Dear Editor,

The AAST patient assessment committee has recently published “Organ injury scaling 2018 update: spleen, liver, and kidney.” We appreciate the continual efforts of the committee to refine and improve the grading system, and we are excited to check a lot of anticipated changes to the nephritic trauma grading once in nearly three decades, addressing a variety of challenges that had become apparent over the years.

Initially outlined in 1989, the Organ Injury Scaling was basically and primarily based on the anatomic findings encountered mostly at the time of open exploration of the eviscerate organ; however, currently, with advances in CT technology and its widespread use, incorporating key radiologic findings within the grading system was logical. Incorporating the vascular supply is additionally a vital addition to the grading system. A depth of parenchymal laceration of 1 cm is used to separate grade 2 and 3 injuries. However, the principle behind selecting this cut for this purpose is unclear, and it should be capricious. Laceration depth offers very little information in predicting the requirement for interventions once further information like hematoma size and vascular distinction extravasation are obtainable. Another ambiguity is the use of segmental vein or artery injury within the organization, it is unclear if segmental injury is the description of vascular anatomy, one among the 5 segmental nephritic arteries supplying the kidneys.

Taken along, these updates within the nephritic injury grading replicate a number of this evidence in management of renal trauma. Anyone is required to know the implications of these changes and learn whether or not this updates grading system can improve the prediction of outcomes. Prophetic tools like normograms and a lot of objective criteria ought to be used in clinical follow-up to pick out patients who would have the benefit of interventions from trauma management after nephritic trauma.

To the Editor:

In late December 2019, several local health facilities reported clusters of patients with pneumonia of unknown cause that were epidemiologically linked to a seafood and wet animal wholesale market in Wuhan, Hubei Province, China. Deep sequencing analysis from lower respiratory tract samples indicated a novel coronavirus, which was named coronavirus disease 2019 (COVID-19). In severe cases, patients with COVID-19 develop a type of acute respiratory distress syndrome (ARDS), sepsis, and multiorgan failure. Moreover, older age and comorbidities are associated with higher mortality. The fibrinolytic system is often suppressed during ARDS where fibrin accumulation can promote hyaline membrane formation and alveolar fibrosis. Depressed pulmonary fibrinolysis is largely due to increased levels of plasminogen activator inhibitor 1 in both plasma and bronchoalveolar lavage fluid. Furthermore, it is observed that an endothelial damage that disrupts pulmonary regulation promotes ventilation-perfusion mismatch (the primary cause of initial hypoxemia) and develops thrombogenesis. Fibrin deposition is the result of an imbalance of the coagulation and fibrinolytic pathways, and several therapeutic strategies have been explored to target the dysfunction of these systems in ARDS. In particular, the use of fibrinolytic therapy (including plasminogen activators) to limit ARDS progression and reduce ARDS-induced death has received strong support from animal models. Human studies are limited, although in a phase 1 clinical trial, Hardaway et al. showed that the administration of urokinase or streptokinase resulted in a significant improvement of PaO2 level in patients with severe ARDS secondary to trauma or sepsis. In this study, these patients had a PaO2 level of less than 60 mm Hg, which increased to 231.5 mm Hg following thrombolytic therapy with an overall 30% survival rate and no incidence of bleeding.

Previous data on fibrinolytic therapy in ARDS associated to the prothrombotic state and clinical findings with pulmonary vascular thromboocclusive disease in COVID-19 suggest that the use of tissue plasminogen activator (tPA) may have an impact in the treatment of severe COVID-19 induced ARDS, when all medical efforts and treatment options were exhausted.

The rational for fibrinolytic therapy is due to the pathologic fibrin deposition that reflects a dysfunctional clotting system, with enhanced clot formation and fibrinolysis suppression, related to tissue factor produced by alveolar epithelial cells and macrophages, and high levels of plasminogen activator inhibitor 1 produced by endothelial cells or activated platelets.

In COVID-19 pneumonia, the thrombi play a direct and significant role in gas exchange abnormalities and in multisystem organ dysfunction. The preserved lung compliance noted early in the course of COVID-19 patients with bilateral airspace opacities suggests that the observed pulmonary infiltrates could represent areas of pulmonary infarct and hemorrhage. Therefore, thrombolysis could improve alveolar ventilation by restoring blood flow to previously occluded regions. This redistribution would reduce blood flow to vasodilated vessels, decreasing the shunt fraction and improving oxygenation.

Currently, there are only case reports and case series showing efficacy of tPA in COVID-19 patients with severe ARDS, demonstrating improvement of PaO2/FiO2 ratio with no bleeding complications. In a recent case series of five COVID-19 patients with severe hypoxemia (PaO2, <80 mmHg) and D-dimer greater than 1.5 µg/mL, all subjects received a protocol including 25 mg of tPA intravenous bolus given for 2 hours, followed by 25 mg continuous infusion for the next 22 hours. Each patient was placed on a weight-based

Fibrinolytic therapy in patients with COVID-19 and acute respiratory distress syndrome: Is this a feasible approach?

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LETTERS TO THE EDITOR

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