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REVIEW

Perioperative coagulopathy monitoring

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Abstract Coagulopathy is common in orthopedic surgery patients either due to acquired factors, such as surgery, trauma, medications, or hemorrhage. Perioperative monitoring of blood coagulation is critical to diagnose the causes of hemorrhage, guide hemostatic therapies, predict the risk of bleeding during surgical procedures, and reduce risk of postoperative cardiac and thromboembolic events. In contrast to previous interventions that measure specific portions of the clotting cascade (such as intrinsic or extrinsic pathways or platelet aggregation), "Point-of-care" coagulation monitoring devices assess the viscoelastic properties of whole blood. These techniques have the potential to measure the entire clotting process, starting with fibrin formation, clot retraction, and fibrinolysis. Furthermore, the coagulation status of patients is assessed in whole blood, allowing the plasmatic coagulation system to interact with platelets and red cells, and thereby providing useful additional information on platelet function. Improved monitoring of coagulopathy is particularly important as new anticoagulant drugs emerge that affect the clotting cascade in novel ways, including the inhibition of intrinsic and extrinsic pathways and platelet function. It is important for orthopedic surgeons to understand the pharmacology and reversal of these drugs in the perioperative setting. The purpose of this review is to review the current techniques to monitoring perioperative coagulopathy and to identify the manner in which novel anticoagulant medications affect the clotting cascade with particular interest in trauma and spine surgery.

Keywords Perioperative coagulation monitoring · Point of care · Orthopedic surgery

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Introduction

For today's physician, the diagnosis and management of perioperative coagulopathies remains a challenge. Perioperative hemostatic dysfunction may necessitate the transfusion of allogeneic blood products and is an independent risk factor for perioperative mortality [1, 2]. Coagulopathies can be multifactorial in nature, a result of disturbances in physiology, dysfunctionalities of hemostatic factors, and abnormalities in blood plasma. Clotting is conventionally tested via the international normalized ratio (INR), activated partial thromboplastin time (aPTT), platelet count, and, in some cases, fibrinogen concentration. However, this battery may be of limited use for the detection, monitoring, and treatment for a majority of perioperative coagulopathies [3, 4]. Laboratory analysis is currently conducted outside the effect of in vivo physiology on hemostasis, and results may not allow for targeted therapy. Conventional coagulation tests do not convey specific information regarding clot stability or fibrinolysis.

In the United States, coagulation test results are also obtained after a significant delay and may not reflect the current state of hemostatic physiology, potentially leading to delayed or inappropriate treatment [5–17]. The use of

bedside tests, called point-of-care (POC) tests, may partly compensate for the methodological limitations and diagnostic shortfalls of conventional coagulation testing [6–17]. While none of the currently available methods of POC coagulation testing can provide an adequate picture of the entire coagulation spectrum alone, multiple methods used together may allow for a comprehensive diagnostic evaluation.

This review article will discuss the use of viscoelastic whole blood testing techniques for the analysis of plasma coagulation, clot stability, and clot fibrinolysis. The discussion will focus on the impact of these techniques on blood loss, clinical outcomes, economic aspects, and the limitations of POC testing.

Review of the clotting cascade

The coagulation cascade consists of two distinct pathways that ultimately result in the formation of a fibrin clot via the activation of various serine proteases. Traumatic injury of bony or soft tissues activates the extrinsic, or tissue factor, pathway. Here, damaged tissue expresses tissue factor (TF), which is exposed to circulating factor VII. Activation of factor VII leads to the formation of the TF-VIIa complex, which serves as a major catalyst for the formation of both factors IXa and Xa. Factor IXa is also synthesized via the intrinsic, or contact, pathway. Exposure to negative charges activates factor XII, called the Hageman factor, forming an enzymatic complex with the addition of high molecular weight kininogen (HMWK). The XIIa-HMWK complex both activates factor XI and converts prekallikrein to kallikrein; kallikrein serves as a positive biofeedback by further cleaving factor XII to XIIa. Activated factor Xia serves as a catalyst to active factor IX. Both pathways combine at the formation of factors IXa and Xa, which is catalyzed by IXa itself. Activated factors Xa and Va are the central gateways to the initiation and formation of thrombin via the synthesis of a prothrombinase enzymatic complex. This protein complex converts prothrombin to thrombin, also called factor II. Thrombin ultimately serves to form the fibrin clot via two mechanisms. First, thrombin cleaves factor XIII to XIIIa. This product combines with fibrin, which is activated from its fibrinogen precursor by thrombin as well. Ultimately, factor XIIIa, fibrin, and fibrinopeptides combine to form clot.

Historical techniques to monitor coagulopathy

The aim of perioperative coagulation testing is to detect the pathomechanisms of dysfunctional hemostasis and to initiate treatment rapidly. Currently, most clinical practices conduct the following routine coagulation screens to guide the diagnosis of and treatment for clotting disorders.

Activated partial thromboplastin time (aPTT)

The aPTT was invented to monitor heparinization in the treatment for thromboembolic disorders. The activation of coagulation factors via the intrinsic coagulation cascade is performed by incubating plasma with partial thromboplastins, calcium, and kaolin powder at 37 °C and a standardized pH. While fibrin strand formation is the endpoint of measurement, the large variation in calibration constants and methods of endpoint detection make standardization very difficult. The aPTT is sensitive to coagulation factors I, II, V, VIII, IX, XI, and XII; heparin; fibrinogen degradation products; hypothermia; and hypofibrinogenemia. Multiple factor deficiencies typically result in a greater prolongation for a given factor level than single factor deficiencies. The empiric cut-off value for therapeutic intervention via fresh-frozen plasma (FFP) or prothrombin concentrate (PCC) is an aPTT >1.5–1.8 above normal upper limit (>60 s) [8].

Prothrombin time (PT)

The prothrombin time was created to monitor and adjust the doses of coumarins. Activation of coagulation factors via the extrinsic coagulation cascade is performed by incubating plasma with tissue thromboplastin and calcium at 37 °C and a standardized pH. Fibrin strand formation is the endpoint. The PT is sensitive to coagulation factors I, II, V, VII, and X. Standardization of PT is based on the responsiveness of a singular type of thromboplastin, which is then measured by its International Sensitivity Index and converted into the INR. Direct INR determination is performed by local calibration using plasma of certified levels of PT. The empirical cut-off value for therapeutic intervention with FFP or PCC is a PT less than 40 % [8].

Platelet count

Platelet counting is a quantitative test performed by automated machines. The empirical cut-off value for platelet transfusion is a platelet count <50–100 G l^{-1} [8].

Fibrinogen concentration

Fibrinogen concentration is determined by two methods: first, by a quantitative determination of fibrinogen molecules, and second, by clottable fibrinogen. In the conventional method, thrombin is added to plasma and the fibrinogen concentration is proportional to the coagulation time measured. This test is affected by heparin and

fibrinogen degradation products. Excessive bleeding has been reported at fibrinogen levels below 50–100 mg dl^{-1} [8, 18, 19], but there recent evidence indicates that higher levels of fibrinogen are required for sufficient fibrin clot polymerization, with target levels at 200–380 mg dl^{-1} [20, 21].

Limitations of historical coagulation testing

The timely and specific monitoring of patients' coagulation profiles is vital in the administration of proper replacement therapy in the perioperative setting. However, current routine testing has several weaknesses. Routine coagulation testing may diagnose hemostatic abnormalities due to single or multiple factor deficiencies, but have little use in the specific identification of dysfunctional factors. Also, the PT and aPTT assess only the speed of fibrin strand formation, not the mechanical or functional properties of the clot over time [8]. The platelet count is purely quantitative and cannot detect preexisting, drug-induced, or perioperatively acquired platelet dysfunction [8]. None of the four screening laboratories describe the fibrinolytic process [8].

Routine coagulation tests are also performed in plasma at a standardized temperature of 37 °C, without the presence of platelets and other blood cells. Therefore, routine laboratory tests consider neither the effect of hypothermia on hemostatic physiology nor the complex interaction of plasma proteins, platelets, and the vessel wall after traumatic or surgical injury [8]. Often, the results of routine hemostatic tests are generally available with a delay of at least 30–60 min; results, therefore, may not be able to provide an accurate picture of the current hemostatic physiology [22]. Hardy et al. [23] concluded that bedside monitors of hemostasis are needed urgently for the management of operative and trauma-associated bleeding [10–19]. The bedside tests for PT and aPTT in whole blood using the CoaguCheck (Roche Diagnostics, Switzerland) attempted to overcome this limitation. However, studies have shown poor correlation of bedside results with central laboratory test results [8]. Fibrinogen concentration, aPTT, PT, and platelet count testing are also susceptible to significant inter-laboratory variability in reagent use and an overall lack of standardization [8].

Although severely abnormal PT and aPTT are predictors of mortality, the poor predictive power of moderately impaired routine coagulation tests has been argued as a major limitation [8, 19]. In the acutely injured trauma patient, an initial abnormal PT increases the adjusted odds of mortality by 35 %, while an initial prolonged aPTT increases adjusted odds of mortality by 326 % [24]. Severe prolongations of aPTT $>$ 1.8 times normal and INR $>$ 1.5–1.8 times normal are associated with bleeding [19]. Several studies demonstrate, however, a poor correlation between the severity of coagulation defects and the necessity or amount of blood transfusion [8, 19]. Platelet count has not been shown to be an independent predictor of mortality in the trauma setting [24].

Finally, routine coagulation tests fail to specifically identify the predominant pathomechanism of bleeding in the trauma or intraoperative setting, potentially leading to ineffective treatment. For example, a prolonged aPTT may be due to an intrinsic coagulation factor deficiency requiring specific substitution, a fibrinogen deficiency requiring fibrinogen substitution, hypothermia requiring rewarming, heparinization requiring protamine reversal, or hyperfibrinolysis requiring anti-fibrinolytic drugs. A false differential diagnosis may mislead necessary therapy. Due to the complex nature of hemorrhage in these settings, physicians require coagulation monitoring strategies sensitive to all major possible pathomechanisms of hemostatic dysfunction. POC coagulation monitoring devices have become available and may ultimately overcome several of the limitations associated with routine coagulation testing [3, 8, 22, 25].

Whole blood testing

The viscoelastic whole blood test was created by Hartert [26] and the American Society of Anesthesiologists have included it in the panel of laboratory monitoring for coagulopathy. Thromboelastography [TEG] (Haemoscope Inc., USA) measures the viscoelastic properties of non-anticoagulated or anticoagulated blood by analyzing the induction of clotting under low shear conditions, resembling in vivo rheologic properties [8]. The pattern of changes in viscoelasticity reflects the kinetics of all stages in thrombus formation, clot stability and firmness, as well as fibrinolysis [8]. Rotational thromboelastometry [ROTEM] (Pentapharm GmbH, Germany) improved the original TEG procedure by reducing vibrational interference and limited transportability. Furthermore, ROTEM not only provides a global picture of the injured patient's hemostatic status, but also permits differential diagnosis of the major underlying pathomechanism of coagulopathy by implementing test modifications. The addition of various coagulation-activating agents and/or platelet-inhibiting agents allows for the detection and quantification of specific coagulation defects, such as defect in clot firmness due to fibrinogen deficiency and thrombocytopenia, prolonged clot generation due to various coagulation factor deficiencies or heparin, and impaired clot stability due to hyperfibrinolysis and factor XIII deficiency [6]. Interpretation of TEG/ROTEM results is simplified by both

graphical and numerical presentation of results, quickly highlighting abnormalities. TEG/ROTEM measurements can be taken at the patient's actual body core temperature between 22 and 42 °C, thus allowing quantitative analysis of the anticoagulant effect of temperature physiology [6, 8]. Transfusion requirements before and after the implementation of ROTEM were statistically significantly lower and clinically more accurate [7]. Recommendation to use viscoelastic point-of-care coagulation monitoring embedded into a management algorithm is high.

Extrinsic thromboelastometry (EXTEM) uses recombinant tissue factor to activate coagulation, causing rapid clot generation. The maximum clot firmness (MCF_{ExTEM}) gives information on the maximum clot strength and stability, which is largely dependent on platelet count and fibrinogen level. Prepared disposable wells containing cytochalasin D, a platelet inhibitor, are used in a derivative test called FIBTEM. MCF_{FIBTEM} represents the contribution of fibrinogen to the clot strength. Critical MCF cut-off values appear within 15 min after test initiation. A low MCF_{FIBTEM} suggests the administration of fibrinogen concentrates, while a normal MCF_{FIBTEM} ($\geq$12 mm) in the presence of a low MCF_{ExTEM} (<50 mm) suggests the need for platelet substitution [6]. Thus, comparing MCF_{FIBTEM} with MCF_{ExTEM} permits the differentiation of coagulopathy secondary to either a low platelet count or dys- or hypofibrinogenemia.

Extrinsic thromboelastometry also tests clotting time (CT_{ExTEM}), providing information about the initial activation and dynamics of clot formation and allowing for the analysis of factor deficiencies. The critical cut-off value for CT, indicating the need for PCC (20–30 IU kg^{-1}) or FFP (30 ml kg^{-1}), appears about 100 s after test initiation [6]. Clotting time is analyzed with a second derivative test utilizing wells containing aprotinin, called APTEM. APTEM permits the quantitative assessment of fibrinolysis and the estimation of the therapeutic benefit from antifibrinolytic agents, such as tranexamic acid. Any improvement in CT or MCF in APTEM, when compared with EXTEM, diagnoses a low-grade hyperfibrinolysis. Such a result indicates the need to first correct the hyperfibrinolysis via the administration of antifibrinolytic drugs, followed by the replacement of the consumed coagulation factors [6].

Intrinsic thromboelastometry [INTEM] utilizes ellagic acid contact activator, comparable to the reagent used for aPTT testing, in order to analyze the patient's general coagulation status. Wells containing heparinase (HEPTEM) or ecarin can be used to detect specific anticoagulant effects. Specifically, comparing CT_{INTEM} to CT_{HEPTEM} permits the quantification of heparin effects at both low and high concentrations. A $CT_{INTEM} > 240$ s and $CT_{HEPTEM}/CT_{INTEM} < 0.66$ suggests protamine administration, and these results can estimate the therapeutic benefit from protamine reversal.

Table 1 breaks down currently used perioperative coagulation monitoring tests and their specific uses.

Platelet function tests are commonly utilized in the preoperative evaluation of patients with a known positive bleeding history, as well as in actively bleeding patients with anti-platelet therapy, known platelet defects, or extracorporeal circulation. These laboratories are especially vital ROTEM, and routine coagulation screening cannot specifically identify the defect in hemostasis. Currently, there does not exist a simple, reliable method for measuring platelet function. Static tests, such as β-thromboglobulin measurement, capture only point data and cannot accurately reflect the dynamic hemostatic process.

Table 1 Common perioperative coagulation monitoring tests

Assay	Indication
Thromboelastograph hemostasis System (TEG) kaolin	Overall platelet function and coagulation assessment
TEG heparinase	Specific detection of heparin
TEG platelet mapping	Platelet function and monitoring antiplatelet therapy
Rotation thromboelastometry (ROTEM) Ex-TEM	Extrinsic pathway; fast assessment of clot formation and fibrinolysis
In-TEM	Intrinsic pathway; assessment of clot formation; and fibrin polymerization
Fib-TEM	Qualitative assessment of fibrinogen levels
Ap-TEM	Fibrinolytic pathway; fast detection of fibrinolysis when used with ex-TEM
Hep-TEM	Specific detection of heparin
Eca-TEM	Management of direct thrombin inhibitors
Tif-TEM	Extrinsic pathway; monitoring recombinant-activated factor VIIa
Sonoclot coagulation and platelet function analyzer (Son ACT)	Large-dose heparin management without aprotinin
kACT	Large-dose heparin management with/without aprotinin
aiACT	Large-dose heparin management with aprotinin
gbACT+	Overall coagulation and platelet function assessment
H-gbACT+	Overall coagulation and platelet function assessment in the presence of heparin; detection of heparin
micro-PT	Extrinsic pathway; monitoring recombinant-activated factor VIIa

Dynamic tests, such as in vivo bleeding time, depict the time-dependent contribution of platelets to overall clot formation. However, this test is poorly standardized, temperature and drug dependent, influenced by vascular disorders, lacking in specificity and sensitivity, and not predictive of bleeding [27]. Furthermore, bleeding time fluctuates significantly during surgery and transfusion and cannot differentiate between bleeding and non-bleeding patients [28].

Sonoclot analysis

The Sonoclot Analyzer (Sienco Inc., Wheat Ridge, CO, USA) is a test of the viscoelastic properties of blood, comparable to TEG/ROTEM, which provides information on the entire hemostasis process, including coagulation factors, fibrin gel formation, platelet function, and fibrinolysis. This device consists of a tubular probe that oscillates vertically within a blood sample. The viscous force of the blood creates impedance to the ultrasonic vibrating probe as it clots, which is converted to an output signal. The electronic signal is processed and reported as the Clot Signal. The Sonoclot Analyzer reports these properties both as quantitative results and by graphically recording the dynamics of clot formation over time, called the Sonoclot Signature. The quantitative results include a lag period (SonACT) corresponding to activated clotting time (ACT) and a wave that occurs as a result of cross-linkage of fibrin (clot rate). Other parameters in the tracing indicate platelet–fibrin binding, fibrin formation, and clot retraction. Hemostatic abnormalities such as platelet dysfunction, factor deficiency, anticoagulant effects, hyperfibrinolysis, and hypercoagulable states can be detected using the Sonoclot [6]. The Sonoclot Analyzer has been criticized because its results were influenced by age, sex, and platelet count [6]. Additionally, studies showed poor reproducibility of some of the measured variables, especially clot rate [6]. However, the Sonoclot Analyzer has demonstrated a precision close to that of thromboelastography [6]. Furthermore, the Sonoclot Analyzer is insufficient in the acutely bleeding patient, limiting its applicability to goal-directed management algorithms [6].

Monitoring anticoagulation

The intraoperative quantification of unfractionated heparin effects during full heparinization, such as during cardiopulmonary bypass, is traditionally performed by the ACT (activated clotting time). Various ACT devices are currently available, but all are fundamentally based on the test principle of aPTT. Unfortunately, ACT is not sensitive enough to monitor low heparin doses, utilized postoperatively for the protection of difficult vascular anastomoses. In these situations, aPTT or thrombin time (TT) is recommended as a poor substitute instrument. However, studies have shown that treatment with low molecular weight heparin and heparinoids can also be assessed with POC viscoelastic tests [6]. Both standard and heparinase-modified tests can be performed to increase the sensitivity of TEG/ROTEM testing, specifically geared to determine the effects of low molecular weight heparin and heparinoids on coagulation [6].

Because platelets play a key role in overall coagulation, the assessment of the platelet function, more than their number, is critical in the perioperative setting [29, 30]; Munoa et al. [31]. Traditional assays, such as turbidimetric platelet aggregometry, are still considered a clinical standard for platelet function testing. However, this process is labor-intensive, costly, time-consuming and requires a high degree of experience and expertise to perform and interpret. Furthermore, platelets are tested under relatively low shear conditions in platelet-rich plasma, conditions that do not accurately simulate primary hemostasis [30]. Viscoelastic POC coagulation analyzers may provide greater information on platelet function. The MA/MCF comparison from TEG/ROTEM reflects overall platelet function and fibrinogen levels. If two different tests, such as EXTEM and FIBTEM, are run simultaneously, comparing the difference between clot firmness will represents the platelet contribution to clot formation. However, since conventional TEG/ROTEM are not sensitive to targeted pharmacological inhibition, a more refined test has been developed for the TEG to specifically determine platelet function in the presence of antiplatelet therapy (Platelet-Mapping^{TM}) [32, 33]. The Platelet Mapping assay measures clot strength as maximal amplitude and enables for the quantification of platelet function, including the contribution of adenosine phosphate and thromboxane A2 receptors to clot formation. The Sonoclot Analyzer has also been shown to reliably detect pharmacological GPIIb/IIIa inhibition [34, 35].

Monitoring procoagulation

The modern practice of procoagulation management is based on the concept of specific component therapy and requires rapid diagnosis and monitoring of the pro-coagulant therapy. Clinical judgment alone, or combined with conventional non-viscoelastic laboratory tests, cannot predict which patient will benefit from a platelet transfusion in the acute perioperative setting. It has been shown that platelet transfusion in the perioperative period of coronary

artery bypass graft surgery is associated with increased risk for serious adverse events [36]. Therefore, the most recent guidelines on perioperative blood transfusion and blood conservation of The Society of Thoracic Surgeons and Society of Cardiovascular Anesthesiologists state that transfusion of coagulation products should be preferably guided by POC tests that assess hemostatic function in a timely and accurate manner [37].

Fibrinogen is a key substrate for clot formation, and isolated fibrinogen substitution in severe models of dilutional coagulopathy has been shown to improve clot strength and reduce blood loss. Supplementary administration of prothrombin complex, concentrate of factor II, VII, IX, X, antithrombin III, and protein C additionally improved initiation of coagulation and reversed dilutional coagulopathy [38]. Fibrinogen levels can be assessed by measuring clot strength (MCF/MA) in the presence of platelet inhibition (e.g., fib-TEM) or by assessing Sonoclot's clot rate [39]. In Europe and the USA, recombinant-activated factor VII (rVIIa) treatment is currently approved for patients with congenital or acquired hemophilia with antibodies to factor VIII or IX, factor VII deficiency, or Glanzmann's thrombasthenia (Europe only). However, rVIIa is increasingly used in off-label indications to control severe bleeding, such as in major trauma, surgical interventions, intracerebral hemorrhage, by locally activating hemostasis at sites of vascular injury. The resulting thrombin burst leads to the formation of a fibrin clot if fibrinogen levels are adequate. Consensus guidelines have been published for these off-label indications, but it is still unclear how to reliably monitor patients receiving rVIIa [39]. To better study the result of thrombin generation, modified TEG/ROTEM parameters based on the first derivative of original TEG/ROTEM tracing have been introduced recently: maximum velocity of clot formation (MaxVel), time to reach MaxVel (tMaxVel), and total thrombus generation (area under the curve) [40, 41]. These parameters may be more sensitive to rVIIa than standard TEG/ROTEM parameters [42].

Economic aspects

Current prospective randomized trials do not allow any definitive conclusions regarding the putative economic savings of POC coagulation testing. A number of retrospective studies have compared the costs of hemotherapy before and after the implementation of POC-based chemotherapeutic algorithms [43–45]. Most studies involved cardiothoracic surgery patients and produced conflicting results [43–45]. These studies do indicate that POC-based coagulation therapy indeed lowers the rate of transfusion of allogenic blood products, mostly by lowering FFP and PC transfusion rates. However, there was a simultaneous increase in the use of clotting factor concentrates, mostly fibrinogen and prothrombin–proconvertin–Stuart factor–antihemophilic factor B. The economic savings from the reduced use of allogenic blood products may compensate for or outweigh the increased expenditures for clotting factor concentrates.

Limitations of POC

Several concerns have been raised regarding the use of viscoelastic POC coagulation tests. While Gorlinger et al. showed fewer postoperative thrombotic/thromboembolic complications in the POC group compared to the control group, the groups did not differ with respect to perioperative mortality [43–45]. In fact, five prospective randomized trials with various lengths of postoperative follow-up have shown no beneficial effect of POC-based coagulation therapy on postoperative mortality [43–45]. The blood collection site, method of sampling processing, patient age, and patient gender may significantly affect the results of these tests [46]. Furthermore, equipment, activators, and other test modifications will alter the assay specificity [46]. All these factors render inter-laboratory standardization difficult, potentially limiting the use of result comparisons.

As with all POC devices, there is a concern that the devices are not adequately maintained, supervised, and without regular quality control. Non-laboratory personnel may be conducting POC tests, which may lead to further errors without adequate training. To minimize these problems and release the operating room/intensive care unit personnel, some hospitals have moved POC coagulation analyzers into the central laboratory (Ganter et al. 2008). Here, a trained individual conducts the viscoelastic coagulation test and results are submitted real time to the patient's site.

Monitoring coagulation in spine surgery

Coagulation disorders that occur during major spinal surgery have been published in several accounts [47, 48]. Patients undergoing spinal fusion often sustain a large amount of perioperative blood loss [47, 48]. This blood loss is replaced by crystalloids, colloids, and blood products. As described earlier, massive transfusions in response to high blood loss places patients at an increased coagulopathy from a dilutional thrombocytopenia [47]. There are also theories describing increased bleeding due to the activation of the fibrinolytic system due to bone as a source of tissue plasminogen activator and urokinase [48]. Lastly, a preexisting coagulation defect may be present in

patients with idiopathic scoliosis or ankylosing spondylitis [48].

Mayer et al. [48] showed that there is a definitive fall in fibrinogen level and platelet count in every patient with major spinal reconstructive procedures. Prothrombin time was found to increase in each patient, although the amount was unpredictable. Changes in quantitative fibrinogen levels were decreased in all cases. Platelet counts were initially variable in number intraoperatively or immediately postoperatively but in all cases dropped below preoperative levels within the first 24 h, with the lowest fibrinogen and platelet levels reached by the third or fourth day after surgery [48].

Horlocker et al. [47] found that the intraoperative coagulation tests with the highest sensitivity and specificity were the international normalized ratio, PT, and aPTT. They found that the intraoperative INR and PT were able to differentiate patients with clinical evidence of excessive bleeding from those with normal hemostasis at cut points of approximately 1.5 times the control values. They found that TEG values were of marginal use, and among those values, the TEG MA (maximal amplitude), which is a reflexion of clot strength, had the highest accuracy [47]. Ultimately, they concluded that TEG values were documented to be useful in cardiothoracic surgery but never in orthopedic surgery, let alone spine surgery.

Conclusions

In summary, perioperative coagulation monitoring includes the assessment of bleeding history and routine laboratory testing. Further evaluation of hemostatic status is warranted in patients with history of coagulopathy or if procedure-specific risk factors for bleeding are anticipated. Routine coagulation tests, such as aPTT, PT, platelet count, and fibrinogen concentration, have major limitations to guide management. Furthermore, time lost while awaiting results aggravate coagulopathy, blood product requirements, time of surgery, and morbidity. Instead, POC coagulation tests deliver specific test results within minutes and permit an early goal-directed intervention. While TEG and Sonoclot Analyzer describe a global picture of the hemostatic process, ROTEM extends this feature with a repertoire of test variations, permitting differential diagnosis of major pathomechanisms of inherent or acquired coagulopathies. POC testing also permits accurate monitoring of the effects of either anti- or pro-coagulation therapies. Previous studies have shown that an algorithm-based use of viscoelastic POC testing may lead to less perioperative blood loss and a decreased rate of allogeneic blood product transfusion. However, the literature does not indicate any effect of POC testing on perioperative morbidity and mortality. Further study regarding critical questions of standardization, utilization algorithms, outcomes, and economic viability is warranted.

Conflict of interest None.

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