Hemostasis and Hemodilution: A Quantitative Mathematical Guide for Clinical Practice

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Quantitative changes of hemostasis during hemodilution remain unclear. With the increasing popularity of artificial blood substitutes (ABS), which solely provide oxygen-transport capacity, this issue becomes even more complex. We developed a mathematical model to quantitatively analyze hemostasis during hemodilution and validated it by recalculating patient data. We calculated and compared maximal allowable blood losses (MABL) related to minimal acceptable hematocrit, platelet concentration, and plasma fibrinogen concentration. MABL is the maximal blood loss that can be tolerated without any additional blood products. The variable with the smallest MABL thus limits hemodilution foremost. Hemodilution included isovolemic replacement of blood loss with colloid or acute normovolemic hemodilution (ANH) followed by isovolemic replacement of blood loss with colloid and ABS. We also related our findings to preoperative patient data (n = 204). The decline in platelet concentrations rarely (<2% of all patients) limits hemodilution. By contrast, critical plasma fibrinogen (<100 mg/dL) concentrations can often (<20% of all patients) limit hemodilution if their initial concentrations are within the lower normal range (<300 mg/dL). These findings become more frequent if ANH is combined with ABS. Under those circumstances ANH blood products are solely required for stabilization of hemostasis, thereby defeating the original purpose of combining ANH with ABS. (Anesth Analg 2003;96:929–35)

Controversies surrounding hemostasis during hemodilution are as old as hemodilution itself. There are ample data with respect to the qualitative effects of different infusion solutions on hemostasis during hemodilution, both in vitro and in vivo. Only a few studies have prospectively addressed quantitative changes in hemostasis during extreme hemodilution (1–8). Most of these studies differ in the type of blood products used. In these situations, clinicians are therefore left with almost nothing for the management of hemostasis except for general, nearly formula-like replacement strategies (8–11). The strength of these strategies is partially limited by the fact that some are based on retrospective data, on data obtained from whole-blood studies, or on data with small sample sizes. Additionally, the strategies give recommendations only for average patients, i.e., patients with normal hemostatic profiles.

No study has evaluated patients with abnormal hemostatic profiles or with profiles within the lower normal range. It is thus unknown whether critical hemostatic levels can become primary transfusion triggers, i.e., whether the need for hemostatic factors occurs before that for red blood cells (RBC).

These problems could become even more overt when artificial blood substitutes (ABS) are included. At this point, ABS have been developed only to provide oxygen-carrying capacity; none of the currently known ABS have predetermined, prohemostatic activity (12). Many clinical trials have also combined ABS with acute normovolemic hemodilution (ANH) (see Ref. 12 for further reference) to further extend the blood loss, which can be tolerated without retransfusion of ANH blood. This idea has even led to a new ANH concept (13). However, this approach might be dangerous in certain situations. Exchange of whole blood during ANH removes not only RBC, but also platelets and coagulation factors from the patient into a plastic bag. Patients then enter surgery with significantly smaller RBC, platelet, and coagulation factor

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concentrations. During surgery, ABS temporarily maintain oxygen-carrying capacity and thereby compensate for the continuing loss of RBC. Hemostasis, however, is not preserved, because platelets and coagulation factors are persistently lost, and ABS do not provide any prohemostatic activity. One can speculate that in certain situations, critical hemostatic levels might be reached before critical hematocrits (Hct). In this case, one would have to retransfuse ANH blood solely because of impaired hemostasis and not because of compromised oxygen-carrying capacity. No study has specifically addressed this problem.

These issues can be resolved only by prospective, randomized clinical trials. However, to obtain strong and conclusive data, study design and protocol must be based on valid considerations that can identify hemostatic profiles or forms of hemodilution, which eventually result in critical hemostatic levels as primary transfusion triggers. A mathematical model before prospective, randomized clinical trials allows for various manipulations of all variables and even for simulation of extreme conditions. The data generated then provide sound information to identify relevant subgroups of patients, forms of hemodilution, or both.

To this end, we developed a mathematical model to quantitatively analyze hemostasis during hemodilution. Hemodilution also included the combination of ANH with ABS. We further validated the mathematical principle of our model by recalculation of individual clinical data from patients undergoing intraoperative hemodilution. Finally, we related our findings to preoperative patient data to judge their clinical relevance.

**Methods**

The exponential decline of blood particle concentration during isovolemic hemodilution, as first described for Hct by Bourke and Smith (14), provides the mathematical foundation for our model.

\[ \text{Hct}_{BL} = \text{Hct}_{INIT} \times e^{-\frac{BL}{EBV}} \]  

(1)

All abbreviations used to simplify formulas are listed in Appendix 1.

The development of formulas to calculate red cell mass/Hct during isovolemic hemodilution, including ANH, have been extensively described elsewhere (15,16).

Because platelets are also corpuscular blood particles, their concentration during isovolemic hemodilution is given by the following equation:

\[ \text{Plt}_{BL} = \text{Plt}_{INIT} \times e^{-\frac{BL}{EBV}} \]  

(2)

Unlike the volume of distribution of RBC or platelets, the actual volume of distribution of fibrinogen does not equal whole-blood volume, but only plasma volume. Consequently, the volume of distribution of fibrinogen increases during hemodilution as Hct declines. For that reason, one cannot simply use the equations given above and change them accordingly. Instead, one first has to mathematically convert plasma fibrinogen concentration into whole-blood fibrinogen concentration, as follows:

\[ \text{Fib}_{BLOOD} = \text{Fib}_{PLASMA} \times (1 - \text{Hct}) \]  

(3)

Because the volume of distribution for whole-blood fibrinogen is equal to the blood volume and remains unchanged during isovolemic hemodilution, one can now substitute \( \text{Fib}_{BLOOD} \) for Hct in Equation 1 and determine whole-blood fibrinogen concentration during hemodilution:

\[ \text{Fib}_{BLOOD-BL} = \text{Fib}_{BLOOD-INIT} \times e^{-\frac{BL}{EBV}} \]  

(4)

Any given whole-blood fibrinogen concentration can be calculated back into its corresponding plasma fibrinogen concentration:

\[ \text{Fib}_{PLASMA} = \frac{\text{Fib}_{BLOOD}}{1 - \text{Hct}} \]  

(5)

The maximal allowable blood loss (MABL) is the blood loss that can be compensated for without the need for (allogeneic) blood products. Comparing MABL with respect to minimal acceptable Hct, platelet concentration, and fibrinogen concentration reveals if and under which circumstances each variable actually limits isovolemic hemodilution.

On the basis of our previous studies (15,16) and the formulas given above, one can determine MABL by following equations:

\[ \text{MABL}_{HCT} = \text{EBV} \times \ln \left( \frac{\text{Hct}_{INIT}}{\text{Hct}_{MIN}} \right) \]  

(6)

\[ \text{MABL}_{PLT} = \text{EBV} \times \ln \left( \frac{\text{Plt}_{INIT}}{\text{Plt}_{MIN}} \right) \]  

(7)

\[ \text{MABL}_{FIB-BLOOD} = \text{EBV} \times \ln \left( \frac{\text{Fib}_{BLOOD-INIT}}{\text{Fib}_{BLOOD-MIN}} \right) \]  

(8)

To validate the underlying mathematical foundation of our model, we recalculated prospectively collected individual data from 20 patients undergoing knee replacement surgery under combined spinal-epidural anesthesia. Data were originally collected during a prospective, randomized clinical study to explore the effects of perioperatively administered colloids and crystalloids on hemostasis (17). Here, data were taken from 20 ASA physical status I–III patients who exclusively received hydroxyethyl starch (200/0.5) for volume replacement, in addition to a baseline infusion of lactated Ringer’s solution (5 mL·kg⁻¹·h⁻¹). Aside from a continuous infusion rate of 3 mL·kg⁻¹·h⁻¹, hydroxyethyl starch was infused to...
compensate for surgical blood loss (1:1) and to correct suspected hypovolemia (2.3 mL/kg as a fluid bolus). To recalculate a patient’s Hct, platelet concentration, and plasma fibrinogen concentration during intraoperative hemodilution, the following variables were used: body weight, total amount of colloid infused, surgical blood loss, and initial values for Hct, platelet concentration, and plasma fibrinogen concentration.

To determine the relevance of our results in routine clinical practice, we sought to assemble data from an everyday patient population and relate them to our findings. Therefore, we collected preoperative data on Hct, platelet concentration, and plasma fibrinogen concentration from 204 consecutive patients (ASA status I–III) undergoing elective surgery (general, trauma, or gynecological) in a community hospital. We next identified those patients who presented with certain preoperative hemostatic profiles (see Results) and expressed their number as a percentage of all patients. Because these data were part of routine preoperative assessment, were obtained from patients’ records during the postanesthesia care unit stay, and were anonymized thereafter, neither approval by an ethics committee nor informed consent was required.

All computations were based on the following assumptions to mimic routine clinical practice as closely as possible:

1. A “model” patient with a 70-kg body weight and an estimated blood volume (EBV) of 70 mL/kg body weight.
2. Isovolemic volume substitution/replacement with cell-free, colloidal solution (volume effect 1.0).
3. Colloid without direct effect on hemostasis.
4. A 200-mL colloidal preload to compensate for preoperative hypovolemia.

The combination of ANH with intraoperative application of ABS (modified according to Refs. 13 and 18) was performed as follows:

1. ANH consisted of a preoperative exchange of 2000 mL of blood versus colloid.
2. Intraoperative isovolemic replacement of surgical blood loss with colloid until Hct reached 24% and, thereafter, replacement with ABS to maintain an oxygen-carrying capacity equivalent to an Hct of 24%.
3. Retransfusion of ANH-blood if the actual Hct was <16%.

All calculations were performed with standard computer software (MS-Excel®; Microsoft, Redmond, WA).

**Results**

Table 1 shows the validation of the mathematical foundation underlying our model. We individually recalculated Hct, platelet concentration, and plasma fibrinogen concentration from 20 patients during intraoperative hemodilution and compared them with those actually measured. As emphasized by very small margins of error, our model can precisely calculate Hct, platelet concentration, and plasma fibrinogen concentration during intraoperative hemodilution. Figure 1 shows MABLs depending on minimal acceptable values for Hct, platelet concentration, and plasma fibrinogen concentration.

Compared with Hct, critical platelet concentrations will trigger the need for adequate blood products first only if the following conditions are met: PltINIT ≤ 150,000/µL, PltMIN = 100,000/µL, HctINIT ≥ 36%, and HctMIN = 24%. Three (1.5%) of 204 patients demonstrated this constellation of conditions before surgery.

In the following clinical situations, critical fibrinogen concentrations will determine the administration of blood products: (a) FibINIT ≤ 270 mg/dL, FibMIN = 100 mg/dL, and HctINIT ≥ 40% (HctMIN = 18%); (b) FibINIT ≤ 250 mg/dL, FibMIN = 100 mg/dL, and HctINIT ≥ 40% (HctMIN = 21%); and (c) FibINIT ≤ 210 mg/dL, FibMIN = 100 mg/dL, and HctINIT ≥ 40% (HctMIN = 24%). Before surgery, 42 patients (20.6%) presented with a Type a profile, 8 (3.9%) with a Type b, and 1 (0.5%) with a Type c.

Compared with regular isovolemic substitution of surgical blood losses, integration of ANH plus ABS into the management of surgical blood losses greatly reduces MABL regarding the minimal acceptable platelet or plasma fibrinogen concentration (Fig. 2). PltMIN will be reached before the minimal acceptable Hct if PltINIT is ≤240,000/µL and PltMIN = 100,000/µL (HctINIT >40%), as illustrated in Figure 3 (left panel) or if PltINIT is ≤210,000/µL and PltMIN = 100,000/µL (HctMIN >35%). Before surgery, 40 (19.6%) of 204 patients and 23 (11.3%) of 204 patients, respectively, demonstrated these profiles.

Before passing the minimal acceptable Hct, FibPLASMA-MIN will be reached first if FibINIT is ≤270 mg/dL (FibMIN = 100 mg/dL) and HctINIT is ≥35% or if FibINIT is ≤335 mg/dL (FibMIN = 100 mg/dL) and HctINIT is ≥40% (Fig. 3, right panel). Twenty-nine (14.2%) and 75 (36.8%) of 204 patients, respectively, presented with these findings.

**Discussion**

Despite numerous *in vitro* and *in vivo* studies on quantitative changes of hemostasis during hemodilution, sound clinical data addressing quantitative changes of hemostasis during (extreme) hemodilution are rare and sometimes even outdated. In situations in which quantitative changes of hemostasis during hemodilution become highly relevant (i.e., extreme hemodilution due to massive blood loss), clinicians are left with only general
recommendations. With increasing popularity of ABS, the situation becomes even more complex, especially when combined with ANH. ABS have been designed to reduce blood transfusions. However, they solely provide oxygen-carrying capacity, but no hemostatic activity. Thus, with continuing blood loss, ABS stabilize oxygen-carrying capacity but further dilute hemostatic factors.

To guide future clinical trials by providing reliable estimates and identifying potentially critical constellations, we developed, validated, and executed a realistic

Table 1. Model Validation by Recalculation of Prospectively Collected Clinical Data

<table>
<thead>
<tr>
<th>Variable</th>
<th>Data before surgery</th>
<th>Clinical data at the end of surgery</th>
<th>Model data at the end of surgery</th>
<th>Margins of error (%) model versus clinical data</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hematocrit (%)</td>
<td>38.2 (30.3–45.2)</td>
<td>32.3 (24.2–39)</td>
<td>30.2 (23.8–34.8)</td>
<td>±4.5 (0.2–12.9)</td>
</tr>
<tr>
<td>Platelets ($\times 10^{12}/\mu$L)</td>
<td>222 (132–366)</td>
<td>189 (109–321)</td>
<td>193 (114–322)</td>
<td>±5.1 (0.6–14.7)</td>
</tr>
<tr>
<td>Plasma fibrinogen (mg/dL)</td>
<td>327 (223–408)</td>
<td>241 (175–325)</td>
<td>230 (173–302)</td>
<td>±7.0 (1.7–18.1)</td>
</tr>
</tbody>
</table>

Body weight: 76 kg (60–91 kg). Intraoperative blood loss: 300 mL (150–600 mL).
All data are given as median (range).

Figure 1. Maximum allowable blood loss (MABL) during isovolemic hemodilution, depending on hematocrit (Hct)$_{MIN}$, platelet (Plt)$_{MIN}$, and fibrinogen (Fib)$_{PLASMA-MIN}$. MABL are given for various initial concentrations each. Perpendicular lines indicate the minimal acceptable values for each variable below which transfusion of blood products becomes necessary.

Figure 2. Decline of platelets (Plt) (left), plasma fibrinogen concentration (right), and hematocrit (Hct) (right $y$ axis in each panel) when acute normovolemic hemodilution (ANH) is combined with artificial blood substitutes (ABS) to manage surgical blood losses. After ANH (2000 mL), colloids are used first to compensate for surgical blood loss. When the hematocrit decreases to less than 24%, ABS (downward-scattered area) are infused to maintain oxygen-carrying capacity (equivalent to an Hct of 24%), whereas platelet and plasma fibrinogen concentration continue to decrease; the actual Hct also decreases. When the actual Hct reaches 16%, retransfusion of ANH-blood plus colloid (upward-scattered area) stabilizes all variables at their current level. Dotted lines represent critical platelet (left panel) and plasma fibrinogen concentrations (right panel). The double-headed arrow indicates the blood loss at which fibrinogen (Fib)$_{PLASMA-MIN}$ limits hemodilution before Hct$_{MIN}$.
mathematical model to quantitatively describe hemostasis during hemodilution. Our model is based on the exponential decline of corpuscular particles during isovolemic hemodilution, as first described by Bourke and Smith (14) and subsequently used by our group and others (for further reference, see Ref. 15). Nonetheless, a mathematical model can be only as good as it can be validated by clinical data. Consequently, we have recalculated prospectively collected individual clinical data from 20 patients undergoing intraoperative hemodilution with hydroxyethyl starch for small to moderate blood loss. We found a very close agreement between clinically measured and mathematically calculated data, as emphasized by small margins of error. Previously, we obtained similar validation results for the underlying mathematical assumptions (15,19) when recalculating pooled hemodilution data from independent studies with larger hemodilution volumes (<1800 mL). As for fibrinogen, our mathematical approach is also well supported by a previous clinical study that analyzed hemostasis during extreme hemodilution, i.e., blood loss exceeding estimated blood volume (EBV) up to twofold (20). Despite an exponential decline during isovolemic hemodilution, clinical platelet concentrations in this study exhibited moderate, less predictable variations from the calculated value when blood losses exceeded 150% of EBV. However, for blood losses smaller than 150% of EBV, our calculations also appear to occur in close range (±10%) to those clinically measured. As seen in our model and in clinical studies (1,20), blood losses going beyond 150% of the EBV are possible only with prior decreases of Hct and Fib to less than accepted critical levels, thereby primarily limiting hemodilution. The inaccuracies above are therefore of only minor relevance to our study, because we aimed to identify variables and their concentrations that limit hemodilution foremost. On the basis of results from both our own studies and independent studies, we therefore consider our mathematical approach sufficiently well validated to execute the model and obtain compelling information.

This is the first clinically validated model that systematically evaluates quantitative changes of hemostasis during hemodilution, including the application of ANH together with ABS. A few studies have developed formulas to fit their clinical data (1,7) or to generally describe hemostasis during isovolemic hemodilution (10). However, no previous study has addressed or found distinct hemostatic profiles that could result in critical hemostatic levels as primary transfusion triggers.

Dilutional thrombocytopenia and dilutional coagulopathy are the universal hemostatic defects associated with extreme hemodilution, as seen during massive blood loss (21). Hypofibrinogenemia is also the first factor deficiency to occur during massive transfusion (10). We therefore selected platelets and plasma fibrinogen as representative key variables for our calculations. Both variables are also part of routine clinical tests. Besides its utmost importance for stabilization of the initial platelet-rich plug during secondary hemostasis, plasma fibrinogen was also chosen because of its prolonged half-life time and its almost exclusive restriction to the intravascular space (21). Plain dilution explains at least 90% of fibrinogen change during hemodilution (7). These characteristics make plasma fibrinogen an ideal coagulation variable for model calculations. Other factors, such as factor VII or IX, demonstrate short half-life times or limited restriction to the intravascular space (21). Thus, they are nonetheless of great importance for maintenance of adequate hemostasis. Our model shows that platelets will be the primary transfusion trigger and thus limit isovolemic hemodilution only if a rather high PltMIN of 100,000/µL is required. High PltMIN is necessary only under special, less frequent circumstances, e.g., neurosurgery (22). If PltMIN is set at 50,000/µL, a well accepted critical level in most surgical cases (10,22), clinically relevant Hct
levels will always be reached first; i.e., HctMIN will be the actual transfusion trigger. Moreover, the above-mentioned preoperative profiles, which could lead to critical PltMIN before critical HctMIN, do not seem to occur as often, as indicated by a very small incidence in our common patient population (1.5%).

Inclusion of ANH plus ABS into the management of intraoperative blood losses leads to the following situations. First, transfusion of platelets still does not seem to be necessary before that of RBC as long as PltMIN is set at 50,000/μL. Second, when high PltMIN (100,000/μL) is demanded, one has to give platelets only under certain conditions. These preoperative conditions appear to be more frequent, as suggested by their incidence in our patient population (19.6% and 11.3%, respectively). Here, inclusion of ABS into ANH management would defeat its purpose of enabling larger intraoperative blood losses. One would have to use (homogeneous) platelet units or ANH blood only to compensate for platelet losses.

During simple, intraoperative isovolemic hemodilution, critical fibrinogen concentrations could trigger the application of blood products first. As opposed to platelets, the effect and clinical relevance of fibrinogen-limited hemodilution largely depends on selected HctMIN levels. The lower the HctMIN, the more likely that critical plasma fibrinogen concentrations will limit hemodilution and will thus affect more patients. In our patient population, a substantial percentage of patients (±20.6%) presented with a preoperative profile that could potentially limit hemodilution because of critical plasma fibrinogen concentrations. If ANH is combined with ABS to cope with large anticipated surgical blood losses, even more patients in our population will potentially be affected by fibrinogen-expected surgical blood losses, even more patients in our patient population (19.6% and 11.3%, respectively). Here, inclusion of ABS into ANH management would defeat its purpose of enabling larger intraoperative blood losses. One would have to use (homogeneous) platelet units or ANH blood only to compensate for platelet losses.

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Considering this large number of patients affected, one has to concentrate on the differentiated use of ANH together with ABS in future clinical trials. Inclusion of such patients without special attention to their hemostasis profile might jeopardize the power and success of these studies.

Despite convincing validation, our model still has its limitations and can by no means replace well-designed, randomized, prospective clinical trials. Our model can only describe quantitative changes of hemostasis during hemodilution. The effect of qualitative changes during hemodilution, such as interactions between colloids/crystalloid and hemostasis, cannot be analyzed. Also, even though our patient population originated from a general community hospital and included more than 200 consecutive patients, it cannot be considered truly representative. Our patient population was accumulated over only a few weeks and did not include ASA status IV and V patients or patients undergoing emergency surgery. Moreover, because of special clinical circumstances not considered in our data analysis, some patients might not have tolerated our (theoretical) minimal acceptable Hct levels. The actual percentage of patients who might have presented with hemostasis-limited hemodilution could therefore be smaller.

In conclusion, we developed, clinically validated, and executed a realistic mathematical model to quantitatively describe major hemostatic changes during intraoperative isovolemic hemodilution. We demonstrated that, under certain circumstances, platelet and plasma fibrinogen concentrations, in particular, could decrease below critical threshold values and thus limit hemodilution even before Hct does. The combination of ANH with current ABS strongly aggravates the intensity and relevance of this phenomenon, i.e., platelets and fibrinogen become more likely to limit hemodilution under these conditions.

Thus, we conclude that during extreme hemodilution, one not only should focus on critical Hct levels as potential transfusion triggers, but should also pay close attention to each patient’s individual hemostatic profile. The application of simple substitution guidelines to replace hemostatic factors could be deleterious in certain situations. Substitution guidelines, however, could be improved by taking into account different types of hemostatic profiles. Future clinical trials combining ANH with current types of ABS should likewise pay more attention to individual preoperative hemostasis to avoid hemostasis-limited hemodilution, because it would clearly defeat the idea of this strategy.

Appendix 1: List of Abbreviations Used

- Hct = hematocrit
- HctBL = hematocrit after defined blood loss
- HctINIT = initial hematocrit
- PltBL = platelet concentration after defined blood loss
- PltINIT = initial platelet concentration
- FibPLASMA = plasma fibrinogen concentration
- FibBLOOD = blood fibrinogen concentration
- FibBLOOD-INIT = initial blood fibrinogen concentration
- FibBLOOD-BL = blood fibrinogen concentration after defined blood loss
- BL = defined blood loss
- EBV = estimated blood volume
Hct_MIN = minimal acceptable hematocrit (transfusion trigger)
Plt_MIN = minimal acceptable platelet concentration (transfusion trigger)
Fib_MIN = minimal acceptable plasma fibrinogen concentration (transfusion trigger)
Fib_BLOOD-MIN = minimal acceptable blood fibrinogen concentration
MABL_HCT = maximal allowable blood loss depending on Hct_MIN
MABL_PLT = maximal allowable blood loss depending on Plt_MIN
MABL_FIB_BLOOD = maximal allowable blood loss depending on Fib_BLOOD-MIN

References