Poster #1

NO TIME TO PLAY: RAPID POLICE TRANSPORT FOR URBAN PENETRATING TRAUMA

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Introduction: Rapid police transport (RPT) in the urban setting has shown promise in decreasing mortality associated with penetrating trauma, especially in the setting of acute hemorrhage. This is likely related to reduced response, scene, and transport times. The goal of this study was to quantify police transport times and characterize police officers (PO) participation.

Methods: A sample of PO transporting penetrating trauma patients was conducted from 4/1/2025 to 10/1/2025. Surveys and RPT times were obtained from PO at the time of drop off. Descriptive statistics summarized qualitative data. Mann-Whitney U tests compared RPT data with Emergency Medical Services (EMS) transport times over the designated period.

Results: There were 127 RPTs and 37 (29%) PO completed the survey. A majority of PO (28, 76%) had been in the department for $\geq$ 5 years and participated in > 10 RPTs. Most PO (28, 76%) had additional medical training as Stop the Bleed (9, 24%) or First Responder (19, 51%). Almost all PO (34, 92%) believe the RPT program improves PO relationships with the community. Other PO were present in 29 (78%) RPT, with EMS on scene in 3 cases (8%). Signs of life were present on hospital arrival in 29 (80%) RPT. Most PO (34, 92%) feel RPT is part of their job and find the practice rewarding. Median RPT times are noted in Table 1. Of included RPT, 32 (87%) were less than 10 minutes from notification to trauma center arrival. Compared to 38 penetrating trauma transports by EMS, median RPT to scene, to trauma center, and total times were significantly shorter (Table 1).

Conclusion: PO are invested in RPT policies and find the practice rewarding. In a majority of cases, RPT leads to transport times of less than 10 minutes between injury and definitive care. This reinforces the concept of scoop and run. Future research is needed to evaluate how this practice can be replicated in other urban areas with potential for ultra-short transport and faster time to hemorrhage control.

	RPT	EMS	p-value
Notification to Scene Time (min), median (IQR)	1 [1,2]	9 [7,15]	$p < 0.01$
Scene to Hospital Time (min), median (IQR)	5 [4,8]	8 [7,12]	$p < 0.01$
Total Response Time (min), median (IQR)	7 [5,9]	24 [18,37]	$p < 0.01$

Poster #2

GEOSPATIAL ANALYSIS OF NON-SCENE TRAFFIC FATALITIES IN RELATION TO BLOOD CENTERS IN THE UNITED STATES

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Introduction: Hemorrhage is the leading cause of early preventable mortality after motor vehicle collisions. Survival depends on timely and balanced resuscitation but there are known gaps in blood product distribution particularly in the rural US. The objective of this study was to evaluate variation in rates of non-scene traffic fatalities by region and proximity to blood centers (BCs) and trauma centers (TCs).
Methods: 30- and 60-minute drive time from level I-III TCs and accredited BCs (defined as blood banks and transfusion centers), as well as coordinates for non-scene traffic fatalities (01/2018-01/2024) were mapped in ArcGIS. Death rates per 100,000 total population were calculated and stratified by region and proximity to BCs and TCs.
Results: 102,611 non-scene traffic fatalities were recorded. Death rates in the South were consistently highest (47.2 in areas beyond 60 minutes from a BC compared to 22.3 in the Northeast) (Table 1). To account for potential confounding by proximity to TCs, death rates were calculated for areas within 60 minutes of a TC and BC (29.7) and compared to death rates for areas within 60 minutes of a TC but beyond 60 minutes from a BC (39.3), as well as areas beyond 60 minutes from a TC (40.5). Regional differences persisted, with a 32% increase in the death rates in the South and West when moving beyond 60 minutes from a BC compared to 17% and 13% increases in the Midwest and Northeast, respectively.
Conclusion: Non-scene traffic fatalities differ by region in the US. While distance from TCs and BCs are likely correlated, distance from a BC seems to play an independent role in mortality. Timely resuscitation with prehospital blood transfusion may offer a solution in rural areas.

Table 1. Death rate by region and proximity in minutes to a blood center

	Overall	Within 30	Beyond 30	Within 60	Beyond 60
Total	30.4	27.3	36.8	29.2	37.9
Midwest	28.3	26.7	31.1	27.5	33.0
Northeast	22.2	21.4	25.9	22.2	22.3
South	39.0	34.6	45.3	37.0	47.2
West	24.2	22.1	30.3	22.8	34.0

Poster #3

MECHANISM MATTERS: NATURAL LANGUAGE PROCESSING-DERIVED PHYSIOLOGIC SIGNALS REVEAL DISTINCT ERROR PATHWAYS IN TRAUMA TRIAGE

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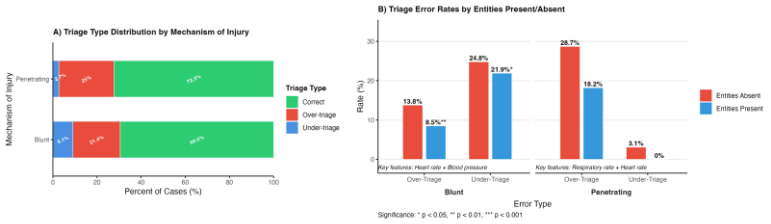
Introduction: Trauma triage errors, and the communication drivers of these errors, remain poorly understood. Pre-hospital handoffs occur under time pressure and often contain incomplete information. In this study, we applied computational language processing to EMS-to-hospital telephone communications to identify mechanism-specific triage "error signatures."
Methods: We analyzed 1,350 recordings of EMS-to-hospital trauma activation handoffs from an adult Level 1 trauma center. Automated speech recognition (*Whisper,* ~2-5% benchmark word error rate) converted audio to written transcripts. Injury Severity Score (ISS), determined post hoc, was used as the reference standard for triage level and compared with clinician activation decisions. Automated text analysis then identified clinical details present in transcripts and was captured as both numerical and qualitative representations of each entity.
Results: Triage performance differed by injury mechanism, with penetrating trauma demonstrating frequent over-triage (25.0%, CI 20.2–30.6%) but minimal under-triage (2.7%, CI 1.3–5.4%), whereas blunt trauma showed the inverse pattern; high under-triage (21.4%, CI 19.5–23.5%) and lower over-triage (9.1%, CI 7.8–10.6%) (Figure 1a). In penetrating trauma, the communication of respiratory rate and heart rate together reduced over-triage from 28.7% to 18.2% (p = 0.085). In blunt trauma, both heart rate and blood pressure together reduced over-triage from 13.8% to 8.5% (p=0.007) and under-triage from 24.8% to 21.9% (p=0.026) (Figure 1b).
Conclusions: Trauma triage errors follow structured, mechanism-specific patterns. Blunt trauma errors are linked to missed hemodynamic instability, while penetrating trauma errors relate to ventilatory risk. Identifying these communication "error signatures" supports development of mechanism-focused handoff checklists to reduce triage error.



Poster #4

POST-INJURY IMMUNE DYNAMICS IN PATIENTS WITH BLUNT TORSO TRAUMA: A PROSPECTIVE COHORT STUDY

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Introduction: Cytokines regulate hemodynamic and immune responses post-trauma to maintain homeostasis. Dysregulated cytokine release triggers systemic inflammation. However, individual variation often complicates the predictive model, as patients with comparable injury severity exhibit divergent clinical trajectories and initial treatment responses. This study evaluates early immune dynamics in blunt torso trauma by characterizing temporal changes in plasma cytokines and their associations with injury severity.

Methods: In this prospective study, 45 hemodynamically stable patients with blunt torso trauma were analyzed. Plasma samples were collected on days 1, 3, and 5 post-injury and analyzed to assess a Human essential 13-cytokine panel using flow cytometry bead-based methods using BD FACSAria TM Fusion Cell sorter. The complement anaphylatoxins C3a and C5a were measured using ELISA.

Results: The severe injury cohort (ISS>15, n=25)was significantly younger (median 30 years) and primarily involved in road traffic injuries. Severe injuries were associated with longer hospital stays, elevated leukocyte counts, and neutrophil-predominant responses with an elevated neutrophil to lymphocyte ratio (NLR) of 8.3. Plasma cytokine levels revealed a time-dependent inflammatory signature, marked by early elevations in IL-6, IL-8, IL-10, MCP-1, TGF-β1, and TNF-α. The cytokines showed an association with severity analysis. The complement anaphylatoxin C3a were also increased.

Conclusion: Early cytokine profiling reveals distinct, time-dependent immune alterations following blunt torso trauma. IL-6, IL-10, IP-10, TNF-α, TGF-β1, and IL-8 were elevated in severe chest injury, and MCP-1 for abdominal and combined injuries. This suggests that molecular signatures could enhance clinical precision. Integrating these markers with clinical parameters may allow early identification of patients at risk for immune dysregulation.

Poster #5

SAME STORM, DIFFERENT DATASET: REPRODUCIBILITY OF DYNAMIC LEUKOCYTE TRANSCRIPTOME CHANGES AFTER BLUNT TRAUMA

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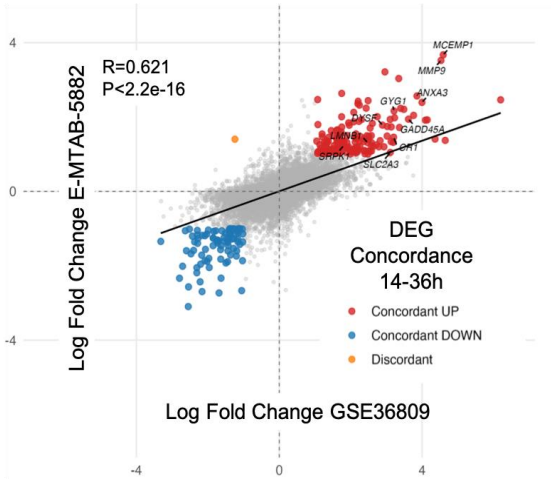
Introduction: Dysregulation of the inflammatory "genomic storm" in response to severe trauma is thought to contribute to multiple organ failure, yet reliable molecular features distinguishing maladaptive from appropriate responses remain unclear. We hypothesized that a core, conserved molecular program of dynamic transcriptional changes would emerge across independent human datasets.
Methods: Secondary analysis of three publicly-available transcriptomic datasets was performed. Severe trauma patients with ISS ≥15 and evidence of shock on arrival were included. Mild trauma patients with ISS<15 were analyzed separately. Time points were binned as 0-6h (immediate), 14-36h (early) and 60-90h (late) postinjury. Differentially-expressed genes (DEGs) were defined by log2 fold change ≥1 and p-value <0.05. The intersection between DEGs independently identified in each dataset maximized specificity. In parallel, a meta-analytic approach maximized sensitivity for detecting shared injury-associated transcriptional changes.
Results: 372 severe trauma patients were included. Overlap between leukocyte datasets was highly significant (immediate, p=2.85e-64, early, p=1.76e-179, late p=3.39e-125). Specific genes implicated immediate increased neutrophil function and decreased interferon-gamma signaling, with increased adaptive immune function and hypoxia-inducible factor signaling and decreased T cell function at early and late timepoints. An attenuated response was seen in mild trauma patients. Notably, genes previously implicated in neutrophil invasiveness were consistently upregulated not only in neutrophil-specific datasets but also in whole blood leukocyte transcriptomes, suggesting that neutrophil-driven programs are detectable at the level of bulk circulating immune cells.
Conclusion: There is high concordance in gene expression changes within 90 hours of severe blunt trauma, despite differences in patient population, transcriptional assay platform, and evolution of resuscitation techniques over time. This conservation reveals a distinct, early and reproducible transcriptional program that may help distinguish regulated and maladaptive inflammation after severe injury.



Poster #6

POLYTRAUMA AND INNATE IMMUNE CELL DYSFUNCTION DELAY WOUND HEALING IN A MODEL OF SOFT TISSUE INJURY AND INFECTION

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Introduction: While the dynamics of wound healing have been extensively studied, the contribution of polytrauma has not been well established. In this study, we characterize the effect of intraabdominal trauma on the immune process of soft tissue healing of a remote wound.

Methods: C57Bl6 and G-Protein Receptor-2 (GRK2)-myeloid-specific null mice underwent a 6mm dorsal punch wound ± *S. aureus* inoculation with and without standardized liver crush injury. Wound healing was measured by calipers daily over 14 days with future plans to include automated image-based measurement of wound size, depth, tissue classification, temperature, and presence of bacterial autofluorescence. Skin was harvested for further analyses.

Figure 1

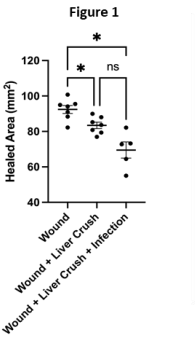
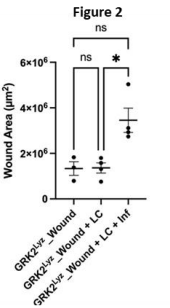


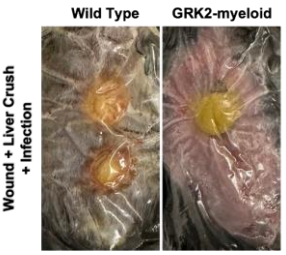
Figure 2



Results: Animals subjected to soft tissue injury with concomitant liver crush injury showed significantly delayed wound healing with less healed wounds at 14 days compared to soft tissue injury alone with a trend (p=0.0930) to even slower healing in the polytrauma+ inoculated mice (**Figure 1**). To study the contribution of GRK2, a GPCR critical for signaling in innate immune cell function, GRK2-myeloid cell null mice were studied. In the absence of GRK2, application of *S. aureus* significantly impaired wound healing (**Figure 2&3**).

Conclusion: In a novel 2-hit skin punch wound+liver crush injury that wound healing was significantly slowed compared to wound only which was further slowed in the presence of a bacterial infection. Further, GRK2 in myeloid cells (macrophages and neutrophils) plays an important role in wound healing in the presence of superficial infection. Based on this and published work, we find that sterile tissue injury releases Danger Associated Molecular Pattern molecules (DAMPs) that render the innate immune response dysfunctional, leaving barrier sites like the skin susceptible to bacterial infection. Ongoing work is investigating specific functional changes in innate immune cells caused by DAMPs that lead to poor wound healing.

Figure 3



Poster #7

IMMEDIATE POST-INJURY EXTRACORPOREAL CO2 REMOVAL AS A PH STABILIZATION INTERVENTION ASSOCIATED WITH SURVIVAL IN A HIGH-MORTALITY POLYTRAUMA MODEL

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Introduction: Increased Lactate (Lac) is associated with mortality, and its clearance with survival. We evaluated Lac clearance trajectories in injured swine, with or without immediate post-injury extracorporeal CO_2 removal ($ECCO_2R$), and hypothesized that failure to achieve Lac<2 mmol/L by or before 24 h is associated with increased mortality.
Methods: Anesthetized mechanically ventilated swine (56.14 ± 0.74 kg, n=53) received ICU care alone (n=19), or ICU + bilateral pulmonary contusion (PC, n=22), or PC + traumatic brain injury (TBI, n=12). Animals were randomized to $ECCO_2R$ n=42 or non-$ECCO_2R$, n=11. Statistics: Mann–Whitney U test; Fisher's exact test with odds ratios (OR); multivariable logistic regression.

Results:

Variable	Overall (S: 68% NS: 32%)	$ECCO_2R$ (S: 74% NS: 26%)	Non-$ECCO_2R$ (S: 45% NS: 55%)
Baseline Lac (mmol/L)	S 2.08±0.83 vs NS 2.64±2.52; ns	S 2.13±0.84 vs NS 2.41±1.19; ns	S 1.78±0.72 vs NS 3.07±4.15; ns
Peak Lac (mmol/L)	S 8.68±4.47 vs NS 11.16±4.05; ns	S 8.43±4.33 vs NS 10.47±4.19; ns	S 10.2±5.52 vs NS 12.42±3.81; ns
pH	S 7.50±0.09 vs NS 7.41±0.14; *	S 7.51±0.08 vs NS 7.44±0.13; *†‡	S 7.43±0.09 vs NS 7.34±0.14; *†‡
pCO_2 (mmHg)	S 30.22±8.22 vs NS 32.17±9.04; *	S 28.55±6.92 vs NS 28.71±6.55; †‡	S 39.67±8.63 vs NS 39.62±9.18; †‡
Lac <2 mmol/L by 24 h	OR 0.23 [CI:0.07, 0.81] *	OR 0.13 [CI:0.03:0.65] *	OR 0.5 [CI:0.06, 6.66] ns
Lac <2 mmol/L by 18 h	OR: 0.21 [CI:0.07, 0.74] *	OR 0.13 [CI:0.03:0.65] *	OR 0.67 [CI:0.08, 6.43] ns
	Multivariable Logistic Regression		
Survival Model	$ECCO_2R$ (vs Non-$ECCO_2R$)	OR: 4.74 [CI:1.06, 21.27] *	
	Time to Lac <2 mmol/L (per h)	OR: 0.88 [CI:0.78, 0.99] *	
Acid/Base-Adjusted Model	$ECCO_2R$ (vs Non-$ECCO_2R$)	OR: 1.09 [CI:0.10, 11.69] ns	
	Time to Lac <2 mmol/L (per h)	OR: 0.94 [CI:0.80, 1.10] ns	
	min pH (per 0.1 pH units)	OR: 2.69[CI:1.13, 6.36] *	
	mean pCO_2	OR: 0.95 [CI:0.80, 1.13) ns	

Key
S: Survivors; NS: Non-Survivors
* $p < 0.05$ (w/in cohort S vs NS)
† $p < 0.05$ (S $ECCO_2R$ vs S Non-$ECCO_2R$)
‡ $p < 0.05$ (NS $ECCO_2R$ vs NS Non-$ECCO_2R$)
ns, $p > 0.05$
p-values are unadjusted

Conclusion: Lac<2 mmol/L by 18 and 24h, and $ECCO_2R$ were significantly associated with survival. In a multivariate regression model, pH normalization was a predictor of survival. $ECCO_2R$ is an early potent pH stabilization intervention in trauma with severe lactic acidosis and should be considered for rapid metabolic correction in polytrauma. Studies are ongoing to demonstrate the lifesaving potential of early $ECCO_2R$.

Poster #8

PHYSIOLOGY MATTERS: WHOLE BLOOD OUTPERFORMS PACKED RED BLOOD CELLS IN PREHOSPITAL TRAUMA

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Introduction: Recently, prehospital care has shifted towards early resuscitation with blood products. However, literature lacks adequate comparison between the various products in the prehospital setting. We compared the use of prehospital whole blood (WB) to packed red blood cells (pRBC) and hypothesized that WB would better normalize shock index and reduce overall mortality.

Methods: This was a retrospective analysis of prehospital blood (PHB) transfusion data (2021-2025) from two urban EMS systems serving multiple level I trauma centers. Adult patients who sustained any traumatic injury and received PHB were included. Patient and injury variables, change in shock index (SI) from prehospital to hospital, prehospital time intervals, and mortality were abstracted. Multivariate regression controlled for differences in baseline characteristics.

Results: A total of 626 patients (WB=252; pRBC=374) were included for analysis. The pRBC group was older (30 vs 38 years, p<0.01), more frequently White (9.1 vs 25.8%, p<0.001), and more frequently bluntly injured (6.0 vs 30.7%, p<0.01). There was no statistical difference in injury severity or prehospital/ED vital signs between groups. Median change in SI was significantly lower in the pRBC group (-0.54 vs –0.33, p=0.01). 24-Hour mortality (19.0 vs 27.0%, p=0.02) and in-hospital mortality (23.4 vs 34.8, p<0.01) were lower in the WB group. On subgroup analysis of penetrating injuries only, 24-Hour mortality (19.0 vs 31.7, p<0.01) and in-hospital mortality (22.4 vs 35.5, p<0.01) remained lower in the WB group. When controlling for age, injury mechanism, and prehospital SI, WB significantly reduced odds of in-hospital mortality when compared to pRBC (OR 0.52; p<0.01).

Conclusion: Prehospital resuscitation with either WB or pRBC normalizes shock index at ED arrival following injury. However, WB more significantly lowers shock index and reduces odds of in-hospital mortality.

Variable	Whole Blood (n=252)	PRBC (n=374)	p-value
Male, n (%)	216 (85.7)	323 (86.4)	0.818
Blunt, n (%)	15 (6.0)	115 (30.7)	<0.001
Injury Severity Score	18 (10-30)	22 (12-34)	0.061
PH GCS	13 (7-15)	13 (3-15)	0.053
PH SBP	80 (69-91)	82 (66-107)	0.182
PH Heart Rate	108 (87-121)	102 (70-130)	0.161
PH Shock Index	1.30 (0.97-1.65)	1.20 (0.88-1.50)	0.011
Average (SD) Units PHB	1 (1)	2 (1)	0.042
ED GCS	15 (13-15)	15 (12-15)	0.671
ED SBP	107 (80-128)	102 (63-130)	0.354
ED Heart Rate	82 (106-62)	84 (47-110)	0.889
ED Shock Index	0.74 (0.62-1.09)	0.84 (061-1.06)	0.300
Average (SD) transfusion requirements			
pRBC	3 (6)	6 (12)	<0.001
FFP	2 (5)	6 (12)	<0.001
Platelets	2 (1)	4 (1)	0.002
24 Hour Mortality	48 (19.0)	101 (27.0)	0.022
Penetrating Only	45 (19.0)	82 (31.7)	0.001
In-hospital Mortality	59 (23.4)	130 (34.8)	0.002
Penetrating only	53 (22.4)	92 (35.5)	0.001

Poster #9

MACHINE LEARNING-BASED CORRELATION OF VENOUS MORPHOLOGY WITH HEMORRHAGIC SHOCK STAGES USING VOLUMETRIC M-MODE ULTRASOUND IN A SWINE MODEL

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Introduction: Hemorrhagic shock remains a leading cause of preventable death, with recognition often delayed until compensatory failure. We sought to define reproducible central venous static and dynamic physiologic signatures of hemorrhage and resuscitation. To enable quantitative multi-vessel assessment, we developed volumetric M-Mode (VMM), a novel ultrasound method generating continuous venous datasets via lateral sweep and tilt.

Methods: Thirteen female Yorkshire pigs underwent staged hemorrhage (15%, 30%, 40%, 50% blood loss) followed by crystalloid resuscitation and fluid overload. The subclavian (SV), inferior vena cava (IVC), internal jugular (IJV), and femoral veins (FV) were imaged with VMM at baseline and each stage. Static collapse was defined as percent change in mean diameter from baseline; dynamic compliance as respiratory distensibility. Stage effects were analyzed across vessels.

Results: Progressive hemorrhage (Stages 0–4) produced a monotonic decline in venous diameter (−6% per stage; $p<0.001$), with maximal reductions of ~25–40% at Stage 4, most pronounced in the IJV and IVC; diameters partially normalized after resuscitation. Respiratory distensibility varied by stage (global $p=0.026$), increasing during intermediate-to-severe shock in the IJV and IVC, with minimal FV change, and decreasing after resuscitation and fluid overload. VMM datasets also permitted extraction of heart and respiratory rates consistent with intraoperative measurements.

Conclusion: Progressive hemorrhage and resuscitation generate reproducible central venous static and dynamic signatures that track shock severity across vessels. VMM provides a volumetric, operator-independent platform to quantify these patterns, supporting its potential for earlier hemorrhage detection and physiologic monitoring in trauma.