Chapter 1

Introduction

1.1 General Remarks

Atherosclerosis, defined by the World Health Organization in its *Technical Bulletin* in 1958, is 'a variable combination of changes of the intima of arteries consisting of focal accumulations of lipid, complex carbohydrates, blood and blood products, fibrous deposits and calcium deposits associated with medical changes'. It is the largest single cause of death in the western world [1]. Despite its comparatively short existence as a nosological entity (Marchand coined the term atherosclerosis in 1904), the process does not belong to our time only. Fallopio [2] described 'degeneration of arteries into bone'; and Cowper (quoted by Long [3]), perceptively noted that 'the passage of blood is impeded into thickened arteries'. However, the pathology of atherosclerosis is still not completely understood, despite continuous efforts over many years.

Among various hypotheses and theories, it is clear that hemodynamic factors make important contributions to both the initiation and localization of atherosclerosis [4,5].

Extensive topographical data relating the distribution of atherosclerosis lesions has been summarized by Woolf [1], and interpreted in a variety of fascinating ways. Texon [6,7] has been a strong protagonist of the view that regions where there appears to be a predilection for atherosclerotic lesions to occur are the segmental zones of diminished lateral pressure produced by forces generated by flowing blood. The common pathological mechanism at work in all these anatomical situations is held by Texon to be a suction
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action in the vessel wall that is produced by the localized decrease in lateral pressure. In his view, such zones are characterized in anatomical terms by curvature, branching, tapering, etc. Caro and his associates [8] maintain that the maximum possible reduction of pressure that can occur from the stagnation value by ‘Bernoulli’ mechanism is approximately 15 \( \text{mmHg} \) (assuming a maximum aortic blood velocity of 200 \( \text{cm} \cdot \text{s}^{-1} \)). In their view, it is impossible to see that reduction of the positive intraluminal arterial pressure from 100 to 85 \( \text{mmHg} \) could exert any inwardly directed suction force on the endothelium.

Measurement of shear stress in vivo is a matter of some considerable difficulty. Fry [9] has succeeded in designing an ingenious system in which the histological changes in the endothelium, which are associated with increases in blood velocity can be studied and quantified. In his study, there appears a ‘critical’ value of time-smoothed shearing stress to which the endothelial cell ‘yield’ and develop altered structured and chemical characteristics. This critical value is in the region of 380 \( \text{dyn} \cdot \text{cm}^{-1} \cdot \text{s}^{-1} \) and the yielding process has a relatively short time constant (probably less than 1 hour). Shearing stress less than the critical value for cell erosion can also produce more subtle alteration in the endothelial cells which might promote atherogenesis.

Whether shearing stress of this magnitude can occur in the normally functioning vascular bed is unknown. One of the contradictory observation is that at points of branching, the flow divider edge, which is chronically exposed to high shearing stresses, tends to be spared, at least in the early stages of plaque formation [5]. Fry [10] pursued this apparent paradox and argued that certain structural and functional changes in the arterial intima occur as a result of the mechanical stress exerted on the vessel lining by the adjacent blood flow. These responses are modulated by the magnitude and, no less importantly, the stability of the stress pattern as well as the duration of exposure to the stress.
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Caro, Fitz-Gerald and Schroter [11] found that fatty streaks and small fatty plaques tend to occur in those regions in which wall shear rate is expected to be low and the development of such lesions is inhibited or at least retarded in those areas where wall shear rates are expected to be high. It was suggested by Caro et al. [8] that this was due to the effect of shear rate on mass transport of lipid-rich macromolecules out of the vessel wall into the lumen.

A dynamic factor that might influence platelet adhesion without necessarily producing a direct effect on the arterial endothelium is stagnation, which can occur downstream of side-branch openings or distal to stenoses. Fox and Hugh [12] studied the occurrence of such stagnation points and maintain that microthrombus formation corresponds with these regions. Muller-Mohnssen, Kratzer and Baldauf [13] proposed that stagnation point flow has a thrombogenic effect by increasing the chances of collision between platelets and other formed elements of blood and the underlying endothelium. Other studies [14] suggested that the 'dwell time' of a platelet in the vicinity of an endothelial surface is a function of the local blood velocity gradient (shear rate). Areas of low shear rate and flow separation have particular long 'dwell time' compared with areas with high shear rates suggesting that it will be much more likely in the low-shear areas if the platelet endothelium interaction is to occur.

Although blood flow is mostly laminar under normal physiological conditions, unstable and turbulent flow patterns might be expected where the velocity is increased, for example, at points of narrowing of the arterial lumen. These turbulent patterns are held to be responsible for a number of diverse pathological changes such as the formation of intimal microthrombi [15], modification of endothelial permeability [16] and the stimulation of intimal smooth-muscle proliferation [17,18]. Therefore, the increased collision between the formed elements of the blood and between these formed elements and the vessel wall may speed up the atherogenesis process if it is not responsible for the initiation of the
atherosclerosis.

We must be very careful about the theories or hypotheses mentioned above since there is no 'direct' evidence for the involvement of fluid dynamics in the atherosclerotic process [19]. Although it is known that the abdominal aorta, the carotid and coronary arteries and the peripheral arteries, such as the ciliae and femorals, as well as regions of arterial branching and sharp curvature, have the greatest predilection for the disease [5], it is not enough to identify these regions generally. Theories are inconsistent, sometimes contradictory due to our limited knowledge of the detailed pattern of the disease and understanding of the fluid mechanics. For example, the haemodynamic mechanism has been thought to be increased lateral pressure [20,21,22]; reduced pressure [6,7]; reduced shear stress [8] and increased shear [5,9]. Thus, any effort to add more information about the flow field will be useful if it results from a consistent, although greatly simplified, flow model.

1.2 Fluid Dynamics in Stenosed Arteries

Many researchers have contributed to the study of flow in constricted tubes. Young and Tsi [23] have studied some of the flow characteristics in models of arterial stenosis under steady and pulsatile flow conditions. Their experiments yielded a description of the extent of separated flow regions, and a measure of pressure losses across the constriction. Lee and Fung [24] obtained numerical solutions to the Navier-Stokes equations for flow through a constricted tube. The steady flow field was calculated for Reynolds number from 0 to 25, and separation was predicted at very low Reynolds number. Calculations were not extended to higher Reynolds numbers due to instabilities in the numerical procedure. This may have been caused by the improper treatment of the boundary condition
when the stream function-vorticity formulation was used. Morgan and Young [25] employed momentum and energy integral equations, based on the work of Forrester and Young [26], to find approximate solutions to the axial velocity component of flow in the region of stenoses. This was extended to higher Reynolds number covering physiological regions by Fukushima et al. [27]. Agreement with experiment data [23] for separation and reattachment points and for pressure drop across the stenoses was reasonably good. However, the method did not give the radial velocity component, and the axial momentum equation was simplified by neglecting the second derivatives of velocity in the axial direction. Deshpade et al. [28] presented a calculation for the steady Navier-Stokes equations in a symmetric constricted tube using a stream function-vorticity formulation. The results were given for several stenosis models with different area reductions and the Reynolds number in the practical region. Reasonable agreement with previous investigations was achieved. However, the treatment of the constriction geometry and related boundary condition was not very accurate.

The study of blood flow in stenosed arteries may provide the necessary information to verify theories of localization and initiation of atherosclerosis. Regardless of the exact initiating factors, the development of localized arterial stenosis may lead to disordered blood flow within and downstream of the constricted regions. Thus, the ability to describe flow through a partial occlusion adds to the insight needed to solve the puzzle of the pathology of atherosclerosis. There is also widespread interest in determining whether the disordered flow patterns can be used to detect localized arterial disease in its early stages, particularly before it becomes clinically significant [29,30]. Beyond that, introducing a stenosed flow condition by implementing a coarctation in arteries in quite common in animal experiments [31,32]. It will be useful to combine the fluid dynamic factors and the results from in vivo experiments to make a reasonable conclusion.

Information on pulsatile flows is rather limited. Daly [33] presented results on pulsatile
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flow through a stenosed canine femoral artery. A Lagrangian-Eulerian procedure was employed to solve the integral type of Navier-Stokes equation. An interesting discussion was given for the relationship between flow quantities such as time averaged peak wall shear stress and pressure drop across the stenosis, and the degree of stenosis height. But the data was not complete for the most severe stenosis and the information was related to a specific flow situation. O'Brien and Ehrlich [34] have also considered pulsatile flow in constricted arteries by solving the unsteady Navier-Stokes equations in stream function-vorticity form. The stenosis was handled by using conformal mapping techniques. Flow through stenoses with mild degree of area reduction was investigated. This is the most complete numerical study so far, in the sense that the full Navier-Stokes equations were employed in the calculations.

In the mean time, more experimental investigations with more sophisticated techniques have been carried out. The Laser Doppler Anemometer has been used to measure the velocity field in a constricted tube [35,36]. Although data on the velocity was very accurate away from the solid wall, the LDA system has its limitation for obtaining accurate results near the wall. The separation length data by Young and Tasi [23] from flow visualization are still considered as the most reliable results.

Considerable efforts have been made to understand the problem of flow in constricted arteries. However, the results are usually either inaccurate or incomplete, and the numerical methods used have certain limitations arising from the form of the governing equations and boundary conditions.

1.3 Flow Model

Blood, which is a fluid containing a suspension, is highly non-Newtonian in the small blood vessels but can be treated as Newtonian in the larger arteries. The flow is high
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pulsatile and almost entirely laminar in a healthy circulatory system, although the peak
Reynolds number can be of the order of 10,000. The flow is usually three dimensional in
the distensible passageways, with an average Reynolds number under 1,000 [37]. Thus
the blood flow in the arterial system can be characterized as laminar, three dimensional,
pulsatile flow in distensible blood vessels [38].

The general flow in the circulatory system is very complicated, and a full description
is out of the question. Simplifications must be made to obtain useful information. Here
we treat the blood vessel as rigid, since the induration of arteries usually happens in
atherogenesis [4]. Although the flow is generally three dimensional, the two dimensional
simulation will certainly reflect some characters of the general three dimensional flow.
Although the in vivo flow of blood in the circulatory system is unsteady, it is often
necessary to begin by studying the steady flow. Studies of this type are very useful and
indicate the distributions of flow properties at a given instant in the unsteady flow cycle.

Hence, our primary numerical simulation of blood flow in stenosed arteries is for in-
compressible, two-dimensional flow through rather simple rigid geometries. A reasonable
balance between unsteady and steady flow problems has to be considered. We will fur-
ther assume a Newtonian fluid since we will focus on flow in large arteries. Thus the
full Navier-Stokes equations have to be utilized since an extensive separated flow region
occurs in the problems of interest here.

1.4 Computational Method and Difficulties for the Incompressible Navier-Stokes Equations

The equations of motion of an incompressible fluid are:

\[ \mathbf{v}_t + \mathbf{v} \cdot \nabla \mathbf{v} - \frac{1}{Re} \nabla^2 \mathbf{v} + \nabla p = 0, \]  \hspace{1cm} (1.1)

\[ \nabla \cdot \mathbf{v} = 0, \]  \hspace{1cm} (1.2)
where \( \mathbf{v} \) is the velocity vector, \( p \) is the pressure, \( Re \) is the Reynolds number. Equation (1.1) is the momentum equation for velocity, and the continuity equation (1.2) is an implicit equation for pressure. The pressure is a mysterious quantity in incompressible flow. Firstly, there is no explicit equation for pressure. It is due to the assumption of incompressibility. In this limit of compressible flow, the time derivative of pressure disappears from the continuity equation. Secondly, as pointed by many researchers [39, 40], this produces a major difficulty in defining proper boundary conditions.

There are two approaches to resolving the problem of having no explicit equation for the pressure. Either an explicit equation can be derived from the Navier-Stokes equations and the incompressibility constraint (see for example Harlow and Welch [41]) or the pressure may be eliminated from the Navier-Stokes equations to obtain a vorticity transportation equation. However, there is no boundary conditions available for either pressure or vorticity. Although these two approaches are related, the problem appears quite differently, and we will discuss them separately.

The finite difference methods for obtaining an explicit pressure equation can be classified into three major types, these are: the methods of artificial compressibility [42], pressure correction [43] and pressure Poisson equation [41].

In the artificial compressibility method, Chorin [42] used the discretized continuity equation on the boundary to provide the boundary condition for pressure in his original work. The continuity equation is then satisfied everywhere. Patankar and Spalding [43] derived an approximate equation from the continuity and momentum equations for pressure during the iterative procedure by using the Mark-and-Cell method [41]. The algorithm, known as SIMPLE, has been widely used in practice. The pressure Poisson equation derived by taking the divergence of the momentum equation (1.1) with a substitution of continuity equation (1.2) gives another alternative way to obtain a explicit equation for pressure. But the fully equivalence of the pressure Poisson equation and the
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continuity equation is always a concern in this context. In the context of the pressure Poisson equation method, several other interesting developments have been reported. Recently, a finite difference Galerkin method has been developed, details can be found in Stephen, Bell, Solomon and Hackerman [44], Bell, Colella and Glaz [45], Goodrich and Soh [46].

Since the equation for pressure is originally implicit, the major difficulty for methods using an explicit pressure equation is the lack of a pressure boundary condition. The simple fact, which is often overlooked, is that the boundary condition problem and the problem of finding the correct explicit pressure equation are closely related. As Gresho and Sani [40] discussed in their well written paper, the correct boundary condition for the pressure Poisson equation is simply the momentum equation. It is, however, not quite correct to apply the momentum equation as the pressure boundary in the projection method [39]. The projection method, originally proposed by Chorin [47], sets up an auxiliary velocity fields \( v^* \) and solves the Navier-Stokes equations in two fractional steps. The pressure operator,

\[
\nabla p = \frac{1}{\Delta t} (v^* - v) \tag{1.3}
\]

project \( v^* \) into a divergence free velocity field \( v \). It, therefore, transform the problem of pressure boundary into the problem of boundary condition for the auxiliary velocity \( v^* \). It will not be correct to apply the momentum equation as the boundary condition if the boundary value of \( v^* \) is not handled properly. Alfrink [48] offered an explicit description of the required compatibility condition to ensure the full equivalence between the pressure Poisson equation and the continuity equation. It is very important that the equivalence between the derived equation for the pressure with related boundary condition and the incompressible constraint is maintained. The influence matrix method by Kleiser and Schumann [49] gives the correct pressure boundary condition by ensuring the necessary
enforcement of the divergence-free condition at the boundary in solving the pressure Poisson equation.

Although these methods have been successfully applied in the computation of flow problems, there are some disadvantages. First of all, the discretization of the continuity equation on the boundary in the first approach will result in an inefficient solution procedure and inaccurate solutions. Secondly, the equation for the pressure correction is an approximation of the original continuity equation, and the accurate discretization near the boundary is difficult, especially for complicated geometries. The available variations of the third approach either are too complicated for practical use or fail to ensure the divergence-free condition.

Another approach, for two-dimensional flows, is to solve the vorticity transport equation by eliminating the pressure from the momentum equations, and consider the vorticity as a primary unknown:

\[ \nabla^2 \psi = -\zeta, \]  
\[ \frac{1}{Re} \nabla^2 \zeta = \psi_y \cdot \zeta_x - \psi_x \cdot \zeta_y. \]  

This stream function-vorticity method has been widely used for calculating two-dimensional flows. However, the boundary condition at a solid boundary is given for the stream function \( \psi \) only, as

\[ \psi_s = f(s), \quad \frac{D\psi}{Dn} = g(s), \]  

where \( n \) is the outward unit vector normal to the boundary and \( s \) is the measure along the boundary. There is no boundary value available for the vorticity \( \zeta \). This indicates that the usual discretized equations will be over determined for the stream function.

The existing methods which have been proposed to resolve this problem can be classified into four types: constructing a biharmonic function, using an implicit vorticity condition, using integral constraints, and influence matrix method. The first approach is
to derive a biharmonic equation for \( \psi \) by eliminating \( \zeta \) from equations (1.4)-(1.5) as

\[
\frac{1}{Re} \nabla^4 \psi = \psi_y \nabla^2 \psi_x - \psi_x \nabla^2 \psi_y. \tag{1.7}
\]

This is a nonlinear fourth-order equation with enough boundary conditions for \( \psi \). The problem of vorticity boundary conditions is completely avoided. But the discretization of such an equation gives a relatively full algebraic system and the solution procedures are sometimes unstable [50]. Schreiber and Keller [51] presented a stable algorithm to solve such a system by LU decomposition. But it takes more computer effort than simply solving the coupled system of \( \psi \) and \( \zeta \). The second approach make use of the second or higher order form of the implicity vorticity condition at the boundary, and was first suggested by Woods [52]. The method produces the explicit vorticity condition by combining a Taylor expansion and the stream function equation (1.5) at the boundary. Accurate results were obtained by Ghia \textit{et al}. [53] who employed a third-order form of vorticity condition in their multigrid procedure. This approach is accurate but complicated for complex geometries. The third approach provides integral constraints for the vorticity and stream function, and was introduced Quartapelle [54], and reviewed by Dennis and Quartapelle [55]. The method makes use of Green's theorem to transform a local implicit boundary condition into a global integral constraint for the vorticity, and has been used successfully for the problem of flow around a circular cylinder and other problems. The fourth approach was introduced by Kleiser and Schumann [49] and has since been widely used in spectral methods [56]. This approach use the idea of decomposition and source functions to obtain a boundary condition for the vorticity. Both integral constraint and influence matrix methods are mathematically simple and beautiful, but they become either complicated or computationally consuming for problems in two or three dimensions and complex geometries.

The major difficulty involved in solving flow problems for both the primitive variable
and stream function-vorticity equations are essentially similar. The difficulties can be resolved by using the new approach proposed in this thesis. We call this method the method of necessary constraints (NC). By carefully examining the governing discretized equations for incompressible flow, we found that there exists a need to impose necessary constraints for solving the Navier-Stokes equations in primitive variable, and in stream function-vorticity forms, and theses are always provided by either inaccurate or complicated ways. The constraints to obtain a determined discretization system or to ensure the equivalence of the pressure Poisson equation to the continuity equation can be provided by several ways. Generally, they can be divided into two types: boundary Constraints (BC) by providing the constraints at the boundary and interior constraints (IC) by giving the constraints inside the domain. Compared with previous methods, IC method will provide the boundary conditions for pressure and vorticity in a much simpler way. The calculation is greatly simplified since no boundary value is needed on a solid boundary. This is particularly important when the geometry of the boundary is complicated.

1.5 Scope of Present Investigation

Because our level of understanding of physiological flow problems, such as flow in stenosed arteries, is relatively poor, numerical solutions to simplified problems are helpful in providing a foundation for approaching the real situation. The lack of reliable experimental methods in determining the wall shear stress and the size of any recirculation region indicating that numerical simulation is useful tool in obtaining such flow information.

Numerical simulation by utilizing the full Navier-Stokes equations, however, encounters the major difficulty of finding a proper pressure or vorticity boundary conditions at a solid boundary, especially when the geometry is irregular. Existing techniques which have been applied in this problem result a complicate algorithms, which might be responsible
for limitation on the Reynolds number and the severity of stenosis.

The objectives of this investigation were (i) to devise and test a simple computational method for the incompressible Navier-Stokes equations for general flow problems which resolves the boundary condition difficulty, (ii) to compute the flow in simplified stenosed arteries under steady and unsteady flow conditions, and (iii) verify some of the computational results by experimental measurement. The first objective was met successfully by proposing a new finite difference method for both primitive variable and stream function-vorticity formulations. Encouraging computational results were obtained both steady and unsteady flow. The third objective was partially achieved.