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A small amount can make a difference: a prospective human study of the paradoxical coagulation characteristics of hemothorax

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- Autotransfusion
- Hemothorax
- Coagulation
- Pleural blood
- Disseminated intravascular coagulation

Abstract

BACKGROUND: The evacuated hemothorax has been poorly described because it varies with time, it has been found to be incoagulable, and its potential effect on the coagulation cascade during autotransfusion is largely unknown.

METHODS: This is a prospective descriptive study of adult patients with traumatic chest injury necessitating tube thoracostomy. Pleural and venous samples were analyzed for coagulation, hematology, and electrolytes at 1 to 4 hours after drainage. Pleural samples were also analyzed for their effect on the coagulation cascade via mixing studies.

RESULTS: Thirty-four subjects were enrolled with a traumatic hemothorax. The following measured coagulation factors were significantly depleted compared with venous blood: international normalized ratio (INR) (0.9 vs 1.1) (P < .001) and activated partial thromboplastin time (aPTT) (180 vs 24.5 seconds) (P < .001). Mixing studies showed a dose-dependent increase in coagulation dilutions through 1:8 (P < .05).

CONCLUSIONS: An evacuated hemothorax does not vary in composition significantly with time and is incoagulable alone. Mixing studies with hemothorax plasma increased coagulation, raising safety concerns.

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Recent evidence has shown that the use of packed red blood cell (RBC) units is directly related to increased 30-day mortality, acute respiratory distress syndrome, nosocomial infection rates, and hospital length of stay. As a result, both the Eastern Association for the Surgery of Trauma and the Society of Critical Care Medicine suggest autotransfusion as an alternative to avoid allogeneic RBC use.

Our prior research showed that an evacuated hemothorax in the setting of trauma contains a predictable hematocrit, decreased platelets, and questionable coagulation factor concentration. We and others have shown that an evacuated hemothorax cannot clot on its own, likely because of defibrination. In addition, we found a highly elevated D-dimer
level in an evacuated hemothorax compared with concurrent venous samples, which is concerning for activated fibrinolysis. Other work with unwashed shed blood in the settings of cardiothoracic surgery and orthopedics mirror these findings and also have found evidence of activated coagulation factors, fibrin degradation products, and elevated concentrations of inflammatory mediators.4,7–9

Practical applications of using shed blood, with the intent of using less banked blood, have been considered, particularly in orthopedic and cardiothoracic surgery.10,11 This also potentially avoids exposing patients to the increased risks of acute respiratory distress syndrome, transfusion-associated lung injury, blood-borne pathogens, and infection risks associated with allogeneic blood transfusion.10–13

The effect of autotransfusion on coagulation has been investigated previously in laboratory and clinical settings. Although it has been shown to possess little fibrinogen, shed blood has been shown to contain activated clotting factors as well as tissue plasminogen activator and antithrombin III.4,5,14 Mixed results have been shown when examining the impact of autotransfusion on coagulation. Some authors have reported increases in clot formation,4,15 no effect,5.16,17 or decreased coagulation as evidenced by increased wound drain output.7

Our previous work characterized an evacuated hemothorax 4 hours after evacuation because that is the limit for the autotransfusion of shed blood recommended by the American Association of Blood Banks. However, in clinical practice, when we have used autotransfusion, we have typically done so in a timelier manner, usually within 1 hour of evacuation. The evolution of the characteristics of an evacuated hemothorax over time has not been described previously.

Our purpose was to further observe the effect of time on evacuated hemothoraces from trauma patients to identify hematologic changes that might affect transfusion decisions. We hypothesized that there would be no significant change in the hematologic, electrolyte, or coagulation parameters over time between 1 and 4 hours after evacuation. A secondary objective was to further evaluate the effect that an evacuated hemothorax, previously defined as incoagulable, may have on hematologic, electrolyte or coagulation parameters over time has not been described previously.

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Materials and Methods

Patient selection

The study was conducted during a 15-month period at University Hospital, San Antonio, TX, an American College of Surgeons–verified level 1 trauma center. The study was approved by the University of Texas Health Science Center at San Antonio Institutional Review Board. Subjects were considered for enrollment in the study if greater than 135 mL of hemothorax was drained in the first hour after tube thoracostomy. Subjects were excluded if they were less than 18 years, pregnant, a known prisoner at the time of tube thoracostomy, or had tube thoracostomy at any facility other than University Hospital. The evacuated hemothorax was collected from either an autotransfusable collecting unit (Oasis 2050 ATS; Atrium Medical Corporation, Hudson, NH) or a nonautotransfusable collecting unit (Oasis 3650 ATS, Atrium Medical Corporation) with the type of collection device determined by nonstudy providers.

Sample collection

A sterile 50-mL syringe was used to withdraw 35 mL hemothorax from the thoracostomy collection chamber needleless access port at 1, 2, 3, and 4 hours after chest tube thoracostomy. After removal from the thoracostomy collection chamber, the evacuated hemothorax was transferred into laboratory collection tubes containing either no preservative or 1 of the following: sodium citrate, lithium heparin, or EDTA. Of the 35 mL collected, half was sent to the hospital core laboratory for coagulation, hematology, and electrolyte profile analysis as presented in Tables 1 and 2. The second half was spun twice at 3,000 g for 15 minutes, and plasma supernatant was frozen for subsequent mixing study analysis. Concurrent venous blood values were obtained from the subjects’ charts based on laboratory values obtained in the emergency center at the time point closest to chest tube thoracostomy. This venous sample was then compared with the evacuated hemothorax sample. The Injury Severity Score and calculated probability of survival using the Trauma and Injury Severity Score were subsequently obtained from the hospital trauma database (Digital Innovations Incorporated, Forest Hill, MD).

Mixing studies

Frozen citrated evacuated hemothorax plasma was thawed at 37°C. Normal pooled plasma (NPP) was mixed with evacuated hemothorax plasma at dilutions of 1:1, 1:2, 1:4 up to 1:128 (parts evacuated hemothorax plasma:parts total). Dilutions were performed by hand using a precision micro-pipette. These mixtures were analyzed for the international normalized ratio (INR) (RecombiPlasTin; Instrumentation Laboratories, AH Breda, The Netherlands) and activated partial thromboplastin time (aPTT) (SynthAsil, Instrumentation Laboratories) using an ACL TOP (Instrumentation Laboratories) with NPP (Precision Biologic; Nova Scotia, Canada) run alone to act as a control.

Data analysis

Data are expressed as medians with 25% and 75% quartiles. Comparisons between groups were analyzed using analysis of variance and SigmaPlot 11.0 software (Systat Software Incorporated, San Jose, CA). Venous blood values were compared with evacuated hemothorax values using the Kruskal-Wallis 1-way analysis of variance on ranks for multiple comparisons. Mixing study data were initially compared using the Friedman repeated measures
<table>
<thead>
<tr>
<th>Parameter</th>
<th>Evacuated hemotherax</th>
<th>Evacuated hemotherax</th>
<th>Evacuated Hemothorax</th>
<th>Evacuated Hemothorax</th>
<th>Patient IV (IQR)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1 h (IQR)</td>
<td>2 h (IQR)</td>
<td>3 h (IQR)</td>
<td>4 h (IQR)</td>
<td></td>
</tr>
<tr>
<td>INR</td>
<td>&gt;9 (invariant) (P &lt; .001)*</td>
<td>&gt;9 (invariant) (P &lt; .001)*</td>
<td>&gt;9 (invariant) (P &lt; .001)*</td>
<td>&gt;9 (invariant) (P &lt; .001)</td>
<td>1.1 (1.0–1.2)</td>
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<tr>
<td>aPTT (sec)</td>
<td>&gt;180 (invariant) (P &lt; .001)*</td>
<td>&gt;180 (invariant) (P &lt; .001)*</td>
<td>&gt;180 (invariant) (P &lt; .001)*</td>
<td>&gt;180 (invariant) (P &lt; .001)</td>
<td>24.5 (22.0–29.0)</td>
</tr>
<tr>
<td>Fibrinogen (mg/dL)</td>
<td>&lt;50 (invariant) (P &lt; .001)*</td>
<td>&lt;50 (invariant) (P &lt; .001)*</td>
<td>&lt;50 (invariant) (P &lt; .001)*</td>
<td>&lt;50 (invariant) (P &lt; .001)</td>
<td>309.5 (263.0–352.0)</td>
</tr>
<tr>
<td>D dimer (ng/mL)</td>
<td>&gt;7,360 (invariant)</td>
<td>&gt;7,360 (invariant)</td>
<td>&gt;7,360 (invariant)</td>
<td>&gt;7,360 (invariant)</td>
<td>Not measured</td>
</tr>
<tr>
<td>WBC (K/μL)</td>
<td>10.1 (6.5–17.8)</td>
<td>12.3 (6.3–16.5)</td>
<td>9.2 (6.6–17.0) (P = .001)</td>
<td>13.1 (6.4–16.8)</td>
<td>15.8 (9.3–23.9)</td>
</tr>
<tr>
<td>Hematocrit (%)</td>
<td>36.3 (33.4–37.6)</td>
<td>34.8 (31.6–37.9)</td>
<td>33.8 (30.8–37.3) (P = .002)</td>
<td>33.6 (29.7–37.6) (P = .002)</td>
<td>39.5 (34.1–41.9)</td>
</tr>
<tr>
<td>Hemoglobin (g/dL)</td>
<td>12.5 (11.2–13.2)</td>
<td>11.7 (10.8–13.3)</td>
<td>11.7 (10.5–12.7) (P = .002)</td>
<td>11.2 (10.1–12.9) (P = .002)</td>
<td>13.5 (11.8–14.4)</td>
</tr>
<tr>
<td>Platelet (K/μL)</td>
<td>47.0 (24.0–76.0) (P &lt; .001)*</td>
<td>48.0 (20.5–70.8) (P &lt; .001)*</td>
<td>49.0 (24.0–79.0) (P &lt; .001)*</td>
<td>51.0 (27.3–80.0) (P &lt; .001)*</td>
<td>224.0 (197.0–251.5)</td>
</tr>
<tr>
<td>Na (mmol/L)</td>
<td>141 (138–142)</td>
<td>141 (138–143)</td>
<td>141 (139–143)</td>
<td>141 (138–143)</td>
<td>142 (140–143)</td>
</tr>
<tr>
<td>K (mmol/L)</td>
<td>4.8 (4–5.6) (P &lt; .001)</td>
<td>4.4 (3.9–5.3) (P &lt; .001)</td>
<td>4.3 (3.9–5.4) (P &lt; .001)</td>
<td>4.5 (3.9–5.2) (P &lt; .001)</td>
<td>3.7 (3.4–3.8)</td>
</tr>
<tr>
<td>Cl (mmol/L)</td>
<td>106 (104–108)</td>
<td>106 (104–108)</td>
<td>106 (104–108)</td>
<td>106 (104–108)</td>
<td>107 (104–110)</td>
</tr>
<tr>
<td>Bicarbonate (mmol/L)</td>
<td>20 (17–22)</td>
<td>19 (17–21)</td>
<td>19 (17–20)</td>
<td>19 (17–21)</td>
<td>23 (20–26)</td>
</tr>
<tr>
<td>Anion gap</td>
<td>14 (10–17) (P &lt; .001)*</td>
<td>15 (13–19) (P &lt; .001) *</td>
<td>15 (12–18) (P &lt; .001)*</td>
<td>15 (12–18) (P &lt; .001)</td>
<td>8 (6–11)</td>
</tr>
<tr>
<td>BUN (mg/dL)</td>
<td>15 (10–18)</td>
<td>15 (11–18)</td>
<td>14 (11–18)</td>
<td>14 (12–19)</td>
<td>13 (10–17)</td>
</tr>
<tr>
<td>Ca (mg/dL)</td>
<td>8.1 (7.7–9.1)</td>
<td>7.9 (7.6–8.9)</td>
<td>8.0 (7.7–8.6)</td>
<td>8.0 (7.6–8.7)</td>
<td>8.5 (7.7–8.9)</td>
</tr>
<tr>
<td>AST (mg/dL)</td>
<td>847 (173–1,642) (P &lt; .001)*</td>
<td>597 (231–1,647) (P &lt; .001)*</td>
<td>593 (204–1,416) (P &lt; .001)*</td>
<td>607 (211–1,477) (P &lt; .001)*</td>
<td>65 (35–190)</td>
</tr>
<tr>
<td>ALT (U/L)</td>
<td>195 (66–357) (P &lt; .001)*</td>
<td>169 (63–361) (P &lt; .001)*</td>
<td>173 (78–357) (P &lt; .001)*</td>
<td>183 (74–358) (P &lt; .001)*</td>
<td>41 (27–142)</td>
</tr>
</tbody>
</table>

ALT = alanine aminotransferase; AST = aspartate aminotransferase; aPTT = activated partial thromboplastin time; BUN = blood urea nitrogen; INR = international normalized ratio; IQR = interquartile range; WBC = white blood cell.

*No P value is generated for P < .001 because of statistical package limitations.
analysis of variance on ranks. Further analysis used the Dunn method, with NPP serving as the control group, and the Bonferroni and Tukey tests were used for multiple comparisons of each dilution. A \( P \) value less than .05 was considered to be statistically significant for all analyses.

Results

Patient population

Thirty-four patients were enrolled over the 15-month period. Ninety-one percent were men, and the median age was 34 years (range 25 to 48 year). Fifty-nine percent of the hemothoraces were caused by blunt injury. The median Trauma and Injury Severity Score (TRISS) was 21 (range 10 to 29), and the median Injury Severity Score was .956 (range .377 to .984). Because patient enrollment was determined 1 hour after arrival to the hospital, 3 patients died after enrollment but before completion of the study collection period. Their collection chamber was disconnected, and samples were collected and analyzed per the study protocol.

Hematology

The evacuated hemothorax white blood cell count was shown to be reduced when compared with patient venous laboratory values at hours 2 and 3. The white blood cell counts at hours 1 and 4 were not significantly different from the venous blood specimens. The evacuated hemothorax hematocrit and hemoglobin concentrations were reduced compared with patient venous blood samples at 3 and 4 hours; hours 1 and 2 were not significantly different. Platelet counts were substantially reduced at all time points compared with concurrent patient venous blood samples. Evacuated hemothorax sample data did not show a significant difference at any measured time point when compared with any other collected time point nor was a notable trend identified across the 4 measured time points (Table 1).

Table 2  Mixing study results

<table>
<thead>
<tr>
<th>Dilution ratio(^1)</th>
<th>1-h aPTT mixing study (s) (IQR)</th>
<th>4-h aPTT mixing study (s) (IQR)</th>
<th>1-h INR mixing study (s) (IQR)</th>
<th>4-h INR mixing study (s) (IQR)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1:2</td>
<td>24.7 (23.3–26.6) ( (P &lt; .001)^* )</td>
<td>24.1 (19.4–25.1) ( (P &lt; .001)^* )</td>
<td>.99 (.97–1.02)</td>
<td>.98 (.97–1.00)</td>
</tr>
<tr>
<td>1:4</td>
<td>25.0 (23.9–26.3) ( (P &lt; .001)^* )</td>
<td>23.7 (19.5–25.2) ( (P &lt; .001)^* )</td>
<td>.89 (.86–.93)</td>
<td>.88 (.86–.91)</td>
</tr>
<tr>
<td>1:8</td>
<td>26.4 (25.4–27.4) ( (P &lt; .001)^* )</td>
<td>25.5 (20.9–26.4) ( (P &lt; .001)^* )</td>
<td>.90 (.87–.93)</td>
<td>.89 (.86–.91)</td>
</tr>
<tr>
<td>1:16</td>
<td>28.3 (26.9–29.1) ( (P &lt; .001)^* )</td>
<td>27.3 (22.5–27.8) ( (P &lt; .001)^* )</td>
<td>.92 (.88–.96)</td>
<td>.90 (.87–.93)</td>
</tr>
<tr>
<td>1:32</td>
<td>29.9 (28.2–30.6)</td>
<td>28.5 (24.4–29.0)</td>
<td>.94 (.91–.96)</td>
<td>.93 (.90–.95)</td>
</tr>
<tr>
<td>1:64</td>
<td>31.1 (29.0–31.9)</td>
<td>29.1 (25.7–30.0)</td>
<td>.95 (.92–.98)</td>
<td>.93 (.92–.95)</td>
</tr>
<tr>
<td>1:128</td>
<td>32.1 (29.4–32.8)</td>
<td>29.9 (27.4–30.4)</td>
<td>.96 (.94–.99)</td>
<td>.95 (.93–.96)</td>
</tr>
<tr>
<td>Control</td>
<td>30.9 (29.1–31.1)</td>
<td>30.9 (29.1–31.1)</td>
<td>.98 (.95–.98)</td>
<td>.98 (.95–.98)</td>
</tr>
</tbody>
</table>

\( aPTT = \) activated partial thromboplastin time; \( INR = \) international normalized ratio; \( IQR = \) interquartile range.

\(^*\)No \( P \) value is generated for \( P < .001 \) because of statistical package limitations.

\(^1\)Dilution ratio represents parts hemothorax plasma to parts total.

Coagulation

INR and aPTT were immeasurably high in all collected data samples. The D-dimer level was immeasurably high in all samples except those from 1 subject. Fibrinogen was undetectable in any sample. These findings were unchanged across all measured time points.

Chemistries

Evacuated hemothorax sodium, chloride, bicarbonate, and blood urea nitrogen were not significantly different from patient venous blood samples. Potassium, aspartate aminotransferase, and alanine aminotransferase were markedly and significantly elevated compared with patient venous specimens at all measured time points. Anion gap and calcium were also significantly elevated compared with patient venous samples at measured time points; however, both were still within physiologic ranges. No chemistry value changed significantly over the measured time points.

Mixing studies

Two- and 3-hour mixing study data were not obtained because preliminary data analysis suggested no difference between 1, 2, 3, and 4 hours after evacuation. Analysis on the first 14 patients showed no difference in mixing study results comparing the 1- and 4-hour samples; thus, mixing studies were not performed on the 4-hour samples after the first 14 as a cost-saving measure.

Mixing studies using 1-hour samples showed a significant acceleration in the INR (reduced time to clot formation) at both the 1:4 and 1:8 dilutions versus NPP (Fig. 1). Similarly, 4-hour data showed a decrease in INR at 1:4, 1:8, and 1:16 compared with NPP. At 1 hour, the dilutions of 2, 4, and 16 were all found to be significantly different from each other, indicating a dose-dependent relationship. The INR at the
1:8 dilution was found to be statistically lower than that of the 1:16 ratio.

aPTT mixing studies on 1-hour samples showed accelerated coagulation (reduced time to clot) at 1:2, 1:4, and 1:8 when compared with NPP (Fig. 2). Mixing studies performed on 4-hour samples showed enhanced coagulation compared with NPP at dilutions of 1:2 through 1:16. At 1 hour, aPTT mixing studies showed that dilutions of 1:4, 1:8, and 1:16 showed dose-dependent acceleration in coagulation (a decrease in aPTT). A dilution of 1:2 was found to be different from 1:8 and 1:16 but indifferent from 1:4, again indicating a dose-dependent relationship (Fig. 2).

Comments

The primary objective of this study was to compare the coagulation, hematologic, and electrolyte profiles of evacuated hemothoraces from acutely injured patients because they change with time after being evacuated from the pleural cavity. We have shown that the coagulation composition, hematologic profile, and clinical chemistry levels of evacuated hemothoraces do not change significantly between 1 and 4 hours after evacuation. Comparing these results with the patients’ venous blood, we were able to confirm previously shown differences between evacuated hemothoraces and circulating venous blood.

Consistent with our prior work, evacuated hemothoraces are defibrinated, thrombocytopenic, and mildly anemic and contain elevated levels of D dimer. These prospective data characterize evacuated hemothoraces with respect to hematologic, coagulation, and electrolyte composition over time.

The secondary objective of our study was to show the effects of evacuated hemothorax plasma on coagulation through in vitro mixing studies using NPP. In doing so, we showed a paradoxical hypercoagulability that dose dependently lowered the INR and aPTT. Despite being unable to form a thrombus after evacuation from the pleural cavity, as
measured via aPTT and INR in the laboratory, an evacuated hemothorax has a dose-dependent acceleration of thrombus formation when mixed with normal pooled plasma, a source of fibrinogen. We theorize that this acceleration in coagulation is caused by the activation of coagulation factors in the evacuated hemothorax, resulting in the consumption of fibrinogen, creating plasma in the collection chamber that is rich in activated coagulation factors without a substrate on which to act. This is further supported by our findings that no sample had measurable fibrinogen levels, and each had immeasurably high levels of D dimer.

Although we cannot directly extrapolate these findings to clinical care, these data do raise some safety concerns regarding direct autotransfusion of evacuated hemothoraces. When autotransfusion has been performed at our institution, a range of 1 to 2 L has been used. As we showed, as little as 1:16 dilution of evacuated hemothorax plasma mixed with NPP causes increased coagulation. That means that using as little as 300 mL of evacuated hemothorax during a traumatic resuscitation could lead to a hypercoagulable condition, which may worsen with the increasing volume of the evacuated hemothorax autotransfused. This study raises concerns that autotransfusion of an unwashed evacuated hemothorax, which likely contains both activated fibrinogenic and fibrinolytic factors, could, despite the natural hemostatic inhibitors in the blood, activate both coagulation and fibrinolysis in vivo, leading to a disseminated intravascular coagulation (DIC)-like process.

From these data, shed hemothorax has been quantified between 1 and 4 hours after evacuation; has been shown to have a measureable, stable hemotologic, coagulation, and electrolyte profile; and remains a viable source of RBCs. However, in vitro data show that an evacuated hemothorax has the potential to induce a highly active coagulative state, raising safety concerns that may not be proportional to the possible benefit of autotransfusion, particularly if one has access to adequate supplies of banked component blood. If a whole evacuated hemothorax must be autotransfused, the coagulation status should be monitored closely.

References


Discussion

Sharmila Dissanaike, M.D. (Lubbock, TX): This is a very interesting study; I congratulate you on critically looking at something that we all intuitively would accept as a good idea. The suspicion would have been that autotransfusion would make you hypocoagulable, but instead you have shown that it is actually making you hypercoagulable. Do you think there is still a role for transfusion? Obviously, the hypercoagulable state can be modified by anticoagulants. So, should we still be considering it or should we just drop the idea completely?

W. Zachary Smith, B.S. (San Antonio, TX): We are actually further investigating this with the next stage of our study and instead of using normal pooled plasma, we are looking at the actual patient’s physical blood as the control for our mixing study. We believe that this needs to be further investigated because we are not exactly sure why it becomes hypercoagulable. Once we can elucidate that, we will be better able to make recommendations. At this time, we believe the risks most likely outweigh the benefits.

John Aucar, M.D. (Tyler, TX): This is a very interesting paper. I commend you for that. I did not hear you mention
much about clinical outcomes (ie, actual banked blood transfusion requirements, packed cells or red cells, or thrombotic complications). It is a very interesting laboratory approach, but could you comment on that for us please?

Mr Smith: Certainly. This was not set out to be a clinical experiment in which we actually autotransfused patients. This was designed to look at in vitro modeling that could be extrapolated to in vivo studies in the future. So, we actually did not transfuse evacuated hemothoraces into any patients. This was purely a laboratory study.

Steve Smith, M.D. (Columbia, SC): I have one of those feared hypothetical questions for you. Let’s make the assumption that a transfused hemothorax actually does create a hypercoagulable state. Could we not use this to our advantage? Many of our multisystem injured trauma patients who have had significant blood loss are hypocoagulable. So, could we not potentially use this to correct that hypocoagulable state, which we are trying to do with current transfusion strategies anyway?

Mr Smith: Sure. That is exactly something that we have discussed in our group. We found that when measuring the patients’ laboratory coagulation studies including INR and aPTT, they are actually hypercoagulable marginally when they first roll into the trauma bay, indicating to us that they have not reached that hypocoagulable state (coagulopathy of trauma) yet. To us, we would most likely transfuse at about 1 hour after the injury, between 30 and 60 minutes, and they would most likely not be hypocoagulable yet. The concern is that you drive them toward hypercoagulability, potentially even some sort of DIC-like effect because you just dumped a lot of activated factor into their system. In conclusion, we do not really know what would happen. You are right. They could become normocoagulable, hypercoagulable, or even develop a full-blown DIC like process.

Fred Pieracci, M.D. (Denver, CO): That was a nice talk. Are these phenomena specific to the pleural space or have you looked at intra-abdominal hemorrhage as well?

Mr Smith: We have not looked at intra-abdominal specifically. We have looked at other studies that have done this with cardiothoracic surgery taking it from the pericardial space as well as some orthopedic studies that have looked at this. Their outcome did not measure laboratory studies. They measured wound output, and they found that it was highly variable in their studies. Some studies would show an increase in wound output; some studies showed no increase. Some studies showed a decrease when they autotransfused intraoperatively. No, we have not looked at abdominal specifically.

Dr Dissanaike: Actually one of the articles later on in this session discusses the hypocoagulable state of traumas; we have 2 very interesting articles in the same topic. Thank you very much. Very nicely done.