https://www.scirp.org/journal/jbise
J. Biomedical Science and Engineering, 2022, Vol. 15, (No. 7), pp: 187-197

Electromagnetism, Blood Flow and Coagulation

Merab Beraia[1], **Guram Beraia**[2]

[1]MRI Department, Institute of Clinical Medicine, Tbilisi, Georgia; [2]Tbilisi State Medical University, Tbilisi, Georgia

Correspondence to: M. Beraia, mberaia@hotmail.com

Keywords: Viscoelastic Blood Flow, Coagulation, Parcel Resident Time, Electromagnetic Force, Information Filter
Received: May 31, 2022 **Accepted:** July 18, 2022 **Published:** July 21, 2022

ABSTRACT

There is a lot of uncertainty in the theory of hemodynamics. The amount of work need to displace the blood in the systemic circulation, exceeds the work done by the left ventricle. With this, blood recovers increased flow resistance between the heartbeats with the Womersley number alterations in the rhythm of the accompanying electrocardiogram (ECG). Viscoelastic transformation is heavily expressed in coagulation. There must be a relationship between the ECG and blood transient flow resistance. The influence of the electromagnetic field on blood coagulation was studied. Venous blood was affected by the oscillated electromagnetic field (500 - 5000 Hz), with the square wave input signal in 25 healthy individuals (15 males, 10 females in the age 18 - 57 years). Electromagnetic irradiation (EMI) time of the sample 3 - 10 min. Hypocoagulation in normal blood samples was revealed (decreased quantity of Platelets up to 10 - 23 × 10^9/L, Prothrombin index up to 9% - 10%, Fibrinogen concentration up to 0.20 - 0.21 g/L) and thrombolysis after the blood stasis. Ac electric field from the myocardial depolarization initiates electroacoustic phenomena. An emerging repulsing electromagnetic force acts on the red blood cells (RBC) and in addition to the pulse pressure from the heart, promotes blood motion and viscoelastic changes. The alterations of the blood inertial and elasticity, in addition to hemodynamics, are facilitated by the magnetic features of the hemoglobin. The external electromagnetic signal can manage the blood coagulation process, including thrombolysis.

1. INTRODUCTION

Now is accepted that the heart is two separate pumps that pump blood through the lungs and the peripheral organs [1]. While blood is a specialized body fluid, that passively flows. But the energy of the heart is insufficient to carry out the transporting function.

Blood is a viscoelastic, thixotropic substance. It changes its high flow resistance very quickly, literally between heartbeats. For that reason, this property has been renamed transient flow resistance. Thixotropy

has been shown to be caused by the extra work needed to align red cells into the plane of flow as the flow begins or restarts. Transient resistance is recovered rapidly because elastic red cells move out of the plane of flow as flow slows or stops as they relax elastically. A large part of blood's transient resistance is mediated by growing contact between red cells, as they lose the orientation they maintained during flow [2].

Viscoelasticity is a property of human blood that is primarily due to the elastic energy that is stored in the deformation of red blood cells as the heart pumps the blood through the body [3]. Another part of the energy is dissipated by viscosity, and the remaining energy is stored in the kinetic motion of the blood.

The aggregatable and deformable nature of RBCs plays a significant role in blood rheology. RBC aggregation causes a large increase in viscosity at low shear rates. The size of RBC aggregation is a function of RBC concentration and shear rate [4]. The existence of the aggregation also depends on the presence of fibrinogen and albumin proteins in plasma. Fibrin has both elastic and viscous properties. Perhaps the most remarkable rheological feature of the fibrin network is its extremely high elasticity and stability despite very low protein content [5].

Changes in viscoelasticity are distinctly expressed in blood coagulation. "Virchow's triad" has been suggested to describe the three factors necessary for the formation of thrombosis: stasis of blood, vessel wall injury, and altered blood coagulation [6]. Coagulation/clotting is the process by which blood changes from a liquid (sol) to a gel, forming a blood clot. Blood in the primary hemostasis can more accurately be described as a fluidized suspension of elastic cells. The dispersed phase in a gel is a liquid. The gel contains the covalent polymer networks formed through the physical aggregation and were caused by hydrogen bonds, crystallization, helix formation, complexation, etc. that results in regions of local order acting as the network junction points [7].

Arterial thrombosis has long been held to be largely a phenomenon of platelet activation, whereas venous thrombosis is largely a matter of activation of the clotting system. Arterial thrombosis occurs under high shear flow when platelet-rich thrombi are formed around ruptured atherosclerotic plaques and damaged endothelium. Venous thrombosis occurs under low shear flow and mostly around the intact endothelial walls. However, there is evidence that this dichotomy is an oversimplification. The shared initial coagulation events are suggested to treat the whole process of arterial and venous thrombosis as a single entity [8].

The characteristic time for primary hemostasis in a healthy person is 1 - 3 minutes. Actually, blood coagulation (secondary hemostasis, hemocoagulation, plasma hemostasis) is a complex biological process of the formation of fibrin protein strands in the blood, which polymerizes and forms blood clots, as a result of which the blood loses its fluidity, acquiring a curdled consistency. Blood clotting in a healthy person occurs at the site of the primary platelet plug formation. The characteristic time of fibrin clot formation is 3 - 8 minutes [9, 10].

The coagulation and fibrinolytic systems are highly regulated and interrelated through mechanisms that ensure balanced hemostasis [11]. There are reports concerning a decrease of blood fibrinolytic activity in humans during electromagnetic storms and sudden cardiac death in locomotive drivers, staying in areas of electromagnetic waves [12]. It is known the effect of homogeneous magnetic fields as low as 0.005 T on platelets, blood coagulation, and fibrinolysis in guinea pigs [13]. There are dates about the influence of a uniform static magnetic field (8 T) on normal erythrocytes, reports on the orientation of fibrinogen, retinal cells, sickled cells, etc. [14].

Time-varying electric fields can induce several biophysical responses when applied to living cells. One phenomenon of interest is electroporation, also known as electro permeability, in which a rapidly time-varying field increases the permeability of the plasma membrane. Another phenomenon is dielectrophoresis—fields of a particular frequency separate the cells or the orientate and manipulate nanoparticles and nanowires [7].

Low and high-frequency electric fields are involved in the functional alteration of the cell structures. Erythrocytes oscillate during their tank treading motion with high-frequency oscillations 1.2 MHz. This oscillatory motion drastically affects the dielectric and electrical proerties of red blood cells [15].

The mechanisms responsible for electromagnetic field-induced blood viscoelastic effects are not fully

understood and have been the subject of debate. Possible ways electromechanical activity of the blood are discussed.

2. METHODS AND MATERIALS

To create conditions for the ac current flow in the blood, the silicone catheter coil on the toroid transformer was filled by the blood and irradiated by the electromagnetic field. On a primary electric coil of the transformer was applied voltage from an electrical signal generator, in a square waveform with the frequencies 500 - 5000 Hz (Figure 1).

The cardiomyocyte action potential curve is an analogy to the square wave, the sharp spikes of the wavefront. Cardiomyocyte depolarization time is 2 msec (frequency 500 sec^{-1}).

The oscillating external magnetic field, inside the blood sample, forms particle polarization. At the ends of the catheter were placed cooper electrodes connected by the external resistor (external load). Ac current flows inside the blood. Signals were controlled by the digital oscilloscope. Blood was taken from 25 healthy individuals (15 males, 10 females in the age 18 - 57 years). Electromagnetic irradiation (EMI) time of the sample 3 - 10 min.

In the initial stage of the experiments, blood at the irradiation was moved, by the syringes on the ends of the silicone catheter coil. Next, the blood was left in the catheter without motion for 30 min. for the thrombi formation and then irradiated by the electromagnetic field (Table 1, Table 2).

3. DISCUSSION

Hemodynamics in arteries and blood viscoelastic alterations.

From the law of energy conservation, heart work must be enough for the blood displacement in the vessels, structural changes in the shear blood flow, and heat production.

Table 1. Blood cell count.

Investigation	Data before EMI	Data after EMI	Referral date
White blood cells (WBC)	5.29 - 6.58	3.63 - 6.77	4 - 10 × 10^9/L
Red blood cells (RBC)	4.13 - 4.95	4.47 - 5.51	3.8 - 5.9 × 10^{12}/L
Hemoglobin (HGB)	127 - 170	134 - 170	116 - 170 g/L
Hematocrit (HCT)	39.0 - 47.5	39.9 - 49.0	35% - 50%
Mean cell volume (MCV)	94.4 - 96.0	88.9 - 89.3	80 - 97 fl
Platelets (PLT)	199 - 260	10 - 23	150 - 400 × 10^9/L
Plateletcrit (PCT)	0.19 - 0.25	0.01 - 0.03	0.12% - 0.35%
Mean platelet volume (MPV)	9.5 - 9.8	10.2 - 11.3	6.5 - 12 fl
Platelet distribution width (PDV)	10.3 - 11.1	8.3 - 19.9	9.9 - 16.1 fl
Neutrophils (NEUT)%	53.9 - 60.7	59.2 - 63.9	50% - 70%
Lymphocytes (LYMPH)%	28.4 - 35.2	30.0 - 33.7	20% - 40%
Monocytes (MONO)%	6.8 - 8.1	5.2 - 5.9	4% - 10%
Eosinophils (EO)%	2.4 - 3.9	0.6 - 0.9	1% - 4%
Basophils (BASO)%	0.3 - 0.6	0.3 - 0.4	0% - 1%

Table 2. Secondary hemostasis.

Imaging	Data before EMI	Data after EMI	Referral date
Activated partial thromboplastin clotting time (APPT)	30.4 - 37.5	180	20 - 37 sec
Prothrombin time (PT)	13.0 - 13.2	90.0 - 90.1	9 - 16 sec
Prothrombin index (PI)	100	9 - 10	75% - 110%
International normalized ratio (INR)	1.0	0.00	0.9 - 1.3
Fibrinogen concentration (FIB)	2.81 - 3.81	0.20 - 0.21	2 - 4 g/L
Thrombin time (TT)	15.7 - 19.4	240 - 240.1	14 - 20 sec

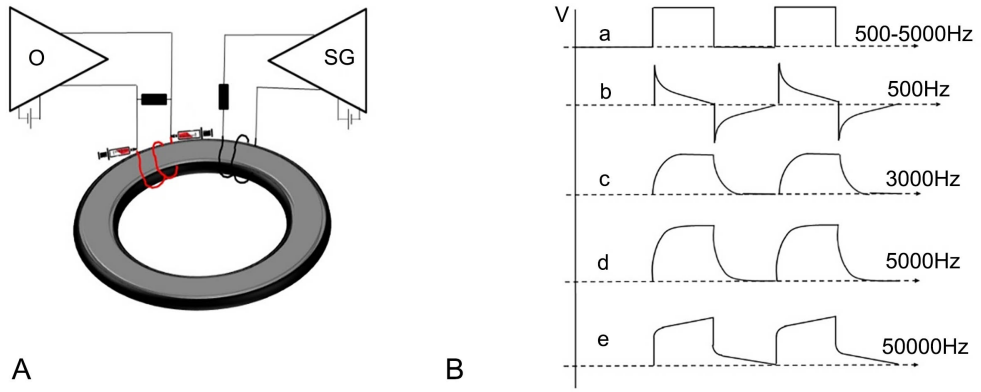

Figure 1. A. The signal generator (SG) sends the square form electric signals to the primary coil of the high-frequency transformer. Induced voltage uptakes from the secondary coil resistor and registered by the oscilloscope (O). The secondary coil is the catheter, filled with the blood. B. Signals in the primary coil: a—500 - 50,000 Hz. Inducting signals from the blood look like voltage oscillations from the RC Differentiator-high pass filter at b—500 Hz, c—3000 Hz, d—5000 Hz, e—50,000 Hz. At 5000 Hz the voltage drops are highest across the blood capacity alternating between charging up to Vc. and discharging down to zero according to the input voltage.

Due to the multifaceted nature of the studying problem, some simplifications should be done. Calculation of the heart work can be successful, in presenting the circulatory system as the compression pump without friction. By examining the physical parameters in the ventricle of the heart and blood vessels, it is possible to calculate the work done at the different segments of the hydraulic pump.

Work done in the left ventricle is about 0.99 J., while full work done for blood flow in systemic arteries and capillaries 2.3 J.

Under the specified conditions, work for the blood displacement is directly proportional to the pressure (*P*) *and volume* (*V*) *of the displaced mass.*

$$W = P \times V = F \times D$$

Amount of the force to the per unit of the area, perpendicular to the surface of the object is a pressure.

$$P = -F/A$$

P—pressure, F—magnitude of the normal force opposite to the pressure, A—area of the surface on contact, D—displacement.

Mean pressure in ascending aorta 100 *mmHg* = 1.33×10^4 N/m^2.

Mean inner diameter of the ascendant aorta 1.5 *cm. Mean area of ascending aorta* 1.77 cm^2.

Volumetric (75 *ml*) *blood mean displacement in ascending aorta in each heart cycle* = 42.46 *cm.*

Force made by the left ventricle is $F = P \times A = 1.33.10^4$ $N/m^2 \times 1.77 \times 10^{-4}$ $m^2 = 2.35$ *N.*

Work done by the left ventricle for the blood displacement in the ascending aorta in each heart cycle $W = 2.35$ $N \times 0.42$ $m = 0.99$ *J.*

The average capillary hydrostatic pressure 17 *mmHg* = 22.66×10^2 N/m^2 [3].

Mean diameter of the capillary 0.0006 *cm. Mean length of the capillary* 0.06 *cm.*

Single capillary volume = 2.83×10^{-11} $m^2 \times 6.10^{-4}$ $m = 16.98.10^{-15}$ $m^3 = 16.98.10^{-9}$ cm^3

Quantity of capillaries filled by the 75 *ml of blood* = 75 $cm^3/16.98 \times 10^{-9}$ cm^3 = *about* 4.42×10^9 *open capillaries.*

Hydrostatic pressure force in the single capillary $F = P \times A = 22.66 \times 10^2$ $N/m^2 \times 2.83 \times 10^{-11}$ $m^2 = 64.13 \times 10^{-9}$ *N.*

Work done for the blood displacement in the singe capillary in each heart cycle $W = 64.13 \times 10^{-9}$ $N \times 6.10^{-4}$ $m = 79.14 \times 10^{-12}$ *J.*

Work in blood distribution in all systemic open capillaries (*without filtration*) *in each heart cycle is* $W = 79.14 \times 10^{-12}$ $J \times 4.42 \times 10^9 =$ **0.39** ***J.***

In electronic–hydraulic analogy, hydraulic equations can be expressed by the electrical. With this work (*W*) *done on the blood flow in systemic arteries and capillaries can by express as: W = UIT.*

From the Ohm's law voltage U = IR.

T-flow time, U voltage (*pressure—in hydraulic*), *current I* (*Q-flow—in hydraulic*), *R—resistance.*

Net flow and the flow time in the arteries and capillaries are the same. Pressure drop (*U*) *in arterial network, up to capillaries* (83 *mmHg*) *is* 4.9 *times higher than that in capillaries* (17 *mmHg*).

Work done in the systemic arterial system must be **0.39** $J \times$ **4.9 = 1.91** ***J, and full work done in systemic arteries + capillaries must be about =*** **2.3** ***J. It is higher than that the work done by the left ventricle.***

Besides the displacement, structural rearrangement of the blood from the aorta to the systemic capillaries is associated with the energetically cost. Here it is necessary to take into account also the additional energy for the venous blood flow and heat production. *i.e.* the work for the blood displacement in the systemic circulation must be many times more, than the work done by the left ventricle and there should be an additional energy source for this.

Blood flow is pulsatile—propagates in the waveform. Mass is displaced by the transversal and later, longitudinal pressure waves, forming together the rotating surface waves across the interface between differing media (**Figure 2**). In protodiastola, due to the frequency dispersion, surface waves destroy the cell aggregates in arterial flow, decreasing inertial/elastic properties, while at the vessel wall, the reflection, can damage the endothelial sheet, forming the denudation—initial factor for atherosclerosis [16].

Electromagnetism in hemodynamics and blood viscoelasticity.

It can be proved, that additional energy in the blood flow is formed by electromagnetic forces. They arise from the heart rotating dipole and transmit to all the living body cells by the ac current. It is provided by the electroacoustic phenomena of the colloid system, RBC electromagnetism and oxygen electronegativity in the cell mitochondria. The electric impulse from the heart initiates an oscillating electric field around the red blood cells, and an emerging repulsing electromagnetic force promotes the blood flow, in addition to the pulse pressure from the myocardial contraction. Outstripping the flow, the modulated signal ECG initiates the viscoelastic changes in the blood (**Figure 3**).

In the isolated system, linear momentum is constant and subject to the principle of conservation [17]. Here energy transfer between the inertial masses is elastic. Inertial forces in the pulsating fluid motion can be characterized by the Womersley number. High Womersley's number in the aortic blood shows its high

inertial and elasticity.

In the flow from the elastic artery to capillaries, blood loses inertial/elastic features and viscous interactions are more evident. On the contrary, at the flow from capillaries to the veins, the blood becomes elastic. Here crucial role should be played coagulation system of the blood, providing, but not obstructing blood flow by affecting the parcel residence time.

Residence time is an indicator of flow trajectory and mixing. Non-uniformed wall shear stress variations in regions with secondary or disturbed near-wall flows, especially in the vicinity of vessel bifurcation, branching junctions, and sharp bends increase the blood residence time (*i.e.* slow-moving flows). Blood low flow velocity is natural for the venous blood, mostly at the stasis, and associated with thrombus formation.

In the structural rearrangement of the blood and a central role can behave the erythrocyte's controllability by the electromagnetic field. The erythrocyte can be viewed as a toroidal dielectrophoretic electromagnetic field-driven cell that maintains its zeta potential via a dielectric constant (chloride anion) that resides between a negatively charged membrane surface and a positively charged Stern layer. There are ferromagnetic (iron) and ferroelectric (chloride anion) influences that may be crucial to the maintenance of this zeta potential [18] that protects cells from aggregation. The functional properties of hemoglobin such as the binding of O_2, the Bohr effect, and the cooperativity are explained based on the magnetic correlations. This analysis suggests that magnetism could be involved in the functioning of hemoglobin [19].

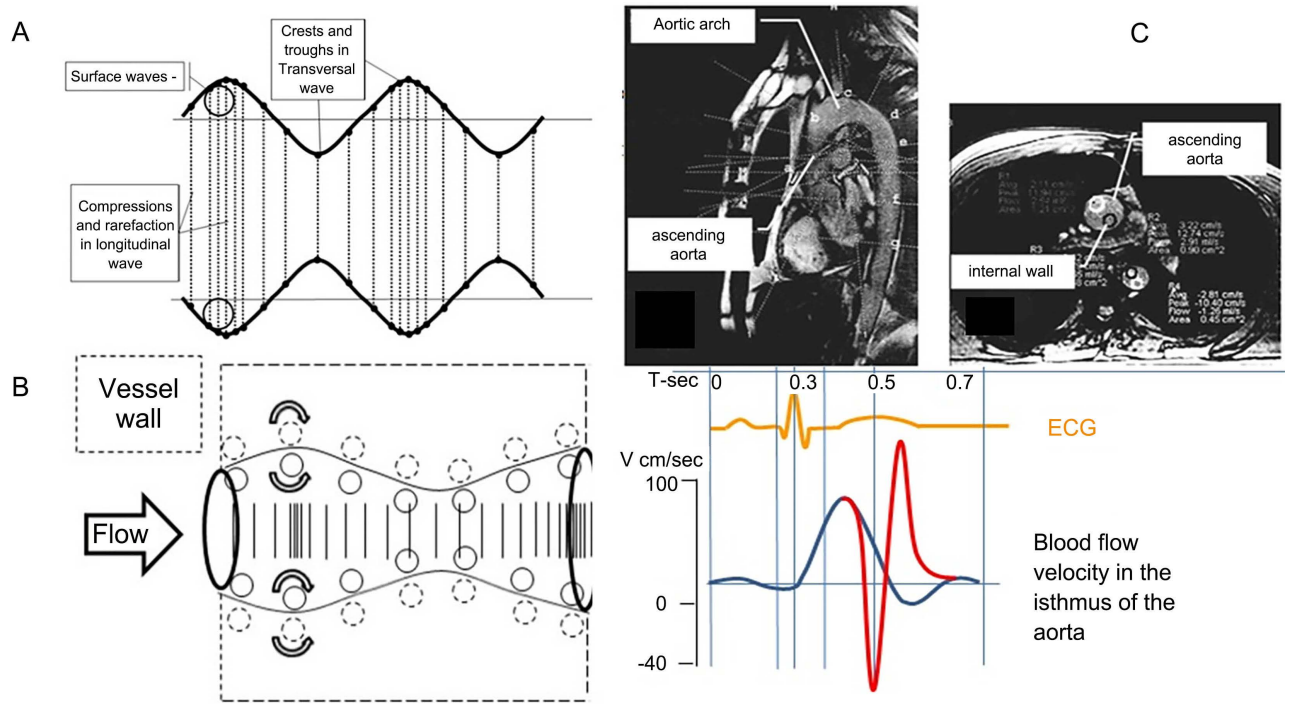

Figure 2. A. Transverse wave is made up of crests and troughs, while a longitudinal wave is made up of compressions and rarefaction. B. Surface wave formation at the boundary layer. C. Blood rotates at the circle flow sites and bifurcations. In the initial diastole, the flow separates due to the retrograde pressure drop. It accompanied with the ECG-T potential. At the external wall of the isthmus of the aorta, retrograde flow acceleration, in the initial diastole is 11.6 times higher (red line) than the forward acceleration in the initial systole (blue line). Opposite-directed waves have different frequencies. Blood flow at the reflection from the wall, denudates the vessel wall by the frequency dispersion. ECG's signal affects the blood flow.

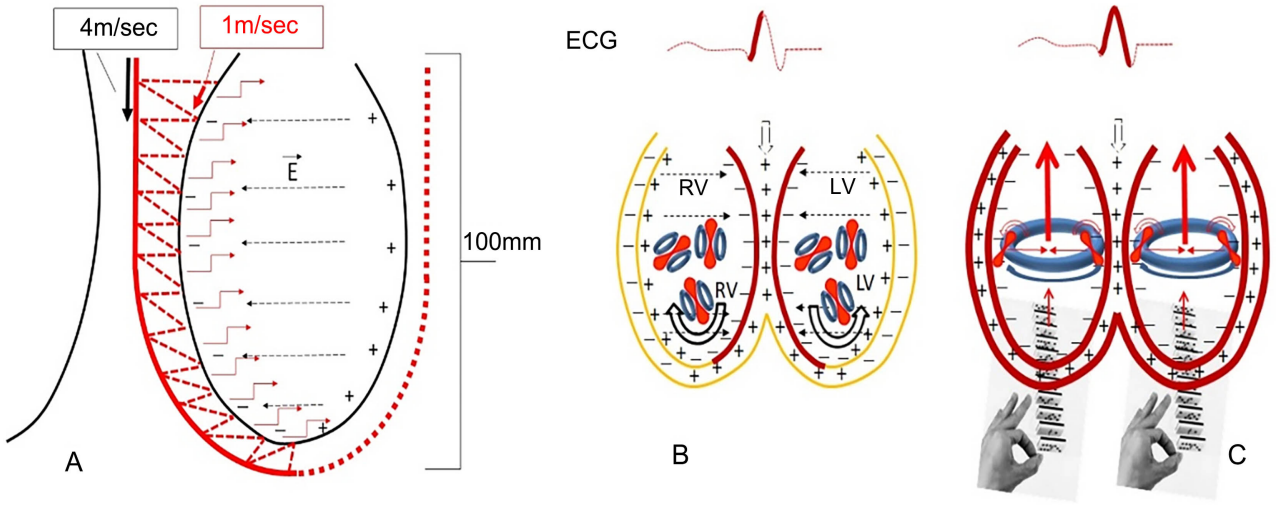

Figure 3. A. Cardiomyocytes initially depolarize in the intraventricular septum. The oscillating electric field between the septum and free walls of ventricles forms the dielectric polarization of the intraventricular substance with the displacement current. With the septal depolarization, an amplitude-modulated wave of depolarization—QRS of the ECG is formed. An evoked signal is directed to all body cells. Cardiomyocyte depolarization time 0.002 sec-frequency 500 Hz. The left bundle branch ramifies into the Purkinje network. Bundle branches and Purkinje fibers have high conduction signal velocity up to 4 m/sec. The electric surface charge will be sharply increased by the cardiomyocyte group by the synchronous depolarization increasing the depolarization frequency ≫ 500 Hz. In addition to this, electroacoustic waves from the RBC (1.2 MHz), propagate in the blood. B. Ac electric field on the RBC promotes the ultrasound oscillation of the cell membrane and electroacoustic phenomena. Rotation of the charges in the area of the Z-potential around the RBC forms repulsing electromagnetic forces (blue circle—direction of the magnetic field), which are compensated by the ventricular wall tension. C. Repulsing electromagnetic forces, acting on the RBC perform the extra work needed to align red cells into the plane of flow as the flow begins or restarts and forms transversal pressure waves. With this blood platelets and leukocytes are displaced/segregated to the near-wall region and are "marginated". At the end of the QRS electric oscillation, initially contracted the apical heart muscles. It destroys the delicate balance between the electromagnetic repulse force and wall tension and facilitates propelling the blood out of the chambers in the large arteries, as the domino effect. Process in the waveform is distributed in the arteries.

In the venous blood, electromagnetic forces contribute to the mutual attraction of the RBCs due to the deoxygenated (paramagnetic) hemoglobin, increasing blood elasticity. With this, zeta potential is the critical control mechanism and offers the stability of colloidal dispersions among the blood components. The venous blood is elastic (Womersley number in inferior vena cava-8.8), while the distance between RBC is determined by the surface negative charge.

In systemic venous circulation, the blood flow direction is provided by the venous valves. Flow is formed by the capillary venous pressure, contraction of the muscle, and negative pressure at the breath. Here elasticity is a necessary factor for distal transmitting of the proximally formed attractive force (at the rest only breath is active). Blood coagulation system and venous compliance also are promoted to this: In venous stasis thrombi are high elastic mass with the fibrin rich, encapsulating a large number of red blood cells in addition to activated platelets.

In pulmonary circulation, venous blood is mixed with the oxygen-rich pulmonary lymph. With these, electromagnetic forces from the ECG simplify the uptake of the oxygen by the deoxygenated paramagnetic hemoglobin in RBC, because oxygen is paramagnetic too. Elastic venous blood is flowing in the elastic pulmonary artery by the right ventricle—a low-pressure elevator. Blood inertial and elasticity in the pulmonary artery is the highest in the circulatory system (Womersley number—15). That is why the blood acceleration, the velocity of the blood flow, and the length of the pulmonary circulation are low.

To reduce the oxygen loss from the diamagnetic oxygenated hemoglobin in the pulmonary venous (as in systemic arterial circulation, rotational heart dipole influences this), blood flows over a short distance. Herewith, electromagnetic force facilitates the RBC repulsion and at the pulmonary venous compliance, helps to the filling of the left atrium.

In the arterial blood (Womersley number in ascending aorta—13.2) electromagnetic forces on the oxygenated (diamagnetic) hemoglobin facilitate mutual repulsion of RBC and freeling of oxygen in plasma. In addition to this, surface wave frequency dispersion in protodiastole, destroys the cell aggregates and increases the entropy of the system up to arterioles, providing to proceed spontaneous biochemical processes in capillaries (Womersley number—0.005). Increasing resistance of the flow at the arterial turbulence and resistance paradox in arterioles is associated with additional energy costs for the viscoelastic transformation. *I.e.* magnetic field can be modifying the inertial properties of the blood, due to the magnetic features of the hemoglobin (attractive-paramagnetic and repulsive-diamagnetic). Herewith elasticity of the substance is controlled with the magnetism.

Coagulation highly affects the substance's elasticity [20]. The mechanism of blood coagulation involves activation, adhesion, and aggregation of platelets, as well as deposition and maturation of fibrin. As it seems from our experimental study, if there is no electromagnetic impact, in the catheter thrombus are formed after 10 - 15 min. When the frequency of electromagnetic oscillation is 500 - 5000 Hz, hypocoagulation is revealed. Herewith the electromagnetic oscillation destroys the formed thrombus. There can be different causes for the hypocoagulation: Coagulation is a fermentative process—based on the redox transformations of compounds through the action of enzymes. By the external ac electric field, fermentation environments are controlled (electro-fermentation). Thrombolysis may be a consequence of the violation in the redox transformations of the compounds by electro-fermentation.

Herewith as the erythrocyte is a toroidal electromagnetic field-driven cell, electromagnetic interaction (500 - 5000 Hz) increases erythrocyte Z potential, decreases PLT/PI/FIB, and can facilitate the stability of the colloid system.

Besides this, time-varying electric fields can induce electroporation of the plasma membrane, modulate the conformational states of membrane proteins, and have been demonstrated extensively for pumps in the outer plasma membrane [21]. To this process, as it seems, most of all the platelets are timed. Their number is significantly decreasing. So, electromagnetic field impacts primary and secondary coagulation. The structure and function of the RBC and leukocytes were not affected.

It should also be noted the presence of ultrasonic cavitation-formation, growth, and subsequent collapse of the bubbles in water by using ultrasonic irradiation with the releasing high energy into the liquid.

It is known that altered flow conditions (stasis, turbulence) affect blood coagulation. In the turbulence flow (shear rate 5000 sec^{-1}), the rotational and elongational flow components of motion are superimposed, parcel resident time increases and affects from different directions, and coiled Von Willebrand factor (VWF) is unfolding [22].

Herewith the mathematical dimension for the shear rate (sec^{-1}) is the same as for the electromagnetic wave oscillation frequency. It expresses the rotational character of the motion of the measured component, (surface wave rotation in turbulence flow and/or the dipole rotation).

In our study, ac electric field in the blood for the dielectrophoresis is made by the external oscillating magnetic field. With the dielectrophoretic activity, the shear force must activate the VWF at 5000 Hz. While it can be in coiled form at 500 Hz. It seems, that the ac electric field with the conjunction of the RBC electroacoustics, changes the coagulation.

Blood after the electromagnetic irradiation expresses the dates of the disseminated intravascular coa-

gulation (DIC). It is involving the abnormal, excessive generation of thrombin and fibrin in the circulating blood. During the process, increased platelet aggregation and coagulation factor consumption occur. DIC is specified by demonstrating thrombocytopenia, an elevated partial thromboplastin time and prothrombin time, and decreasing plasma fibrinogen levels.

Although electromagnetic influence is carried out on the arterial and venous blood, structural changes with the increasing entropy are specified only for the pulsatile blood. It must be associated with the frequency dispersion of the diamagnetic blood at the boundary reflection. ECG-T wave ac potential facilitates to this. Activation of the coagulative system at the low shear rate in the vena decreases the system entropy.

By the external ac field, all of the substances and body cells can be potentially polarized, while the process of the charge displacement/current flow is expressed as the system impedance. Materials or systems exhibiting multiple phases, such as the human body, commonly show a universal dielectric response. The characteristic dielectrophoretic frequencies of oscillation and relaxation motions of the cell envelopes and as a whole be within the range of 10^3 - 10^6 Hz [21]. The theoretical analysis of natural oscillations of the bilayer lipid membrane, considered a thin film of a viscous fluid, estimates the lower frequency limit to be ~100 Hz for the oscillations of the bilayer thickness and ~5×10^3 Hz for the bending oscillations of the bilayer at an invariable thickness [23]. Cells' metabolic and cell-to-cell long-range electromagnetic signaling activities are accompanied by the generation of endogenous electromagnetic fields. Dynamic variations in the length of cytoskeleton microtubules, attached to membrane microdomains were identified as a driving force of cell membrane mechanical vibrations [24].

The selective influence on the blood structures under the electromagnetic influence indicates the predispositions to the specific information. Passing the different electric signals, the studied chemical substance and cell structures may appear as filters, with energy storage and dissipation properties. Herewith energy storage (capacitance) is associated with the reactance whose value changes with respect to the applied frequency. In the electric circuit, the type of the filter is determined depending on which way around it connects the resistor and the capacitor with regards to the output signal. Resulting in a Low/High Pass Filter (**Figure 4**). In cases when the input signal is a "square wave" shaped (almost vertical step input as in the cardiomyocyte depolarization curve), the output response of the filter produces another type of the output signal (a circuit of the integrator or differentiator). In the body cell, they can selectively activate different biological processes.

Electromagnetic fields are complex phenomena, which transport energy and information across space. Information can be imposed into electromagnetic waves through various forms of modulation.

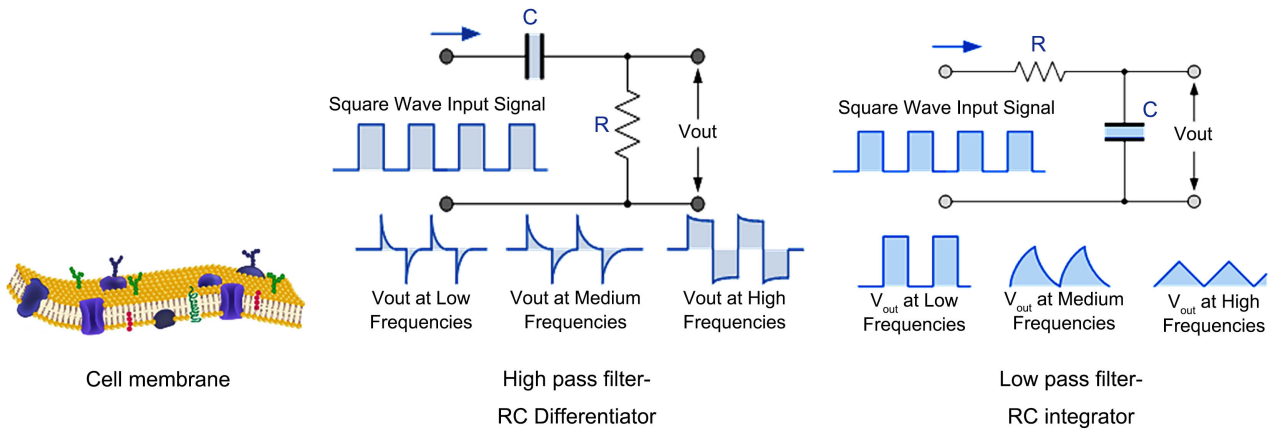

Figure 4. The cell membrane looks like a capacitor. The function of energy storage and reactance can be found in amino acids, proteins, and saline (our experiments, were not shown in the tables). The difference will be expressed in the outcome signal according to the applied frequency.

Magnetic fields appear through the relativistic motion of electric fields, which is why electricity and magnetism are so closely linked [25].

Chemical reactions can be induced at a distance due to the propagation of electromagnetic signals during intermediate chemical stages. Although it is well known at optical frequencies, e.g. photosynthetic reactions, electromagnetic signals, as the energy/information carrier, true for much lower frequencies [26].

The point of view that the living system consists of energy and information is commonly accepted. The metabolism of the organism is in essence a cost of information processing. Information handling is considered the most essential feature of the living system. Energy and information components are acting upon and within the living system on the level of microphysical events through their additive values of negative and positive entropy. Consideration of the examples of the function of a biological membrane and its models, calculations of experimental equivalents of information negentropy, and lowering the energy cost of biological reactions during onto—and phylogenesis lead us to the conclusion that lowering the energy cost of biological reactions decisive in the emergence of the living system and its biological evolution [27].

While the many questions stay open for discussion, the next advance in medicine will be to discern and apply electromagnetic signaling parameters acting to promote wellness of the normal biological processes, with the decreasing reliance on the pharmaceuticals. Investigation of the information basis in pathophysiological processes must be performed by a group of scientists with the adapted laboratory. The author is open to cooperation.

4. CONCLUSIONS

Ac electric field from the myocardial depolarization initiates electroacoustic phenomena. An emerging repulsing electromagnetic force acts on the RBC and in addition to the pulse pressure from the heart, promotes blood motion and viscoelastic changes.

The alterations of the blood inertial and elasticity, in addition to hemodynamics, are facilitated by the magnetic features of the hemoglobin.

The external electromagnetic signal can manage the blood coagulation process, including thrombolysis.

CONFLICTS OF INTEREST

The authors declare no conflicts of interest regarding the publication of this paper.

REFERENCES

1. Hall, J.E. (2011) Guyton and Hall. Textbook of Medical Physiology. 12th Edition, Elsevier, Amsterdam.
2. Tanenberg, R.J. and Donofri, P.D. (2008) Hemorheology: Principles and Concepts. In: Levin, M.E., *et al.*, Eds., *Levin and O'Neals's Diabetic Foot*, Seventh Edition, Mosby/Elsevier, Philadelphia, p. 627.
3. Prado, G., Farutin, A., Misbah, C. and Bereau, L. (2015) Viscoelastic Transient of confined Red Blood Cells. *Biophysical Journal*, **108**, 2126-2136. https://doi.org/10.1016/j.bpj.2015.03.046
4. Mader, E., Skinner, S., Romana, M., Fort, R., *et al.* (2019) Blood Rheology: Key Parameters, Impact on Blood Flow, Role in Sickle Cell Disease and Effects of Exercise. *Frontiers in Physiology*, **10**, Article No. 1329.
5. Weissel, J.W. and Litvinov, R.I. (2017) Fibrin Formation, Structure and Properties. *Subcellular Biochemistry*, **82**, 405-456. https://doi.org/10.1007/978-3-319-49674-0_13
6. Garmo, C., Bajwa, T. and Burns, B. (2021) Physiology, Clotting Mechanism. StatPearls, Treasure Island.
7. Berg, J.C. (2010) Introduction to Interfaces and Colloids, an: The Bridge to Nanoscience. World Scientific Publishing, Singapore.
8. Prandoni, P. (2009) Venous and Arterial Thrombosis: Two Aspects of the Same Disease? *Clinical Epidemiology*,

1, 1-6. https://doi.org/10.2147/CLEP.S4780

9. Palta, S., Saroa, R. and Palta, A. (2014) Overview of the Coagulation System. *Indian Journal of Anaesthesia*, **58**, 515-523. https://doi.org/10.4103/0019-5049.144643
10. Furie, B. and Furie, B.C. (2005) Thrombus Formation *in Vivo*. *The Journal of Clinical Investigation*, **115**, 3355-3362. https://doi.org/10.1172/JCI26987
11. Chapin, J.C. and Hajjar, K.A. (2015) Fibrinolysis and the Control of Blood Coagulation. *Blood Reviews*, **29**, 17-24. https://doi.org/10.1016/j.blre.2014.09.003
12. Kazimierska, E. (2001) The Effect of Electromagnetic Fields on Blood Coagulation and Fibrinolisys in Humans. *Polski Merkuriusz Lekarski*, **10**, 9-11.
13. Gprczynska, E. and Wegrzynowicz, R. (1983) The Effect of Magnetic Fields on Platelets, Blood Coagulation and Fibrinolysis in Guinea Pigs. *Physiological Chemistry and Physics and Medical NMR*, **15**, 459-468.
14. Higashi, T., *et al*. (1993) Orientation of Erythrocytes in a Strong Static Magnetic Field. *Blood*, **82**, 1328-1334. https://doi.org/10.1182/blood.V82.4.1328.bloodjournal8241328
15. Abdalla, S. (2011) Effect of Erythrocytes Oscillations on Dielectric Properties of Human Diabetic Blood. *AIP Advances*, **1**, Article ID: 012104. https://doi.org/10.1063/1.3556986
16. Beraia, G. and Beraia, M. (2017) Frequency Dispersion of the Surface Wave on the Vessel Wall—Primary Reason of Atherosclerosis. *Journal of Integrative Cardiology*, **3**, 1-7. https://doi.org/10.15761/JIC.1000212
17. Halliday, D., Resnick, R. and Walker, J. (2011) Fundamentals of Physics. John Wiley & Sons, Hoboken.
18. Purnell, M.C. and Ramsey, R.D. (2019) The Influence of the Golden Ratio on the Erythrocyte. In: Tombak, A., Ed., *Erythrocyte*, IntechOpen, London, 1-13.
19. Mayda, S., Kandemir, Z., Bulut, N. and Maekawa, S. (2020) Magnetic Mechanism for the Biological Functioning of Hemoglobin. *Scientific Reports*, **10**, Article No. 8569. https://doi.org/10.1038/s41598-020-64364-y
20. Kou, J.H., Li, Y.H., Yang, P.Y. and Zhang, J.T. (2017) A Novel Blood Coagulation Measuring Method Based on the Viscoelasticity of Non-Newtonian Fluid. *World Journal of Engineering and Technology*, **5**, 134-151. https://doi.org/10.4236/wjet.2017.54B015
21. Vajrala, V., Claycomb, J.R., Sanabria, H. and Miller, J.H. (2015) Effects of the Oscillatory Electric Fields on Internal Membranes: An Analytical Model. *Biophysical Journal*, **94**, 2043-2052.
22. Rana, A., Westein, E., Niego, B. and Hagemeyer, C.E. (2019) Shear-Dependent Platelet Aggregation: Mechanisms and Therapeutic Opportunities. *Frontiers in Cardiovascular Medicine*, **6**, Article No. 141. https://doi.org/10.3389/fcvm.2019.00141
23. Kononenko, V.L. and Shimkus, J.K. (2000) Spontaneous and Forced Oscillations of Cell Membrane of Normal Human Erythrocytes: Absence of Resonance Frequencies in a Range of 0.03 - 500 Hz. *Membrane and Cell Biology*, **14**, 363-382.
24. Axelrod, A. (2017) Cells Membrane Vibrations as a Mechanism of Endogenous ELF Magnetic Field Generation in Biosystems.
25. Yeap, K.H. and Hirasawa, K. (2019) Electromagnetic Fields and Waves. IntechOpen, London.
26. Widom, A., Swain, J., Srivastava, Y.N. and Sivasubramanian, S. (2012) Electromagnetic Signals from Bacteria DNA.
27. Pol, A.T. (1990) Life: Energy-Information Relationship within Material Systems. General Outline of the Concept. *Computers & Mathematics with Applications*, **20**, 269-285. https://doi.org/10.1016/0898-1221(90)90333-F