predictable benefits of analgesia on respiratory function. I am concerned that treatment decisions may have been made using an incorrect interpretation of the results of thromboelastography and that as a result of this article physicians might repeat this mistake.

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(Accepted for publication July 1, 2013.)

Standard Thromboelastography Should Not Be Used to Assess Candidacy for Neuraxial Procedures in Patients Taking $P2Y_{12}$ Inhibitors

To the Editor:

We read with interest and concern the case scenario by Ahn *et al.*[1] describing the use of the thromboelastograph to guide thoracic epidural placement in an elderly patient with chest trauma taking clopidogrel and aspirin. Despite presumed platelet inhibition, this patient's thromboelastograph demonstrated a slightly hypercoagulable state with an increased maximum amplitude of 76.1 mm. We agree that trauma-induced inflammation and acute-phase reaction are possible causes of the reported thromboelastograph findings in this patient. However, we are concerned about the authors' use of standard, kaolin-activated thromboelastography as a method to assess platelet function in the setting of a $P2Y_{12}$ antagonist as well as aspirin.

Thrombin is, by far and away, the most potent activator of platelets.[2] This activation is accomplished through thrombin-mediated cleavage of the protease-activated receptors. Adenosine diphosphate, in contrast, is a relatively weak activator of platelets.[3] Adenosine diphosphate agonism of the $P2Y_{12}$ receptor serves to amplify the platelet in response to thrombin and to stabilize platelet aggregates. This results in a critical issue that practitioners using the thromboelastograph to guide interventions need to understand; kaolin-activated coagulation generates thrombin in quantities that are sufficient to overcome the effects of $P2Y_{12}$ antagonists on platelet function as assessed by thromboelastography.[4] In a similar manner, platelet inhibition by aspirin is also masked in kaolin-activated thromboelastography.

Platelet inhibition can be assessed by a modified thromboelastograph assay known as TEG Platelet Mapping® (Haemonetics, Niles, IL). The details related to this assay can be found elsewhere. Despite the fact that authors' discussion correctly identifies the need for this modified thromboelastograph assay to assess platelet inhibition from clopidogrel and aspirin, the assay was not used in the presented case. Instead, a supranormal maximum amplitude result from a standard thromboelastograph assay was incorrectly interpreted as representing a safe environment for neuraxial intervention.

Fortunately, the patient described in this scenario did not appear to suffer any consequence. As the authors themselves note, several studies have found the risk of epidural hematoma to be extraordinarily low even in patients taking clopidogrel on the day of placement. Despite this, we strongly discourage the use of results from standard, kaolin-activated thromboelastography as evidence of a safe hemostatic milieu for neuraxial anesthesia or analgesia in a patient receiving any $P2Y_{12}$ antagonist. Instead, we recommend using TEG Platelet Mapping® or another assay capable of assessing platelet inhibition from these medications.

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(Accepted for publication July 1, 2013.)