Jo Fields: Welcome to Trauma Loupes, the Journal of Trauma and Acute Care Surgery’s monthly podcast. This is Jo Fields. Today, Dr. Gene Moore, our editor-in-chief, and Dr. Angela Sauaia, the Journal’s biostatistician will comment on a few selected articles. First, Dr. Moore and his must reads for the month...

Dr. Gene Moore: Thank you Jo, Jennifer and Angela. Welcome to the May issue of the Journal. One of our goals this year is to add important research addressing common emergency surgery issues.

The CME article by Dr. Jason Farrah and colleagues at Wake Forest is a superb example. They reviewed their outcome of emergent bowel resection in 231 patients over four years to determine whether stapled versus handsewn anastomoses were superior. Forty-three percent of the anastomoses were handsewn. In sum, after multivariate logistic regression, the stapled technique was associated with a 2.6 odds ratio for anastomotic failure. Interestingly, these finds are remarkably similar to the Western Trauma Association study, where you would anticipate the same challenge of edematous, friable gut due to reperfusion. The question of handsewn versus stapled anastomoses also continues in elective surgery because there is a dearth of adequate powered randomized trials. To confuse the matter, a meta-analysis suggests stapled anastomoses are better for ileocolic reconstruction following Crohn’s disease. Thus, the conduct of randomized multicenter trials in this arena is a conspicuous opportunity for the AAST, including single versus two-layer hand sewn anastomoses.

Another important controversy addressed in this issue is the appropriate volume of fluid resuscitation for the seriously injured patient. Interestingly the two manuscripts, both presented at the AAST this year, were based on the same well-known Glue Grant database and arrived at seemingly disparate conclusions.

The first report by Dr. Josh Brown et al was designed to address the hypothesis that the outcomes resulting from prehospital crystalloid resuscitation would depend on the presence of prehospital hypotension defined as SBP < 90 mm Hg. High volume resuscitation was defined as >500 ml, and propensity scoring was used to control for group disparity. Of the 1216 selected study patients, 51% had prehospital hypotension.

In sum, high crystalloid resuscitation was associated with an improvement in SBP at ED arrival, and each 1 mm increase of SBP was associated with a 2% increase in survival; whereas, high volume
resuscitation in patients without prehospital hypotension was associated with increased coagulopathy and increased mortality.

In the accompanying report by Dr. George Kasotakis et al, the hypothesis was that the volume of crystalloid during the first 24 hours postinjury would not affect in-hospital mortality. Of note, patients were excluded if they died within the first 48 hours. In sum, the amount of crystalloid resuscitation did not impact mortality, but higher volume was associated with days on the ventilator and ICU LOS as well as a variety of complications including ARDS, MOF, compartment syndrome, and infections. Thus, collectively, these studies suggest overzealous crystalloid resuscitation can be deleterious but that adhering to hypotensive resuscitation to < 90 mm Hg might also have adverse consequences. Thus, we clearly have great opportunities to research to clarify these important issues.

Dr. David Hoyt was asked to place these reports into perspective and, in typical fashion, reminded us that these studies are limited by secondary analyses without clear protocols to guide fluid management. But, at the same time, he strongly agrees that we have yet to define the optimal fluid resuscitation for trauma.

The other must-read manuscript is by Dr. Matt Kutcher and colleagues from the San Francisco General who hypothesize principle component analysis would identify clinically distinct patterns of clotting factor deficiency. Using a prospectively collected database of 163 seriously injured patients in whom blood was sampled at the time of ED arrival, nineteen percent were coagulopathic defined as an INR exceeding 1.3. Three patterns of coagulation system derangement were found: Number one, the most common, was essentially global clotting factor deficiency; and this pattern correlated with severity of coagulopathy and mortality. Number two, the next most frequent pattern, reflected pronounced activated protein C and fibrinolysis. Interestingly this pattern—typically described as the acute coagulopathy of trauma—was not associated with conventional laboratory markers of coagulopathy, but did correlate with subsequent ARDS, MOF, VAP, and mortality. Pattern number three, the least common, was dominated by factor VIII deficiency and a smaller contribution of activated protein C. This pattern did not correlate with clinical outcome. In sum, this innovative study suggests there are distinct patterns of coagulopathy that warrant emphasis on different aspects of the coagulation system, including fibrinolysis. Additionally, analyzing the clinical factors, such as ISS, magnitude and depth of shock, TBI, and pre-existing comorbidity, may provide mechanistic information to be pursued at a basic level. We have similarly observed distinct patterns with TEG, and this may offer new
insights as well. In addition to these interesting articles, there are many other timely contributions in this issue. Happy reading!

Jo Fields: Thank you, Dr. Moore. Now we will turn the microphone over to Dr. Angela Sauaia to hear more about the statistical side of the May issue’s offerings...

Dr. Angela Sauaia: Thank you Jo, thank you Gene and Jen. Hello everyone, happy May! It is May, and Denver is finally starting to look like spring. As usual, I like to highlight a statistical method, and this month two methods competed for my attention: principal components analysis and propensity scoring. Let’s talk this month about principal components and next month we can chat about propensity scoring.

Einstein is quoted with saying that “you do not really understand something unless you can explain it to your grandmother.” Although a little chauvinistic, after all, Marie Curie was a grandmother, I think we all get the message. We need to be able to explain our methods so clearly that even someone not as intelligent and brilliant as a trauma surgeon can understand it. So, let’s try that with principal components analysis.

Principal Components Analysis or PCA belongs to the family of statistical methods that also includes factor analysis, exploratory factor analysis. It is a multivariate statistical technique used to reduce the number of variables in a data set into a smaller number of “dimensions” or “factors” or “components”. In mathematical terms, from an initial set of n correlated variables, PCA creates uncorrelated components, where each component is a weighted combination of the initial variables. Like separating pieces of a large puzzle into three or four buckets based on some commonality they have (with color, shape, etc.) that will then help us put the whole puzzle together. In this case, we are placing variables that are correlated in different components as opposed to buckets. A sentence in the Introduction of the article named “Principal component analysis of coagulation after trauma” helps us understand this method: “PCA can be thought of as a multivariate correlation analysis, in which the best-fit line is replaced by a best-fit plane,” where more than one line can exist. Each line is defined by a group of variables that have a high correlation amongst themselves.

A concept that is important within PCA is the Eigenvalue. In plain English, eigenvalue is a measure of the strength of a component in a principal components analysis. It is one measure of the percent of the variance if all of the observed variables explained by that component.
PCA loadings are equivalent to Pearson correlations between each original variable. In addition, the PCA scores can be calculated for each patient by multiplying the raw variable values by the PC loadings and then summing these weighted variables. There, now we know that all variables included in this principal components analysis of coagulation can fit somewhat comfortably into three buckets, a big bucket named PC1, a medium size bucket named PC2, and a small little bucket named PC3. Now we can look at how each of these components correlated with risk factors such as age, ISS, etc. and also with outcomes such as death, organ failure, ARDS.

Once the components are defined and characterized in terms of risk factors and outcomes, it is time to let the data talk to you. I like to compare this phase to looking to a stereogram, those pictures hidden within a picture that you need to stare intently at the picture to find the hidden image. Using previous knowledge about biological mechanisms, we can then understand why those variables loaded together. OK, enough math for one podcast.

Ok, also, exciting, albeit in a sad way, is the review on disparities in trauma. The systematic review and meta-analysis beautifully conducted and reported using the MOOSE and PRISMA statements, showed some disturbing, but not surprising disparities by insurance status. Although trauma care is, so to speak, a universal access system, non-insured persons come to the trauma bay with untreated comorbidities. Although results suggested that disparities by race and ethnicity were independent from socio-economic status, this adjustment was incomplete. As noted by the authors in the discussion, most studies are limiting use limited measures of socio-economic status such as median income, which reflect just a very narrow piece of the poverty story. Poverty has a generational effect, also called cumulative advantage in social science. This means that the descendants of the poor are more likely to remain poor. Even if they move up in the social ladder, they are more vulnerable to financial problems and more likely to return to poverty than the descendants of the affluent. Measures of wealth or net worth are necessary to more completely reflect the socio-economic status. Much of the discussion of this important paper focuses exactly on the need for “more accurate and sensitive data” on socio-economic status. Only with this type of serious discussion, we can possibly address injustice in healthcare. I stand up and applaud Dr. Haider and colleagues for delving deeply into the real mechanisms of health disparities and I applaud our Journal for publishing the study. We rock!

Jo Fields: Thank you, Dr. Sauaia. We will be back in a month with highlights from the June 2013 issue. In the meantime, please send any questions or requests to info@jtrauma.org. Thanks for listening!