Interactive Influential Papers of 2019

19 November 2019

Facilitated by Dr Brian Doyle, FACEM

Objectives

- Go over 3 papers in 90 minutes
- Brief 5 minute overview of the paper (big picture)
- Interactive audience discussion

CRASH-3 Trial

Published online: 14 October 2019

- CRASH-1 trial?
- CRASH-2?
- CRASH-3…
- CRASH-4?

Effects of tranexamic acid on death, disability, vascular occlusive events and other morbidities in patients with acute traumatic brain injury (CRASH-3): a randomised, placebo-controlled trial

*The CRASH-3 trial collaborators**

Summary

Background Tranexamic acid reduces surgical bleeding and decreases mortality in patients with traumatic extracranial bleeding. Intracranial bleeding is common after traumatic brain injury (TBI) and can cause brain herniation and death. We aimed to assess the effects of tranexamic acid in patients with TBI.

Methods This randomised, placebo-controlled trial was done in 175 hospitals in 29 countries. Adults with TBI who were within 3 h of injury, had a Glasgow Coma Scale (GCS) score of 12 or lower or any intracranial bleeding on CT scan, and no major extracranial bleeding were eligible. The time window for eligibility was originally 8 h but in 2016 the protocol was changed to limit recruitment to patients within 3 h of injury. This change was made blind to the trial data, in response to external evidence suggesting that delayed treatment is unlikely to be effective. We randomly assigned (1:1) patients to receive tranexamic acid (loading dose 1 g over 10 min then infusion of 1 g over 8 h) or matching placebo. Patients were assigned by selecting a numbered treatment pack from a box containing eight packs that were identical apart from the pack number. Patients, caregivers, and those assessing outcomes were masked to allocation. The primary outcome was head injury-related death in hospital within 28 days of injury in patients treated within 3 h of injury. We prespecified a sensitivity analysis that excluded patients with a GCS score of 3 and those with bilateral unreactive pupils at baseline. All analyses were done by intention to treat. This trial was registered with ISRCTN (ISRCTN15088122), ClinicalTrials.gov (NCT01402882), EudraCT (2011-003669-14), and the Pan African Clinical Trial Registry (PACTR20121000441277).

Results Between July 20, 2012, and Jan 31, 2019, we randomly allocated 12 737 patients with TBI to receive tranexamic acid (6406 [50·3%] or placebo [6331 [49·7%], of whom 9202 (72·2%) patients were treated within 3 h of injury. Among patients treated within 3 h of injury, the risk of head injury-related death was 18·5% in the tranexamic acid group versus 19·8% in the placebo group (855 *vs* 892 events; risk ratio [RR] 0·94 [95% CI 0·86–1·02]). In the prespecified sensitivity analysis that excluded patients with a GCS score of 3 or bilateral unreactive pupils at baseline, the risk of head injury-related death was 12·5% in the tranexamic acid group versus 14·0% in the placebo group (485 *vs* 525 events; RR 0·89 [95% CI 0·80–1·00]). The risk of head injury-related death reduced with tranexamic acid in patients with mild-to-moderate head injury (RR 0·78 [95% CI 0·64–0·95]) but not in patients with severe head injury (0·99 [95% CI 0·91–1·07]; p value for heterogeneity 0·030). Early treatment was more effective than was later treatment in patients with mild and moderate head injury (p=0·005) but time to treatment had no obvious effect in patients with severe head injury (p=0·73). The risk of vascular occlusive events was similar in the tranexamic acid and placebo groups (RR 0·98 (0·74–1·28). The risk of seizures was also similar between groups (1·09 [95% CI 0·90–1·33]).

Published Online October 14, 2019 https://doi.org/10.1016/S0140-6736(19)32233-0

See Online/Comment https://doi.org/10.1016/S0140-6736(19)32312-8

*Members listed at end of paper

For the Arabic translation of the abstract see Online for appendix 1

For the Chinese translation of the abstract see Online for appendix 2

For the French translation of the abstract see Online for appendix 3

For the Hindi translation of the abstract see Online for appendix 4

For the Japanese translation of the abstract see Online for appendix 5

For the Spanish translation of the abstract see Online for appendix 6

For the Urdu translation of the abstract see Online for appendix 7

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CRASH 3 trial

- Does tranexamic acid work in patients with TBI?
- RCT placebo controlled trial in 175 hospitals in 29 countries!
- **Inclusion**: Adults with moderate or severe TBI (GCS ≤12) within 3 hours of injury or any haemorrhage on CT
- **Excluded:** major extracranial haemorrhage

(Some changes to the study design during recruitment)

CRASH 3: methods

- Good randomisation and blinding
- **Intervention**: 1gm tranexamic acid over 10 minutes followed by 1gm infusion over 8 hours vs. matched placebo
- **Primary outcome**: head injury related death by 28 days
- Prespecified analysis that excluded GCS 3 or bilateral unreactive pupils
- Secondary outcomes: lots- including safety outcomes

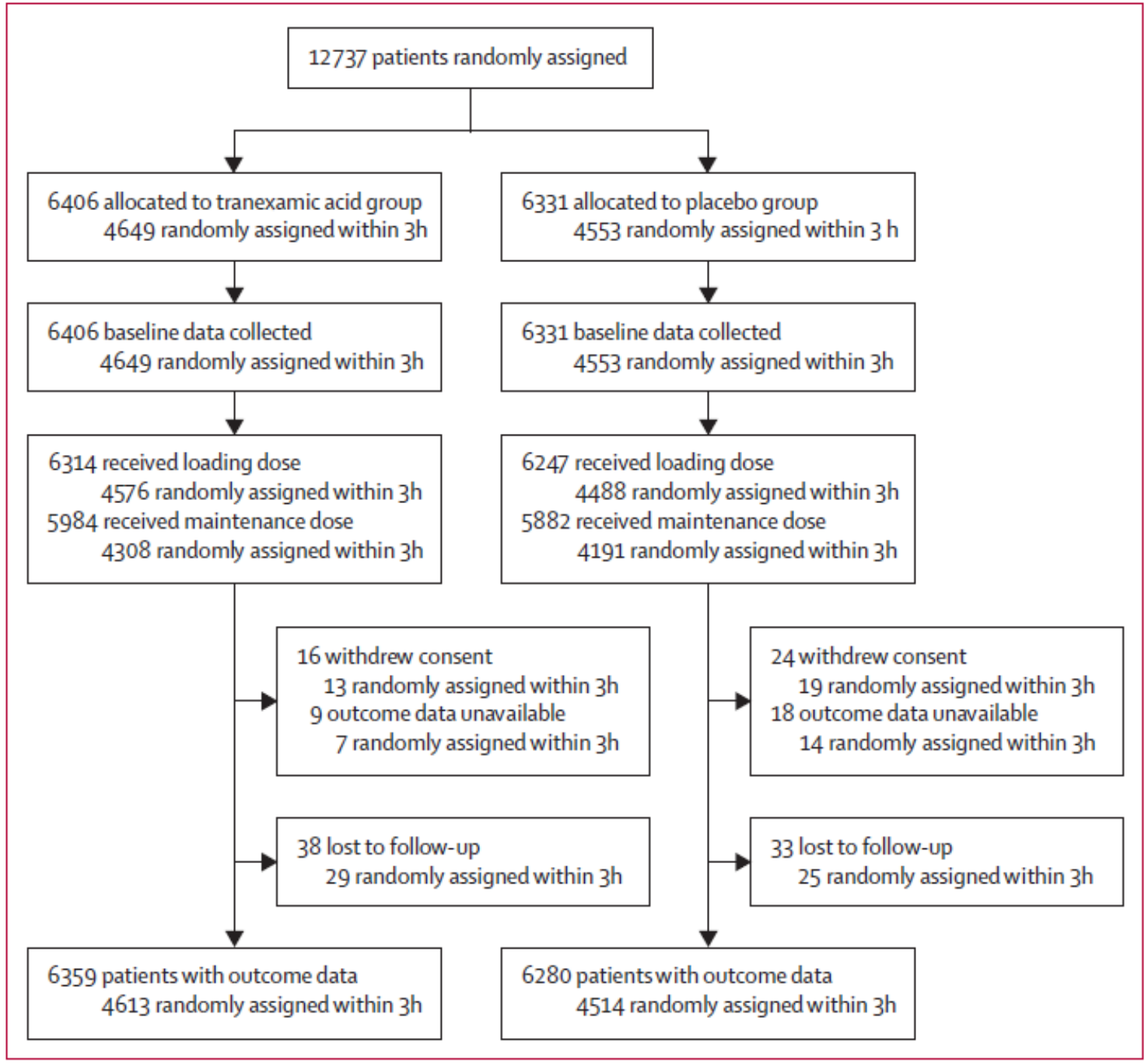


Figure 1: **Trial profile**

CRASH 3: participating countries

- **Pakistan (4567)**
- **UK (3143)**
- Malaysia (1567)
- Georgia (771)
- Spain (425)
- Nigeria (409)
- Colombia (335)
- Nepal (225)
- Albania (214)
- Japan (165)
- UAE (126)
- Myanmar (121)
- Cameroon (116)
- Afghanistan (87)
- Mexico (79)
- Italy (72)
- Iraq (55)
- Cambodia (45)
- Zambia (44)
- Romania (35)
- El Salvador (28)
- Egypt (20)
- Slovenia (15)
- Ireland (12)
- PNG (10)
- Canada (7)
- Jamaica (7)
- Indonesia (6)
- Kenya (1)

CRASH 3 results

	Tranexamic acid	Placebo	Risk ratio (95% CI)
All	855/4613 (18·5%)	892/4514 (19·8%)	0·94 (0·86–1·02)
Excluding patients with GCS score of 3 or bilateral unreactive pupils*	485/3880 (12·5%)	525/3757 (14·0%)	0·89 (0·80–1·00)

GCS=Glasgow Coma Scale. *Prespecified sensitivity analysis.

Table 2: **Effect of tranexamic acid on head injury-related death in patients randomly assigned within 3 h of injury**

- NNT 77 or 66
- No difference in safety outcomes
- Some limitations reported

CRASH 3 Trial:

- *"…given the absence of any adverse effects in this trial, the implications of wrongly concluding that tranexamic acid is ineffective are likely to be far more consequential than are those of wrongly concluding that tranexamic acid is effective."*

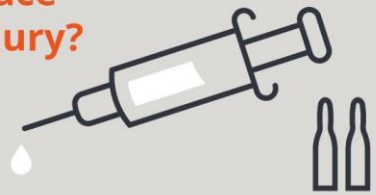
CRASH 3: Authors conclusion

“Our results show that tranexamic acid is safe in patients with TBI and that treatment within 3 hours of injury reduces head injury-related death. Patients should be treated as soon as possible.”

CRASH 3: Thoughts?

Can tranexamic acid (TXA) reduce death from traumatic brain injury?

TXA is a drug that prevents bleeding by stopping blood clots from breaking down



CRASH 3 Trial

12,737 Patients

29 Countries

175 Hospitals

Results

TXA could save **1 in 5** people who would have died following a mild or moderate head injury

Time is vital - TXA is more effective the earlier it is given

Every **20 minute delay** leads to a **10%** reduction in effectiveness

TXA is **safe to give**, there's no evidence of side effects and no increase in disability

CRASH-3 Trial (2019)

Find out more at: crash3.lshtm.ac.uk

High sensitive Troponin:

5 Nov 2019

Circulation

ORIGINAL RESEARCH ARTICLE

A Randomized Trial of a 1-Hour Troponin T Protocol in Suspected Acute Coronary Syndromes

The Rapid Assessment of Possible Acute Coronary Syndrome in the Emergency Department With High-Sensitivity Troponin T Study (RAPID-TnT)

BACKGROUND: High-sensitivity troponin assays promise earlier discrimination of myocardial infarction. Yet, the benefits and harms of this improved discriminatory performance when incorporated within rapid testing protocols, with respect to subsequent testing and clinical events, has not been evaluated in an in-practice patient-level randomized study. This multicenter study evaluated the noninferiority of a 0/1-hour high-sensitivity cardiac troponin T (hs-cTnT) protocol in comparison with a 0/3-hour masked hs-cTnT protocol in patients with suspected acute coronary syndrome presenting to the emergency department (ED).

METHODS: Patients were randomly assigned to either a 0/1-hour hs-cTnT protocol (reported to the limit of detection [<5 ng/L]) or masked hs-cTnT reported to <29 ng/L evaluated at 0/3-hours (standard arm). The 30-day

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April 2011: Roche 5th generation hsTnT

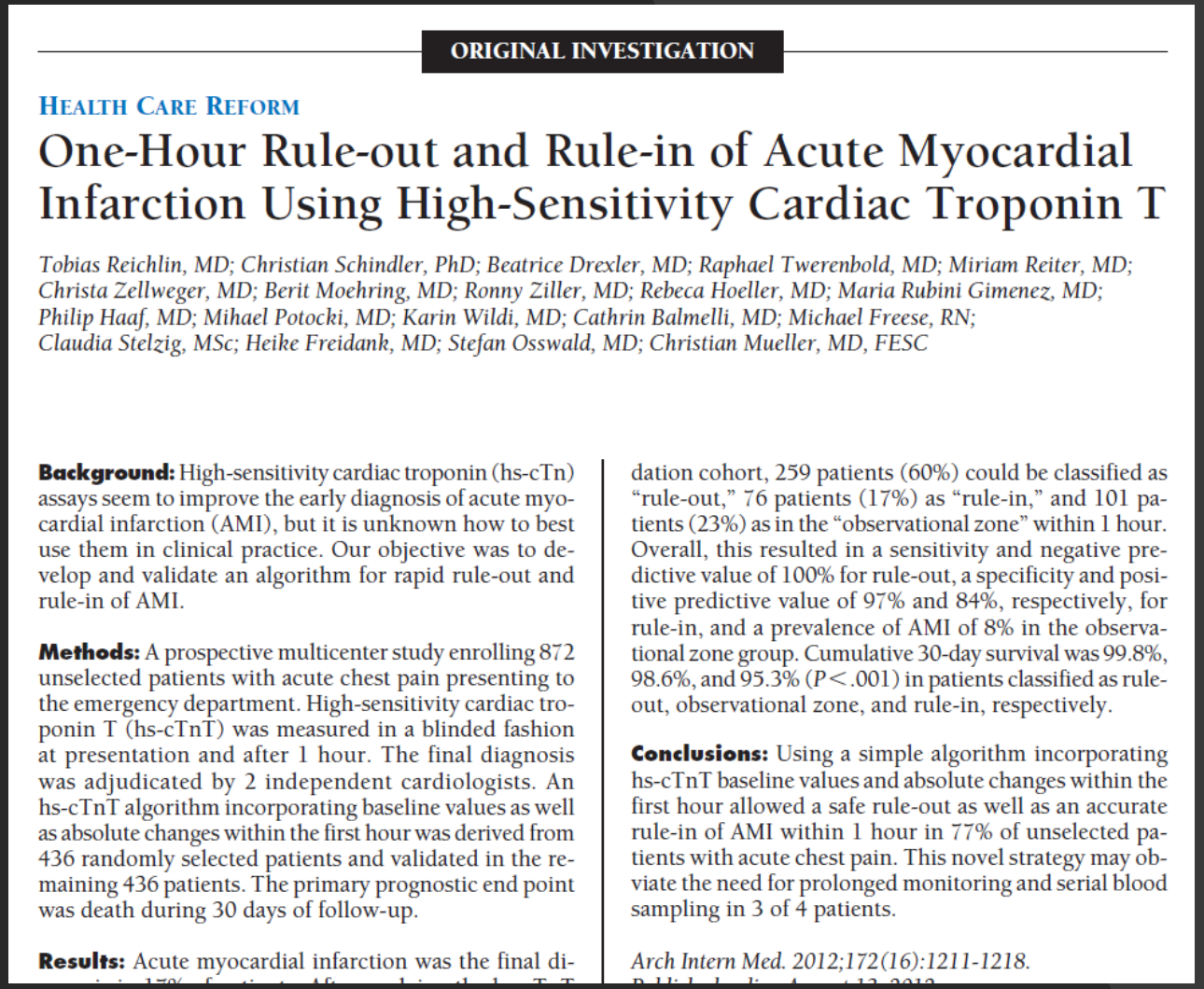
“Routine practice” in South Australia

- 5th generation Troponin “masked” and reported to **<29ng/L**
- State-wide chest pain protocol
  - Troponin measured at **baseline** then at **3** (and/or 6) hours
  - **Admission** if: positive troponin, ongoing chest pain, known CAD
  - **Discharge** if: negative troponin, & outpatient functional testing if age >65 or >3 cardiac risk factors. Otherwise GP follow up

What if…

- Troponin at 0 and 1 hour *(but at least >3 hours after symptom onset)*
- Report troponin to <5ng/L

- Change protocol to:
  - Rule out & **discharge** if <5ng/L
  - Rule out & **discharge** if ≤12ng/L and change in troponin over 1 hour <3ng/L
  - Rule in & **admit** if >52ng/L or change ≥5ng/L
  - "Continued observation with repeat testing and possible hospital admission" for the others.



ORIGINAL INVESTIGATION

HEALTH CARE REFORM

One-Hour Rule-out and Rule-in of Acute Myocardial Infarction Using High-Sensitivity Cardiac Troponin T

Tobias Reichlin, MD; Christian Schindler, PhD; Beatrice Drexler, MD; Raphael Twerenbold, MD; Miriam Reiter, MD; Christa Zellweger, MD; Berit Moehring, MD; Ronny Ziller, MD; Rebeca Hoeller, MD; Maria Rubini Gimenez, MD; Philip Haaf, MD; Mihael Potocki, MD; Karin Wildi, MD; Cathrin Balmelli, MD; Michael Freese, RN; Claudia Stelzig, MSc; Heike Freidank, MD; Stefan Osswald, MD; Christian Mueller, MD, FESC

Background: High-sensitivity cardiac troponin (hs-cTn) assays seem to improve the early diagnosis of acute myocardial infarction (AMI), but it is unknown how to best use them in clinical practice. Our objective was to develop and validate an algorithm for rapid rule-out and rule-in of AMI.

Methods: A prospective multicenter study enrolling 872 unselected patients with acute chest pain presenting to the emergency department. High-sensitivity cardiac troponin T (hs-cTnT) was measured in a blinded fashion at presentation and after 1 hour. The final diagnosis was adjudicated by 2 independent cardiologists. An hs-cTnT algorithm incorporating baseline values as well as absolute changes within the first hour was derived from 436 randomly selected patients and validated in the remaining 436 patients. The primary prognostic end point was death during 30 days of follow-up.

Results: Acute myocardial infarction was the final diagnosis in 87 patients (20%) in the derivation cohort and 92 patients (21%) in the validation cohort. In the validation cohort, 259 patients (60%) could be classified as "rule-out," 76 patients (17%) as "rule-in," and 101 patients (23%) as in the "observational zone" within 1 hour. Overall, this resulted in a sensitivity and negative predictive value of 100% for rule-out, a specificity and positive predictive value of 97% and 84%, respectively, for rule-in, and a prevalence of AMI of 8% in the observational zone group. Cumulative 30-day survival was 99.8%, 98.6%, and 95.3% ($P < .001$) in patients classified as rule-out, observational zone, and rule-in, respectively.

Conclusions: Using a simple algorithm incorporating hs-cTnT baseline values and absolute changes within the first hour allowed a safe rule-out as well as an accurate rule-in of AMI within 1 hour in 77% of unselected patients with acute chest pain. This novel strategy may obviate the need for prolonged monitoring and serial blood sampling in 3 of 4 patients.

Arch Intern Med. 2012;172(16):1211-1218.

Methods

- **Population**: Low risk patients suitable for discharge if ruled-out
- Randomise patients to either strategy: 0/1 vs. standard protocol
- **Primary outcome measure**: incidence of composite all-cause mortality or new MI within 30 days
- **Secondary** end points: representation, readmission, coronary revascularization, etc
- Noninferiority margin was set for a number needed to harm of 200 (0.5%)
- *(Study ended enrolment in April 2019 due to lack of equipoise)*

Summary so far…

Results

- 3288 patients analysed and followed to 30 days
- Median age 59
- Median HEART score 3
- **Primary outcome**: 1% miss in both arms (non-inferior)

Secondary outcomes

- Patients in the 0/1 hour arm **more likely to be discharged** from ED **45%** vs. **32%**
- LOS less in 0/1 hour arm - 4.6hr vs. 5.6hr
- 0/1 hour arm had less referral for functional testing *(stress test etc)*
- More patients in subgroup with initial troponin ≤29ng/L had greater rate of coronary angiography (7.1% vs. 5.3%) and revascularisation (2.5% vs. 1%)

Authors conclusion

CONCLUSIONS: This in-practice evaluation of a 0/1-hour hs-cTnT protocol embedded in ED care enabled more rapid discharge of patients with suspected acute coronary syndrome. Improving short-term outcomes among patients with newly recognized troponin T elevation will require an evolution in management strategies for these patients.

Thoughts?

Vitamin C for Sepsis

The craze…

[Original Research **Critical Care**]

CHEST

CHEST 2017; 151(6):1229-1238

Hydrocortisone, Vitamin C, and Thiamine for the Treatment of Severe Sepsis and Septic Shock

A Retrospective Before-After Study

PODCAST

Paul E. Marik, MD, FCCP; Vikramjit Khangoora, MD; Racquel Rivera, PharmD; Michael H. Hooper, MD; and John Catravas, PhD, FCCP

BACKGROUND: The global burden of sepsis is estimated as 15 to 19 million cases annually, with a mortality rate approaching 60% in low-income countries.

METHODS: In this retrospective before-after clinical study, we compared the outcome and clinical course of consecutive septic patients treated with intravenous vitamin C, hydrocortisone, and thiamine during a 7-month period (treatment group) with a control group treated in our ICU during the preceding 7 months. The primary outcome was hospital survival. A propensity score was generated to adjust the primary outcome.

RESULTS: There were 47 patients in both treatment and control groups, with no significant differences in baseline characteristics between the two groups. The hospital mortality was 8.5% (4 of 47) in the treatment group compared with 40.4% (19 of 47) in the control group ($P < .001$). The propensity adjusted odds of mortality in the patients treated with the vitamin

Vitamin C has to be good…

- Vit C deficiency common in sepsis
- Plasma levels are associated with degree of organ failure
- Plays a critical role in many physiologic processes typically deranged in sepsis
- Small trials and retrospective cohort studies of vitamin C have demonstrated promising results among patients with burns, sepsis, and septic shock

JAMA | Preliminary Communication | CARING FOR THE CRITICALLY ILL PATIENT

Effect of Vitamin C Infusion on Organ Failure and Biomarkers of Inflammation and Vascular Injury in Patients With Sepsis and Severe Acute Respiratory Failure
The CITRIS-ALI Randomized Clinical Trial

JAMA. 2019;322(13):1261-1270.

Alpha A. Fowler III, MD; Jonathon D. Truwit, MD; R. Duncan Hite, MD; Peter E. Morris, MD; Christine DeWilde, RN, PhD; Anna Priday, BS, MS; Bernard Fisher, BS, MS; Leroy R. Thacker II, PhD; Ramesh Natarajan, PhD; Donald F. Brophy, PharmD; Robin Sculthorpe, RPh; Rahul Nanchal, MD; Aamer Syed, MD; Jamie Sturgill, PhD; Greg S. Martin, MD, MSc; Jonathan Sevransky, MD, MHS; Markos Kashiouris, MD, MPH; Stella Hamman, RN, MSN; Katherine F. Egan, BSN, RN, CCRC; Andrei Hastings, MD; Wendy Spencer, RN, CPN; Shawnda Tench, BBA, CCRP; Omar Mehkri, MD; James Bindas, MBA; Abhijit Duggal, MD; Jeanette Graf, BS, CCRP; Stephanie Zellner, MS, CCRC; Lynda Yanny, RN, BSN, CCRC; Catherine McPolin, RN, BSN, CCRP; Tonya Hollrith, RT, MR; David Kramer, MD; Charles Ojielo, MD; Tessa Damm, DO; Evan Cassity, MS; Aleksandra Wieliczko, RN; Matthew Halquist, PhD

IMPORTANCE Experimental data suggest that intravenous vitamin C may attenuate inflammation and vascular injury associated with sepsis and acute respiratory distress syndrome (ARDS).

OBJECTIVE To determine the effect of intravenous vitamin C infusion on organ failure scores and biological markers of inflammation and vascular injury in patients with sepsis and ARDS.

DESIGN, SETTING, AND PARTICIPANTS The CITRIS-ALI trial was a randomized, double-blind, placebo-controlled, multicenter trial conducted in 7 medical intensive care units in the United States, enrolling patients (N = 167) with sepsis and ARDS present for less than 24 hours. The study was conducted from September 2014 to November 2017, and final follow-up was January 2018.

INTERVENTIONS Patients were randomly assigned to receive intravenous infusion of vitamin C (50 mg/kg in dextrose 5% in water, n = 84) or placebo (dextrose 5% in water only, n = 83) every 6 hours for 96 hours.

Visual Abstract
Editorial page 1257
Supplemental content

The question…

Does Vitamin C help patients with severe sepsis and severe acute respiratory failure

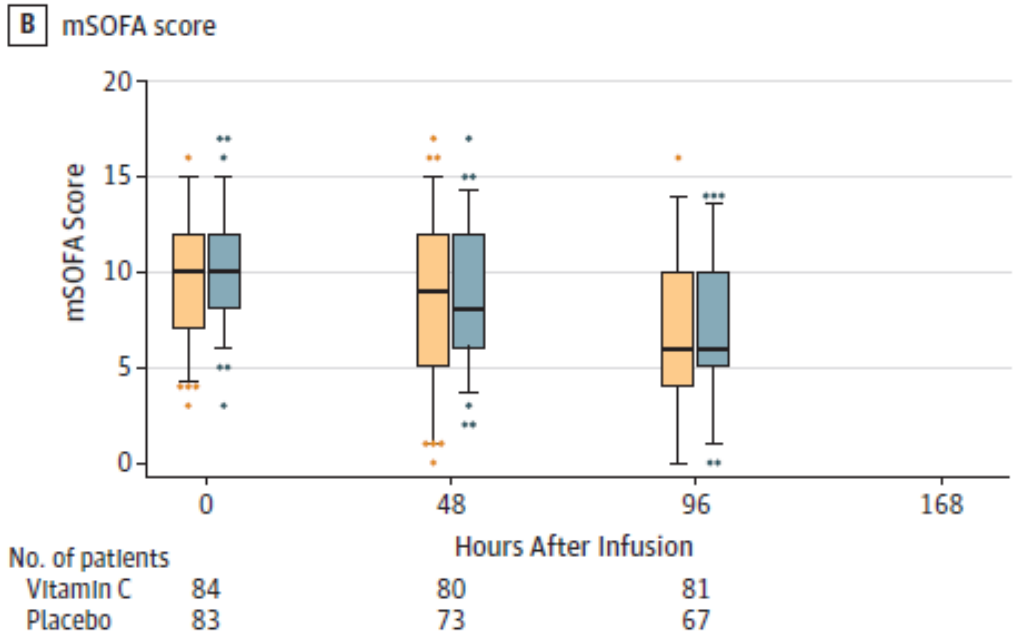
Methods

- Randomized double-blind placebo controlled trial
- 7 ICU's in the USA *(2014-2017)*
- **Inclusion**: Sepsis plus ARDS present for less than 24 hours
- **Intervention**: IV infusion of Vit C (50mg/kg) q6hrs for 96 hours vs. placebo
- **3 co-primary outcomes**: change in mSOFA, CRP and thrombomodulin at 0, 48, 96 and 168 hours
- 46 prespecified secondary outcomes

Results

Results

- 1262 patients screened
- 170 patients randomised
- No difference in any primary outcome



Secondary outcomes

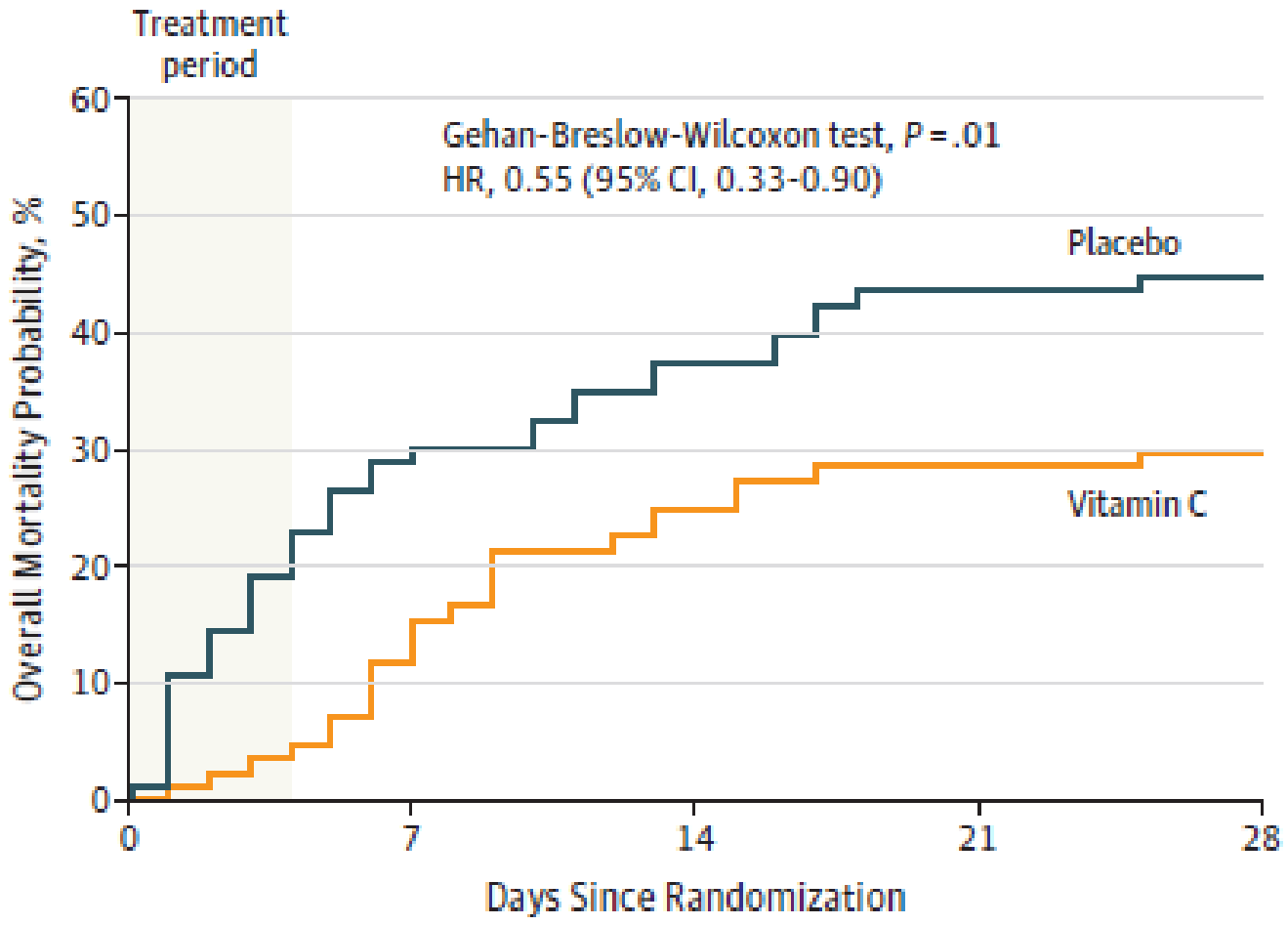
- 43 of the 46 outcomes were not significantly different (p >0.05)
- **28 day mortality 46% vs. 30%** (p=0.03)
- Between group difference 16% [95% CI, 2%-31%]

Table 2. Secondary Outcomes in a Trial of Vitamin C Infusion in Patients With Sepsis and Severe Acute Respiratory Failure

		Vitamin C			Placebo			Difference, Coefficient (95% CI)	P Value
Variable	Hour	No.	Median or %	IQR	No.	Median or %	IQR		
Angiopoietin-2, median (IQR), ng/mL[a]	0	83	8.8	14.9	83	11.6	17.5	−5.1 (−10.9 to 0.6)	.08
	48	80	6.9	11.5	70	9.7	16.9	−2.4 (−9.0 to 4.2)	.48
	96	74	5.7	9.5	63	5.2	7.3	3.8 (−2.5 to 10.0)	.24
	168	53	5.3	6.4	51	3.9	5.5	2.9 (−2.0 to 7.9)	.24
Procalcitonin, median (IQR), ng/mL[b]	0	83	2.4	8.5	83	3.7	19.7	−14.3 (−30.0 to 1.5)	.07
	48	80	1.1	5.2	70	1.7	5.5	−1.3 (−7.4 to 4.8)	.68
	96	74	0.7	2.5	63	0.7	2.2	−1.0 (−3.6 to 1.6)	.44
	168	53	0.5	1.6	51	0.5	1.3	−1.5 (−4.6 to 1.7)	.36
RAGE, median (IQR), ng/mL[c]	0	83	4.0	5.2	83	5.0	6.2	−0.9 (−2.5 to 0.7)	.26
	48	80	2.8	4.2	70	3.3	4.0	0.3 (−0.9 to 1.4)	.67
	96	74	2.1	4.0	63	2.0	3.4	0.8 (−0.3 to 1.8)	.15
	168	53	1.7	2.9	51	1.5	3.2	0.3 (−0.6 to 1.1)	.55
TFPI, median (IQR), ng/mL[d]	0	83	36.7	52.4	83	36.3	33.7	3.4 (−6.2 to 13.0)	.48
	48	80	31.3	34.5	70	34.3	33.4	−0.5 (−10.8 to 9.8)	.92
	96	74	36.2	39.0	63	36.7	30.6	−2.0 (−11.2 to 7.2)	.66
	168	53	30.5	51.0	51	30.2	31.7	1.6 (−11.1 to 14.2)	.81
Vasopressor use, %	0	84	64.3		83	71.1		−6.8	.35
	48	82	54.9		72	50.0		4.9	.55
	96	80	30.0		65	27.7		2.3	.76
	168	72	22.2		59	10.2		10	.07
Oxygenation index,[e] median (IQR)	0	76	0.082	0.090	80	0.089	.077	0.129 (−0.096 to 0.353)	.26
	48	53	0.058	0.062	57	0.056	.054	0.004 (−0.016 to 0.023)	.71
	96	42	0.079	0.084	33	0.045	.040	0.016 (−0.017 to 0.049)	.33
	168	28	0.052	0.051	16	0.074	.066	−0.003 (−0.050 to 0.044)	.90
VE-40[f] to median (IQR)	0	78	0.126	0.044	81	0.115	.058	0.013 (−0.002 to 0.028)	.09
	48	59	0.109	0.049	60	0.110	.061	0.001 (−0.017 to 0.019)	.94
	96	46	0.129	0.060	39	0.105	.047	0.036 (−0.015 to 0.086)	.16
	168	29	0.114	0.043	17	0.118	.057	−0.020 (−0.055 to 0.015)	.26
Net fluid balance to median (IQR), mL	0	83	1604	2927	79	1901	3034	−3759 (−1123 to 373)	.32
	48	81	768	2471	73	473	1797	545 (−255 to 1345)	.18
	96	76	134	2168	66	−659	2560	792 (208 to 1376)	.01
	168	57	190	2076	54	−380	2213	496 (−206 to 1198)	.16
All-cause mortality to day 28, %			84	29.8	38	82	46.3	−0.17	.03
Ventilator-free days to day 28, median (IQR), d		84	17	24	82	8	22	2.5 (−0.9 to 5.9)	.15
ICU-free days to day 28, median (IQR), d		83	11	21	82	0	18	3.2 (0.3 to 6.0)	.03
Hospital-free days, to day 60, median (IQR), d		82	22	46	80	0	39	7.0 (0.3 to 13.8)	.04
Bilirubin, total, median (IQR), mg/dL	0	77	0.7	2.8	79	1.2	1.7	0 (−1.3 to 1.2)	.96
	48	72	0.8	2.0	68	0.8	2.3	0.1 (−1.3 to 1.5)	.87
	96	62	0.8	2.5	64	0.8	1.7	0.6 (−1.1 to 2.2)	.50
	168	45	0.6	1.0	42	0.8	1.0	0.6 (−1.6 to 2.7)	.60
Creatinine, median (IQR), mg/dL	0	84	1.4	1.3	83	1.7	1.8	−0.1 (−0.6 to 0.3)	.49
	48	82	1.6	1.4	73	1.2	1.0	0.2 (−0.3 to 0.6)	.41
	96	78	1.1	1.1	65	1.1	.9	0.1 (−0.2 to 0.5)	.47
	168	64	1.0	1.2	55	1.2	1.6	0.1 (−0.5 to 0.6)	.82
Platelets, median (IQR), $\times 10^3/\mu L$	0	83	147	174	83	160	137	−5.1 (−45.0 to 34.8)	.80
	48	82	137	166	73	116	153	23.8 (−15.9 to 63.4)	.24
	96	77	144	183	63	146	175	15.2 (−29.5 to 59.9)	.50
	168	64	183	209	55	219	149	−2.4 (−59.9 to 55.0)	.93

(continued)

Figure 3. All-Cause Mortality From Randomization (Day 0) to Day 28 Among Patients With Sepsis-Associated Acute Respiratory Distress Syndrome



No. at risk					
Placebo	83	59	53	47	45
Vitamin C	84	74	65	61	59

Author’s conclusion

CONCLUSIONS AND RELEVANCE In this preliminary study of patients with sepsis and ARDS, a 96-hour infusion of vitamin C compared with placebo did not significantly improve organ dysfunction scores or alter markers of inflammation and vascular injury. Further research is needed to evaluate the potential role of vitamin C for other outcomes in sepsis and ARDS.

NEWS RELEASE 1-OCT-2019

New study links vitamin C therapy to better survival rates after sepsis

Exploratory 3-year study finds that treating septic patients with intravenous vitamin C could lead to decreased mortality, quicker recovery

VIRGINIA COMMONWEALTH UNIVERSITY

IMAGE: AN EXPLORATORY STUDY AUTHORED BY VCU'S ALPHA A. "BERRY " FOWLER III, M.D., FINDS THAT TREATING SEPTIC PATIENTS WITH INTRAVENOUS VITAMIN C COULD LEAD TO DECREASED MORTALITY AND QUICKER RECOVERY.... view more ›

CREDIT: VIRGINIA COMMONWEALTH UNIVERSITY

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http://www.vcu.edu

More on this News Release

New study links vitamin C therapy to better survival rates after sepsis
VIRGINIA COMMONWEALTH UNIVERSITY

JOURNAL
JAMA

FUNDER
National Institutes of Health

MEETING
32nd Annual Congress of the European Society of Intensive Care Medicine

KEYWORDS

CLINICAL TRIALS
CRITICAL CARE/EMERGENCY MEDICINE
INTERNAL MEDICINE
MEDICINE/HEALTH
PULMONARY/RESPIRATORY MEDICINE

Thoughts?

Summary take home points

- Tranexamic acid for TBI?
- High sensitive troponin?
- Vitamin C for sepsis?