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Korteveg-de-Vries Soliton Equation in Pulse Wave Modelling

S. V. Vasylyuk, D. V. Zaitsev, and A. V. Brytan

Abstract In the early stages of blood flow research, the widely used Bernoulli equation was applied to describe blood behavior in large vessels, while the Poiseuille formula was used to estimate capillary flow behavior within a single vessel, accounting for the significant resistance of blood. These expressions were derived assuming a steady laminar incompressible flow, with fluid particles moving along constant streamlines and the velocity profile in the radial direction u(r) remaining constant in time and not changing in the axial flow direction at a given point [1]. However, such models fail to capture the emergence and movement of arterial pulse waves, which are a critical and versatile physiological phenomenon that have garnered significant interest for investigation and modeling. In this section, we will explore the use of soliton theory for modeling pulse waves. The underlying concept is that the behavior of a real pulse wave shares many similarities with solitons. In the realm of mathematics, solitons are defined as localized stationary solutions to nonlinear partial differential equations or their generalizations, such as differential-difference or integro-differential equations. Notably, various physical situations and phenomena can be described by the same equations, including the Korteweg-de Vries equation, the Sine-Gordon equation, and the Schrödinger nonlinear equation. Specifically, the elastic interaction between solitons and local perturbations plays a crucial role in this context. The method of pulse waves modeling based on soliton solution of Korteweg-de Vries equation is considered in this work, and test calculations in Maple 8 environment are made. The results obtained allow us to speak about the

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applicability of soliton theory for pulse wave modeling. It is planned to analyze the possibility of further application of N-soliton solution for medical purposes.

1 Introduction

Historically the beginning of the investigation of blood flow takes off the implementation famous Bernoulli equation for describing blood behavior in large vessels. Also for initial estimating of capillary flow behavior with significant blood resistance inside single vessel Poiseuille formula is used. Both expressions are obtained for a case of the velocity profile calculating for a steady laminar incompressible flow, by solving the force balance applied to the fluid. In this assumption, the fluid particles move along constant streamlines while the velocity profile in the radial direction $u(r)$ does not change in the axial flow direction and for a certain point it is constant in time [1]. However, such types of models cannot allow to describe the emergence and subsequent movement of arterial pulse waves. By the way, the importance and versatility of this physiological phenomenon cause the significant interest in its investigation and modeling [2].

From the physical point of view, blood flow is a rather complex process; many researchers even call it the third mode of flow. Difficulties in modeling this process are caused by many factors: both purely "physical" (e.g., the fact that blood itself is non-Newtonian fluid, despite it flows through blood vessels) and the need to take into account various regulatory functions. For this reason, most models based on the theory of mathematical hemodynamics are quite complicated and difficult to apply.

There is a huge amount of mathematical models for behavior modeling of arterial pulse wave. Actually, this set can be divided into two branches. First is applied to the initiation and propagation of pulse wave in aorta and cardiovascular system in general. One of the simplest models in this area is the Windkessel model [3] which gives the expression for pressure in aorta during cardiac cycle as a function of cardiac stroke time dependence form. This is also known as two element's model or analog model. According to the form of mathematical apparatus used in this model, it is possible to draw analogy between blood and electric circuit. This idea became a basis of consideration of cardiovascular system as analog of electric circuit. One of the classic models is the model proposed by Westerhof [4]. Westerhof has interpreted the pressure and flow pulses as complex waves consisting of a forward traveling and a backward traveling component. The forward wave is associated with the ejection of blood from the heart and the backward wave is associated with physical reflections caused by the mechanical discontinuities in the arterial tree. In the diastolic phase, both waves are considered to have destructive superposition, whereas in the systolic phase, both waves are considered to have constructive superposition. The constructive superposition can also explain the increase of peak pressure of the waveform during its propagation in some arteries which is referred to as steepening. Such representation of cardiovascular system further also was developed into approach of lamped [5, 6] and distributed [7] parameter system One of the main goals of such

modeling is obtaining a realistic and detailed pulse wave profile during cardiac cycle in aorta and explain its further propagation. The use of this approach makes it possible to simplify the consideration of such an effect as the expansion of the vessel lumen during the movement of a pulse wave.

But, if we consider the blood flow and wave propagation in a separate vessel, the application of the hydrodynamic equation becomes a more convenient and rigorous approach [8]. Simultaneously for completing this system of equations, it is necessary to add the elasticity equation for tube wall motion under varying internal pressure. The vessel walls are often assumed to be Hookean material, namely linear relation between stress and strain. However, taking into account the interaction of blood flow with a deformable vessel wall significantly complicates the distribution of this problem, which forces us to use various model representations [9].

This part considers the approach of modeling pulse waves using soliton theory. The basic idea is that the nature of a real pulse wave is very similar to the properties of solitons. The central role is played by the properties of the elastic interaction between solitons and solitons with local perturbations [10–12].

2 Properties of Solitons. The Possibility of Using KdV Equation to Describe the Pulse Wave

Soliton is a localized stationary or stationary on average perturbation of a homogeneous or spatially periodic nonlinear medium [10]. Up to the beginning of the 1960s, soliton was called a soliton wave—a wave packet of a constant shape, propagating with a steady speed over the surface of a heavy liquid of a finite depth and in plasma. Nowadays, many different physical objects fall underneath the definition of soliton. The first classification of soliton can be made according to the number of spatial dimensions, along which the stationary perturbation of a nonlinear medium is localized. The one-dimensional soliton includes classical soliton waves in liquids, domain walls in ferro- and antiferromagnetics, 2p-pulses and envelope solitons in nonlinear optics [13–15].

In mathematical terms, solitons are localized stationary solutions of nonlinear partial differential equations or their generalizations (differential-difference, integro-differential, etc., equations). In many cases different physical situations and phenomena are described by the same equations, e.g., the Korteweg-de Vries equation, the Sine–Gordon equation, the Schrödinger nonlinear equation, the Kadomtsev–Petviashvili equation [10–14]. Linear equations (except the one-dimensional wave equation) have no localized stationary solutions. Solitons are essentially nonlinear objects whose behavior and properties are fundamentally different from the behavior of wave packets of small amplitude. The difference is especially strong if the soliton has a topological charge; i.e., if the configuration of the wave field in the presence of the soliton is topologically different from the configuration of the unperturbed state. So, a number of equations having soliton solutions belong to the class of equations

where the inverse scattering problem is applicable and most of them are integrable Hamiltonian systems [15–18].

In [10], the process of fluid flow through an elastic thin-walled tube is considered. The nonlinear differential equations of these processes are derived; the modeling of a solitary (pulse wave) is performed on the basis of the Korteweg-de Vries models and the modified nonlinear Schrödinger equation. The main advantage of these models is that the pulse wave propagation process is considered quite deeply when they are derived. These models describe only some of the phenomena observed in the cardiovascular system due to the complexity of the blood flow process.

Here we consider the pulse wave without taking into account the processes of blood flow regulation; we consider the pulse wave as the propagation of a solitary (pulse) wave in an elastic thin-walled tube. Based on this, we chose the Korteweg-de Vries equation as a model:

$$u_t + 6uu_x + u_{xxx} = 0. \tag{1}$$

The dimensional equation KdF for the velocity $u(t, x)$ for perturbation in a channel with a rectangular cross section has the form.

Equation (1), as one of the partial solutions, has the so-called soliton solution of the form:

$$u(t, x) = \frac{A}{ch^2[b(x - vt)]}, \tag{2}$$

in which A, b and v are parameters determined by the direct substitution (2) in (1). In this case, A and v have the dimension of velocity (v is the speed of the soliton wave), and b has the dimension of length.

In Eq. (1), h is the dimensional depth of the channel. As for the speed u_0, which is included in (1), there are two versions of its physical content.

This is the speed of its own flow in the channel, which is considered a given value. In the case of the cardiovascular system of its own speed, independent of the system itself, no, so this content is not suitable for speed u_0.

This is the speed that is a consequence of the desired speed $u(t, x)$ and can be interpreted as the speed of capture (speed of entrainment). It must be defined together with the solution $u(t, x)$ and in this sense is a certain analog of eigenvalue. This case will be considered as one that forms the blood flow.

We will simulate the pulse wave with a soliton solution of the KdF equation. Since no one has deduced the KdF equation for a channel with a round cross section, we will use a simple analogy, as a result of which we can postulate the equation:

$$u_t + uu_x + u_0 S u_{xxx} = 0, \tag{3}$$

in which S is the cross-sectional area of the aorta in the immediate vicinity of the heart (hereinafter we can take into account that SS can be a function of u due to

vascular elasticity, and velocity u_0 can be a function of x in the transition from aorta to arteries, capillaries, etc.).

For solution (2), we consistently find:

$$u_t = 2Abv\frac{sh[b(x-vt)]}{ch^3[b(x-vt)]};$$

$$uu_x = -2A^2b\frac{sh[b(x-vt)]}{ch^5[b(x-vt)]};$$

$$u_{xxx} = -8Ab^3\frac{sh[b(x-vt)]}{ch^3[b(x-vt)]} + 24Ab^3\frac{sh[b(x-vt)]}{ch^5[b(x-vt)]}.$$

Substituting the right-hand sides of these three relations in (3), we find the equation:

$$\left\{2Abv - 8Ab^3u_0S\right\}\frac{sh[b(x-vt)]}{ch^3[b(x-vt)]} + \left\{24Ab^3u_0S - 2A^2b\right\}\frac{sh[b(x-vt)]}{ch^5[b(x-vt)]} = 0,$$

which can be satisfied by equating to zero both curly braces. As a result, we obtain two ratios:

$$v - 4b^2u_0S = 0; \tag{4}$$

$$12b^2u_0S - A = 0, \tag{5}$$

which includes four unknown quantities: A, b, v, and u_0. Equation (4) makes it possible to immediately find the blood flow velocity u_0, which is stimulated by the soliton velocity v:

$$u_0 = \frac{v}{4Sb^2}. \tag{6}$$

Substituting this in Eq. (5), we can find the amplitude factor A:

$$A = 3v. \tag{7}$$

Now only two values remain undefined: the pulse rate of the soliton v and the parameter b, which determines the width of the soliton.

As for the parameter b, it is quite obvious from Eq. (6) that in the general case it can be sought in the form:

$$b = \frac{\beta}{\sqrt{S}}, \tag{8}$$

where β is a dimensionless indefinite parameter that can be interpreted as a dimensionless representation of factor b. Taking into account (8), the relation (6) takes the form:

$$u_0 = \frac{v}{4\beta^2}. \tag{9}$$

Equation (9) shows that the parameter β satisfies the condition $\beta > \frac{1}{2}$, since the blood flow velocity u_0 is always less than the pulse velocity of the soliton v.

Solution (2), taking into account (7) and (8) takes the form:

$$u(t, x) = \frac{3v}{ch^2\left(\beta \frac{x-vt}{\sqrt{S}}\right)}. \tag{10}$$

We will determine the two remaining parameters (β and v) based on known physiological facts.

In particular, it is known that the pulse velocity of the soliton v in the aorta (at the exit of the heart) is 5-8 m/s [1-4] (while the blood flow velocity u_0 in it is 0.5-1 m/s [1-4] (I found the figure in [1-4]). That is, the pulse rate v at the exit of the heart is 10/6 times greater than the current velocity u_0. For certainty, we will use the value $(v/u_0) = 16$, which corresponds to the pulse rate of the soliton $v = 8$ m/s.

$$\beta = \frac{1}{2}\sqrt{\frac{v}{u_0}},$$

Therefore, using the formula, obtained from (9), we can find: $\beta = 2$.

The last thing left to do is check the results, knowing the normal heart rate—1 beat per second. This means that between two consecutive pulse waves in the form of a soliton (between two heartbeats) the period should be: $T = 1$ s. The test comes down to the fact that with such a pulse, the solitons should not intersect with great accuracy (then the condition of separation (solitude) of the soliton wave is met). To perform this test, use the solution (10).

Before we put this in (10) $x = 0$, on the grounds that the point of exit of the aorta from the heart will be considered the origin. Then from (10) we obtain a special (not arbitrary) soliton-like boundary condition:

$$u(t, 0) = \frac{3v}{ch^2\left(\frac{\beta vt}{\sqrt{S}}\right)}, \tag{11}$$

which in this consideration is considered to form a physiologically healthy heart rhythm, and any deviations from this limit condition will lead to arrhythmias (more on this later). If we put $t = T$ in the argument of this boundary condition, then this argument takes the form:

$$\frac{\beta v T}{\sqrt{S}}. \tag{12}$$

The only value in this argument that has not yet been discussed is the cross section S of the artery directly at the exit of the heart (at the point $x = 0$). For it, we use the value $S \sim 4 \times 10^{-4}$ m^2. Then the value of argument (12) can be estimated by substituting the numerical values of all quantities: $\beta = 2$, $v = 8$ m/s, $T = 1$ s, $S = 4$. As a result, we will have

$$\frac{\beta v T}{\sqrt{S}} = 800.$$

If we use the analytical or graphical representation of the hyperbolic cosine, it is obvious that as the value of argument (12) increases, the function $ch\ (x)$ increases in proportion to e^x. That is, for the value of $ch\ (800) \to \infty$, or $1/(ch^2(800) \to 0)$ obtained here. This means that the solution (10) in the form of a sequence of one-soliton pulse waves at the obtained parameters is physically correct, because with a good margin provides separation of each soliton, as well as blood flow velocity $u_0 = 0.5$ m/s at the beginning of the aorta.

If the boundary condition (11) is violated, i.e., it ceases to be soliton-like, then other solutions of Eq. (3) are realized: multi-soliton solutions with different pulse velocities v, or solutions in the form of cnoidal waves. The latter, however, also require a special boundary condition.

3 Soliton Solution of the Korteweg-de Vries Equation by Perturbation Method

The Korteweg-de Vries equation is considered:

$$u_t + 6uu_x + u_{xxx} = 0 \tag{13}$$

In [10, 11], the method of forming the N-soliton solution of this equation is presented. The general solution of the Korteweg-de Vries equation consists of a soliton and a non-soliton part. In our case, we consider a solution in which the local perturbations (non-soliton part) are negligibly small. That is, we are building a somewhat idealized model that does not take into account small local perturbations. Hirota [11] showed that in general the N-soliton solution has the form:

$$u = 2\frac{\mathrm{d}^2}{\mathrm{d}x^2} \ln F, \tag{14}$$

where F is the determinant of some matrix [11].

Substituting (14) into (13), once integrating and assuming the integration constant equal to zero, we obtain:

$$F_{xt}F - F_xF_t + F_{xxxx}F - 4F_{xxx}F_x + 3F_{xx}^2 = 0. \tag{15}$$

For further analysis, it is convenient to enter the operator:

$$D_x^m D_t^n ab = (\partial_x - \partial_{x'})^m (\partial_x - \partial_{x'})^n a(x,t)b\big(x',t'\big)\Big|_{\substack{x'=x\\ t'=t}}. \tag{16}$$

So, Eq. (15) can be rewritten as:

$$\big(D_xD_t + D_x^4\big)F \cdot F = 0. \tag{17}$$

Next, suppose that the function F could be represented as a formal series:

$$F = 1 + \varepsilon f^{(1)} + \varepsilon^2 f^{(2)} + \cdots, \tag{18}$$

where

$$f^{(1)} = \sum_{i=1}^{N} e^{\eta_i}, \quad \eta_i = k_i x - \omega_i t + \eta_i^0, \tag{19}$$

where $k_i, \omega_i, \eta_i^{(0)}$—are constants.

In the case of the Korteweg-de Vries equation, this formal series ends. Indeed, substituted (18) into (17), we found

$$\big(D_xD_t + D_x^4\big)\big(1 + \varepsilon f^{(1)} + \varepsilon^2 f^2 + \cdots\big)\big(1 + \varepsilon f^{(1)} + \varepsilon^2 f^2 + \cdots\big) = 0$$

And equated to zero, the coefficients for each degree of ε, we obtained

$$O(1)\colon 0 = 0 \tag{20a}$$

$$O(\varepsilon)\colon 2\big(\partial_x\partial_t + \partial_x^4\big)f^{(1)} = 0 \tag{20b}$$

$$O\big(\varepsilon^2\big)\colon 2\big(\partial_x\partial_t + \partial_x^4\big)f^{(2)} = -\big(D_xD_t + D_x^4\big)f^{(1)}f^{(1)} \tag{20c}$$

$$O\big(\varepsilon^3\big)\colon 2\big(\partial_x\partial_t + \partial_x^4\big)f^{(3)} = -2\big(D_xD_t + D_x^4\big)f^{(1)}f^{(2)}. \tag{20d}$$

Equation (20b) is a homogeneous equation. As a solution of this equation, we took Eq. (19). If we try to continue the calculations of the next parts of the series, starting with solution Eq. (7) for an arbitrary random N, we could encounter the

analytical difficulties. More often, we can obtain equations solutions for $N = 1, 2$, and then hypothesize the structure of the solution for an arbitrary random N and prove it by induction method. For $N = 1 f^{(1)} = e^{\eta_1}$. Then it follows from Eq. (8b) that $\omega_1 = -k_1^3$. We could obtain $f^{(2)}$ from the relation (8c), which reduces to $\left(\partial_x \partial_t + \partial_x^4\right) f^{(2)} = 0$.

So $f^2 = 0$. and the series sequence breaks off. Therefore, for $N = 1$ we have:

$$F_1 = 1 + e^{\eta_1}, \quad \omega_1 = -k_1^3, \quad u = \frac{k_1^2}{2} \operatorname{sech}^2 \frac{1}{2}\left(k_1 x - k_1^3 t + \eta_1^{(0)}\right)$$

For $N = 2$, we take Eq. (20b) as a solution

$$f^{(1)} = e^{\eta_1} + e^{\eta_2}, \quad \eta_i = k_i x - k_i^3 t + \eta_i^{(0)}$$

Then (20c) reduces to the equation:

$$2\left(\partial_x \partial_t + \partial_x^4\right) f^{(2)} = -2\left((k_1 - k_2)(-\omega_1 + \omega_2) + (k_1 - k_2)^4\right) e^{\eta_1 + \eta_2}$$

That is have a solution [3]

$$f^{(2)} = e^{\eta_1 + \eta_2 + A_{12}} e^{A_{ij}} = \left(\frac{k_i - k_j}{k_i + k_j}\right)^2 \tag{21}$$

(note that $k_1 \neq k_2$). Substituting $f^{(1)}$, $f^{(2)}$ in (20d), we make sure that the right-hand side of (8d) is zero, so let's take it $f^{(3)} = 0$. Thus, for $N = 2$

$$F_2 = 1 + e^{\eta_1} + e^{\eta_2} + e^{\eta_1 + \eta_2 + A_{12}}$$

The function $u = 2d^2(\ln F_2)/\mathrm{d}x^2$ corresponds to the two-soliton solution of the Korteweg-de Vries equation. Performing similar calculations for $N = 3$, we obtain:

$$F_3 = 1 + e^{\eta_1} + e^{\eta_2} + e^{\eta_1 + \eta_2 + A_{12}} + e^{\eta_1 + \eta_2 + A_{13}} + e^{\eta_2 + \eta_3 + A_{23}} + e^{\eta_1 + \eta_2 + \eta_3 + A_{12} + A_{13} + A_{23}} \tag{22}$$

where the coefficients A_{ij} are determined by Formula (21) (Function u dependence of the coordinate is obtained on the basis of Eqs. (14) and (22) is shown on Fig. 1).

Based on the above, it is hypothesized that the structure of the general N-soliton solution has the form [3]:

$$F_N = \sum_{\underline{\mu}=0,1} \exp\left(\sum_{i=1}^{N} \mu_i \eta_i + \sum_{1 \leq i < j}^{N} \mu_i \mu_j A_{ij}\right),$$

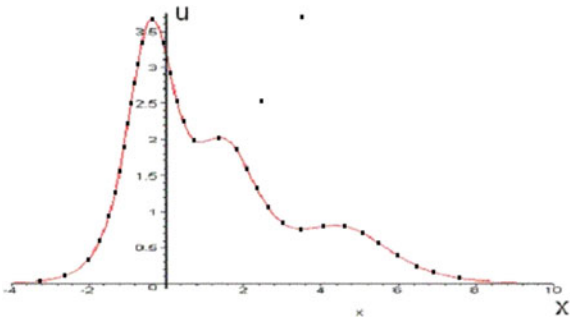

Fig. 1 Function u dependence of the coordinate is obtained on the basis of Eqs. (14) and (22)

where the sum of $\underline{\mu}$ runs on all sets μ_i, $i = \overline{1, \ldots, N}$. Note that μ_i, $i = \overline{1, \ldots, N}$—they are associated with the phase shift of solitons during scattering.

4 Application of the Theory of Solitons to Detailed Modeling of a Pulse Wave

It is assumed that the pulse wave is a set of pulses interacting with each other in time. Since a soliton is a solitary wave that elastically interacts with arbitrary local perturbations, it makes sense to consider the system of interacting solitons as a model. That is, we put the correspondence between: 1 pulse—for soliton. So, the N-soliton solutions of the Korteweg-de Vries equation are used as the analytical form of soliton waves [10].

$$u = 2\frac{\mathrm{d}^2}{\mathrm{d}x^2} \ln F_N$$

The potential u is a complex function represented as a combination of exponential functions with base e. The obtained solution u includes $3N$ parameters, through which variables $\eta_i = k_i x + \omega_i t - h_i^{(0)}$ are determined, where k_i, ω_i, $\eta_i^{(0)}$ are the parameters of this system.

The following properties of solitons were used:

(1) The amplitude of the ith soliton, which does not closely interact with other solitons, is directly proportional to the corresponding parameter k_i, namely

$$u_{\max i} = \frac{1}{2}k_i^2.$$

(2) The argument of the point of maximum of the ith soliton is determined by the following expression:

$$x_{\max i} = \frac{-\omega_i t + h_i^{(0)}}{k_i}.$$

(3) The velocity in phase c is defined as the ratio of the coefficients at $\boldsymbol{x}$ and $\boldsymbol{t}$. For the ith soliton, it is equal.

$$c_i = {}^{\omega_i}\!/_{k_i}$$

For this system, all phase velocities are considered to be the same, since we assume that the real pulse wave does not change in time or, at least, for some period of time. That is $c_1 = c_2 = \cdots = c_N$

Using these properties, we obtain a system of equations as in [3]:

$$\begin{cases} \frac{1}{2}k_i^2 = u_i, \quad i = \overline{1, N}; \\ -\frac{-\omega_i t + h_i^{(0)}}{k_i} = x_i, \quad i = \overline{1, N} \\ \frac{\omega_i}{k_i} = \frac{\omega_{i+1}}{k_{i+1}}; \quad i = \overline{1, N-1} \end{cases}$$

As the values of the local maxima, we take the values of the coordinates of the vertices of each "hump" of the real pulse wave obtained experimentally.

5 The Soliton Theory Application to Intermittent Pneumatic Compression; Influence on Thrombus Release

The resulting system consists of $3N - 1$ equations, and we have $3N$ unknowns; therefore, one of the parameters we choose arbitrarily. Take $\omega_1 = 1$, then this system is solved definitely. Solving this system, we could obtain the solution of Eq. (1) that is agreed with the theory in [10] ideas of applying the apparatus of mathematical modeling in medicine of pulse waves and in pneumatic device.

A recent survey of healthcare practitioners in North China revealed that the main concern with intermittent pneumatic compression (IPC)—supply is the fear of a thrombus release due to the soliton waves appearing in veins. This was expected by 35% of respondents [18]. And this is actually one of the first objections to discuss when getting acquainted with IPC in Ukraine. To assess the incidence of symptomatic pulmonary embolism (PE) in patients undergoing IPC therapy we performed a literature review searching the MEDLINE database with no language restrictions from January 1, 2017, until December 31, 2020. We consider two scenarios: when IPC starts after the onset of thrombosis, and when thrombosis occurs after IPC starts.

The first option is more often in unfavorable conditions, when an adequate diagnosis of thrombosis meets difficulties. These can be cases with mute blood clots, with low scores on the thrombotic risk scale, when it is not possible to perform routine ultrasound diagnostics, or when some vessels are less visible on the sensor.

Table 1 Incidence of symptomatic PE in patients undergoing IPC therapy

	Patients with IPC		Patients without IPC	
	Total	Symptomatic PE cases	Total	Symptomatic PE cases
Neurosurgery, neurology [20, 21]	3870	10 (0.26%)	3218	37 (1.15%)
Orthopedic, traumatology [22–24]	607	2 (0.33%)	1238	15 (1.21%)
Oncology [25–27]	688	5 (0.73%)	370	7 (1.9%)
Other [27, 28]	20,324	6 (0.03%)	10,819	6 (0.06%)
Total	25,489	23 (0.09%)	15,645	65 (0.42%)

In 2015, the CLOTS-3 study report appeared. In stroke, thromboprophylaxis by IPC begins post-factum, when the thrombotic risk is already increasing. Although the authors excluded patients with symptoms of pre-existing thrombosis, the risk of having a thrombus was not entirely low. Initial ultrasound was not performed, and the control one was unable to fully visualize the veins in almost half of the patients. Commenting on this, the authors noted: "There was a concern that the application of IPC to patients who may already have a deep vein thrombosis might displace the thrombus and increase the risk of PE. However, this potential risk has not been documented in the randomized controlled trials so far. We have not identified any case reports that provide convincing evidence that this has occurred" [19].

The second scenario is more typical when the IPC is used for thromboprophylaxis. We identified nine trials with 40,667 participants, and the main results are presented in Table 1.

Although thrombosis is more common with IPC than with heparin, dangerous complications such as clinical or fatal PE occur in less than 1% of cases. Moreover, some sources [20, 21, 24, 28], show that the risk of PE with heparin may be higher than with IPC. This is probably because the IPC mimics physical activity. A thrombus that grows during IPC therapy is adapted to motor load, while the anatomical structure of a "heparin" thrombus may not be strong enough for soliton fluctuations. Another reason may be that during IPC, the thrombus progresses mainly in those veins unreachable to the external mechanical pressure. Therefore, IPC therapy just does not interfere with such a blood clot, in particular, does not break it.

6 Conclusion

At the moment, there are many systems analyzing pulse waves (in most cases, mathematical statistics methods are used for the analysis). The idea of applying the apparatus of mathematical modeling in this topic seems to us auspicious, but most of mathematical models are quite complicated and difficult to apply in practice.

The method of pulse waves modeling based on soliton solution of Korteweg-de Vries equation is considered in this work, test calculations in Maple 8 environment are made. The results obtained allow us to speak about the applicability of soliton theory for pulse wave modeling. It is planned to analyze the possibility of further application of *N*-soliton solution for medical purposes.

A significant causal relationship between PE and IPC procedures has not yet been established. The incidence of symptomatic PE developing during IPC therapy is 0.03–0.73% and varies depending on the patient profile. However, caution should be when prescribing IPC therapy for ones with suspected venous thrombosis. More thorough further research is desirable in soliton wave's usage in medicine.

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