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DOUBLE POROUS MEDIUM MODEL OF BLOOD AND INTERSTITIAL FLOW IN THE LIVER

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1 Introduction

The liver is a complex organ whose function is vital to human health. Liver disease is increasingly common in developed countries, and it frequently leads to an early death or poor quality of life, as well as placing a heavy burden on health services. It often leads to a high rate of production of interstitial fluid, which drains from the liver, collecting in the peritoneal cavity and leading to ascites. This condition is uncomfortable and can lead to additional complications, such as hyponatremia (sodium imbalance), bacterial peritonitis and hepatorenal syndrome.

Histological analysis shows that the liver is divided into functional units called lobules. Each lobule has a polygonal cross-section, with a central vein along the axis of the polygon and a number (typically between six and eight) of portal tracts at the vertices.

We develop a mathematical model of the microcirculation and interstitial flow in the liver, which we solve to estimate the uptake of lymph in the liver and the rate of collection of interstitial fluid in the peritoneal cavity. The model is based on earlier models developed by Bonfiglio *et al.* and Ricken *et al.* [1, 2], who treated the sinusoidal space as a homogeneous porous medium. Bonfiglio *et al.* were motivated by the effect of the high pressures that can develop after partial resection of the liver, and they used a two-dimensional model to investigate the flow within the cross-section of the lobule, considering in particular the effect of possible anisotropic permeability within the lobule, the fact that blood is non-Newtonian and the effect of tissue compliance. Ricken *et al.* also used an anisotropic model with dynamic remodelling of the vessels to investigate the regrowth of sinusoids after a partial resection.

The model is described in detail in [3]. We treat the sinusoidal space as a porous medium, and we treat the interstitial space as a separate porous medium. The two spaces are connected, that is, fluid flows from one to the other at a rate proportional to the pressure difference. We also account for uptake of interstitial fluid by the lymphatic vessels within the sinusoidal space. At the portal tracts and central veins we prescribe the blood pressure, whilst at the outer walls of the liver, we allow the interstitial fluid to flow out at a rate proportional to the pressure difference across the wall. We show that the presence of the boundaries only affects the flow and pressure to a depth of a few lobules into the liver; in the interior of the liver, the flux of interstitial fluid across lobule boundaries is insignificant. We therefore consider two models, firstly a model of a single lobule well into the interior, from which we estimate the total rate of uptake by the lymphatic vessels inside the liver (the effect of regions near the outer boundaries is insignificant). Secondly, we use a model of a group of lobules near the liver wall to estimate the total flux of interstitial fluid across the walls and into the peritoneal cavity.

The model predicts that, if the pressures within the liver are increased, such as would occur after resection and may occur as a result of impaired flow during disease, both the uptake of lymph from the liver and the rate of interstitial flow into the abdominal cavity increase significantly.

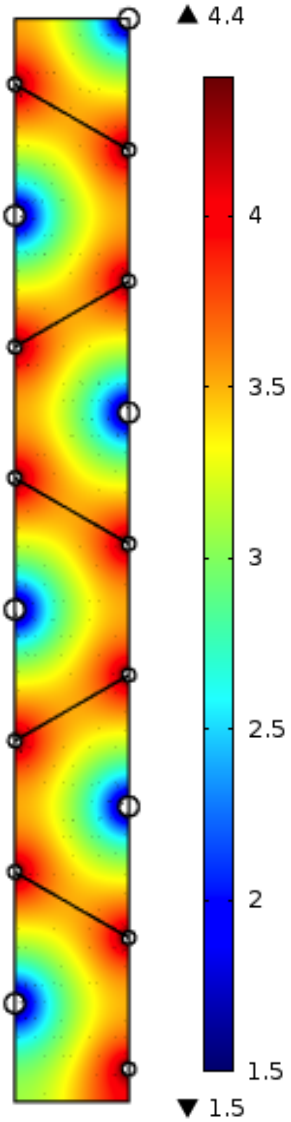

Figure 1: Predicted interstitial pressure near the Glissonian–peritoneal membrane, which in the plot is represented by the lower horizontal boundary of the domain (values in mmHg).

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