Tarabrin O., Sukhonos R., Kirpichnikova K., Buhayenko Ye., Hladkykh I., Suslov O. Clinical utility of low-frequency piezoelectric thromboelastography for global hemostasis assessment and prediction of thrombo-hemorrhagic complications. Journal of Education, Health and Sport. 2025;85:67617. eISSN 2391-8306. https://dx.doi.org/10.12775/JEHS.2025.85.67617
https://apcz.umk.pl/JEHS/article/view/67617
https://zenodo.org/records/17989184

The journal has had 40 points in Minister of Science and Higher Education of Poland parametric evaluation. Annex to the announcement of the Minister of Education and Science of 05.01.2024 No. 32318. Has a Journal's Unique Identifier: 201159. Scientific disciplines assigned: Physical culture sciences (Field of medical and health sciences);
Health Sciences (Field of medical and health sciences). Punkty Ministerialne 40 punktów. Załącznik do komunikatu Ministra Nauki i Szkolnictwa Wyższego z dnia 05.01.2024 Lp. 32318. Posiada Unikatowy Identyfikator Czasopisma: 201159. Przypisane dyscypliny naukowe: Nauki o kulturze fizycznej (Dziedzina nauk medycznych i nauk o zdrowiu); Nauki o zdrowiu (Dziedzina nauk medycznych i nauk o zdrowiu).© The Authors 2025;

The authors declare that there is no conflict of interests regarding the publication of this paper.
Received: 22.08.2025. Revised: 28.08.2025. Accepted: 19.09.2025. Published: 25.09.2025.

UDC 612.115.3–073.97:616.151.5–005.6–089

Clinical utility of low-frequency piezoelectric thromboelastography for global hemostasis assessment and prediction of thrombo-hemorrhagic complications

O. Tarabrin, R. Sukhonos, K. Kirpichnikova, Ye. Buhayenko, I. Hladkykh, O. Suslov

International Humanitarian University, Odesa, Ukraine

Abstract

Background. Venous thromboembolism (VTE), including deep vein thrombosis (DVT) and pulmonary embolism (PE), remains a major cause of preventable in-hospital morbidity and mortality worldwide. Conventional coagulation tests (prothrombin time, activated partial thromboplastin time, fibrinogen, D-dimer) provide only a static snapshot of hemostasis and do not adequately reflect the global hemostatic potential (HP) or hypercoagulable states, particularly in high-risk surgical patients. There is growing interest in "global" viscoelastic methods such as thromboelastography (TEG) and low-frequency piezoelectric thromboelastography (LPTEG) for dynamic, point-of-care assessment of the Blood Aggregation Regulation System (RAS) and hemostatic potential.

Objective. To describe the methodological principles and analytical parameters of low-frequency piezoelectric thromboelastography and to evaluate its potential for dynamic assessment of hemostasis and prediction of thrombo-hemorrhagic risk, including in combination with the functional stress test "double local hypoxia of the upper limb" (DLHUL).

Methods. LPTEG records changes in the viscoelastic properties of whole blood during hemocoagulation, from a liquid to a solid-elastic state, yielding an integrated curve (A0–A6, t1–t5). Primary analytical parameters include contact coagulation intensity (CCI), intensity of coagulation drive (ICD), constant of thrombin activity (CTA), intensity of clot polymerization (IPC), maximum amplitude (MA), intensity of total coagulation (ITC), intensity of clot retraction and lysis (IRCL), and coefficient of total anticoagulation activity (CTAA). Pre-analytical conditions were standardized (venous blood sampling with a 1.0-mL silicone syringe without tourniquet, time from sampling to cuvette ≤ 20 s). The study included 40 healthy volunteers (Group 1) and 120 surgical patients with risk factors for thrombosis (Group 2). All participants underwent a functional DLHUL test (two brief episodes of arterial and venous occlusion of the upper limb separated by a 20–25-minute interval), with LPTEG recorded before and after the test.

Results. In Group 1 (without clinical predictors of thrombotic risk), two distinct patterns of hemostatic response to DLHUL were identified: a compensated type (decrease in platelet-vascular indices, enhanced fibrinolysis, shift of HP toward hypocoagulation) and a subcompensated type (increase in CCI, shortening of clotting time, shift of HP toward hypercoagulation). These patterns occurred with similar frequency (20/40 each. In Group 2 (patients with thrombotic risk factors), baseline LPTEG already showed increased platelet aggregation, activation of the coagulation link (elevated ICD and MA), and reduced fibrinolytic activity (decreased IRCL). After DLHUL, CCI increased by 21.07%, A0 by 5.87%, ICD by 8.51%, and MA by 8.17%, while IRCL decreased by 23.67%, indicating further progression toward hypercoagulation and suppression of fibrinolysis. Most patients in Group 2 demonstrated decompensated (n = 98) or exhausted (n = 22) hemostatic responses, with limited adaptive reserve of the anticoagulant and fibrinolytic systems.

Conclusions. LPTEG provides a comprehensive, real-time assessment of hemostatic potential that surpasses conventional laboratory tests by simultaneously characterizing platelet-vascular, coagulation, fibrinolytic, and anticoagulant components. The combination of LPTEG with the DLHUL functional test enables identification of compensated, subcompensated, decompensated, and exhausted hemostatic response types, facilitates stratification of perioperative thrombo-hemorrhagic risk, and supports individualized thromboprophylaxis and anticoagulant therapy. Low-frequency piezoelectric thromboelastography is a promising tool for dynamic hemostasis monitoring in surgical and critically ill patients.

Keywords: **low-frequency piezoelectric thromboelastography; LPTEG; thromboelastography; hemostatic potential; Blood Aggregation Regulation System; RAS; venous thromboembolism; deep vein thrombosis; pulmonary embolism; hypercoagulability; double local hypoxia test; DLHUL; perioperative risk; thromboprophylaxis; viscoelastic testing**

Introduction

The prothrombotic phase may result in the formation of venous and arterial thrombosis, leading to high morbidity and mortality worldwide. According to the US Centers for Disease Control and Prevention (CDC), venous thromboembolism (VTE), which includes deep vein thrombosis (DVT) and pulmonary embolism (PE), affects up to 900,000 individuals annually in the United States alone, and approximately 60,000–100,000 patients die from VTE-related complications each year [1]. VTE is among the leading causes of potentially preventable in-hospital death; 50–70% of cases are associated with recent hospitalization, surgery, or prolonged immobilization [1, 2]. In the general population, the lifetime cumulative risk of VTE approaches 8%, meaning that nearly one in twelve individuals will experience VTE during their lifetime [2].

Global Burden of Disease data demonstrate that PE ranks among the major causes of cardiovascular mortality alongside ischemic heart disease and stroke. Age-standardized PE mortality rates have decreased in high-income countries but remain stable or are increasing in low- and middle-income regions [3]. This highlights the need for improved strategies for early diagnosis and prevention of VTE, particularly in high-risk populations such as surgical, oncological, orthopedic, and critically ill patients.

The biological mechanism regulating blood clotting, known as the Blood Aggregation Regulation System (RAS), is critically involved in thromboembolic events. Thromboembolism is primarily attributed to dysfunction of RAS subsystems [4]. This dysfunction impairs the appropriate discretization of hemostatic potential (HP) in different vascular segments, thereby preventing the formation of clots where and when they are physiologically needed. Arterial thrombosis typically occurs following erosion or rupture of an atherosclerotic plaque, resulting in platelet-mediated thrombus formation that can cause ischemic damage, particularly in tissues at the end of the vascular bed [5]. Acute coronary syndrome and ischemic stroke are severe and common consequences of atherothrombosis, driven predominantly by tissue ischemia, which may develop gradually through progressive atherosclerosis or acutely due to thrombus embolization in blood vessels or cardiac chambers.

VTE is a frequent vascular complication after acute myocardial infarction and stroke, as well as in surgical patients. Clinical studies indicate that the incidence of in-hospital DVT ranges from approximately 10–40% in patients undergoing general surgery and 40–60% after major orthopedic procedures in the absence of adequate thromboprophylaxis [6, 7]. DVT affects deep veins in 25–30% of cases, potentially leading to PE. PE occurs in roughly 10% of surgical and orthopedic patients and is a significant cause of in-hospital mortality [6, 7]. In patients with multiple risk factors (malignancy, prolonged immobilization, obesity, advanced age, systemic inflammation), the risk of VTE is markedly increased, while standard laboratory tests frequently fail to identify subclinical hypercoagulability in a timely manner.

Clinically, VTE presents as DVT or PE, with PE often arising as a complication of DVT. Thrombus formation and propagation are determined by the integrity of the vascular wall, disturbances in blood flow, and activation of coagulation factors, collectively known as Virchow's triad. Blood flow disturbances or venous stasis may occur due to prolonged immobilization, extended bed rest, or patient positioning during surgery. Perioperative risk factors for VTE include the type and extent of surgery, postoperative anastomotic failure, history of smoking, immobility, trauma, obesity, cardiovascular and respiratory pathology, estrogen use, malignancy, age over 40 years, acquired hypercoagulable conditions, and hereditary thrombophilia [4, 8–10].

Despite extensive research on the coagulation system and the availability of validated clinical risk scores (e.g. Caprini, Padua, Khorana), the incidence of thromboembolic complications in high-risk patients remains substantial, especially during surgery and in the postoperative period, where the surgical intervention itself acts as a triggering factor for thromboembolism. Current international guidelines emphasize that conventional coagulation tests (prothrombin time, activated partial thromboplastin time, fibrinogen, D-dimer) insufficiently reflect global HP and, in particular, hypercoagulable tendencies, limiting their predictive value for VTE risk [3, 5, 10]. Consequently, there is increasing interest in "global" methods for hemostasis assessment, such as thromboelastography (TEG) and rotational thromboelastometry (ROTEM), which provide an integrated, dynamic picture of coagulation and fibrinolysis.

At the same time, thrombosis is a largely preventable complication through timely diagnosis, risk stratification, and implementation of appropriate prophylactic measures, including both pharmacological and mechanical methods. In patients scheduled for elective surgical procedures who are at increased risk of thromboembolic events, angiosurgical interventions may be considered when clinically indicated [5, 7]. For individualization of

thromboprophylaxis and early detection of latent hyper- or hypocoagulation, dynamic hemostatic assessment is of particular importance, especially low-frequency piezoelectric thromboelastography (LPTEG), which can refine the individual hemostatic profile and guide therapeutic decisions.

When assessing prothrombotic and thrombotic conditions in surgical candidates, a typical diagnostic work-up includes Doppler imaging of lower limb vessels, echocardiography, routine laboratory tests, and, when available, TEG. TEG, which has gained prominence in cardiac and vascular surgery, allows for detailed, dynamic evaluation of all components of the hemostatic system. However, current laboratory methods mainly provide a static "snapshot" of the hemostatic system at the time of blood sampling, without informing on the reserve capacity of platelet-vascular, coagulation, and fibrinolytic components. In managing patients at risk of thrombo-hemorrhagic disorders, it is necessary not only to quantify markers of the RAS but also to understand their functional interplay in maintaining an optimal HP within the vascular bed. Such a comprehensive approach enables characterization of RAS functional activity and its responsiveness to changes in HP induced by various factors, as well as assessment of the compensatory abilities of the coagulation and fibrinolytic systems [4, 5].

Thromboelastog raphy is an important method for hemostasis assessment that measures blood coagulation parameters in real time. It allows evaluation of clot formation quality, bleeding risk, and thrombus formation potential. TEG can rapidly and accurately assess a patient's hemostatic status at the point of care, which is especially important in critical conditions such as trauma, massive bleeding, and other critical states associated with thrombo-hemorrhagic disorders. In addition, TEG is useful in monitoring anticoagulant therapy, in pregnancy, and in patients with thrombotic diseases. While routine laboratory tests reflect isolated parameters at a single time point, TEG depicts the dynamic process of coagulation, offering a global assessment of thrombus formation and allowing more precise identification of the hemostatic link requiring pharmacologic intervention.

Recently, significant attention has been paid to "global" tests for rapid and integrative evaluation of plasma and cellular components in whole blood, especially within the Point-of-Care Testing paradigm. These components play a crucial role in fibrinogenesis. Hemostatic potential (HP), a key dimension of hemocoagulation, can be assessed using such "global" TEG-based tests, which are particularly informative in the late stages of fibrinogenesis — lateral fibrin folding, formation of cross-linked fibrin (PSF), clot stabilization, and subsequent lysis.

Low-frequency piezoelectric thromboelastography (LPTEG) analyzes changes in the viscoelastic properties of blood during hemocoagulation, as it transitions from a liquid to a solid-elastic state. The dynamics of this process are determined by changes in the aggregate state of blood and are recorded as an integrated curve, where each point (Ai) reflects the state of the system at a given time (Ti).

LPTEG evaluates the following parameters:

A0 – initial amplitude at time t0;

t1 – reaction time (minutes from the start of the test to the minimum LPTEG amplitude A1);

A1 max t1 – decrease in amplitude during the reaction time t1;

t2 – time to reach amplitude A2;

A2 – increase in LPTEG amplitude by 100 arbitrary units;

t3 – clotting time (CTT), corresponding to the gelation point (TJ), in minutes, determined automatically when the tangent of the curve angle changes by 60%;

A3 – LPTEG amplitude at the gelation point TJ, in relative units;

A4 – LPTEG amplitude 10 minutes after reaching the maximum amplitude;

A5 – maximum LPTEG amplitude within 10 minutes;

t5 – time to reach maximum amplitude (MA, A5), i.e. time of formation of the fibrin-platelet clot structure;

A6 – amplitude 10 minutes after reaching MA.

Calculated LPTEG indices include:

Contact coagulation intensity (CCI) = (A1–A0)/t1; this mainly reflects the aggregation activity of blood cellular elements, phases I and II of coagulation, or suspension stability (SSC).

Intensity of coagulation drive (ICD) = (A3–A1)/t3; this predominantly characterizes the proteolytic stage of phase III hemocoagulation. The segment of the LPTEG curve near the gelation point (≈60% change in tangent angle) reflects the onset of polymerization, leading at the gelation point to fibrin gel formation — the primary structural framework of the hemostatic clot.

Constant of thrombin activity (CTA) = A2/(t2–t1), where A2 is set at 100 relative units; this provides a universal criterion for evaluating the intensity of the proteolytic stage of fibrin formation.

Intensity of clot polymerization (IPC) = (A4–A3)/10 minutes; this primarily characterizes the polymerization stage of phase III hemocoagulation. Because changes in clot

viscoelastic properties during fibrin polymerization and formation of transverse intermolecular (covalent) bonds are relatively slow and the transition to the stabilization phase is somewhat arbitrary, a fixed 10-minute interval from the gelation point is used to standardize LPTEG analysis and evaluate the early post-gel polymerization stage.

Maximum amplitude (MA) = (A5–A1), in relative units; this reflects the maximum clot density determined by platelet activity and the quantitative/qualitative properties of cross-linked fibrin (PSF), and corresponds to the completion of formation of a retracted cross-linked fibrin clot.

Intensity of total coagulation (ITC) = MA/t5; this index assesses the overall intensity of fibrinogenesis.

Intensity of clot retraction and lysis (IRCL) = (A5–A6)/A5×100%; this reflects the activity of plasmin, leukocyte proteases (granulocyte elastase, cathepsin G, monocytic cathepsin D, complement), and erythrocyte kinases present in the 0.5-mL blood sample. To improve accuracy, the analysis time may be extended to 20–30 minutes.

Coefficient of total anticoagulation activity (CTAA) = ICD/IPC; this represents the global anticoagulant activity of blood, dependent on several groups of inhibitors: disaggregants (NO_2, PGI_2, cAMP/cGMP), specific and non-specific serine protease inhibitors (α_2-macroglobulin), tissue factor pathway inhibitor (TFPI), coenzyme inhibitors (proteins C and S, thrombomodulin), and fibrin degradation products. This index is useful because peak activity of the regulatory system is expressed predominantly in phases I and II of coagulation and during the proteolytic component of phase III prior to the onset of active clot polymerization.

To standardize the pre-analytical stage, venous blood is drawn using a three-component silicone syringe with a rubber cuff (volume 1.0 mL), without tourniquet application. The interval between blood sampling and transfer to the disposable cuvette should not exceed 20 seconds. The plastic cuvette, positioned in the device thermostat, is filled to the calibration mark (~0.45 mL), after which the test is initiated.

The amplitude of the process is evaluated along the ordinate axis (A, in relative units), while time is plotted along the abscissa axis (T, in minutes).

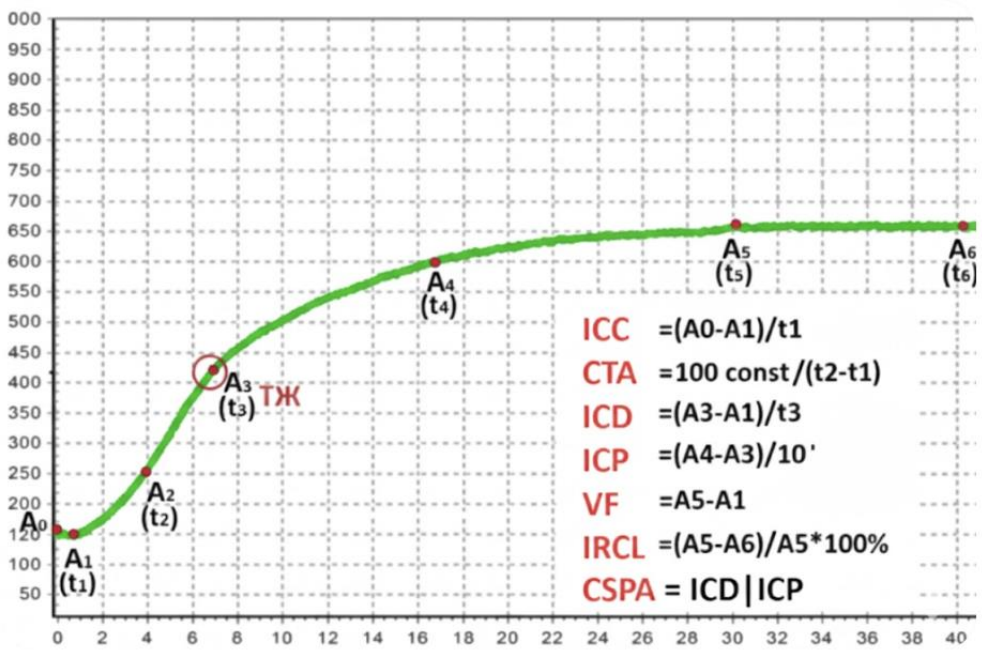

Figure 1. LPTEG of whole blood from a healthy volunteer. A0–A6 — LPTEG amplitudes (relative units) at stages of PSF formation, retraction and lysis; t1–t5 — time intervals of fibrinogenesis stages; JP (t3) — gelation point; MA — maximum clot density.

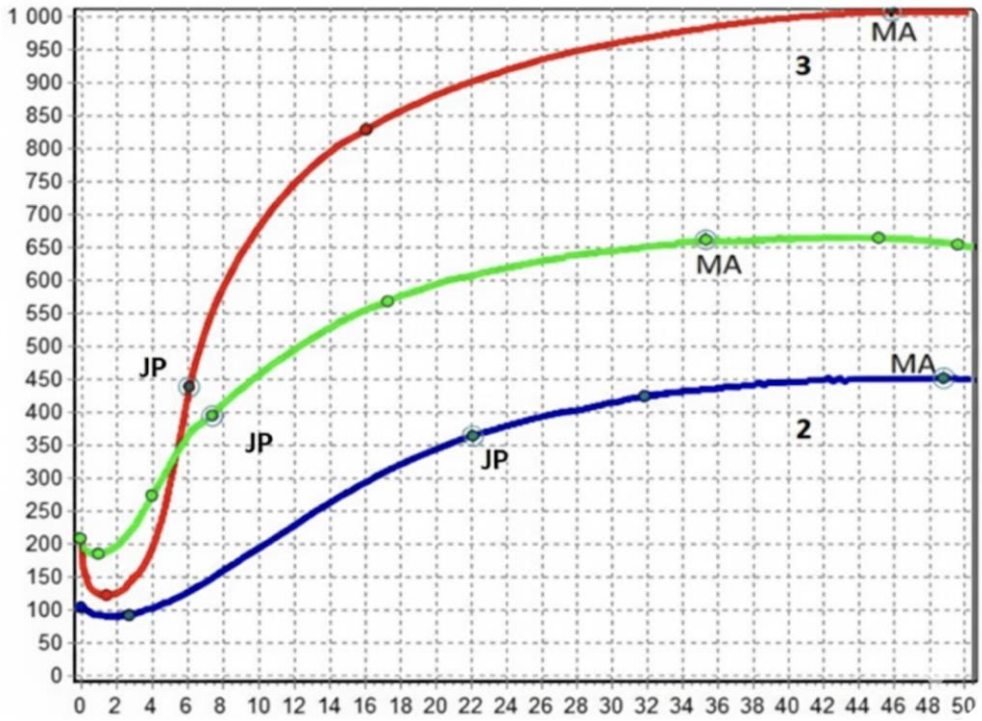

Figure 2. LPTEG analysis in hypo- and hypercoagulable RAS states based on comparison of recorded curves with reference normocoagulant values: LPTEG in normo- (1), hypo- (2), and hypercoagulable (3) conditions.

Thus, this technology enables not only assessment of a temporal “slice” of HP but also monitoring of the effectiveness of antithrombotic therapy.

To confirm the feasibility of using LPTEG to evaluate the antiplatelet effect of a COX-1 inhibitor at a relatively low dose (75 mg acetylsalicylic acid), Figure 3 presents data from 10 healthy volunteers, demonstrating a pronounced increase in t1 following drug administration.

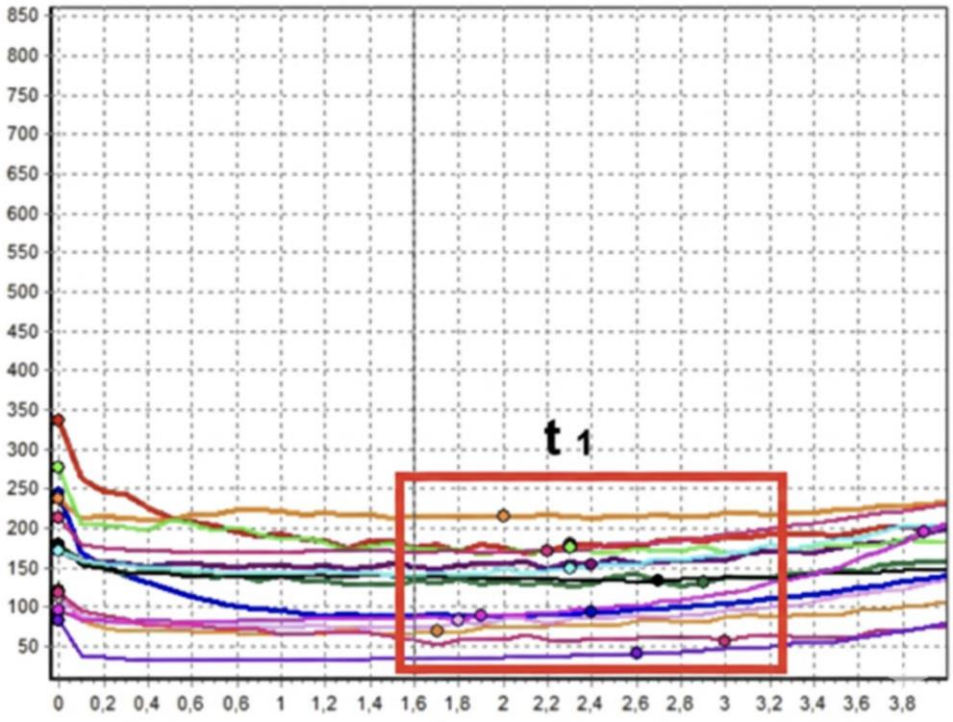

Figure 3. Scaled baseline LPTEG curves for 10 healthy volunteers before (left) and 12 hours (right) after ingestion of 75 mg aspirin.

The effect of heparin on the hemostasis system, as assessed by low-frequency piezoelectric thromboelastography, is also illustrative. In a study involving 10 conditionally healthy volunteers (Figure 4), LPTEG showed a comparable response at the time of maximum drug effect. Assessment of the proteolytic stage of fibrinogenesis was performed by comparing t1, t2 and CTA — a universal criterion for this stage of hemocoagulation.

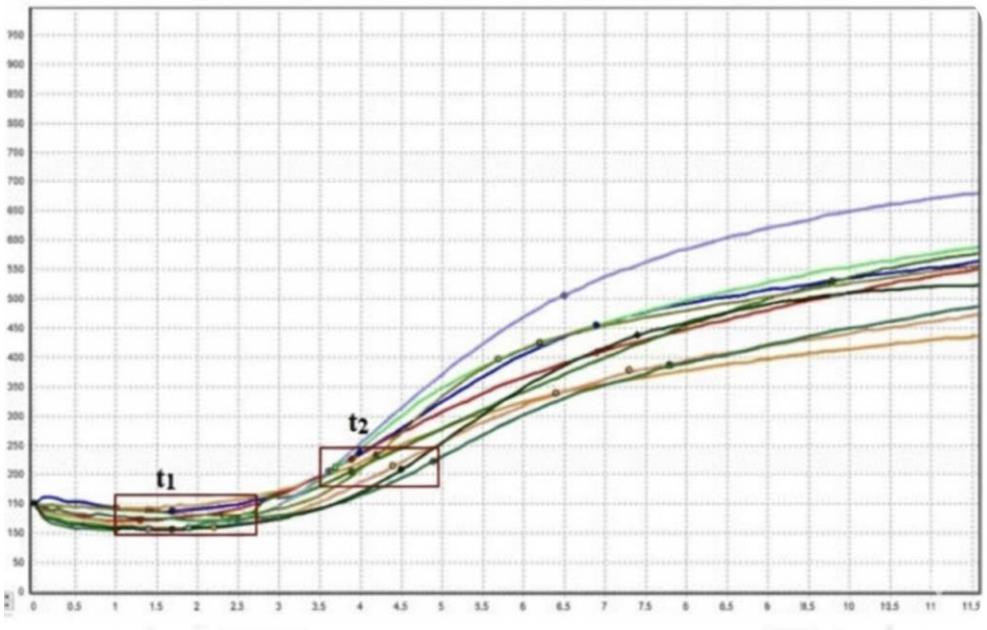

Figure 4. Scaled LPTEG curves recorded in 10 healthy volunteers before (left) and 10 minutes (right) after administration of 5000 IU heparin.

Use of LPTEG with a functional stress test (DLHUL)

LPTEG is used not only to detect overt hemostatic disorders during a clinical event but also as a tool to identify thromboembolic risk. For this purpose, the functional stress test "double local hypoxia of the upper limb" (DLHUL) is applied. This test is particularly useful in patients with existing predictors and risk factors for thrombo-hemorrhagic complications who are scheduled for elective surgery. Determining the degree of thrombogenicity and assessing dysfunction in specific components of the hemostasis system allows for an appropriate choice of preventive therapy, thereby reducing perioperative complications.

A randomized prospective study was conducted. Patients were divided into two groups according to the presence of thrombotic risk factors. Group 1 comprised healthy volunteers (n = 40) without risk factors for thrombosis. Group 2 comprised patients with established thrombotic risk factors (n = 120) scheduled for elective surgery. Inclusion criteria for Group 2 were: smoking history, history of VTE, paralysis of the lower limbs, trauma (e.g. fractures of the lower limb bones), morbid obesity (body mass index > 35 kg/m^2), concomitant cardiovascular and respiratory disorders (acute myocardial infarction, atrial fibrillation, congestive heart failure, previous ischemic stroke, obliterating atherosclerosis, chronic respiratory failure, chronic obstructive pulmonary disease), use of estrogens in pharmacological doses (e.g. oral contraceptives, hormone replacement therapy), malignancy,

age > 40 years, and acquired hypercoagulable conditions, including autoimmune diseases. Exclusion criteria were current antiplatelet and/or anticoagulant therapy.

All patients underwent DLHUL with TEG-based hemocoagulation assessment. The method is based on inducing Virchow's triad (vascular wall damage, blood flow obstruction, and altered blood rheology) in a localized vascular segment. The main purpose of the test is to elicit a response delineating the hemostatic limits of the system and to observe the onset and duration of adaptive and compensatory reactions within the hemostasis system.

Double local hypoxia of the upper limb is achieved by temporary occlusion of arterial and venous vessels using a tourniquet for approximately 5–6 minutes, with a 20–25-minute interval between occlusions. TEG readings are recorded before and after the test to assess various aspects of hemostasis, including aggregate blood state (A0), contact coagulation intensity (CCI), coagulation drive intensity (ICD), maximum clot density (MA), and fibrinolytic activity represented by the clot retraction and lysis index (IRCL).

Table 1. Results of LPTEG during DLHUL

Indicator	GROUP 1				GROUP 2			
	before		after		before		after	
	M	±σ	Compensated type	Subcompensated type	M	±σ	M	±σ
Aggregate state of blood (A0)	225,22	13,32	211,31 ± 20,64*	269,56 ± 17,15*	435,02	22,44	462,13	30,01
Intensity of contact coagulation (CCI)	86,32	1,01	75,54 ± 1,12*	91,01 ± 1,01*	142,17	2,44	180,12	3,46*
Intensity of coagulation drive (ICD)	21,15	0,62	20,65 ± 0,46*	21,37 ± 0,41*	41,07	1,12	44,89	1,66*
Maximum clot density (MA)	513,51	31,44	490,11 ± 31,01*	600, 03 ± 33,42*	878,01	60,99	956,13	42,44
IRCL	15,55	0,42	21,04 ± 0,42*	15,66 ± 0,44*	7,47	0,77	6,04	0,45*

Notes: * $p < 0.05$ — statistically significant difference between baseline and post-test values within the group; ** $p < 0.05$ — statistically significant difference between groups after DLHUL.

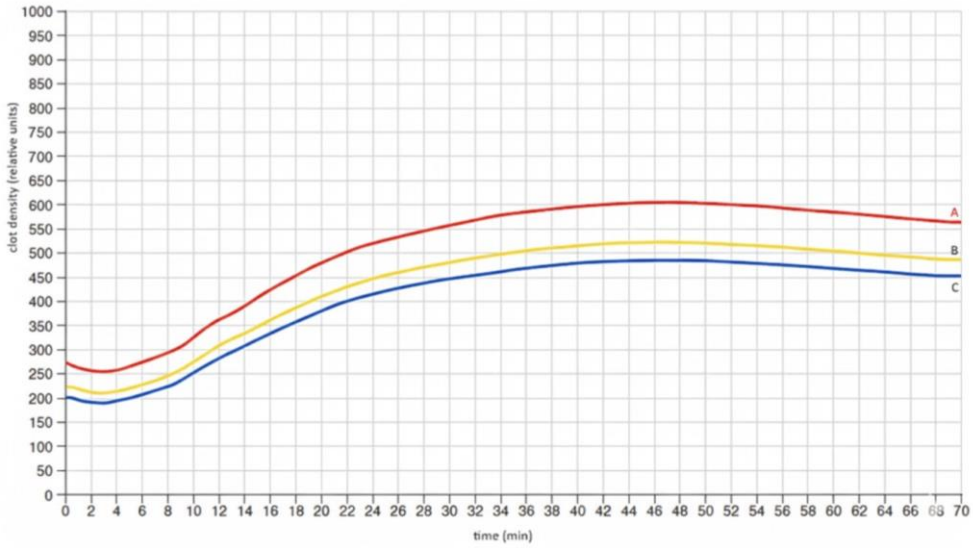

Figure 5. Changes in the hemocoagulation system in Group 1 before and after DLHUL:

A — subcompensated type;

B — compensated type;

C — before DLHUL.

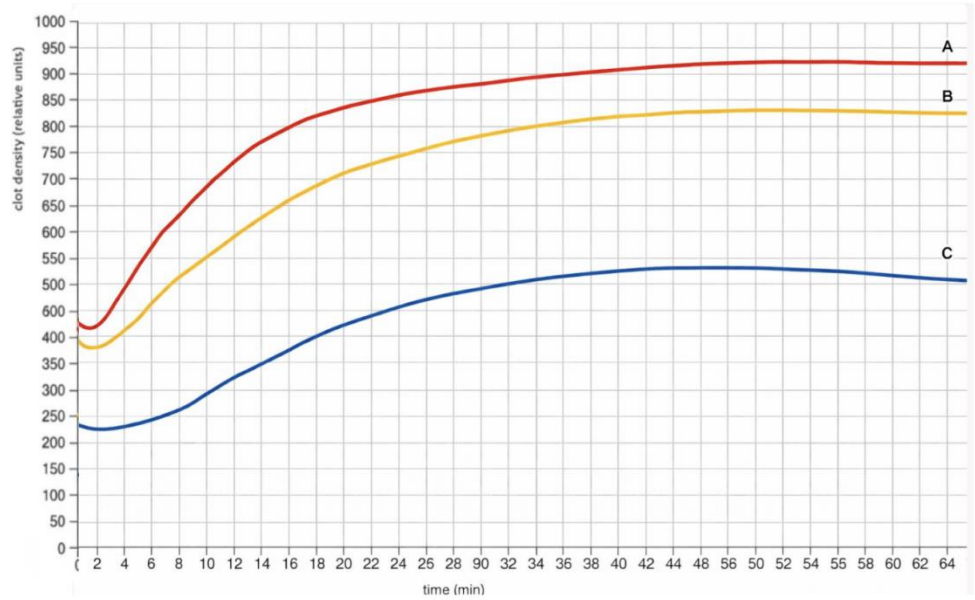

Figure 6. Thromboelastograms of changes in hemostatic potential in Group 2 (patients with thrombotic risk factors) before and after DLHUL: C — averaged thromboelastogram for Group 1; B — Group 2 before DLHUL; A — Group 2 after DLHUL.

Analysis of TEG data after DLHUL in Group 1 (without thrombotic risk predictors) identified two types of hemostatic response:

Compensated type, characterized by decreased platelet-vascular indices;

Subcompensated type, characterized by increased platelet-vascular indices.

These types exhibit distinct TEG patterns corresponding to compensated and subcompensated responses and occur with equal frequency (n1 = 20; n2 = 20).

In Group 1 volunteers with a subcompensated response, DLHUL induced an increase in CCI and a decrease in clotting time, indicating enhancement of the external pathway of prothrombinase formation. Overall, the response of the procoagulant arm of the coagulation system in this subgroup reflects a shift of HP toward hypercoagulation.

In contrast, Group 1 volunteers with a compensated response demonstrated increased fibrinolytic activity, decreased ICD compared with the subcompensated type, and prolonged clotting time after DLHUL, suggesting reduced activity of the external mechanism of prothrombinase synthesis and a shift of HP toward hypocoagulation.

In Group 2 (patients with thrombotic risk factors), DLHUL elicited marked changes in HP across all components of the hemostasis system. In the platelet-vascular component, platelet aggregation increased significantly in response to the stimulus. TEG data (Table 1) revealed statistically significant deviations from normal values for A0 and CCI, which characterize platelet aggregation properties. CCI after DLHUL exceeded baseline by 21.07%, while A0 increased by 5.87%. ICD increased by 8.51% and MA by 8.17%, indicating activation of the coagulation link. Fibrinolytic activity, as reflected by IRCL, decreased by 23.67%, indicating inhibition of fibrinolysis after the functional test.

In most Group 2 subjects, DLHUL revealed decompensated (n1 = 98) or exhausted (n2 = 22) types of response. That is, in the presence of enhanced platelet aggregation, hypercoagulation, and suppression of anticoagulant mechanisms and fibrinolysis already at baseline, DLHUL further aggravates these disturbances toward hypercoagulation: platelet aggregation increases, the coagulation link is further activated, and fibrinolysis becomes more depressed. However, the relative magnitude of change after the trigger is less pronounced than in Group 1, reflecting the limited adaptive reserve of an already overstressed system.

In individuals without a history of factors provoking a hypercoagulable state, two types of RAS response to DLHUL are possible: compensated and subcompensated. In this cohort, the risk of perioperative thrombotic complications is relatively low.

In contrast, in subjects with a history of hypercoagulability-provoking factors, two other RAS response types predominate: decompensated (more common) and exhausted (less common). These patients have a high risk of perioperative thrombotic events and a

non-negligible risk of thrombo-hemorrhagic complications, including disseminated intravascular coagulation (DIC).

Conclusions

Thromboelastography, and particularly low-frequency piezoelectric thromboelastography (LPTEG), is an effective method for assessing hemostatic potential. Unlike standard laboratory tests, LPTEG provides a detailed graphical and numerical representation of the hemostatic system. Its broad array of primary and derived indices enables identification of disturbances in specific hemostatic components (platelet-vascular, coagulation, fibrinolytic, and anticoagulant links).

The use of LPTEG in combination with the DLHUL functional test allows:

detection of latent hyper- or hypocoagulable states;
evaluation of adaptive and compensatory capacities of the hemostasis system;
stratification of patients by perioperative thrombo-hemorrhagic risk;
individualization of thromboprophylaxis and anticoagulant therapy.

Thus, low-frequency piezoelectric thromboelastography is a promising tool for dynamic hemostasis assessment, guiding therapeutic strategy and evaluating the risk of thrombo-hemorrhagic disorders in patients with altered hemostatic potential, especially in the settings of surgery and intensive care.

References

1. Centers for Disease Control and Prevention. Venous Thromboembolism (Blood Clots): Data and Statistics [Internet]. Atlanta (GA): CDC; [cited 2025 ___]. Available from: https://www.cdc.gov/blood-clots/data-research/index.html

2. Heit JA. Epidemiology of venous thromboembolism. Nat Rev Cardiol. 2015;12(8):464–474.

3. Raskob GE, Angchaisuksiri P, Blanco AN, Büller H, Gallus A, Hunt BJ, et al. Thrombosis: a major contributor to global disease burden. Arterioscler Thromb Vasc Biol. 2014;34(11):2363–2371.

4. Tarabrin O, Shcherbakov S, Gavrychenko D, Saleh O, Lyoshenko I, Kushnir O. Can we use the low-frequency piezoelectric tromboelastography for diagnosis coagulation disorders? Eur J Anaesthesiol. 2013;30(Suppl 51):92.

5. Turpie AGG, Chin BSP, Lip GYH. Venous thromboembolism: pathophysiology, clinical features, and prevention. BMJ. 2002;325(7369):887–890.

6. Turpie AG, Bauer KA, Eriksson BI, Lassen MR. Fondaparinux vs enoxaparin for the prevention of venous thromboembolism in major orthopedic surgery: a meta-analysis of 4 randomized double blind studies. Arch Intern Med. 2002;162(16):1833–1840.

7. Hyers TM, Agnelli G, Hull RD, Morris TA, Samama M, Tapson V, et al. Antithrombotic therapy for venous thromboembolic disease. Chest. 2001;119(1 Suppl):176S–193S.

8. Walker ID, Greaves M, Preston FE. Guideline: investigation and management of heritable thrombophilia. Br J Haematol. 2001;114(3):512–528.

9. Previtali E, Bucciarelli P, Passamonti SM, Martinelli I. Risk factors for venous and arterial thrombosis. Blood Transfus. 2011;9(2):120–138.

10. Gerhard-Herman MD, Gornik HL, Barrett C, Barshes NR, Corriere MA, Drachman DE, et al. 2016 AHA/ACC guideline on the management of patients with lower extremity peripheral artery disease: executive summary. J Am Coll Cardiol. 2017;69(11):1465–1508.

11. World health statistics 2012. Geneva: World Health Organization; 2012. p. 34–37.

12. World Health Statistics 2014. Geneva: World Health Organization; 2014. p. 21–24.

13. Tverdovskyi IV. The use of a test with double local hypoxia of the upper limb in patients with obliterating atherosclerosis of the lower limb vessels. Medytsyna neotlozhnykh sostoyanyy. 2019;8(103).