guide timing of definitive fixation [2-4]. These thresholds indicate physiological recovery; they do not require **complete lactate normalisation**.

The controversy is whether elevated lactate should delay source-control surgery for an open fracture. Damage-control orthopaedics separates urgent abbreviated procedures from definitive fixation or reconstruction. In an unstable patient, initial care may consist of antibiotics, haemorrhage control, adequate but time-conscious debridement/lavage, temporary stabilisation and planned second look. No included study directly tested a lactate-normalisation strategy before debridement. Therefore, the clinical question is whether lactate should be a **gatekeeper for debridement** or a guide to resuscitation and operative magnitude.

Section 2: Data extraction table

S.no	Author, year, Country	Study Design	Sample Size	Study population (Inclusion and exclusion criteria)	Groups compared	Results
1	Malhotra et al., 2014, USA [1]	Retrospective cohort; Level I trauma-centre registry.	404 patients; 415 blunt open extremity fractures (upper 129, lower 286/287). D&I <=8 h n=328; D&I >8 h n=87.	Adult blunt open extremity fractures over 72 months. Included blunt open extremity fractures presenting to a state-designated and ACS-verified Level I trauma centre. Excluded transfers, isolated wrist/ankle fractures and first D&I >24 h.	First operative debridement and irrigation <=8 h versus >8 h from presentation.	Deep infection 52/415 (13%). Infection was higher after delayed D&I: 17/87 (19%) vs 35/328 (11%), p=0.04. Lower-extremity infection: 27% after >8 h vs 10% after <=8 h, p=0.002. Delay >8 h independently predicted infection overall (OR 2.035, 95% CI 1.022-4.054) and in lower-extremity fractures (OR 3.006, 95% CI 1.280-7.059). Baseline lactate was similar between early and delayed groups (3.3 vs 3.4 mmol/L, p=0.77). Follow-up: radiographic healing or 6 months.
2	Vallier et al., 2013, USA [2]	Retrospective single-centre database; prognostic modelling for Early Appropriate Care (EAC).	1,443 adults: pelvis 291, acetabulum 399, spine 102, proximal/diaphyseal femur 851.	High-energy mechanically unstable pelvis, acetabulum, spine and proximal/diaphyseal femur fractures treated surgically. Excluded low-energy injuries, pathological fractures and skeletally immature patients.	Definitive fixation within 24 h (n=869) versus after 24 h (n=574); serial pH, base excess and lactate up to 72 h.	Any complication: 149/869 (17.1%) within 24 h vs 170/574 (29.6%) after 24 h. Pneumonia: 5.9% vs 11.7%; ARDS: 1.2% vs 4.0%; sepsis: 1.2% vs 4.0%. Lactate <4.0 mmol/L, pH >=7.25 or BE >=-5.5 mmol/L were proposed as practical resuscitation thresholds. This addresses definitive fixation, not debridement timing. Follow-up: initial episode of care; labs up to 72 h.
3	Vallier et al., 2015, USA [3]	Prospective EAC protocol implementation cohort with historical comparison.	335 patients; 380 fractures (femur 173, pelvic ring 71, acetabulum 57, spine 79); historical cohort 1,443 patients.	Skeletally mature patients with ISS >=16 and mechanically unstable femur, pelvis, acetabulum or spine fractures requiring fixation. Excluded low-energy, neoplastic and skeletally immature patients.	Definitive fixation <=36 h versus >36 h after EAC criteria. EAC: lactate <4.0 mmol/L, pH >=7.25 or BE >=-5.5 mmol/L and no pressor requirement.	All 335 reached EAC criteria within 36 h. Complications: 44/269 (16.3%) with <=36 h fixation vs 22/66 (33.3%) with delayed fixation, p=0.0009. ICU LOS 4.35 vs 11.6 days; hospital LOS 9.52 vs 17.3 days; ventilation 2.57 vs 7.6 days. Protocol explicitly allowed urgent debridement/lavage with splinting or external fixation for open extremity fractures. Follow-up: in-hospital outcomes; some deaths after discharge noted.
4	Weinberg et al., 2015, USA [4]	Prospective cohort using EAC; prognostic analysis of time to resuscitation.	332 adults with ISS >=16; 376 fractures (femur 171, acetabulum 56, pelvic ring 70, cervical spine 6, thoracolumbar spine 73).	Major trauma patients managed prospectively with EAC at a Level I centre. Excluded low-energy injuries, skeletally immature patients, ISS <16, nonoperative definitive treatment and fixation before EAC resuscitation.	Time from injury to EAC resuscitation criteria as a continuous exposure; patients with complications compared with those without complications.	Mean time to EAC resuscitation 6.8 +/- 7.8 h (range 0.3-35.3). Complications in 66/332 (19.9%). ISS and time to EAC resuscitation independently predicted complications; each 1 h delay increased odds by 3.2% (OR 1.032, 95% CI 1.001-1.063, p=0.041). This is physiologic timing evidence, not debridement evidence. Follow-up: 6-month postoperative period.
5	Richards et al., 2020, USA [5]	Retrospective cohort from two academic trauma centres; propensity-weighted Cox analysis.	279 ICU patients with multisystem trauma and femoral shaft fractures; early fixation n=160, late fixation n=119.	Age 18-89 years, ISS >15, direct trauma-centre admission, ICU admission, femoral shaft fracture treated with internal fixation. Excluded head AIS >3, fewer than two lactate values within 24 h, ICU LOS <2 days, nonoperative/pathological femur fractures.	Early femur fixation <24 h versus late fixation >=24 h; admission lactate and 24-h time-weighted lactate.	Median admission lactate 3.0 mmol/L (IQR 1.9-4.3); median 24 h time-weighted lactate 2.3 mmol/L (IQR 1.6-3.2). 24 h time-weighted lactate predicted late fixation (OR 1.66 per 1 mmol/L, 95% CI 1.24-2.21, p<0.001). MOF occurred in 40/279 (14.3%); late fixation associated with MOF (HR 3.21, 95% CI 1.48-7.00, p<0.01). Follow-up: 28 days for MOF.
6	Sari et al., 2026, Northern Cyprus/Turkiye [6]	Retrospective observational cohort / prognostic factor study.	227 orthopaedic polytrauma patients; 111 underwent surgery (damage control 33, definitive surgery 78); open fracture 33.	Orthopaedic polytrauma patients admitted to a Level I trauma centre from January 2020 to December 2022. Included patients meeting at least one of six polytrauma criteria with admission pH, lactate, base deficit and haemoglobin recorded. Excluded incomplete records, isolated head injuries, missing key admission blood gas parameters and one treatment refusal.	Composite in-hospital complication versus no complication; ROC-derived laboratory thresholds; damage-control versus definitive surgery described.	Complications 61/227 (26.9%): mortality 30/61, wound infection 19/61, nonunion 5/61, circulatory problems 2/61, contractures 2/61, implant failure 1/61. Mean lactate 3.1 mmol/L (range 0.8-16.7). Lactate AUC 0.647; cut-off >3.4 mmol/L. In multivariable analysis, ISS and haemoglobin remained associated; lactate was not independently significant. Follow-up mean 8.92 months, but primary outcome was index hospitalization.
7	Oladipo et al., 2024, USA [7]	Retrospective single-centre cohort / prognostic factor study.	401 trauma activation patients undergoing tibial and/or femoral fixation; femur 201, tibia 138, multiple lower-extremity long-bone fractures 64; ballistic 154, blunt 247.	Trauma activation patients aged 16-65 years undergoing operative tibial and/or femoral fixation with initial lactate and blood alcohol levels available. Excluded age <16 or >65 years and absence of both lactate/alcohol levels.	Initial lactate groups: normal <2.5 mmol/L, intermediate 2.5-4.0 mmol/L, high >4.0 mmol/L; blunt versus ballistic subgroup analysis.	Postoperative morbidity 18/401 (4.5%); mortality 6/401 (1.5%). High lactate group morbidity 8/128 (6.3%) vs 3/137 (2.2%) in normal lactate. Higher lactate independently predicted postoperative morbidity (OR 1.305 per 1 mmol/L, p=0.015), longer LOS, higher cost and lower home disposition. Ballistic patients had higher lactate than blunt (4.0 +/- 2.4 vs 3.4 +/- 1.9, p=0.004), but lactate was not independently associated with morbidity in ballistic subgroup. Follow-up: index hospitalization.

Section 3: Results summary write-up (up to 500 words)

Seven cohort studies comprising 3,421 patients were included. One study directly evaluated first operative debridement and irrigation (D&I) timing in blunt open extremity fractures [1]. The remaining six studies evaluated lactate or metabolic acidosis as markers of physiological risk and timing of definitive fracture fixation in orthopaedic polytrauma or lower-extremity long-bone fracture cohorts [2-7].

Only Malhotra et al. defined early versus delayed debridement: first operative D&I <=8 h versus >8 h from presentation; cases with first D&I >24 h were excluded [1]. No included study compared debridement performed before lactate normalisation with debridement deliberately delayed until lactate normalised. Lactate was measured in Malhotra et al., but baseline lactate was similar in early and delayed D&I groups (3.3 vs 3.4 mmol/L) and was not used as an operative gatekeeper [1].

Lactate thresholds varied across studies. EAC studies used lactate <4.0 mmol/L, pH >=7.25 or base excess >=-5.5 mmol/L before definitive fixation [2-4]. Oladipo et al. stratified lactate as <2.5, 2.5-4.0 and >4.0 mmol/L [7]. Sari et al. identified a ROC-derived lactate cut-off >3.4 mmol/L [6]. Richards et al. used admission and 24 h time-weighted lactate as continuous measures of shock burden [5].

Infection outcomes were most directly reported by Malhotra et al.: infection occurred in 19% after D&I >8 h versus 11% after D&I <=8 h, with a stronger effect in lower-extremity open fractures (27% vs 10%) [1]. Union, reoperation and functional outcomes were not consistently reported. Mortality was reported in the polytrauma/fixation cohorts but not in relation to lactate-normalisation-based debridement. Overall, findings were heterogeneous in population and outcome, but consistently showed that lactate is a risk marker rather than a tested reason to defer debridement.

Section 4: Discussion (<1000 words)

4.1 Principal findings

The available evidence does not support deferring open-fracture debridement until serum lactate has normalised. Only one study directly evaluated timing of first operative D&I in open extremity fractures, and it found higher infection when D&I was delayed beyond 8 h, particularly in lower-extremity fractures [1]. The other studies consistently show that lactate and acidosis are important markers of systemic risk, but they address definitive fixation or in-hospital risk prediction rather than debridement itself [2-7].

4.2 Interpretation of evidence

Debridement/source control and definitive fixation/reconstruction are separate decisions. Debridement reduces bacterial burden, removes devitalised tissue and contamination, and allows staged control of the wound. Definitive fixation, flap reconstruction and prolonged reconstruction are larger physiological insults and should be timed according to resuscitation status. EAC evidence supports proceeding with definitive fixation after improvement of acidosis, not after complete lactate normalisation [2-4].

4.3 Biological rationale

Lactate rises when oxygen delivery is inadequate relative to tissue demand, commonly due to haemorrhage, shock or severe injury. Persistent hyperlactataemia identifies patients at risk of organ dysfunction and poor tolerance of prolonged surgery. However, an open fracture has an additional local biological problem: contamination and devitalised tissue. Delaying debridement may permit progression of necrosis and infection. The most coherent strategy is simultaneous resuscitation and source control: correct shock aggressively, but do not deliberately leave a contaminated open fracture untreated while waiting for lactate to normalise.

4.4 Strengths of current evidence

The included studies represent clinically relevant trauma cohorts. EAC studies used objective laboratory thresholds and clinically important outcomes [2-4]. Richards et al. used 24 h time-weighted lactate to describe shock burden [5]. Malhotra et al. directly studied debridement timing, used multivariable regression and analysed upper- and lower-extremity open fractures separately [1].

4.5 Limitations of current evidence

No randomized or prospective comparative study tested debridement delayed until lactate normalisation. Most evidence is observational and vulnerable to confounding by injury severity: sicker patients are more likely to have both elevated lactate and delayed surgery. Lactate thresholds varied across studies. Timing protocols also varied. Most studies did not report union, repeat debridement, reoperation or functional outcomes as primary outcomes for this question.

4.6 Applicability to clinical practice

A. Polytrauma patients: Elevated lactate should trigger urgent resuscitation, haemorrhage control and limitation of operative magnitude. It may justify postponing definitive fixation, flap reconstruction or prolonged procedures. It should not, by itself, justify deferring all debridement. In unstable patients, initial management should be damage-control debridement/lavage with temporary stabilisation and planned second look.

B. Isolated long-bone open fractures: In isolated patients without systemic instability, there is no evidence-based reason to wait for lactate normalisation before debridement. The direct open-fracture evidence suggests that avoidable delay, especially beyond 8 h in lower-extremity fractures, may increase infection [1].

4.7 Future research priorities

Future studies should prospectively enrol open long-bone fracture patients, record serial lactate clearance, document reasons for delay, and separate polytrauma from isolated injuries. Required outcomes should include deep infection, repeat debridement, reoperation, nonunion, flap failure, amputation, mortality, validated function and cost. Protocols should distinguish abbreviated source-control debridement from definitive fixation and reconstruction.

4.8 Overall interpretation

Lactate is a patient physiology marker, **not** a wound-cleanliness marker. The evidence supports lactate-guided resuscitation and staging of major definitive surgery. It does **not** support waiting for lactate normalisation before urgent debridement of open long-bone fractures.

Section 5: Risk of bias of included studies

Risk of bias was assessed using the JBI Critical Appraisal Checklist for Cohort Studies and visualised using ROBVIS. JBI score was calculated as Yes responses divided by applicable items x 100. Risk categories were: >=80% low risk, 60-79% moderate risk, and <60% high risk.

Study	JBI score	Risk category	Main reason / impact on recommendation
Malhotra 2014 [1]	81.8% (9/11)	LOW	Direct debridement evidence; retrospective registry design and incomplete follow-up details remain limitations.
Sari 2026 [6]	72.7% (8/11)	MODERATE	Retrospective single-centre design, broad composite outcome and incompletely described follow-up/missing-data handling; indirect to debridement.
Weinberg 2015 [4]	81.8% (9/11)	LOW	Prospective EAC cohort with objective exposure and independent adjudication; indirect to debridement.
Vallier 2013 [2]	81.8% (9/11)	LOW	Large retrospective cohort with objective physiological measures; residual confounding and incomplete follow-up details remain; indirect to debridement.
Vallier 2015 [3]	63.6% (7/11)	MODERATE	Non-randomised protocol implementation with surgeon/operational protocol violations and historical comparison; important but indirect.
Richards 2020 [5]	81.8% (9/11)	LOW	Two-centre cohort with advanced adjustment; ICU femoral shaft population limits directness to debridement.
Oladipo 2024 [7]	81.8% (9/11)	LOW	Retrospective cohort; missing data were not imputed and rare outcomes were underpowered; indirect but supportive lactate evidence.

ROBVIS interpretation: Across 77 domain-level judgements, 60 were low risk (77.9%), 16 were moderate/some concerns (20.8%) and 1 was high risk (1.3%). At overall study level, 4/7 studies were low risk (57.1%), 2/7 moderate/some concerns (28.6%) and 1/7 high risk (14.3%). The commonest concerns were follow-up completeness and strategies for incomplete follow-up. These concerns limit causal inference, but they do not invalidate the direction of the recommendation.

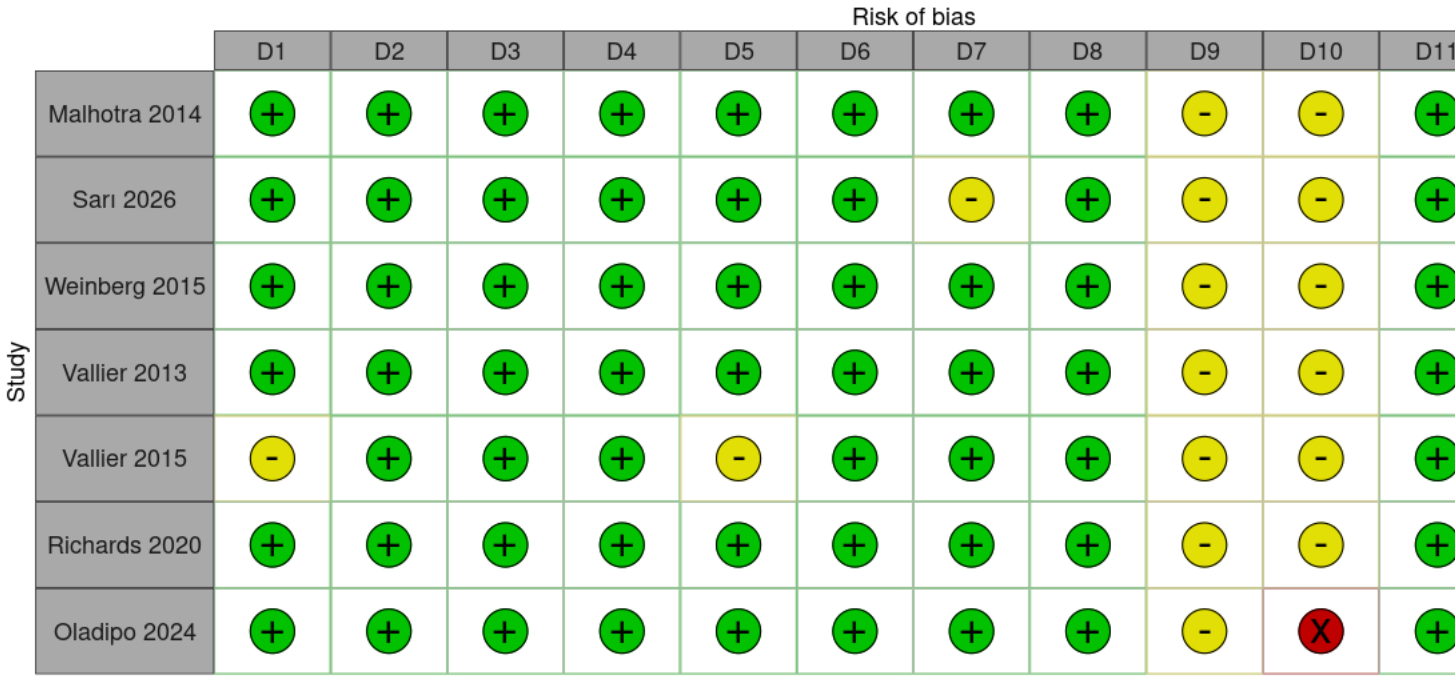

Figure 1. ROBVIS risk-of-bias visualisation for included cohort studies using mapped JBI cohort domains.

Section 6: Response / Recommendation

Debridement should not be deferred solely until serum lactate has normalised. The evidence supports using lactate to assess shock burden and to guide resuscitation and operative magnitude, but **not as a stand-alone prerequisite** for urgent open-fracture source control.

Section 7: Level / strength of evidence

LOW. Seven cohort studies were included, but only one directly evaluated timing of debridement in open extremity fractures [1]. The remaining studies provide indirect but clinically relevant evidence on lactate, acidosis, resuscitation and timing of definitive fracture fixation [2-7]. Evidence is limited by observational design, confounding by injury severity, heterogeneity in lactate thresholds, incomplete follow-up reporting and lack of direct comparative data on debridement delayed until lactate normalisation. Consistency is acceptable for the general principle that lactate is a marker of physiological risk, but directness and precision for the exact review question are low.

References

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