Overall when comparing patients who received TXA to those who did not receive TXA, the TXA group had higher rates of higher ISS (29.1 vs. 10.1, p = <0.001), higher head AIS >3 (32.8% vs. 13.7%, p = <0.001), higher transfusion requirements (39.5 units vs. 1.2 units, p = <0.001), higher rate of emergent operations (47.1% vs. 42.5%, p = <0.001) and neurosurgical interventions (12.6% vs. 3.7%, p = <0.001), lower initial GCS, and were more likely to have penetrating injuries (93.6 vs. 33.7, p = <0.001). After propensity score matching, however, there were no statistically significant differences between the groups for the above variables. There was an independent association with improved neurological outcomes in patients who received TXA. All of the TXA patients (n=46) were discharged with a GCS of 14-15, compared to only 87% (n=40) in the no-TXA group (p=0.01). There was no significant difference in intubation at discharge or thromboembolic events between the groups, however the TXA group had significantly improved mortality (0% vs. 10.1%, p = 0.028).

The authors of this study concluded that, as previously demonstrated, TXA is associated with improved mortality in severely injured trauma patients. Their findings also supported improved neurological outcomes in patients who are traumatically injured with associated head injuries. The authors acknowledged the limitations of this study given its small cohort size, inherent bias associated with retrospective studies, and the risk of selection bias secondary to omission of incomplete records. They discussed that given that the majority of the patients in the study were active-duty soldiers, it is difficult to translate the results into civilian trauma populations which include older patients with more co-morbidities. Although the results are limited, this study encourages further research with larger prospective trials.

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Comment: This study is a great step into understanding TXA's effect on neurological outcomes in adult patients with TBI. Although this study is limited by several factors as detailed above, TXA definitely appears promising in reducing additional injury for TBI patients. We are hopeful for results of ongoing prospective trials that will add the stronger evidence needed in order for emergency physicians to be routinely using this in practice for head injury.

☐ THE POCUS PULSE CHECK: A RANDOMIZED CONTROLLED CROSSOVER STUDY COMPARING PULSE DETECTION BY PALPATION VERSUS BY POINT-OF-CARE ULTRASOUND.

Badra K, Coutin A, Simard R, et al. *Resuscitation* 2019;139:17-23

Point-of-care ultrasound (POCUS) has become an important adjunct available for cardiopulmonary resuscitation (CPR) in cardiac arrest. CPR depends on high-quality chest compressions with few interruptions, however detection of a pulse has been repeatedly shown to be unreliable and often leads to prolonged pauses in compressions. Ultrasound (US) provides a possible alternative method for pulse checks that could be more reliable than standard methods. The overall objective of this study was to determine whether healthcare providers could perform US pulse checks as quickly as manual pulse checks in cardiac arrest.

The primary outcome of this randomized crossover non-inferiority trial was the amount of time required to perform a pulse check with POCUS as compared to a manual pulse check. Secondary outcomes included number of attempts at pulse check, number of participants who took more than 5 and 10 seconds to complete a pulse check, and confidence levels of the participants. Participants included healthcare professionals 18 years old and older who provided informed consent and were enrolled in an advanced life support (ALS) course. Exclusion criteria included age under 18 and participant not providing informed consent. Participants performed pulse checks on volunteers provided by an ALS course who had their age, sex, weight, height, body mass index, heart rate, blood pressure, neck circumference, and neck length recorded. Study participants included attending physicians, medical students, nurses, and paramedics. Initial confidence levels of participants with pulse detection by US and palpation were recorded as measured by a 100mm visual analogue scale (VAS). Characteristics such as recent BLS or ACLS certification were also recorded. Participants then underwent training to learn to identify the carotid pulse with US using B-mode. Three hours after the US training, each participant performed a pulse check on two separate live models. Each participant was randomized to use palpation or US first and to the model used for testing. A countdown was performed, and participants attempted to identify a carotid pulse using the method to which they had been randomized. Detection of a pulse was confirmed by the investigator palpating a radial pulse while the participant counted in time with the heartbeat. The time it took each participant to detect a pulse was measured by two independent viewers blinded to the group of each participant. Investigators consulted with multiple experts in resuscitation to determine a non-inferiority margin of two seconds.

Investigators enrolled 115 participants, but four were excluded due to incorrect US setup or models prompting participants. Average time to identification of a carotid pulse was 4.22 s (standard deviation (SD) 3.26 s) with US and 4.71 s (SD 6.45 s) manually with a mean difference of -0.49 s (90% CI: -1.77 to 0.39). Manual pulse check had a larger variability in time to identification of a pulse compared to US (p <0.001). Success on the first pulse check attempt was significantly higher in the US group (99.1% vs 85.6%, p=0.0001). There was no significant difference in the number of participants who took greater than 5 seconds and greater than 10 seconds to identify a pulse between groups. However, there were four outliers that took greater than 20 seconds to identify a pulse, all using manual palpation. Recent BLS or ACLS certification had no significant effect on pulse check times for participants. Prior experience with US did lead to quicker pulse identification with a mean of 3.13 s for those with prior US experience and 5.00 s for those without (p=0.003). Staff physicians and residents had faster pulse check times of 3.29 s, with paramedics and medical students (4.04 s), and nurses (5.12 s) having significantly slower times (p=0.02). Prior to the US training course, participants had higher confidence

levels with manual palpation. At completion of the study, participants reported higher confidence levels as measured by the VAS with US (91mm (IQR 82-97) vs 83mm (IQR 72-94), p<0.001).

The authors concluded that pulse checks using ultrasound were not slower than with manual palpation. They felt using ultrasound produced more consistent results and led to a higher first attempt success rate compared with palpation. Despite participants feeling less comfortable with ultrasound initially, after a brief training course, they reported higher confidence levels with US as compared to manual palpation. One advantage of this study was that it included multiple different health professions, which makes it generalizable to a broader population. The authors did note this study was limited by the fact that pulse checks were performed on live, non-bradycardic participants. Pulse checks potentially would take longer in bradycardic patients. There were also questions as to whether these results would hold true in a cardiac arrest, but authors hypothesize that the benefit of POCUS would be even greater in cardiac arrest, due to pulse checks during arrest being unreliable. POCUS remains a promising alternative to manual pulse checks in cardiac arrest.

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☐ APPLICATION OF HIGH-SENSITIVITY TROPONIN IN SUSPECTED MYOCARDIAL INFARCTION.

Neumann JT, Twerenbold R, Ojeda F, et al. *The New England Journal of Medicine* 2019;380:2529-40

Chest pain is universally a leading cause of emergency department visits. Technological advancements have led to the development of high-sensitivity troponin assays. The application, interpretation, and appropriate utilization of their results is still under investigation. Furthermore, the increased sensitivity of these assays raises additional challenges such as determination of true myocardial infarction (MI) versus other causes of myocardial injury, appropriate timing of serial troponin sampling (i.e., from 1 to 6 hours), and prognostic implications of persistently elevated troponin levels. This study sought to develop a calculator capable of identifying the probability of acute MI (AMI) and 30-day outcomes, as well as provide prognostic information on patients identified as not having a myocardial infarction.

The study used data from 15 international cohorts consisting of 23,327 prospectively enrolled patients with suspected myocardial infarction. After exclusion of patients with ST-segment elevation myocardial infarction, 22,651 patients remained for analysis. A derivation data set was derived using 9,604 patients and the remaining 13,047 patients were used for validation of the results. Patients were assigned as either low or high risk based on high sensitivity troponin concentrations at presentation (C1, nanograms per liter) and absolute changes on serial sampling (C2, nanograms per liter). Authors calculated the negative predictive value (NPV), sensitivity, positive predictive value (PPV), and specificity for a variety of combinations of C1 and C2. Using the derivation data set, they were able to evaluate diagnostic performance of various combinations. Results were further examined using the validation data set. Long-term prognostic data was calculated by comparing high-sensitivity troponin values of patients without myocardial infraction to the general population. Patients were matched in a 1:1 ratio using various patient demographics. Using Cox regression analysis, risk of myocardial infarction and death at follow-up were estimated. Median follow up time of the study population was 730 days and 8 years in the general population.

A total of 3,455 (15.3%) of patients in the study population were diagnosed with myocardial infarction. Low high-sensitivity troponin and small changes on serial sampling were associated with high NPV for myocardial infarction and NPV decreased as the initial troponin and absolute changes in serial troponin concentration increased. Conversely, PPV was greater with elevated high-sensitivity troponin concentrations and large absolute changes on serial sampling while PPV decreased as concentrations and absolute changes decreased. Authors classified patients into low risk groups (NPV 100-99.5%, 99.4-99.0%, 98.9-98% and 97.9-97%) and high-risk groups (PPV 80.0% and higher, 79.9-75%, 74.9-70% and 69.9-65%). They provide an interactive calculator at www.compass-mi.com for classification of patients into appropriate groups. In regards to prognostic data, death or myocardial infarction occurred in 3.9% of patients at 1 year and 6.3% at 2 years of the acute study population compared to general population rates of 1% and 2.6% at 1 and 2 years, respectively. Overall risk of myocardial infarction or death in patients presenting to the ED with high-sensitivity troponin I > 10-14 ng/L but diagnosed as not having acute myocardial infraction was 4.8% and 8.1% at 1 and 2 years, respectively, while risk in the general population was 1.4% at 1 year and 3.4% at 2 years.

Authors derived and validated a risk assessment tool, titled The Calculation of Myocardial Infarction Risk Probabilities to Manage Patients with Suspicion of Myocardial Infarction (COMPASS-MI) project, for evaluation of patients with suspected myocardial infarction using high-sensitivity troponin assays. The authors state this tool can risk stratify patients into low -and high-risk groups based on initial troponin and dynamic changes. Such stratification may help identify patients appropriate for discharge versus those at high risk of myocardial infarction or death. Patients identified as neither low- nor high-risk would require further evaluation. Elevated high-sensitivity troponin may provide prognostic information as well since there was a strong correlation with myocardial infarction or death in both study participants diagnosed as not having myocardial infarction and in the general population comparison group. The authors noted limitations, including non-standardized methodology of diagnosis of myocardial infarction between cohorts, heterogenous study populations, potential variability between troponin T and troponin I assays, and use of samples stored for up to two decades to derive general population data.

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