Author’s response

Considerations for a computer model for the hepatic circulation under chronic Budd-Chiari syndrome conditions

Dear Editor,

We appreciate the opinion of Dr Mancuso on our paper [1], which stated that the Budd-Chiari syndrome (BCS) has different aetiology in the West and the East [2]. While this disease is rare in the West and is presented more as acute hepatic venous (HV) thrombosis, it is usually a chronic consequence of HV thrombotic disease in the East [2]. We concur on Dr Mancuso’s viewpoint here, as well as that reported in Qi et al. [2].

As pointed out in our paper, the simulation for BCS reflects the hepatic circulation under acute BCS cases, whereby the right HV flow is blocked ([1], Fig. 4). The poor drainage in the right parenchyma leads to right portal flow congestion, and hence triggers the arterial buffer response [3]. This in turn causes increased right arterial flow ([1], Fig. 5). However, we were uncertain whether the left arterial flow increases. In our simulation it was the case, but the left arterial flow could be reduced due to the rise in the left portal flow. Thus, the total arterial flow could have small or little changes, as may be measured from Doppler ultrasound.

The development for a computer model for chronic BCS would require a significant increase in complexity that may not be able to be captured using a Simulink electric analog model. The need for the time occurrences of these clinical observations are also necessary. If the present model will be used as a foundation then the following thoughts will be very relevant.

Firstly the electric analog components would need to vary with the simulation time parameter. In the first instance, this will be a time-dependent resistor. How much, or the variation in the value of this resistor would probably not be clinically derived and will only be an assumption (e.g. linear). The capacitance and inductive values would be even more difficult to derive;

Secondly, the observation of intrahepatic venous collaterals bypassing the obstruction would mean that additional branches need to be implemented that are also time-dependent and must also be assumed to occur at times where this is completely unknown unless there is significant clinical data. The fact that Doppler ultrasound observes no significant changes in hepatic arterial flow is also likely because of this reason. The acutely increased arterial flow has been distributed to these collaterals.

In summary, indeed the model would need substantial improvements in order for it to model a clinical setting and this can be a future iteration of the model.

Conflict of interest

None declared.

Funding

This work was supported by the Auckland Medical Research Foundation (Project number 3713305).

Ethical approval

Not required

References


H. Ho ∗

Bioengineering Institute, The University of Auckland, Auckland 1010, New Zealand

C. Qiu

Department of Physiology, School of Medical Sciences, The University of Auckland, Auckland 1010, New Zealand

∗Corresponding author.

E-mail address: harvey.ho@auckland.ac.nz (H. Ho)

Received 14 June 2019
Accepted 16 June 2019