Viscoelastic Coagulation Testing: Use and Current Limitations in Perioperative Decision-making

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Rapid diagnosis and therapy of coagulopathy plays a key role in the care of the severely bleeding patient in major trauma, postpartum hemorrhage, or major surgery. Perioperative diagnostic and treatment algorithms are mostly guided by viscoelastic point-of-care coagulation assays. There is convincing evidence that implementing transfusion algorithms based on the results of viscoelastic point-of-care coagulation tests can reduce transfusions and lead to improved patient outcomes (table 1).1–4 In the perioperative period, two assays are most often used: the kaolin thromboelastography (TEG) and the functional fibrinogen TEG as part of TEG-based viscoelastic monitoring and the tissue factor–activated rotational thromboelastometry (ROTEM, also called “EXTEM”) and fibrinogen ROTEM (also called “FibTEM”) assays as part of ROTEM-based viscoelastic monitoring. These assays are designed to help answer basic questions regarding treatment of perioperative coagulopathy, such as when should the clinician transfuse platelets, administer fibrinogen concentrate, or give plasmonic coagulation factors by transfusing fresh frozen plasma (FFP) or prothrombin complex concentrates (PCCs)? And when is the administration of an antifibrinolytic agent justified? However, it is important to realize that the standard TEG/ROTEM assays are not sensitive and specific to adequately detect platelet inhibition,5 effects of direct oral anticoagulants, or inherited bleeding disorders (e.g., hemophilia, von Willebrand disease).6 Thus, the diagnosis of these rather specific conditions is better made preoperatively as part of a routine diagnostic workup.7

In this Clinical Focus Review, we aim to reevaluate the current literature published on viscoelastic point-of-care tests and their impact on clinical decision-making. In particular, we were interested in which fundamental questions affecting routine patient care in the perioperative period could be answered by TEG and ROTEM assays and which could not. Because this is not a systematic review, we did not consider specific methodologic criteria for study selection or analysis. Nevertheless, we agreed on the search criteria and the selection of relevant publications. In addition to randomized controlled trials, systematic reviews, meta-analyses, society recommendations, and guidelines were assessed and implemented if deemed relevant. The interpretations are intended to emphasize the clinical aspects of standard viscoelastic point-of-care assays and to highlight specific areas that warrant further development.

Viscoelastic Testing for Administration of Platelets and Fibrinogen Concentrate

A basic assumption of TEG and ROTEM is that the amplitude of the viscoelastic signal is a composite of the interaction of platelets and fibrinogen. In the functional fibrinogen TEG, abciximab, a glycoprotein IIb/IIIa receptor inhibitor, is used, whereas the fibrinogen ROTEM assay uses cytochalasin D, an inhibitor of actin polymerization in platelets.5,9 The addition of the antiplatelet agent reduces the platelet-mediated clot activation signal to selectively evaluate the fibrinogen component of clot strength. In turn, platelet contribution is calculated by the difference between the viscoelastic amplitude of the tissue factor–activated ROTEM and fibrinogen ROTEM.10

However, two major considerations call into question the validity of the results obtained with the above-mentioned assays. First, there are convincing data showing that there is residual platelet noise in the fibrinogen assays caused by incomplete inhibition of platelet aggregation.4,9 This is more pronounced when a platelet glycoprotein IIb/IIIa receptor inhibitor is used and less pronounced when cytochalasin D is used. The combination of the agents leads to complete inhibition of platelet aggregation and thereby prevents any residual “platelet noise.”10 Second, there is emerging evidence that it is not the amplitude of the viscoelastic signal but the clot elasticity (100 × viscoelastic amplitude [mm]/100 − viscoelastic amplitude [mm]) that more accurately reflects the platelet contribution to clot. Early theoretical considerations in this regard11 have recently been confirmed by studies of blood samples from patients who received platelet receptor P2Y12 inhibitor therapy before cardiac surgery.12 The same research group demonstrated that the correlation of the viscoelastic amplitude with platelet count and adenosine diphosphate–induced platelet aggregation was weak.12 The difference in platelet-specific elasticity between the tissue factor–activated ROTEM and fibrinogen ROTEM,
Table 1. Viscoelastic Testing and Its Association with Transfusion Requirements in Different Settings

<table>
<thead>
<tr>
<th>Reference</th>
<th>Study Type (No. of Patients)</th>
<th>Investigated Devices (vs. Control Group)</th>
<th>Clinical Setting (Number of Studies)</th>
<th>Main Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Whiting et al.¹</td>
<td>Systematic review and cost-effectiveness analysis (cardiac: 1,089; trauma: 4,217; postpartum hemorrhage: 245)</td>
<td>TEG, ROTEM, or Sonoclot (Sienco Inc., USA) (vs. no test or standard laboratory tests)</td>
<td>Cardiac setting: cardiac surgery (11 randomized controlled trials and 3 prediction studies)</td>
<td>TEG/ROTEM-guided management reduced platelet transfusion (relative risk, 0.95; 95% CI, 0.80 to 1.10; six studies), platelet transfusion (relative risk, 0.68; 95% CI, 0.35 to 0.95; five studies)</td>
</tr>
<tr>
<td>Wikkelsø et al.²</td>
<td>Systematic review, meta-analysis and trial sequential analysis (1,185)</td>
<td>TEG or ROTEM (vs. clinical judgment/standard laboratory tests)</td>
<td>Bleeding patients: cardiac surgery (13 randomized controlled trials) liver transplantation (1 randomized controlled trial); burn wound excision (1 randomized controlled trial)</td>
<td>TEG/ROTEM-guided management reduced platelet transfusion [relative risk (95% CI) 0.73 (0.60 to 0.88); I2 = 0%, 10 studies, 382 participants], FFP transfusion [relative risk (95% CI) 0.52 (0.30 to 0.93); I2 = 0%, 10 trials, 382 participants] and overall mortality [3.9% vs. 7.4%, relative risk (95% CI) 0.52 (0.28 to 0.95); I2 = 8%, 8 trials, 887 participants].</td>
</tr>
<tr>
<td>Dias et al.³</td>
<td>Systematic review and meta-analysis (882)</td>
<td>TEG 5000 and 65 (vs. no TEG)</td>
<td>Perioperative setting: cardiac surgery (7 randomized controlled trials) liver surgery (2 randomized controlled trials)</td>
<td>TEG-guided management reduced platelet transfusion (P = 0.049), FFP transfusion (P &lt; 0.001), erythrocyte transfusion (P = 0.12), operating room length of stay (P = 0.005), intensive care unit length of stay (P = 0.04), and bleeding rate (P = 0.002). Mortality remained comparable between the treatment and the control group.</td>
</tr>
<tr>
<td>Meco et al.⁴</td>
<td>Systematic review, meta-analysis, meta-regression, and trial sequential analysis (1,035)</td>
<td>TEG or ROTEM (vs. clinical judgment)</td>
<td>Perioperative setting: cardiac surgery (7 randomized controlled trials)</td>
<td>TEG/ROTEM-guided management reduced FFP transfusion (odds ratio, 0.52; 95% CI, 0.28 to 0.95; I2 = 0%, 8 trials, 717 participants), postoperative bleeding (odds ratio, 0.51; 95% CI, 0.28 to 0.94; I2 = 0%), and mortality (odds ratio, 0.57; 95% CI, 0.18 to 1.74; P = 0.28).</td>
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</table>

Conclusion: TEG/ROTEM-guided hemostatic therapy improves blood product management and patient outcome.

Conclusion: TEG/ROTEM-guided hemostatic therapy is effective in reducing allogenic blood product exposure and postoperative bleeding after surgery and improves patient outcome.

**Viscoelastic Tests: Use and Limitations**

however, provided a strong correlation with platelet count and even a moderate correlation with an adenosine diphosphate–induced platelet aggregation assay.

**Trigger for Platelet Transfusions in Clinical Recommendations**

The United States–based Society of Cardiovascular Anesthesiologists has published a practical approach using both TEG and ROTEM for targeted blood component therapy. Critical thresholds for platelet transfusion were defined for the viscoelastic amplitude after 10 min in ROTEM and the maximum amplitude in TEG (Table 2). The trigger for platelet transfusion, however, was not given as a defined value of these parameters but as a more open synoptic approach also considering a minimum target value for fibrinogen in the functional fibrinogen assays.
Trigger for Fibrinogen Concentrate or Cryoprecipitate in Clinical Recommendations

Recommendations for the replacement of concentrated fibrinogen based on viscoelastic test results are available from the Pan-European multidisciplinary Task Force for Advanced Bleeding Care in Trauma (hereafter referred to as the European trauma guidelines), the Society of Cardiovascular Anesthesiologists, and the Haemostasis and Transfusion Subcommittee of the European Association of Cardiothoracic Anaesthesiology and Intensive Care (table 2). In the European trauma guidelines, target values for fibrinogen plasma levels were only defined for the standard laboratory method. At the same time, viscoelastic tests were considered equivalent. The defined transfusion threshold for fibrinogen administration (less than 1.5 g/l plasma fibrinogen level) in the actively bleeding patient correlated well with a fibrinogen ROTEM maximal clot firmness of less than 10 mm in healthy people.

In the recommendation from the Society of Cardiovascular Anesthesiologists, similarly, a synoptic approach was used. In the European recommendations, trigger values for fibrinogen supplementation with fibrinogen concentrate, as well as fibrinogen plasma thresholds prohibiting administration of exogenous fibrinogen, have been defined. A similar approach was followed for postpartum hemorrhage by the British Society for Haematology (table 2).
Viscoelastic Testing for Repletion of Plasma Coagulation Factors

The clotting time in ROTEM and the reaction time (r) in kaolin TEG reflect the state of the plasmatic coagulation system by measuring the amount of time until the formation of a first fibrin network. The kaolin TEG activates whole blood via the intrinsic contact system, and in this respect is similar to the activated partial thromboplastin time, which is sensitive to deficiencies in the coagulation factors II, V, VIII, IX, and X and fibrinogen, but not in factor VII. In the tissue factor–activated ROTEM assay, tissue factor and the heparin reversal agent hexadimethrine bromide are added, which binds and neutralizes heparin up to a plasma concentration of 4 IU/ml. The tissue factor–activated ROTEM assay therefore shows similarities to the prothrombin time and the international normalized ratio (INR) and is sensitive to deficiencies in the extrinsic and common pathways represented by factors II, V, VII, and X and fibrinogen.

In clinical reality, the decision to transfuse plasma coagulation factors is based on clot time or reaction time. Current four-factor PCCs contain the vitamin K–dependent coagulation factors II, VII, IX, and X. The concentrates were originally developed to reverse the effect of warfarin and other vitamin K antagonists. However, in trauma and cardiac surgery, these concentrates are increasingly used to treat major bleeding and coagulopathy as a “hemostatic resuscitation.” In the tissue factor–activated ROTEM assay, tissue factor and the heparin reversal agent hexadimethrine bromide are added, which binds and neutralizes heparin up to a plasma concentration of 4 IU/ml. The tissue factor–activated ROTEM assay therefore shows similarities to the prothrombin time and the international normalized ratio (INR) and is sensitive to deficiencies in the extrinsic and common pathways represented by factors II, V, VII, and X and fibrinogen.

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In the bleeding patient with preoperative warfarin use, the decision to administer PCC is usually based on the preoperative INR. However, in the severely bleeding patient not taking warfarin, the decision to transfuse FFP and/or PCC should ideally be based on laboratory and viscoelastic test values (see below), which than should also be used to monitoring their effects after administration. Unfortunately, the relevant data supporting PCC use are sparse despite their increasing administration. However, there is evidence suggesting that clotting time in tissue factor–activated ROTEM may be an appropriate tool to use. In ex-vivo studies using blood samples from patients on vitamin K antagonist therapy, the correlation between the clotting time in tissue factor–activated ROTEM and the reaction time in kaolin TEG with INR was investigated.

As a result, clotting time in tissue factor–activated ROTEM correlated with INR, whereas reaction time in kaolin TEG was insensitive. Clotting time in tissue factor–activated ROTEM (reference range, 38 to 79 s) for an INR value between 1.2 and 2.0 clustered around 80 s, for an INR value between 2 and 3 around 100 s, and for an INR of more than 3 around 140 s.

The fact that clotting time in tissue factor–activated ROTEM is sensitive to the loss of the vitamin K–dependent coagulation factors raises the question of whether targeted replacement of these factors by transfusion of PCC can also be monitored with tissue factor–activated ROTEM assay. In a retrospective study of trauma patients in whom coagulation factor concentrates were transfused based on ROTEM results, administration of (median) 1,200 IU PCC, which approximates 15 IU/kg PCC, normalized clotting time in tissue factor–activated ROTEM from (median) 101 to 77 s. In contrast, clotting time in a contact phase–activated ROTEM (comparable to kaolin TEG) remained unchanged.

Trigger for FFP and/or PCC in Clinical Recommendations

In the Society of Cardiac Anesthesiology recommendations, transfusion of 10 to 15 ml/kg of FFP or a low dose of PCC (not defined) is recommended when clotting time in tissue factor–activated ROTEM or the reaction time in heparinase TEG is significantly prolonged (table 2). Of note, the European recommendations for hemostatic resuscitation in trauma recommend a dose of 25 IU/kg of a PCC, whereas in cardiac surgery patients, an initial dose of 12.5 IU/kg (similar to that suggested by the U.S. recommendations) should be considered because of the inherent risk of thromboembolism. In the European trauma guidelines, the authors point out the possible influence of hypofibrinogenemia on clotting time in tissue factor–activated ROTEM. Therefore, PCC should be given only when fibrinogen levels are less than 1.5 g/l (corresponding to a fibrinogen ROTEM maximal clot firmness of less than 10 mm), and clotting time in tissue factor–activated ROTEM is prolonged or remains prolonged after replacement of fibrinogen.

Viscoelastic Testing of Fibrinolytic State

In the viscoelastic tests, clot lysis is determined after 30 or 60 min by calculating the decrease in maximal clot firmness on ROTEM or the maximal amplitude on TEG. The evaluation of the fibrinolytic system is complex because local and systemic fibrinolysis occurs in parallel and involves blood flow and cells, especially platelets. In trauma patients, there is a distinction between hyperfibrinolysis, hypofibrinolysis, and “fibrinolytic shutdown,” a severe condition in which even physiologic fibrinolysis is halted, and disseminated microembolism can occur, damaging multiple organs. This has implications for the patient’s prognosis and presumably for therapy. Study results are, however, conflicting again. In a large prospective cohort study in trauma patients, fibrinolysis was defined as a reduction of tissue factor–activated ROTEM maximal clot firmness of more than 15% measured 60 min after the onset of clot formation. Additionally, plasmin–antiplasmin complexes were measured. The study showed that the tissue factor–activated ROTEM assay could detect hyperfibrinolysis in only 5% of patients when plasmin–antiplasmin complexes were elevated to 30 times normal. However,
in 57% of patients with hyperfibrinolysis detected by plasmin–antiplasmin complexes more than twice the normal level, 28-day mortality was significantly increased compared to patients without evidence of hyperfibrinolysis, as indicated by elevated plasmin–antiplasmin complexes (12% vs. 1%, P < 0.001). These results were recently confirmed in another prospective multicenter observational cohort study in 914 trauma patients. In a large prospective two-center study involving 2,540 severely injured patients, the diagnosis of fibrinolysis was based on the rapid TEG assay, which contains both kaolin and tissue factor. Hyperfibrinolysis was defined as a decrease in maximum amplitude by more than 3% within 30 min, physiologic fibrinolysis as a decrease between 0.8% and 3%, and fibrinolytic shutdown as a decrease in maximal amplitude of less than 0.8%. Patients with hyperfibrinolysis had the worst outcomes, with a mortality rate of 34%, followed by those with fibrinolytic shutdown (22%) and patients with physiologic fibrinolysis (14%; P < 0.0001).

A recent analysis of 549 patients in the randomized controlled Pragmatic Optimized Ration Platelet and Plasma Trial provided evidence that a low lysis value of less than 0.9% at 30 min in the TEG may not reflect shutdown of enzymatic fibrinolysis with hypercoagulability but rather a coagulopathic state of moderate fibrinolysis with fibrinogen consumption and platelet dysfunction that is associated with poor outcomes.

Viewing the available data, it is currently unclear whether the discussed viscoelastic assays can discriminate between different degrees of fibrinolysis or whether test results obtained for clot dissolution are rather an indicator of a general coagulopathy. This may be important for both prognosis and therapeutic decisions and leads to the question of whether the potential of these assays has been fully realized. Timing appears to play a crucial role in fibrinolysis and the narrow therapeutic window. In-vitro and ex-vivo investigations using the tissue factor–activated ROTEM provided convincing evidence that the timing of the onset of lysis, defined as a reduction of clot of more than 15% after the onset of coagulation, provides a faster diagnosis of hyperfibrinolysis than lysis at 30 or 60 min or maximal lysis. Using samples from patients after cardiac arrest, hyperfibrinolysis was diagnosed with the lysis onset time within 7 and 22 min.

The same study showed that functional fibrinogen TEG and fibrinogen ROTEM resulted in the shortest clot lysis time (time from maximal amplitude to 2 mm reduction in viscoelastic amplitude) for the TEG or lysis onset time for the ROTEM system, compared with the other assays. Diagnosis was 1.30 to 2.42 times faster when compared to the other assays. In patients undergoing liver transplantation, the fibrinogen ROTEM assay was shown to have higher sensitivity for detecting hyperfibrinolysis compared to tissue factor–activated ROTEM and kaolin TEG (94% vs. 46% and 24%, respectively; P < 0.001).

Triggers for Antifibrinolytic Therapy in Clinical Recommendations

In the Society of Cardiac Anesthesiology recommendations, antifibrinolytic therapy is generally suggested for patients undergoing cardiac surgery. However, in patients without prophylactic antifibrinolytic therapy, hyperfibrinolysis should be addressed (table 2). The European trauma guidelines recommend administration of a 1-g bolus of tranexamic acid in trauma patients as soon as possible—preferably before reaching the emergency room—followed by an infusion of 1 g over 8 h, instead of waiting for results of viscoelastic tests. According to the British Society of Haematology, antifibrinolytic therapy in trauma and obstetric patients should not be withheld, based on the results of ROTEM or TEG (table 2).

Future Considerations for Viscoelastic Testing in Bleeding Management

The causes of bleeding, particularly in trauma and cardiac surgery, are mostly multifactorial. Thus, for targeted therapy of diffuse bleeding, reliable information about the major components of the coagulation system is crucial. ROTEM and TEG are widely implemented in modern treatment algorithms. However, we believe that further technical considerations in the key assays are necessary. Furthermore, large multicenter studies are needed to validate trigger values for targeted therapeutic interventions (table 3). In view of this, it is also important to investigate the negative predictive value of these tests to rule out a substantial coagulopathy in the bleeding patient. A clear statement in the case of trauma has been published by the British Society of Haematology (table 2). However, a recent meta-analysis highlighted a clear lack of evidence for cardiac surgery.

Future considerations should include minimizing platelet contribution in the fibrinogen assays, because minimal residual platelet noise may have consequences for the decision to transfuse or not transfuse fibrinogen concentrate. All available data show that this can be achieved by dual platelet inhibition in the fibrinogen assays. Further, the question of whether clot amplitude or clot elasticity better signals the contribution of platelets to the clot needs to be answered, because it is perceived that this value is a determinant of platelet function. To date, theoretical considerations are supported by only one study in cardiac surgery and need to be confirmed by additional studies in other clinical settings. This may lead to a fundamental paradigm shift, with clot elasticity used instead of clot amplitude to guide platelet transfusion. In addition, the clotting times of viscoelastic tests should distinguish better between moderate impairment of the clotting system, which can be treated by FFP transfusion alone, and severe impairment, in which hemostatic resuscitation with concentrated clotting factors is indicated instead. To optimize dose and presumably reduce thrombotic complications, these values should serve as a control panel reflecting the effect of these potent clotting factor concentrates. Tissue factor–activated assays that
are sensitive to coagulation factor VII appear to have potential in this regard but need further investigation.

For trauma management, rapid assessment of the fibrinolytic system status is an important issue that needs further investigation. The currently used assays that are based on clot lysis often do not correlate with biomarkers of fibrinolysis, including D-dimers, and the parameters and thresholds for hyperfibrinolysis are arbitrarily defined. This might explain the conflicting results in trauma studies regarding the usefulness of viscoelastic tests. Therefore, a reevaluation of the thresholds should be considered. There is some evidence that viscoelastic fibrinogen assays, especially those with activated tissue factor, are faster and more sensitive than assays with preserved platelet activity. In addition, clot dissolution time appears to be a parameter that can provide information more rapidly and presumably increase sensitivity for the diagnosis of clinically relevant hyperfibrinolysis.

New devices and new assays have recently been developed for viscoelastic point-of-care testing of the effects of antplatelet agents and direct oral anticoagulants and assessment of the fibrinolytic system. The ClotPro (Haemonetics, USA) system is almost comparable to ROTEM, but it already applies dual platelet inhibition in its functional fibrinogen assay. The Quantra hemostasis analyzer (Hemosonics, USA) is based on the technique of sonorheometry. First clinical studies provided convincing evidence that the results obtained by this device strongly correlate with results achieved with thromboelastometry and parameters effectively represent results from multiple standard laboratory tests. The setup of activators and tests used is similar to the other discussed viscoelastic test systems, but Quantra already includes an automated calculation of the platelet contribution to the clot stiffness. Even in light of the development of new diagnostic instruments and a broader line of assays targeting rather specific acquired disturbances of the coagulation system, the basic assays of TEG and ROTEM, including the functional fibrinogen assays, remain the foundation for the vast majority of clinical decisions. In this regard, further improvement and clinical validation of these broadly used basic assays are needed.

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**Table 3. Technical Limitations and Suggested Improvements in Basic TEG/ROTEM Assays**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Affected Part of the Coagulation System</th>
<th>Assay Specification</th>
<th>Technical Limitations</th>
<th>Data Limitation</th>
<th>Suggested Improvements</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clotting time/reaction time</td>
<td>Procoagulant factors</td>
<td>Intrinsic activation</td>
<td>Insensitivity for factor VII</td>
<td></td>
<td>Studies to provide correlation of clotting time with INR</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Extrinsic activation + hexadimethrine bromide for heparin insensitivity &lt; 4 IU/ml</td>
<td>Longer time until result is achieved when compared to tissue-factor activated assay</td>
<td></td>
<td>Development of trigger values for targeted PCC therapy</td>
</tr>
<tr>
<td>Maximum clot firmness/maximum amplitude</td>
<td>Platelets</td>
<td>Intrinsic activation/extrinsic activation</td>
<td>Platelet effect must be calculated: maximum clot firmness/maximum amplitude ± maximum clot firmness</td>
<td>Calculation of platelet contribution to the clot occurs automatically</td>
<td>Studies to provide trigger values for platelet transfusion</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Clot elasticity needs clinical validation</td>
</tr>
<tr>
<td>Fibrinogen</td>
<td></td>
<td>Intrinsic activation/extrinsic activation + cytochalasin D or GPIIb/IIIa inhibitor</td>
<td>Potential for residual platelet noise when using single platelet inhibitor</td>
<td></td>
<td>Dual platelet inhibition</td>
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<tr>
<td>Lysis</td>
<td>Thrombolysis</td>
<td></td>
<td>Critical values unclear:</td>
<td></td>
<td>Recommended thresholds to be confirmed in randomized control trials</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>• Clot lysis at 30 min, 3% or 15%</td>
<td></td>
<td>Parameters need to be defined</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>• Lysis onset time</td>
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</table>

INR, international normalized ratio; PCC, prothrombin complex concentrate; ROTEM, rotational thromboelastometry; TEG, thromboelastography.
Competing Interests

Dr. Levy serves on advisory committees for Instrumentation Laboratory, Werfen Company (Bedford, Massachusetts), Merck (Darmstadt, Germany), and Octapharma (Lachen, Switzerland). The other authors declare no competing interests.

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