I agree completely with Dr. Stone's cited importance of a "team leader" to coordinate the continuum of care through recovery and disposition. But I disagree that any such person needs to be routinely involved in the EMS or initial evaluation of trauma patients, and that such a team leader need necessarily be a general surgeon. I refer interested readers back to my article where the successful nonsurgeon leadership roles in Canadian and European trauma are discussed. Drs. Grossman and Portner refer to their trauma fellowship in which emergency physicians fill this role. Certainly there will always be a vital need for trauma-receiving facilities to maintain an on-call surgical panel to render operative intervention when appropriate. I simply maintain that the hypothesis that patient outcomes are improved through the routine, rapid arrival of surgeons to major trauma alerts (as arbitrarily mandated for trauma center designation by the American College of Surgeons) remains unproven and can be particularly and potently questioned in the setting of blunt trauma.

In all fairness it must be noted that the conclusions of my article do not speak for all emergency physicians. Indeed, more than one member of our specialty has privately objected to what I have put forth—but for entirely different reasons. These emergency physicians have no greater belief than I do that a surgery residency provides any otherwise unattainable expertise on the non-operative aspects of trauma resuscitation; however, they have justifiable and practical concerns about workflow. Following the resuscitative phase many trauma patients require substantial energies to coordinate imaging and consultants, and to collate and implement their results. Despite being time-consuming, it must be noted that these duties are largely busy work and in most teaching hospitals are relegated to junior house staff. Some emergency physicians fear that my comments will give surgeons license to abandon this onerous element of trauma care and reassign it to us. It seems clear that there needs to be a provider with sufficient time available to coordinate this post-resuscitative phase. In the American model this has traditionally been the surgeon, in the European model this is the intensivist, in the Canadian model this may be a physician (surgeon or non-surgeon) on a rotating trauma call panel. One can also imagine a situation in which supervised physician assistants or nurse practitioners could fill this role.

Drs. Grossman and Portner believe that portions of my article are "inaccurate"; however, both examples they cite concern pre-1980 historical context that, while interesting, does not contradict nor appear to substantively relate to any of my main points. I regret that they find my article "polarizing," but believe that fresh outside perspectives from non-surgeons will be useful—if not vital—to any meaningful resolution of the challenges discussed.

Drs. Exadaktylos and Velmahos use this opportunity to voice their support for the concept of "acute care surgery."[6] However, as discussed in my article, this proposal to save trauma surgery faces potent and perhaps fatal challenges. First, neurosurgeons and orthopedists are unlikely to cooperate in ceding operative procedures to trauma surgeons.[4,7,8] Second, the actual implementation of this restructuring is politically at the individual hospital level, and thus widespread change is subject to local politics and unlikely to occur quickly. Third, it is unclear that the American Board of Surgery might ever consider this a distinct enough body of knowledge to qualify as a true subspecialty. Finally, surgeons themselves are not convinced that the acute care surgeon model is financially viable.[9] Emergency physicians have no reason to object to the acute care surgery model, but could rightly desire and expect more substantial and fundamental reform.

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doi:10.1016/j.annemergmed.2009.04.005

Funding and support: By *Annals* policy, all authors are required to disclose any and all commercial, financial, and other relationships in any way related to the subject of this article that might create any potential conflict of interest. The author has stated that no such relationships exist. See the Manuscript Submission Agreement in this issue for examples of specific conflicts covered by this statement.

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Prothrombin Complex Concentrate and Fatal Thrombosis: Poor Evidence to Implicate a Relatively Safe Drug

To the Editor:

We read with great interest the case report on intracardiac thrombosis associated with prothrombin complex concentrate.[1]

Several clarifications should be noted regarding prothrombin complex concentrate. There are 2 types of prothrombin complex concentrates available – activated-prothrombin complex concentrate for treating hemophilia A and B with inhibitors (eg, FEIBA, Baxter) and non-activated-prothrombin complex concentrate (Hemophilia B treatment in the past) licensed for warfarin reversal in Europe and Canada. Furthermore, there are 2 types of non-activated-prothrombin complex concentrates: a 4-factor-prothrombin complex concentrate containing adequate amounts of vitamin K-dependent factors II, VII, IX and X, and a 3-factor-prothrombin complex concentrate containing significantly lower amounts of factor VII (less than one third of factor IX). In the US only 3-factor-prothrombin complex concentrates are available (Profilnine, Grifols and Bebulin, Baxter) and used off label for rapid warfarin reversal. This patient was given Profilnine at 50IU/Kg (4000IU in minimum 30CC) very rapidly - within one minute - (package insert - rate not to exceed 10cc/minute). He was also simultaneously given 24μg desmopressin. Fifty minutes later a pericardiocentesis was performed; immediately thereafter the patient developed pulseless electrical activity. The echocardiogram at that time showed right-ventricular thrombus. Thus, thrombus developed suddenly after the procedure, and more than 50 minutes after the prothrombin complex concentrate and desmopressin infusions. Importantly, desmopressin is more prothrombotic and used routinely to treat bleeding in type 1 von Willebrand disease and mild hemophilia A by releasing endothelial-stored von Willebrand factor and factor VIII. The indication for desmopressin in this patient is not given – presumably it was to counteract aspirin/dipyrimadole. However, with an international normalized ratio (INR) >12, his bleeding was clearly due to warfarin toxicity. Diabetic patients have hyperactive platelets and a potent drug like desmopressin could be thrombotically catastrophic. If there were a hypercoagulable state caused by prothrombin complex concentrate, the thrombus should have developed before pericardiocentesis, not immediately after. Desmopressin likely caused increased von Willebrand factor (peak effect 30-60 minutes post-infusion) and platelet hyperactivity; and, following trauma to the right ventricle during pericardiocentesis, there was formation of a thrombus. Minor trauma to the right-ventricle would be difficult to detect during autopsy. On admission the INR was >12.8; assuming all vitamin K-dependent factors were <10%[2], a dose of 50U/kg would increase factor II by 50%, VII by <10%, factor IX by 16% and X by 26%[3]. The INR during resuscitation remained >12.8, indicating that most factors would have been significantly low. In our experience, Profilnine for warfarin reversal has been a safe drug showing suboptimal correction of INR[3] rather than a hypercoagulable state. We believe this case requires reevaluation to avoid implicating a relatively safe and important life-saving drug in thrombotic complication. The prothrombin complex concentrate-associated thrombogenicity for Hemophilia B treatment was due to cumulative effects of factors II and X with longer half lives than factor IX during a long-term therapy (7-10 days), not to a single dose of non-activated-prothrombin complex concentrate. Thrombotic complications due to activated-prothrombin complex concentrate (eg, FEIBA) are well known in hemophilia patients with chronic liver disease because of presence of several activated factors (eg, factors VIIa and FXa). Non-activated-prothrombin complex concentrate for severe warfarin-induced vitamin K-dependent factors simply restores non-activated factors in a dose-dependent manner and allows for establishment of normal hemostatic mechanisms rather than induce hypercoagulable state.

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doi:10.1016/j.annemergmed.2009.07.004

Funding and support: By *Annals* policy, all authors are required to disclose any and all commercial, financial, and other relationships in any way related to the subject of this article that might create any potential conflict of interest. The authors have stated that no such relationships exist. See the Manuscript Submission Agreement in this issue for examples of specific conflicts covered by this statement.

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In reply:

I would like to thank Drs. Sarode and Matevosyan for an interesting letter regarding our case report, "Massive, Fatal, Intracardiac Thrombosis Associated With Prothrombin Complex Concentrate."[1] Their detailed description of the variety of prothrombin complex concentrates is appreciated. The patient was given desmopressin for correction of possible uremic bleeding, as he presented with acute renal failure, a BUN of 164 mg/dL and a pericardial effusion. Drs. Sarode and Matevosyan suggest that the thrombus formed due to the desmopressin and that desmopressin is particularly prothrombotic. However, clinical literature does not support this claim. A recent meta-analysis looking at the utility of perioperative desmopressin in 2,488 patients found no significant difference in rates of thrombotic complications in patients receiving desmopressin versus placebo.[2] Drs. Sarode and Matevosyan also suggest that there was undetected trauma