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SECTION 17.
PHYSICS AND MATHEMATICS

Vasylyuk Svitlana Viktorivna
researcher,
Physics faculty
Taras Shevchenko National University of Kyiv, Ukraine

Zaitsev Dmytro Valeriovych
research assistant
Kundiiev Institute of Occupational Health of the NAMS of Ukraine, Ukraine

APPLICATION OF SOLITON SOLUTION THEORY TO PULSE WAVE MODELING

Introduction

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 due to many factors: both purely "physical" (for example, the fact that the blood itself is not Newtonian fluid, blood flows through blood vessels and veins) and the fact that you have to take into account various regulatory functions. For this reason, most models based on the theory of mathematical hemodynamics are quite complex and difficult to apply.

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. [1-3]

1. Volobuev's model

In [1] 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 in their derivation the process of pulse wave propagation is considered quite deeply. It is noted that due to the complexity of the blood flow process, these models describe only some of the phenomena observed in the cardiovascular system.

In our work, we consider the pulse wave without taking into account the processes of blood flow regulation; we can say that 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:

$$u_t + 6uu_x + u_{xxx} = 0 \quad (1)$$

Properties of solitons

Soliton is a localized stationary or stationary on average perturbation of a homogeneous or spatially periodic nonlinear medium [1-3]. 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 [1-6].

In mathematical terms, soliton 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. [1-5] Linear equations (except the one-dimensional wave equation) have no localized stationary solutions. S. are essentially non-linear 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 [6-9].

3. Soliton solution of the Korteweg-de Vries equation

The Korteweg-de Vries equation is considered:

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

In [1,2] 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 [2] showed that in general the N-soliton solution has the form:

$$u = 2\frac{d^2}{dx^2} lnF \tag{2}$$

Where F is the determinant of some matrix [2].

Substituting (2) into (1), once integrating and assuming the integration constant equal to zero, we obtain:

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

For further analysis it is convenient to enter the operator:

$$D_x^m D_t^n ab = (\partial_x - \partial_{x`})^m (\partial_x - \partial_{x`})^n a(x,t)b(x`,t`)|_{\substack{x`=x \\ t`=t}} \tag{4}$$

So, the Equation (3) can be rewritten as:

$$(D_xD_t + D_x^4)F \cdot F = 0 \tag{5}$$

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

$$F = 1 + \varepsilon f^{(1)} + \varepsilon^2 f^{(2)} + \ldots \tag{6}$$

Where

$$f^{(1)} = \sum_{i=1}^{N} e^{\eta_i}, \eta_i = k_i x - \omega_i t + \eta_i^0 \tag{7}$$

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 (6) into (5), we found

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

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

$$O(1): 0 = 0 \tag{8a}$$

$$O(\varepsilon): 2(\partial_x\partial_t + \partial_x^4)f^{(1)} = 0 \tag{8b}$$

$$O(\varepsilon^2): 2(\partial_x\partial_t + \partial_x^4)f^{(2)} = -(D_xD_t + D_x^4)f^{(1)}f^{(1)} \tag{8c}$$

$$O(\varepsilon^3): 2(\partial_x\partial_t + \partial_x^4)f^{(3)} = -2(D_xD_t + D_x^4)f^{(1)}f^{(2)} \tag{8d}$$

Equation (8b) is homogeneous equation. As a solution of this equation, we took eq.(7). 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 $(\partial_x \partial_t + \partial_x^4) f^{(2)} = 0$.

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

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

For $N = 2$, we take equation (8b) as a solution

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

Then (8c) reduces to the equation:

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

That is have a solution [3]

$$f^{(2)} = e^{\eta_1 + \eta_2 + A_{12}}$$

$$e^{A_{ij}} = (\frac{k_i - k_j}{k_i + k_j})^2 \qquad (9)$$

(please, note that $k_1 \neq k_2$). Substituting $f^{(1)}, f^{(2)}$ in (8d), 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 $u = 2d^2(\ln F_2)/dx^2$ corresponds to the two-soliton solution of the Korteweg-de Vries equation. Performing similar calculations for $N = 3$, we obtain:

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

where the coefficients A_{ij} are determined by formula (9).

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

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

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

4. Application of the theory of solitons to 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 [1-3].

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

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 $k_i, \omega_i, \eta_i^{(0)}$ are the parameters of this system.

The following properties of solitons were used:

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

$$u_{maxi} = \frac{1}{2} k_i^2$$

2) The argument of the point of maximum of the i-th soliton is determined by the following expression:

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

3) The velocity in phase c is defined as the ratio of the coefficients at ***x*** and ***t***. For the i-th 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 $c_1 = c_2 = \ldots = c_N$

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

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

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 equation (1) that is agreed with the theory in [1-3] 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 [9]. 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.

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" [10].

The second scenario is a more typical when the IPC is used for thromboprophylaxis. We identified 9 trials with 40667 participants, and the main results are presented in the Table 1.

Table 1

The 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 [11, 12]	3870	10 (0.26%)	3218	37 (1.15%)
Orthopedic, traumatology [13, 14, 15]	607	2 (0.33%)	1238	15 (1.21%)
Oncology [16, 17, 18]	688	5 (0.73%)	370	7 (1.9%)
Other [19, 20]	20324	6 (0.03%)	10819	6 (0.06%)
Total	25489	23 (0.09%)	15645	65 (0.42%)

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 [11, 12, 15, 19] 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.

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 Fries 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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