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On the Representation of Turbulent Stresses for Computing Blood Damage

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Abstract

Computational prediction of blood damage has become a crucial tool for evaluating blood-wetted medical devices and pathological hemodynamics. A difficulty arises in predicting blood damage under turbulent flow conditions because the total stress is indeterminate. Common practice uses the Reynolds stress as an estimation of the total stress causing damage to the blood cells. This study investigates the error introduced by making this substitution, and further shows that energy dissipation is a more appropriate metric of blood trauma.

Keywords

turbulence; blood hemolysis; damage; dissipation

1. Introduction

The Food and Drug Administration evaluates medical devices based on the criteria of safety and efficacy. The anchor of safety is biocompatibility, which in the case of blood-wetted medical devices is dictated by the absence of shear-induced blood trauma, thrombosis, and infection (Ailawadi and Zacour, 2009; Barnes, 2008; Sayer, et al., 2009). One of the greatest prevailing challenges in the development of blood-wetted cardiovascular devices is the elimination of unintended flow-induced trauma to the blood, namely hemolysis (the destruction of blood cells) and shear-activation of platelets (Blackshear, 1972; Bluestein, 2004; Wurzinger, et al., 1985).

Because of the cellular composition of blood and the complexity of the flow fields in these devices, it has been impractical to develop a microstructural model of cellular damage. Consequently, investigators over the past several decades have relied upon homogenized models (Apel, et al., 2001; Blackshear, 1972; Bludszuweit, 1995; Giersiepen, et al., 1990; Heuser and Opitz, 1980; Leverett and al., 1972; Yeleswarapu, et al., 1995); mostly empirical relationships between the shear history and an experimentally measured indicator of cell trauma (such as hemoglobin liberated from the red blood cell into the plasma.) These empirical models are generally of the form:

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$$D = C \tau^\alpha t_{\text{exp}}^\beta \quad (1)$$

where D is an index of damage, τ is the scalar shear stress, t_{exp} is the exposure time, and C , α , and β are coefficients (Blackshear, et al., 1965; Boreda, 1995; Einav and Bluestein, 2004; Giersiepen, et al., 1990; Jesty, et al., 2003; Song, et al., 2003; Wurzinger, 1985). (See Table 1.) In addition, there are two theoretical models of significance, the first by Richardson (1975):

$$D = C \eta \dot{\gamma}^2 t_{\text{exp}} = \frac{C}{\eta} \tau^2 t_{\text{exp}}, \quad (2)$$

and a second by Yeleswarapu et al. (1995), who in turn were motivated by Kachanov (1986):

$$\dot{D} = \tau^2 \frac{C}{(1 - D)^\delta}, \quad (3)$$

where η is the viscosity, $\dot{\gamma}$ is the scalar shear rate, and δ is a model constant.

The unknown constants have been determined through various controlled experiments, intentionally designed to cause cellular trauma. The preponderance of these experiments has been laminar, viscometric flows. However, blood flow in cardiovascular devices occurs in both the laminar and turbulent regimes.

Prediction of hemolysis in turbulent flow, therefore, requires an appropriate constitutive model to determine turbulent stresses. The most accurate approaches used in engineering applications include Direct Numerical Simulation (DNS) and Large Eddy Simulation (LES), which for geometries of practical importance are prohibitive. It is therefore common to employ time-averaged equations that describe the mean motion. The most frequently used model turbulent flow in blood-wetted devices has been the Reynolds Averaged Navier-Stokes (RANS) equations.¹ However, the formulation of the RANS results in the loss of the small-scale turbulent features of velocity and stress. To account for these lost stresses, it is common for bioengineers to incorporate the so-called *Reynolds Stress* as a surrogate for the turbulent stresses² when computing blood trauma (Bluestein; 2004, Einav and Bluestein; 2004, Mitamura 2000; Qian, 1989; Song, et al., 2004; Tamagawa, et al., 1996; Walburn, et al., 1985; Walburn, et al., 1985; Wu, et al., 2005). With this approach the k - ϵ model (Ahmadi, 1985) and the algebraic stress model (ASM) have been introduced (Rodi, 1982). However, it has been suggested that the assumptions leading to the k - ϵ model are inadequate for the modeling of turbulence in highly swirling flows (Speziale, 1981; Sloan et al., 1986; Hwang et al., 1991, 1993). Even with the various modifications made for the k - ϵ model, it is still not capable of describing secondary flows in noncircular ducts and it cannot predict non-zero normal-Reynolds-stress differences (Speziale, 1987; Speziale and Ngo, 1988).

¹For a detailed description of the Reynolds-Averaged Navier-Stokes equations see Alfonsi (2009)[21]

²The term *turbulent stress* is sometimes referred to as the *fluctuating stress* in the literature.

This study seeks to investigate the errors introduced by using the Reynolds stress as a surrogate for turbulent stress through arithmetic manipulation of the Navier-Stokes and RANS equations.

2. Mathematical Preliminaries

Assuming blood to behave as a single-phase and incompressible fluid, the conservative form of the linear momentum equation can be represented as:

$$\rho \frac{D\mathbf{u}}{Dt} = \rho \frac{\partial \mathbf{u}}{\partial t} + \text{div}(\rho \mathbf{u} \otimes \mathbf{u}) = -\nabla p + \text{div} \boldsymbol{\tau} + \mathbf{f}, \quad (4)$$

and conservation of mass as:

$$\text{div} \mathbf{u} = 0 \quad (5)$$

where $\mathbf{u}(\mathbf{x})$ is the velocity field, $p(\mathbf{x})$ is the pressure, ρ is the density, and $\mathbf{f}$ represents the body forces. The Navier-Stokes equation, which is the foundation of the Reynolds Averaged Equations, assumes a Newtonian fluid and thus a constitutive relation of the form:

$$\boldsymbol{\tau} \equiv \eta (\nabla \mathbf{u} + \nabla \mathbf{u}^T), \quad (6)$$

where η is a constant representing the shear viscosity of the fluid. Although blood is known to be shear-thinning, it has been shown that it can be treated as a Newtonian fluid for shear rates above 100s^{-1} which is the case for many practical problems.

Reynolds' treatment of turbulent flow decomposes the field variables into their average and time-varying components. For example, velocity can be decomposed into:

$$\mathbf{u} = \bar{\mathbf{u}} + \mathbf{u}', \quad (7)$$

where the over-bar represents the time-average and the prime (') indicates the fluctuating components. The RANS equations are obtained by substituting the decomposed state variables into the original equations and taking the time-average. Substituting Equation (7) into the constitutive equation yields a total stress of:

$$\boldsymbol{\tau} = \eta (\nabla \bar{\mathbf{u}} + \nabla \bar{\mathbf{u}}^T + \nabla \mathbf{u}' + \nabla \mathbf{u}'^T) \quad (8)$$

where the first term defines the mean stress

$$\bar{\boldsymbol{\tau}} = \eta (\nabla \bar{\mathbf{u}} + (\nabla \bar{\mathbf{u}})^T), \quad (9)$$

and second term, the turbulent stress:

$$\boldsymbol{\tau}' = \eta (\nabla \mathbf{u}' + \nabla \mathbf{u}'^T), \quad (10)$$

hence

$$\boldsymbol{\tau} = \bar{\boldsymbol{\tau}} + \boldsymbol{\tau}'. \quad (11)$$

Substitution of Equations (7) and (8) into (4) and (5), when averaged, results in the following linear momentum and mass balance equations:

$$\rho \frac{D\bar{\mathbf{u}}}{Dt} + \text{div}(\rho \overline{\mathbf{u}' \otimes \mathbf{u}'}) = -\nabla \bar{p} + \text{div} \boldsymbol{\tau} + \mathbf{f}, \quad (12)$$

$$\text{div} \bar{\mathbf{u}} = 0 \quad (13)$$

which collectively comprise the RANS equations. The first term of Equation (12) is the acceleration of the mean flow field and the second term represents the acceleration of the fluctuating flow field (later named the *divergence of the Reynolds stress*.) The right hand side represents the average stresses and forces acting on the fluid.

The RANS lacks closure, i.e., has more unknowns than equations due to the presence the turbulent fluctuating terms for velocity. Early models of turbulence eliminated these terms by introducing the concept of a *Reynolds stress*:

$$\tau_{\text{Re}} = -\rho \overline{\mathbf{u}' \mathbf{u}'} \cong \eta_T (\nabla \bar{\mathbf{u}} + \nabla \bar{\mathbf{u}}^T) - \frac{2}{3} \rho k \mathbf{I}, \quad (14)$$

where η_T is the turbulent viscosity, $\mathbf{I}$ is the identity matrix, k is the kinetic energy of the flow:

$$k \equiv \frac{1}{2} \mathbf{u}' \cdot \mathbf{u}'. \quad (15)$$

The Reynolds stress approximation thus consolidates six cross-components of the fluctuating velocity into the three mean velocities.

Various methods have been developed to calculate the turbulent viscosity and turbulent kinetic energy, such as mixing length models, the k- ϵ model (Ahmadi, 1985; Launder and Spalding, 1974), and the algebraic stress model (ASM) (Rodi, 1976). Since several such closure models take on the appearance of a viscous stress, the name “Reynolds stress” has been imparted on this term despite the fact that it actually describes acceleration rather than an actual stress. Accordingly, caution should be exercised when relying on this term to predict the damage caused by true stress on the blood cells. Indeed, even with the various modifications made for the k- ϵ model, it has been shown to be inadequate for modeling

turbulence in certain engineering applications, such as highly swirling flows (Hwang, 1991, 1993; Sloan, et al., 1986; Speziale, 1987) and secondary flows in noncircular ducts, nor is it able to predict non-zero normal-Reynolds-stress differences (Speziale, 1987). Clearly, further investigation of the accuracy of the Reynolds Stress approximation is warranted.

3. Analysis of Error in Blood Trauma Estimation Due to Reynolds Stress Assumption

The error introduced by the use of Reynolds stress as a surrogate for turbulent stress, i.e.

$$\boldsymbol{\tau}' = \kappa \boldsymbol{\tau}_{\text{Re}} \quad (16)$$

may be investigated by comparison to the exact solution in Equation 10. It is observed that the mean of $\boldsymbol{\tau}'$, in Equation 10, is the zero tensor. Therefore, Equation 16 can hold only if κ is identically zero for all time or all components of Reynolds stress are zero. The former case implies that there cannot be a direct relationship between the Reynolds and total stress. The latter case is only true for laminar flow. Therefore, the use of Reynolds stress to estimate blood trauma introduces a systematic error.

Further insight can be derived by subtracting the RANS equation from the original Navier-Stokes equation:

$$\rho \frac{\partial \mathbf{u}'}{\partial t} + \text{div}(\rho \mathbf{u}' \bar{u}) + \text{div}(\rho \bar{u} \mathbf{u}') + \text{div}(\rho \mathbf{u}' \otimes \mathbf{u}') + \text{div}(\boldsymbol{\tau}_{\text{Re}}) = -\nabla p' + \text{div} \boldsymbol{\tau}', \quad (17)$$

$$\text{div} \mathbf{u}' = 0. \quad (18)$$

Substitution of the approximate stress from Equation 16 into Equation 17 likewise results in a non-zero mean equation, violating the assumptions upon which the Reynolds average equations are based. Hence the approximate relationship given in equation 16 is ill advised.

4. Energy Dissipation for Modeling Blood Trauma

Using independent methods, Jones (1995) and later Quinlen and Dooley (2007) proposed that turbulent stresses are better approximated through the use of turbulent energy dissipation. This approach entails the use of a scalar norm for the total stress tensor, commonly practiced in failure mechanics (Ugural and Fenster, 1975).

Beginning once again with the assumption of a Newtonian fluid, and expressing the constitutive relationship (Equation 6) as:

$$\boldsymbol{\tau} = \eta \mathbf{A}, \quad (19)$$

where

$$\mathbf{A} = \nabla \mathbf{u} + \nabla \mathbf{u}^T, \quad (20)$$

which can be decomposed as:

$$\mathbf{A} = \nabla \bar{\mathbf{u}} + \nabla \bar{\mathbf{u}}^T + \nabla \mathbf{u}' + \nabla \mathbf{u}'^T = \bar{\mathbf{A}} + \mathbf{A}'. \quad (21)$$

Various norms have been proposed for reducing the shear or the stress tensor to a single scalar quantity to compute blood trauma (Bludszweit, 1995; Farinas, et al., 2006) but it has been shown that the specific choice does not greatly effect the estimation of hemolysis (Farinas, et al., 2006). The following commonly used norm for any second order tensor $\mathbf{A}$ based on the inner product may therefore be applied:

$$A = \|\mathbf{A}\| \equiv \sqrt{\frac{1}{2} \mathbf{A} : \mathbf{A}} \quad (22)$$

which leads to the following expression for the norm of the stress:

$$\tau = \|\boldsymbol{\tau}\| = \eta \sqrt{\frac{1}{2} (\bar{\mathbf{A}} : \bar{\mathbf{A}} + 2\bar{\mathbf{A}} : \mathbf{A}' + \mathbf{A}' : \mathbf{A}')}. \quad (23)$$

Since the time average of the scalar norm does not eliminate the unknown cross terms it becomes necessary to take the square of the scalar stress before taking the time average:

$$\tau^2 = \eta^2 (\bar{\mathbf{A}} : \bar{\mathbf{A}} + 2\bar{\mathbf{A}} : \mathbf{A}' + \mathbf{A}' : \mathbf{A}') \quad (24)$$

for which the time average is:

$$\text{mean}(\tau^2) = \eta^2 (\bar{\mathbf{A}} : \bar{\mathbf{A}} + \mathbf{A}' : \mathbf{A}') \quad (25)$$

An approximation of the norm of the total stress can be taken as the square root of the previous mean:

$$\tilde{\tau}_{eda} = \sqrt{\text{mean}(\tau^2)} = \eta \sqrt{\frac{1}{2} (\bar{\mathbf{A}} : \bar{\mathbf{A}} + \mathbf{A}' : \mathbf{A}')}. \quad (26)$$

and hereinafter will be referenced as the *energy dissipation approximation* (EDA). This can be rearranged as:

$$\tilde{\tau}_1 = \sqrt{\frac{1}{2} (\eta^2 \bar{\mathbf{A}} : \bar{\mathbf{A}} + \eta^2 \bar{\mathbf{A}}' : \bar{\mathbf{A}}')} = \sqrt{\bar{\tau}^2 + \eta \rho \varepsilon}, \quad (27)$$

where $\bar{\tau}$ is the norm of the mean stress³, and ε is the energy dissipation due to the turbulent eddies⁴ traditionally defined as:

$$\varepsilon = \frac{1}{2} \frac{\eta \mathbf{A}' : \mathbf{A}'}{\rho}. \quad (28)$$

The value for ε is typically calculated empirically (i.e. using mixing length models) or through parameters of more complex closure models (e.g. k- ε , k- ω). It has been shown previously that the dissipation is nearly constant over time (Pope, 2000).

5. Analysis of Error of Damage Predictions

Without loss of generality, for the purpose of error analysis, the collection of prevailing blood damage models may be represented as a power law equation of the following form:

$$D = D(\eta, t_{\text{exp}}) \tau^2. \quad (29)$$

where D is a (damage) function with dependence on exposure time, t_{exp} , and viscosity, η . The exponent of shear stress is chosen here to be identically equal to 2, which agrees with the statistical accuracy of several empirical models (Blackshear, et al., 1965; Boreda, 1995, Song, et al., 2003; Wurzinger, 1985) and two theoretical blood damage models (Richardson, 1975; Yeleswarapu, et al., 1995).

The absolute error of the predicted damage, based on true stress versus approximate stress, is:

$$\text{Error}(D) \equiv |D_{\text{true}} - D_{\text{approximate}}| = C \left| \tau^2 - \tilde{\tau}^2 \right|. \quad (30)$$

The naïve approximation for stress in turbulent flow employs the mean stress, Equation 9, resulting in the approximate total stress given by:

$$\tilde{\tau}_m = \eta \sqrt{\frac{1}{2} \bar{\mathbf{A}} : \bar{\mathbf{A}}}, \quad (31)$$

where the subscript m refers to this *mean* approximation. Therefore the approximation of damage based on the mean stress, D_m , results in an absolute error of:

³The term *total stress* is the combination of the *mean stress* and *turbulent stress*. It is sometimes referred to as the *true stress*.

⁴Energy quantities are calculated per mass in turbulent flow in contrast to per volume.

$$Error(D_m) = \alpha \frac{1}{2} \eta^2 \left| \overline{\mathbf{A}} : \overline{\mathbf{A}} + 2\overline{\mathbf{A}} : \mathbf{A}' + \mathbf{A}' : \mathbf{A}' - \overline{\mathbf{A}} : \overline{\mathbf{A}} \right| = \alpha \frac{1}{2} \eta^2 \left| 2\overline{\mathbf{A}} : \mathbf{A}' + \mathbf{A}' : \mathbf{A}' \right|. \quad (32)$$

The commonly used approximation of the total stress by the sum of the mean stress and the Reynolds stress gives, as defined in Equation 9 and 14 respectively:

$$\tilde{\boldsymbol{\tau}}_R = \overline{\boldsymbol{\tau}} + \boldsymbol{\tau}_{Re}, \quad (33)$$

and an approximate scalar shear stress of:

$$\tilde{\tau}_R = \sqrt{\frac{1}{2}(\overline{\boldsymbol{\tau}} + \boldsymbol{\tau}_{Re}) : (\overline{\boldsymbol{\tau}} + \boldsymbol{\tau}_{Re})}, \quad (34)$$

where the subscript R will represent this stress approximation. The error becomes:

$$\begin{aligned} Error(D_{Re}) &= \alpha \frac{1}{2} \eta^2 \left| \overline{\mathbf{A}} : \overline{\mathbf{A}} + 2\overline{\mathbf{A}} : \mathbf{A}' + \mathbf{A}' : \mathbf{A}' - \left(\overline{\mathbf{A}} - \frac{1}{\eta}(\rho \overline{\mathbf{u}' \mathbf{u}'}) \right) : \left(\overline{\mathbf{A}} - \frac{1}{\eta}(\rho \overline{\mathbf{u}' \mathbf{u}'}) \right) \right| \\ &= \alpha \frac{1}{2} \eta^2 \left| 2\overline{\mathbf{A}} : \mathbf{A}' + \mathbf{A}' : \mathbf{A}' - (\rho \overline{\mathbf{u}' \mathbf{u}'}) : (\rho \overline{\mathbf{u}' \mathbf{u}'}) + 2\overline{\mathbf{A}} : (\rho \overline{\mathbf{u}' \mathbf{u}'}) \right|. \end{aligned} \quad (35)$$

The error using the approximation $\tilde{\tau}_{eda}$ presented above (Equation 27) is:

$$Error(D_1) = \alpha \frac{1}{2} \eta^2 \left| \overline{\mathbf{A}} : \overline{\mathbf{A}} + 2\overline{\mathbf{A}} : \mathbf{A}' + \mathbf{A}' : \mathbf{A}' - \overline{\mathbf{A}} : \overline{\mathbf{A}} - \mathbf{A}' : \mathbf{A}' \right| = \alpha \frac{1}{2} \eta^2 \left| 2\overline{\mathbf{A}} : \mathbf{A}' \right|. \quad (36)$$

The magnitude of the above error estimates is clearly problem-specific; however, some insight on the accuracy of each method may be obtained by evaluating the respective mean errors, hence systematic errors:

$$\overline{Error(D_m)} = \alpha \frac{1}{2} \eta^2 \left| \overline{\mathbf{A}' : \mathbf{A}'} \right|, \quad (37)$$

$$\overline{Error(D_{Re})} = \alpha \frac{1}{2} \eta^2 \left| \overline{\mathbf{A}' : \mathbf{A}'} - \frac{1}{\eta^2}(\rho \overline{\mathbf{u}' \mathbf{u}'}) : (\rho \overline{\mathbf{u}' \mathbf{u}'}) + \frac{1}{\eta} 2\overline{\mathbf{A}} : (\rho \overline{\mathbf{u}' \mathbf{u}'}) \right|, \quad (38)$$

and

$$\overline{Error(D_1)} = 0. \quad (39)$$

This illustrates that there is a systematic error associated with the mean stress assumption that is proportional to the time-averaged turbulent energy dissipation. The assumption that the total stress is represented as the sum of the mean stress and Reynolds stress results in a systematic error that is a function of the turbulent viscous dissipation and the square of the norm of Reynolds stress. The systematic error associated with the dissipation approximation (Equation 27) is zero: it under-predicts and over-predicts damage equally. Since the Reynolds stress dominates the viscous dissipation term for bounded flow, the error using the Reynolds stress could be significant.

6. Discussion

Standard closure models for turbulent flow of blood are insufficient to accurately calculate total stress experienced by the cells suspended therein. Direct numerical simulation is so computationally expensive that only the simplest of geometries can be evaluated. This difficulty arises from the fact that the RANS, with its associated closure models, were developed to study large-scale flow features (Pope, 2000), whereas hemolysis is induced by stresses acting on the micro-scale (Quinlan and Dooley, 2007). The most common substitute for the *turbulent stress* has been the Reynolds stress, but it was shown here that this substitution is potentially fraught with large errors. When substituted into the most common formulae for hemolysis, the errors in computing turbulent stress are amplified by the exponent of the shear stress, which have been reported as great as 3.075 (Giersiepen, et al., 1990). In this study, the value was assumed to be 2.0. Errors are further compounded by the inaccuracy of the multiplicative term α involving time exposure. The foregoing clearly raises concerns as to reliability of blood damage predictions in turbulent flow that are computed on the basis of Reynolds stress.

The alternative representation of the total scalar stress by the combination of *mean stress* and turbulent dissipation Equation 27 or 28, was shown to be less problematic. Previous studies, using markedly different methods and forms, have shown that energy dissipation is a robust physical model for predicting blood damage in general (Jones, 1995; Quinlan and Dooley, 2007, Richardson, 1975). The earliest study, by Richardson (1975), examined the stress loading on the individual cells using the membrane model of Rand (1964), established that blood damage in laminar flow is functionally determined by viscous dissipation. Jones (1995) based his conclusions on the transfer of energy across streamlines. Quinlan and Dooley (2007) studied turbulence on the cellular level. This study corroborates these conclusions through arithmetic manipulation of the scalar stress equation. Ultimately, turbulent energy dissipation is less heuristic and arbitrary than the use of Reynolds stress because of its physical relationship to stress, i.e. the product of stress and strain rate. Estimates of the mean (systematic) error of the dissipation approximation (Equation 27) is zero irrespective of flow conditions: implying that the over- and under-predictions by this model caused by fluctuations in the true stress field uniquely cancel.

Nevertheless, Reynolds stress has almost universally been used for predicting blood-trauma in practical application involving turbulence. In fact this practice has been so commonplace that terminology *Reynolds stress* and *turbulent stress* have been used interchangeably in literature (Einav and Bluestein, ; Qian, 1989; Song, et al., 2004; Tamagawa, et al., 1996). This practice overlooks foundational principals of the RANS, hence its attendant strengths and weaknesses (Crow, 1968; Pope, 2000). To the best of our knowledge, there has yet to be a published study that employs turbulent energy dissipation to predict hemolysis in a practical application.

Surprisingly accurate results have nevertheless been reported through the substitution of the (laminar) stress with the Reynolds stress in the power law hemolysis formula of Giersiepen

(Bluestein, 2004; Einav and Bluestein, 2004; Mitamura, 2000; Song, et al., 2004; Tamagawa, et al., 1996; Walburn, et al., 1985; Walburn, et al., 1985; Wu, et al., 2005). It is further surprising considering the errors in the coefficients of this model (Paul, et al., 2003). This could possibly be explained by an ad-hoc correlation for the cumulative damage between the Reynolds stress and the turbulent stress for certain problems. For example, in bounded-flow, vorticity is highly correlated ($r=0.999$) to scalar stress, but vorticity is based on the curl of velocity and the stress is related to the symmetric portion of the gradient of velocity tensor (Hund and Antaki, 2004; Kuethe and Chow, 1998). A further possible explanation can be linked to the closure models themselves (Crow, 1968; Pope, 2000). For example, Crow (1968) showed that turbulent closure models for the RANS are inaccurate in contracting nozzles; whereas other researchers have shown that they may be better represented using a viscoelastic formulation (Pope, 2000; Rajagopal, 1999; Speziale, 1999). Until the questions surrounding the accuracy of the Giersiepen model can be sufficiently resolved (Paul, et al.), it is not possible to identify the special cases or guidelines wherein Reynolds stress may yield a accurate prediction of hemolysis.

The forgoing discussion has been based on the assumption of blood as a Newtonian fluid; however, blood is known to exhibit non-Newtonian properties under certain circumstances. Therefore the examination of turbulent flow of blood must be considered in the context of the associated constitutive model. Based on the early work of Rivlin (1957) on the relation between the turbulent flow of a Newtonian fluid and the flow of a non-Newtonian, Speziale (1987) developed a class of non-linear turbulence models which in principle have the same structure as the Rivlin-Ericksen fluid of third grade. In a series of papers, Rajagopal and co-workers have shed more light on the constitutive modeling of turbulence and the similarities between turbulent flow and the flow of non-Newtonian fluids. (See Huang and Rajagopal (1996), Rajagopal et al. (2004), Huang et al. (2003), and Tao et al. (2010)). Future efforts to develop a rational, accurate model of blood trauma would benefit from this fundamental work.

In light of the uncertainty of prevailing models for turbulence-induced blood trauma, further investigation is clearly warranted. In addition to exploration of additional closure models, DNS will prove to be an essential tool for computing the turbulent fluctuating stress, which cannot be determined otherwise.

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Table 1

Comparative values of the power-law coefficients for red blood cells (RBCs) and platelet (Plt) activation and the experimentally measured quantities, i.e. plasma free hemoglobin (pfHb) over total hemoglobin (Hb), concentration of lactate dehydrogenase [LDH], the concentration of serotonin [Ser], and concentration of Thrombin [Thr].

Cell-Measurement	C	a	b	Source
RBC-pfHb/Hb	3.62e-5	2.42	0.785	Giersiepen (1990)
RBC-pfHb/Hb	1.8e-6	1.99	0.765	Song et al. (2003)
RBC-pfHb/Hb	-	2	1	Wurzinger (1986)
RBC-pfHb/Hb	-	2	1	Blackshear et al. (1965)
Plt-[LDH]	3.31e-6	3.08	0.77	Giersiepen et al. (1990)
Plt-[Ser]	1	1	0.492	Boreda et al. (1995)
Plt-[Ser]	1	2	1	Modified (PSF)
Plt-[Thr]		1	1	Bluestein et al. (2004) PAS