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Congress Report on Anesthesia /Critical Care Sessions

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Acute on Chronic Liver Failure

Rajiv Jalan: Acute on chronic liver failure - challenges

Chronic Liver dz →compensated cirrhosis→decompensated cirrhosis (stagewise process of progression from compensated to decompensated cirrhosis). These can be incited by precipitants such as viral infection, medications, alcohol ischemia, surgery, sepsis, or idiopathic. However AASLD working definition is: “acute deterioration of pre-existing chronic liver disease usually related to a precipitating event usually associated with increased mortality at three months due to multisystem organ failure”. Some studies suggest the presence of ACLF may increase mortality rate at 28 and 90 day. Therefore, even if the patient recovers from this disease, they still have an elevated 90 day mortality risk. Multimodal definition (PIRO concept): Predisposition (etiology, age, previous decompensation, comorbidities), Injury (precipitating illness), Response (inflammation/infection), Organ (number and type of failure). In this dz, there may be a host response to injury as compared with other types of cirrhosis. Patients without ACLF have low risk of 28 day mortality regardless of leukocyte count, in stark contrast to patients with ACLF who have significant mortality increase that worsens with increasing leukocyte count.

Challenges:

Prognostic modeling: MELD vs CLIF scoring

CLIF-Organ Failure score, Age and Ln White-cell count are independent risk factors for mortality in ACLF patients. And calculators have been derived using CLIF that demonstrated a better C statistic for ACLF patients compared to child-pugh, meld or meld-Na.

ACLF is a dynamic syndrome. If have ACLF-1 patient progresses to higher ACLF (2 or 3) their mortality increases, whereas if they move to no ACLF, mortality improves. Clif score at day 3-7 provides a good assessment for the mortality outcome of these patients. Clif-C ACLF score improves performance of MELD Na score. Perhaps CLIF-C score should be used to modify meld for organ allocation in these patients. Clif-C ACLF score is nonlinear. With low score there is little increased mortality effect, with high score the mortality is very high. With score of '30/35-70, there is linear direct relationship to compared to mortality outcomes and they represent the group with the greatest potential modifiable risk.

IF transplant these patients with ACLF 2/3 may have good outcomes. However, if patients CLIF-C ACLF score over 70 may have high mortality regardless. Many of these patients die of sepsis.

Conclusion: Diagnostic/prognostic criteria for ACLF are robust (meld less accurate in assessing risk of death), can be used to prioritize pt for liver transplant but more validation required), the main issue with transplanting ACLF patients is timing, ACLF diagnosis/prognosis and a pathophysiology toolkit may help development of new therapies.

Fuat Saner: Sepsis - an always present threat

Sepsis is increasing over time (2003-2011), however sepsis related mortality decreasing. Patients with cirrhosis are prone to sepsis with increasing drug resistant organisms. Pretransplant MRSA/VRE increases risk of postop infection. Risk factors for Carbapenem resistant klebsiella pneumoniae include: lab MELD, HCC, roux en y choledochojejunostomy, bile leak, RRT.

How do we avoid/assess these infections? Procalcitonin (PCT) has been suggested as an early marker for predicting sepsis. PCT with and without sepsis has been evaluated, but much of the data does not demonstrate a relationship in the patients requiring liver transplantation. Fungal infections are another scourge (candida spp. Aspergillus spp.). Incidence 5-40% in high risk, LT patients (ie MELD>30, retransplantation, fulminant liver failure, RRT). One study demonstrated that prophylactic administration of antifungals had significantly lower infection (17.7 vs 32.4%) rates.

However, most studies have not demonstrated a mortality benefit to prophylactic antifungal administration except in certain high risk patients.

Constantine Karvellas: ICU management of Acute on Chronic Liver Failure

Why do ACLF pt end up in ICU? Circulatory failure, septic shock, HE/respiratory failure, variceal bleeding/hemorrhage, AKI/HRS. Hyperdynamic state may mask cardiac dysfunction. ACLF patients generally have lower MAP, higher Scv02 and compromised lactate metabolism, therefore we need other tests to assess Cardiac Output/volume status. Bedside echocardiography (LV/RV function, TR jet, fluid responsiveness), CVP monitoring (CVP 8-12), mixed venous sat (Scv02>70), blood lactate, and other modalities such as non-invasive CO monitoring devices may be useful. Management includes vasopressor administration (norepinephrine, vasopressin, terlipressin) with goal MAP>65 (should we consider MAP>75 in patients with AKI?). Rule out adrenal insufficiency which can be common in these patients and consider 200mg/day of hydrocortisone. ARDS is prevalent and lung protective strategy for ACLF should use guidelines by ARDS network: TV=6ml/kg predicted BW, RR set to normal minute ventilation, target Sa02>88%, plateau pressure <30mmHG, fluid restrictive strategy may be beneficial but must be balanced with renal function. Hepatic encephalopathy in ICU: intubate for HE grade III/IV. Therapies include lactoluse, rifaximin, but also consider PEG (less bowel distention than with lactulose). Bleeding/coagulopathy in ACLF is difficult to assess in the absence of viscoelastic testing given decrease in pro and anticoagulants (INR therefore not useful measure). Transfusion strategies for acute upper GIB should be restrictive once bleed is controlled with a goal of maintaining hgb>7. In variceal bleeding HVPG>20mmHG predicts mortality, can treat with terlipressin (improved bleeding, lower mortality), if not available can use octreotide (no mortality benefit documented), or can consider early TIPS. TEG guided transfusion prior to invasive testing in cirrhotics has been shown to lower transfusion with no increased complications. Cirrhosis associated AKI had 10-fold higher mortality, HRS patients with 90 day mortality of 85% if not transplanted. ATN as cause of AKI before transplant puts patients at risk for post LT mortality increase (and persistent renal dysfunction). Zarbok showed early RRT improved mortality in ACLF patients with AKI. What anticoagulant for RRT? Citrate? Heparin? Prostacyclin? None? Citrate may be detrimental to patients with liver failure. Prognosis and goals of care should be discussed with patient/family. Clif-c ACLF score >80 had a 90-day mortality greater than 90%. Increased utilization of palliative care consultation in patients denied liver transplant listing should occur.

Conclusion: echo is valuable to assess volume status, lung protective strategy in ALI, early appropriate abx impacts outcomes, consider PEG in patients with HE, Hgb >7g/dl transfusion trigger, consider viscoelastic testing to assess coagulopathy, vasoconstrictor therapy associated with reversal of HRS and do not impact mortality in the absence of LT, more studies required on the use of RRT, artificial liver support may be beneficial (however MARS, PROMETHEUS have not demonstrated survival benefit in ACLF), consider palliative care referral in patients with poor prognosis and not LT candidates.

Pavel Trunecka: When to transplant, when not to transplant

Basic assumptions: proceed with transplant governed by thresholds of when a patient is considered too healthy or sick to benefit. Thresholds are difficult to identify and may differ between centers/countries.

Definitions: Too healthy if expected lifetime is greater in absence of Transplant. Too sick if predicted survival lower than minimum acceptable level. LT is the best treatment option for borderline indications (most HCC, well selected CCA, select colon mets, AH not responsive to steroids, etc.). MELD liver allocation developed to guide rationing, but poor tool to assess post-transplant outcomes. There is a conceptual model based on the relationship between MELD, frailty and waitlist outcomes that has been put forth. Frailty not currently incorporated into MELD. Several scoring systems have been designed to predict survival after LT, most frequently the SOFT (survival outcomes following liver transplantation). SOFT score utilizes 13 recipient and 5 donor factors and can be compared with the BAR score. BAR score (balance of risk score) came from Europe based on UNOS database and validated with European database. It utilizes MELD, recipient age, retransplantation status, life support pre-transplant, cold ischemia and donor age. A futility score has also been derived from high MELD (40) patients at UCLA. However, when these scores are applied to other transplant populations, they do not always hold up,therefore caution should be used with application of scores to cohorts outside of validation.

Conclusion: MELD not satisfactory predictor for WL, candidates are endangered by infection, MOF, hemorrhage, respiratory failure and HE. Many mathematical scores could assist in prediction of unacceptably poor outcome. Identification and recognition of predictive factors beyond the MELD score influencing WL outcomes, mortality and perioperative risk, may allow for earlier TX or revised allocation policies, extent of minimal transplant benefit and score validation based on local conditions may be necessary, delisting is still one of the most demanding decision in transplantation!

Anesthesia/Critical Care Abstracts

Rengeine Kiss Timea: Kinetics of Hemostasis Reserve Capacity in Bloodless Liver Transplantation

Hemostatic reserve capacity is a concept in which transfusion management can target the products that are needed. Lab values are obtained at 5 time points: preoperatively, on arrival in the ICU, 12 hours post arrival, 24 hours post arrival and 48 hours post arrival. Pre-described triggers are determined and assessment of levels of fibrinogen (1g/l), factor II, V, VII, X, XIII (30% activity for previous factors), platelets (30G/l), antithrombin III (40% activity) and hematocrit (27%) levels allow for targeted intervention. This process allows for a "focus on the weakest link" in system. The HRC is calculated as: [Blood Volume x (Lab value-trigger level)]/(Lab value + trigger level). These authors examined 26 patients and found Factor I, II, V, VII, X decreased significantly but improved within 48 hours after graft reperfusion in general. By utilizing this modality, in last 2 years (n=170) in Budapest, most patients required no PRBC transfusion (>52%). And these principles can be applied elsewhere, ie to to Jehovah's witness, etc.

Ahmed Mukhtar: Effect of Bacterial Translocation on Hemodynamic and Coagulation Parameters during Liver Transplantation

Bacterial translocation via DNA analysis of patient blood showed cirrhotic patients with increased translocation. Such infections can potentially increase the risk of coagulopathy, such as factor Xa levels. These authors recruited 30 pt with LDLT and Child C cirrhosis. ROTEM performed (extem, intem heptem), anti-FXa, and TNF alpha and IL-17 evaluated looking for incidence of bacterial translocation at time of transplantation. PCR analysis divided patients into those DNA –(20) and +(10) and there were no differences between the groups. However, looking at ROTEM, the DNA+ patients had significant difference in CT, CFT, alpha angle and MCF via extem. Intem and heptem showed significant difference in CFT only. No significant difference in anti Xa activity. TNF alpha and IL-17 both differed significantly between groups. Conclusion: plasma bacterial DNA present in 30% of patient at time of transplantation and those patients had hypocoagulable state based on viscoelastic testing.

Kyota Fukazawa: Incidence and risk factors for death from intracardiac clot and pulmonary embolism during and immediately following orthotopic liver transplantation

24-hour mortality after liver transplantation examining the contribution and risk factors for devastating intraoperative cardiac thrombus formation and/or pulmonary embolism. 65,308 patients from the UNOS Standard Transplant Analysis and Research (STAR) database (2002-2014), excluded fulminant/status 1A, living donor, split liver, multi-visceral transplant. Controls were patients who survived for 30 days, as compared to the study group, those who died within 24 hours. After exclusion 41,237 patients with 54 (0.1%) deaths within 24 hours. Another 2484 (6.0%) died within 1-29 days. Most common cause of death within 24 hour was hemorrhage (20.9%), 2nd was embolism (19.8%) and 3rd was primary graft failure (17%). Risk factors for clot in multivariable model was higher in Asian (HR 3.6), those with history PVT (2.6), DM (1.9) or hx of pulmonary embolism (30.4), poor functional status <19 (karnofsky) (3.2), need for vent support (2.7), and being African American (0.3) was protective. To quantify risk of death, created a DICPEI ROC (AA as donor, Asian as recipient, dm, pvt, PE, vent support, functional status) with an AUC 0.7186. Conclusion: 3 major reasons for postop mortality were identified as hemorrhage, ICPE, primary graft failure. Several predictors emerged: history of PE, severely limited functional capacity, need for preoperative ventilation, history of PVT, Asian ethnicity, and donor ethnicity. The DICPEI has C statistic 0.7 suggesting that these factors can be grossly predictive.

Sherlu Pai: The effect of hypertrophic cardiomyopathy on mortality in liver transplantation

HCM is autosomal dominant, leads to diffuse or segmental patterns of LV thickening and is associated with LVOT obstruction in systole (systolic anterior motion of mitral valve) and sudden cardiac death. HCM treatment includes pharmacotherapy, and ICD placement. A retrospective review of patients with HCM at all Mayo centers from 2000-2015 (Arizona, Minnesota and Florida) looked at patients with prior history of syncope, NSVT, family hx of SCD, HCM treatment history, and HCM Risk-SCD prediction model. Preoperative echo workup evaluated maximal LV septal and posterior wall thickness and increased resting LVOT gradient (>60). Only 29/4128 had preoperative dx of HCM (6 died within 1 year of transplant). Conclusion: HCM should not be absolute contraindication to transplantation, but if there is a high LVOT resting gradient and elevated LV thickness, there is increased postoperative mortality and this should be included in selection of patients for LT.

Chris Wray: Postoperative myocardial injury in liver transplant patients: a single center pilot study

Background: VISION Study: MINS (myocardial injury following non-cardiac surgery: type 2 MI or myocardial oxygen supply/demand imbalance). VISION study examined Troponin T (TnT) elevation within 72 hours following transplantation and the effect on 30 day mortality in large international cohort. LT high risk surgery, these authors looked at retrospective single center in non-consecutive LT patients (12 years, =2010). Similar study using TnI at UCLA. LT candidates are becoming older with elevated CAD risk and we are transplanting with ever increasing MELD scores. Would a consecutive cohort have same incidence of MINS? Dr. Wray examined TnI serial testing in all LT patients over 3 days postoperatively from 2015-2016 (1.5 year). 200 patients with LT were examined, but only 111 received the full complement of TnI levels (72 hours). They demonstrated a MINS prevalence of 76% (85/111) when MINS was defined as TnI>0.1. Patients with MINS demonstrated about a 15% mortality. There was no association with age, MELD, or angiography proven CAD. They reported the highest prevalence of troponin elevation in patients following LT. This raises the question: how should post LT MINS be evaluated and treated?

Sanjeev Aneja: Effect of obesity over the development of acute kidney injury in patients undergoing live donor liver transplantation-prospective observational study

Most patients received LT for viral hepatitis, with the majority of for HCV. 147 patients examined with a mean surgical time >12 hours and a 44% overall incidence of AKI according to AKIN classification. Those with normal BMI (<25) had significantly lower rates of AKI compared to overweight and obese group.

Jacek Cywinski: Experience with the first 10 cases of normothermic machine preservation in human liver transplantation-comparison of matched cohort

Does NMP perfusion offer benefit to postreperfusioin hemodynamic, metabolic and coagulation parameters? NMP in 8 DBD/2 DCD; Control was 32 DBD/8 DCD. Study underpowered for statistical significance so used Absolute Standardized Difference and found a small to medium difference in norepinephrine and epinephrine usage. The NMP group needed less FFP post reperfusion, had better cardiac index and had lower lactic acid and a higher base excess. As enrollment increases, these trends hopefully will reach statistical significance.

Zachary Dietch: Locoregional anesthesia is associated with reduced complications in patient with cirrhosis undergoing open inguinal hernia repair

Inguinal hernia repair with locoregional anesthesia could be associated with fewer complications in patients with cirrhosis. NSQIP database was examined from 2011-2014 and composite outcomes based on MELD (>15) were analyzed. ' 8000 patients most who received general anesthesia ('6500). Patients with GA had higher rates of complex surgery (incarceration/gangrene) and higher rates of respiratory complications and surgical site infections (SSI). Of those with MELD>15, overall and infectious complications were higher in GA group. Locoregional anesthesia resulted in lower odds ratio of complications compared with GA with a more profound effect in patients with MELD>15.

Fulminant Liver Failure

Symposium on May 26, 2017

William Bernal: Acute Liver Failure - managing the brain

Basic neurologic care: intubation/ventilation, adequate sedation, cardiovascular stability, frequent neurologic observations, CO2 control, glucose and sodium control, head elevation at 30 degrees, fever avoidance.

Evidence for this care is lacking but fewer neurologic deaths/complications are occurring over time. Acute hepatic dysfunction results in increased neurotoxins such as ammonia and an increased inflammatory

response. Therefore treatment aims to control or reduce these. To minimize hemostatic disarray, we aim to optimize temperature, **sodium, glucose**, magnesium, phosphate, calcium, ph, **CO2**, and cardiovascular state. Serum sodium control has some evidence to demonstrate that brain swelling can be minimized/delayed with control of sodium levels. Attempt to control ammonia level by decreasing circulating levels (ie. extra corporeal removal) or systemic production. Hemofiltration using CVVH rates may correlate with ammonia clearance. Can attempt to reduce production by protein restriction (ie hold enteral feeding for short periods to lower levels). Induced hypothermia may lower systemic production of ammonia, lower cerebral uptake, lower cerebral metabolism. Data looking at hypothermia in large studies failed to show improved outcomes such as survival in transplanted or non-transplanted, waitlisted patients. We seldom cool patients less than 35C, but can consider doing this in refractory ICH.

Who should have an ICP monitor? High risk demographics are <35yo, acute/hyperacute presentation with persistent elevation of ammonia >150-200, renal/cardiovascular failure. For those patients with elevated ICP, EASL suggests mannitol or hypertonic saline for surges in ICP with short term hyperventilation.

Catherine Paugam-Burtz: ALF management in the ICU

Aim of ICU support is to optimize conditions for liver regeneration, restore organ function, avoid MOF and optimize conditions for going to the OR. Non-specific ICU support requires hemodynamic management: SIRS, vasoplegia, hyperdynamic state (septic like!). Hemodynamic resuscitation should include appropriate monitoring to guide fluid administration (treating hypovolemia and avoiding excessive fluid), and appropriate use of vasopressors. Mechanical ventilation is often required for coma or ARF and protective ventilation (low TV, PEEP, low plateau pressure) should be utilized to ensure normoxia/normocapnia (to avoid brain injury). Acute Kidney Injury (AKI) is very common and RRT often is required to help metabolic control (elevated ammonia levels) and continuous techniques are likely advantageous over intermittent, but data is lacking.

N-Acetylcysteine can be used for paracetamol overdose with possible survival benefit in early stages of ALF in non-paracetamol liver injury. ALF associated with increased risk of infection which is associated with worse prognosis. Delay in abx administration in infected patients is associated with increased mortality. Antimicrobial prophylaxis in ALF is not associated with decreased rates of infection. Should we apply abx prophylaxis to the most severe patients or have high index of suspicion of infection and treat broadly while awaiting microbiologic sampling results? Listed patients should get antifungal prophylaxis since ALT/RRT are major risk factors for fungal infection.

Extracorporeal liver assist device: Biologic (synthetic activity/detoxifying) and non-biologic devices (detoxification using albumin dialysis, elimination of water soluble and protein bound components). No device has yet to show a survival improvement with ALF. Another option is high volume plasma (HVP) exchange: study from 2016 looked at 182 patients who received SMT with or without HVP and half received liver transplantation. Those with plasma exchange had significantly improved survival. Comparing those who were and were not transplanted, the group that did not get transplanted had the greatest survival benefit with this modality. HPV may work as an immunomodulating treatment: removal of circulatory DAMPS, reduction of proimflammatory compounds of innate immune cells, reduction of tissue injury, reduction of organ failure, reduction of SIRS. Conclusion: HVP may improve transplant free survival, no liver assist devices have shown improved survival, consider prophylactic abx if high suspicion for infection, and conduct generic ICU support for ALF patients.

Yaman Tokat: Transplant options for FHF

FHF is characterized by rapid deterioration of liver function and development of hepatic encephalopathy. Etiologies include: viral/drug/vascular/metabolic/miscellaneous. Pregnancy induced hepatic failure has one of the best outcomes as does hepatitis A. Hyperacute (<7 days) has high rate of cerebral edema but has good outcomes, and the late onset (12-24 days) has the worst outcome. LT for ALF is the only therapy with proven benefit, but rapid progression and variable course of ALF limit its use. In the US spontaneous survival/recovery occurs in 45% of patients. Decision making is complex and there exist many criteria guidelines. Reliance entirely upon these guidelines is not recommended as per AASLD position paper (updated in 2011). The decision is influenced by: Etiology and rate of progression. Consider waiting on transplantation for patients with sustained evidence of improvement in prognostic criteria in the absence of clinical deterioration, patients with paracetamol overdose without high grade HE (3/4) regardless of coagulopathy. Do not transplant patients with brainstem damage, invasive fungal infection, rapidly escalating inotrope requirements, or severe pancreatitis.

William Bernal in 2010 published a good evaluation of survival with and without transplantation. In USA 66% with FHF receive graft within 3 days, but in other countries this is not the case. Therefore there must be consideration of live donors, but only if there is a significant chance of successful outcome to the recipient. Several papers suggest LDLT survival similar to DDLT. FHF in Turkey 2011-2015: 395 patients with ALF, 293 emergently listed, 148 had family donation: 110 survived, 37 died and 1 received DDLT. 1 year survival was 75% for LDLT and 64% for DDLT. Turkey data showed patients with longer waitlist time had significantly higher perioperative mortality with LDLT for ALF. LT is a lifesaving operation and LDLT is safe for patients with ALF.

LDLT allows optimal timing of LT and lowers waitlist mortality even in the US. Therefore LDLT may be a superior option as compared to DDLT.

Jim Findlay: Intraoperative challenges

ALF is uncommon (3% LT in US) with little evidence/research behind management. There exists a complex interplay of systems with the need for management of: fluid/CV status, ventilation, coagulation, ICP. Preoperative evaluation: DO IT! Don't get fooled into rushing to transplantation. Perform a full evaluation and necessary testing. Evaluate for rapid progression of disease (MOF/cerebral edema).

When planning for the OR:

-ARDS may be part of the disease process, consider if the anesthesia OR ventilator can handle the ventilator setting (can bring ICU ventilator).

-Dialysis: should we continue RRT in OR or not? Many institutional challenges may present themselves, however if already on RRT likely should continue if possible (no clear evidence). -ICP monitoring when its available use it. Consider how to transport patient to the OR. If patient has elevated ICP (being monitored) on transport the ICP may acutely rise, so continue monitoring! Head up positioning during transport (as in ICU). Consider using a transport ventilator to ensure consistent ventilation. Patient unlikely to have portal hypertension. ICP management: goal ICP>20, CPP>60, if ICP not monitored, consider treating them as if they have ICP elevation.

-IV anesthetic ideal (propofol), avoid volatile agents (cerebral vasodilation), but if used keep MAC<1 and avoid nitrous oxide.

-Maintain head up position, neutral neck position, minimize head down time for CVC placement. Be prepared to treat ICP spikes, can occur with surgical stimulus, recirculation. ---Can treat elevated ICP elevation with propofol, osmotherapy (ie Mannitol), hyperventilation (but maintain CO2>25 to avoid vasoconstriction), can consider adding barbiturate or indomethacin.

-Temperature management should not have prophylactic hypothermia, however if they have been hypothermic in ICU DON'T rewarm them (can lead to elevations in ICP if already cooled!).

-CV: fluids as appropriate but consider the effect on volume in ICP.

-Which pressor to use? Norepinephrine can increase BP, CPP with little effect on ICP. Vasopressin has little direct info, low dose terlipressin can increase ICP. Terlipressin added to NE can increase CPP compared to NE alone, however based on small studies in stable patients with normal ICP and CPP, therefore the applicability is unclear. Vasopressin decreases splanchnic blood flow. Phenylephrine is effective in raising CPP in TBI. Patient may arrive on norepinephrine or phenylephrine and consider adding vasopressin or analogue as needed.

-Coagulation: loss of synthesis of pro and anticoagulants. Despite elevated INR VE testing is usually NORMAL with normal thrombin generation and decreased fibrinolysis often occurs. Recommendation for ICP monitor INR<1.5, plt >50k but no evidence to support this. Perhaps consider VE testing before placement. Ignore INR! Can add platelet count or fibrinogen level if TEG, but can just use ROTEM itself if have. Consider keeping plt>50k and fibrinogen>100.

PCC vs FFP for coagulopathy? PCC great if volume is a concern. For ventilation, maintain lung protective strategy (tidal volume 6-8 IBW, with some PEEP).

-There is an unclear effect of PEEP on ICP. Ventilation will be effected by volume status. ------Euvolemia is ideal

-Maintain serum osmolality <320, Na 145-150.

-Postoperatively return to ICU, may have delayed wakeup (encephalopathy, medications). ICP elevations reported early post-transplant (individualized assessment) and when patient waking up and moving ICP.

ROTEM Session

TEG first described by Hartert in 1948. First published data in LTX in 1985 (reduced transfusion by 33%). Since 1990 development of ROTEM based on TEG. Should continue test for 60 minutes to assess if there is late fibrinolysis. If lysis index is <85% then should consider treating for lysis. A10 can predict MCF and its utilization can expedite clinical decision making. Fibtem has Cytochalasin D to destroy platelets. New coagulation model is based on initiation, propagation and amplification of clots rather than historically described pathways. Clot formation requires a thrombin burst for fibrin formation to occur. ROTEM has no sensitivity for plt inhibition from aspirin or Plavix, also no sensitivity for von willebrand deficiency. SHOT data demonstrated significant morbidity from transfusion of plasma. Song et al in BJA 2013 112; 290 showed rapid diagnosis of coagulopathy critical in treating perioperative bleeding and ROTEM can predict this quickly. Saner in BJA (2017, 0: 1-9) evaluated ROTEM vs Standard lab tests, demonstrating the poor clinical assessment from standard lab tests without viscoelastic testing. If fibrinogen is low, clotting time will always be low as well. Therefore should evaluate fibrinolysis and clot firmness before thrombin generation. Post reperfusion fibrinolysis occurs in significant number of patients but often resolves on own, therefore may be acceptable not to treat unless clinical bleeding. Tanaka and Sakai demonstrated that fibtem most effectively assesses degree of fibrinolysis (BJA 2017). Endogenous heparinoids can happen after reperfusion but is often self limiting.

Transfusion 2014 54, 2760 Gorlinger: Rotem guided treatment with fibrinogen concentrate and/or PCC did not increase occurrence of thrombosis or ischemic events compared to those that did not receive these.

According to the 2016 Cochrane review, ROTEM significantly decreases mortality.