primary fibrinolysis often occur. The reported incidence of that plasminogen activation varies widely, dependant upon the study reviewed and the test utilized. What is clear is that the monitoring tests routinely utilized by most cardiac surgical centers do not assess the two major hemorrhagic diatheses just mentioned. Activated clotting time, prothrombin time, partial thromboplastin time, and the remainder of a routine coagulation profile do not examine platelet function and they offer only small amounts of information about fibrinolysis.

We have begun routine monitoring of patients with a viscoelastic test of whole blood coagulation, utilizing the Thromboelastograph (TEG), in addition to the ACT and coagulation profile. TEG assesses clot strength from the time of initial fibrin formation through clot development and platelet retraction until eventual lysis. Platelet activity (number and function together), primary fibrinolysis, diffuse intravascular coagulation, and hypercoagulation may all be identified with this technique. TEG provides accurate predictions of postoperative hemorrhage (85 percent predictive), whereas routine coagulation testing does not (≥50 percent predictive). In our series of 1,000 patients there was an 11 percent incidence of post-bypass primary fibrinolysis as monitored by the TEG.

If it is assumed that the 11 percent incidence found in our studies is predictive of postoperative hemorrhage, then Del Rossi et al should have seen 17 or 18 patients in their EACA and placebo groups who might have bled because of primary fibrinolysis. If treatment with EACA was effective only in those 18 patients, that would have been sufficient to show a significant difference in postoperative bleeding and transfusion requirements between groups.

In today's environment of transfusion avoidance due to AIDS and hepatitis, any technique that reduces the need for a transfusion is laudable. The use of EACA for the treatment of primary fibrinolysis may be such a technique. The use of D-8-arginine vasopressin (DDAVP) to enhance platelet and factor VIII activity in cardiac surgery has been advanced by some. Could combinations of both produce even more superior results than those demonstrated by Del Rossi et al?

EACA is not an innocuous drug. In the face of diffuse intravascular coagulation (DIC) it may worsen the clinical picture. DIC, thankfully, is rare in cardiac surgery. However, when it occurs it is not quickly differentiated from primary fibrinolysis by routine testing. TEG may, at least in an animal model, differentiate the two. Universal prescribing of any medication is suspect. We do not agree that routine use of EACA is warranted based upon the results of this study, as there are certainly some patients with undiagnosed DIC who will perish. Del Rossi et al state that patients receiving EACA had fewer major complications than the control group; however, no statistical difference was shown, only trends in very small groups. The claim to fewer complications cannot be validated without supporting statistical data.

In summary, this is a useful study which adds a valuable piece of information to our knowledge about coagulation function during cardiopulmonary bypass. We feel that the study demonstrates primary fibrinolysis to be a major cause of clinical hemorrhage. More important, however, the study also demonstrates the inadequacy of intraoperative coagulation monitoring. We feel that a treatment such as EACA should not be advocated for universal application or individually without demonstrable evidence of a disease process (ie, primary fibrinolysis), utilizing appropriate monitoring.

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To the Editor:

We thank Dr. Spiess and his colleagues for their interest in our article, as well as their kind remarks on the importance of the study. They bring up an interesting point regarding hemostatic defects after CPB. Although thrombocytopenia destruction and dysfunction is commonly seen, this appears more prominently at the end of CPB, and its magnitude is directly proportional to the duration of extracorporeal circulation. Hyperfibrinolysis, on the other side, appears to start with sternotomy and pleural and/or pericardial incision and reaches its peak one hour after the initiation of CPB. Unfortunately, the multiplicity of causes of deregulation of hemostasis and the variable course of these conditions makes clinical assessment of which component has more importance very difficult. Although we acknowledge that the bleeding tendency seen after CPB is multifactorial, there is enough literature support to show that fibrinolytic activity is present in up to 90 percent of patients undergoing open heart surgery with CPB. We believe that if the activation of plasminogen is allowed to proceed, multiple hemostatic changes occur, including thrombocytopenia and platelet dysfunction.

We certainly agree that routine hematologic tests do not target specific hemorrhagic diathesis encountered after CPB. Since there are practical problems related to the rapid laboratory evaluation of fibrinolysis and since EACA in therapeutic doses and in the absence of signs of DIC does not produce any detectable hypercoagulability, it seems logical to use it prophylactically.

Since our study tested only the activity of EACA against placebo, we do not know if there is any synergistic effect of DDAVP or any other compound which has been shown to conserve blood loss. Also, we clearly specified that the differences in major complications are not necessarily attributed to the differences in treatment between the two groups.

We agree with Dr. Spiess and his colleagues that intraoperative monitoring of coagulation would bring valuable information; however, until such practical tests develop and gain clinical acceptance, prophylactic use of pharmacologic compounds that may conserve blood loss after CPB should be considered.