distribution. When indicated, confirmation of primary airway disease can be achieved by specific testing of distal lung function and/or by high-resolution CT scanning.

We chose to entitle our manuscript “Lessons From the World Trade Center” to highlight the potential application of our findings to a broader clinical population with intrinsic airway disease and inhalational/environmental injury beyond exposure to WTC dust. Recognition of this pattern when FEV1/VC remains normal and/or by high-resolution CT scanning.

In summary, thrombocytopenia in patients requiring intensive care may not be a contraindication for thromboprophylaxis in most cases. Indeed, a severe degree of thrombocytopenia may actually signify higher thrombotic risk from platelet aggregation rather than bleeding risk.

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REFERENCES


Response

To the Editor:

We thank Dr Thachil for his comments. Although the safety of anticoagulant thromboprophylaxis in patients developing thrombocytopenia was not the subject of our study,1 we agree that this issue is important. In the Prophylaxis for Thromboembolism in Critical Care Trial (PROTECT), patients with thrombocytopenia on enrollment (platelets < 75 x 10^9/L) were excluded.2 If platelet count decreased to < 50 x 10^9/L in the ICU in enrolled patients, the study drug (either unfractionated heparin or dalteparin) was withheld and restarted at the clinician’s discretion. Consequently,

Sighificantly lower ADAMTS13 activity (corresponding with increased platelet aggregation) was observed in patients with severe sepsis in intensive care.3 Additionally, in the most serious complication of disseminated intravascular coagulation in these patients, platelet aggregation as demonstrated by low levels of ADAMTS13 enzyme was observed.4 It is relevant to note that platelet aggregation is a common feature of many diseases prevalent in an intensive care population that would predispose patients to thrombosis rather than to bleeding, and thromboprophylaxis is more likely to be helpful. Of interest, the authors identified severe illness, prior surgery, use of inotropes or vasopressors, and renal replacement therapy, all of which are inherently prothrombotic states where thromboprophylaxis would have been more appropriate than excluded.

We thank Dr Thachil for his comments. Although the safety of anticoagulant thromboprophylaxis in patients developing thrombocytopenia was not the subject of our study,1 we agree that this issue is important. In the Prophylaxis for Thromboembolism in Critical Care Trial (PROTECT), patients with thrombocytopenia on enrollment (platelets < 75 x 10^9/L) were excluded.2 If platelet count decreased to < 50 x 10^9/L in the ICU in enrolled patients, the study drug (either unfractionated heparin or dalteparin) was withheld and restarted at the clinician’s discretion. Consequently,