Introduction

There has been a traditional interest among ophthalmologists in studies of the outflow of aqueous humour because of its dominating role in maintaining a constant level of intraocular pressure. The latter is essential for the visual functions of the eye and for the nourishment of the ocular tissues, in particular, the retina. These studies have been devoted mainly to the recognition of the principal site of the outflow resistance in the aqueous outflow system and to the investigations of its origin and its possible role in the development of a pathological state known as glaucoma.

Aqueous humour leaves the anterior chamber by flowing out through an ocular system comprising the trabecular meshwork, Schlemm’s canal and the collector channels and through another system known as the uveoscleral system (Bill, 1966; Moses, 1975). The former system accounts for 70–80 per cent of the total aqueous outflow and the latter for the remainder. The uveoscleral outflow is independent of pressure (Bill, 1966). Thus, the conventional outflow of aqueous humour is mainly responsible for the regulation of intraocular pressure. A measure of the ease with which the flow of aqueous humour occurs through the aqueous outflow system is the hydraulic conductivity which is also known as ‘outflow-facility’ in ophthalmology. Under pathological conditions, the outflow facility may decrease leading initially to aqueous accumulation in the anterior chamber with a concomitant increase in the intraocular pressure. A sufficiently high, prolonged intraocular pressure may result in glaucoma, with or without impeding retinal blood supply to the optic nerve.

Vision loss resulting from glaucoma is controllable but not curable. Present glaucoma therapies are directed towards maintaining the intraocular pressure at a level which does not cause further damage to the optic nerve head. It is accomplished through reducing elevated intraocular pressure by decreasing aqueous humour formation, or increasing aqueous outflow, or both. To develop more reliable and responsive diagnostic and therapeutic techniques, a more precise knowledge of the aqueous production and outflow and elucidation of the mechanisms of these processes is required.

Although numerous attempts have been made to identify the site of chief outflow resistance in the aqueous outflow system (Grant, 1955; 1958; McEwen, 1958; Rothen, 1960; Bill and Svedberg, 1972; Nomata et al., 1972; Tripathi, 1977; Moses, 1979), it is a matter of controversy to date. The majority of investigators hold the view that the principal site of outflow resistance is the trabecular meshwork (Grant, 1958; McEwen, 1958; Peterson et al. 1971; Tripathi, 1977; Chandler and Grant, 1979), whereas some researchers are of the opinion that the chief resistance to aqueous outflow is offered in the canal of Schlemm (Moses, 1977; 1979; Tandon and Autar, 1989). Despite the number of experimental studies, theoretical studies of the aqueous outflow system have been very few (McEwen, 1958; Weinbaum, 1965; Tandon et al., 1986). Thus, the theoretical understanding of the aqueous outflow phenomenon is quite limited. The present study deals with a theoretical analysis of the aqueous outflow in the trabecular meshwork.
Now it has been well established that aqueous outflow facility diminishes with a rise in the intraocular pressure (Moses, 1977). The ciliary muscle contraction increases the outflow facility (Bill, 1975; Moses, 1977). As mentioned earlier, most of the outflow resistance is encountered in the trabecular meshwork. The outflow facility of the aqueous outflow system is, therefore, mainly determined by the meshwork. The factors involved in the process of aqueous percolation through the meshwork play a role in the determination of the outflow facility (i.e. permeability), which is an important determinant of the intraocular pressure (Van Buskirk, 1982). To develop a suitable mathematical model for the aqueous flow in the meshwork, we have to take into account all the factors involved in the outflow process.

The structure of the trabecular meshwork is very complicated and so it is not readily susceptible to mathematical analysis. Different layers of the meshwork (namely uveal meshwork, corneoscleral meshwork and endothelial meshwork) have been identified, but the functions of different parts of the tissue are not clear (Bill, 1975). The trabecular meshwork as a whole consists of a series of flat perforated sheets or lamellae, lying one on the top of the other, connective tissues containing collagen and elastin fibres, a ground substance, and the endothelial cells. The perforated sheets are connected by tissue strands and endothelial cells. The perforations in the sheets and the space between the sheets provide interstitial space for aqueous passage. Thus, the trabecular meshwork filled with aqueous humour may be described as a biphasic matrix comprising the solid and fluid phases.

Johnstone and Grant (1973) and several other investigators (Grierson and Lee, 1975; Kayes, 1975) observed from their experimental studies that striking structural changes occurred in the meshwork in response to alterations in the intraocular pressure and in the mechanical tension on the meshwork (Moses, 1977), which is provided by ciliary muscle traction (Van Buskirk, 1982). An elevation in the intraocular pressure above the normal level may produce compression in the meshwork diminishing the flow regions.

Aqueous humour flowing through the meshwork will interact with its solid phase giving rise to a drag force, i.e., a force exerted by aqueous humour on the solid phase as it passes through the tissue. A rise in the intraocular pressure forces more aqueous fluid to flow through the meshwork, which in turn exerts a greater drag force on the solid matrix causing compression in the meshwork. Strain will be induced in the elastic solid matrix due to the imposed normal and hoop stresses. The strain can alter the conformation of the interstices which accommodate the aqueous flow and this alters the permeability of the meshwork. The compression caused by the increased intraocular pressure may decrease the intrinsic permeability, which depends upon the dilation of the solid phase. This concept is supported by Lai and Mow (1980). Once a fluid pressure is applied, the viscous drag caused by the fluid flowing through the tissue causes it to compact in a non-uniform manner, thereby decreasing the permeability.

The behaviour of aqueous humour thus depends upon the intrinsic interaction between the deformation of the solid matrix and the motion of the interstitial aqueous fluid. From a fluid mechanical point of view, there are two primary mechanisms for the transport of aqueous humour across the meshwork.

1. The interstitial aqueous fluid may be transported through the poroelastic, permeable meshwork under the influence of a pressure differential across the meshwork.

2. The deformation of elastic solid matrix caused by the drag force directly affects the aqueous outflow phenomenon.

Flow through a deformable, poroelastic trabecular meshwork requires a more detailed description than that provided by Darcy's law. Darcy's law alone says nothing about deformation and the pressure field in the tissue and their transient responses as the intraocular pressure rises. To formulate a mathematical model for aqueous flow in the meshwork, both the solid and fluid phases must be considered and a biphasic continuum model developed so that the interaction between the solid matrix and the motion of aqueous fluid might be incorporated. Other tissues for which similar multiphasic continuum models have been applied successfully include the meniscus (Favnesi et al., 1983), nasal cartilage (Mow and Schoenbeck, 1982), arterial tissue (Rajagopal et al., 1981), corneal stroma (Fat and Goldstick, 1965), porcine skin (Oomens et al., 1984) and articular cartilage (Holmes, 1985).

The mechanism of the ciliary muscle contraction in reducing outflow resistance is not well understood. Also, it has been suggested that the mechanical forces exerted on the meshwork act in a reverse direction to that of the intraocular pressure (Moses, 1977). The magnitude of the mechanical tension as distinct from the intraocular pressure is also not known, to the best of our knowledge. Unfortunately, we could not incorporate the ciliary muscle contraction and/or the mechanical tension directly into our model. Alternatively, one can think that the mechanical tension encounters the intraocular pressure. When its effect increases, the compression in the trabecular meshwork may reduce, which in turn facilitates aqueous outflow through diminishing collapse in the intertrabecular spaces.

The present paper is concerned with the development of a mathematical model for the flow of aqueous humour in the trabecular meshwork. The formulation of the model is based on the 'biphasic mixture theory'. The model assumes that the permeability is a function of the dilatation.

2 Formulation of the problem

The trabecular meshwork filled with continuously moving aqueous humour may be considered as a mixture of two interacting continua: a linear elastic solid phase and a liquid, phase. Before proceeding with the modelling process it may be appropriate to give a brief description of the 'biphasic theory'.

2.1 Biphasic theory

The system under investigation is assumed to consist of a porous, viscoelastic solid matrix which is swollen with moving water. Each constituent of the two-phase immiscible mixture is governed by the physical laws of conservation of mass and momentum with the provision made for interaction between the two phases.

Assuming that the true density of each phase is homogeneous and that each phase is intrinsically incompressible, the conservation of mass of each phase leads to the following equations:

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\frac{\partial \phi^s}{\partial t} + \nabla \cdot (\phi^s \mathbf{u}^s) = 0
\]

\[
\frac{\partial \phi^f}{\partial t} + \nabla \cdot (\phi^f \mathbf{u}^f) = 0
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