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Development of an in-silico cardiovascular model to investigate vascular refilling response during haemodialysis

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Introduction:

Intradialytic hypotension (IDH) is a frequent complication affecting around 30% of haemodialysis treatments, however its pathogenesis is not fully defined. We have developed and implemented a novel cardiovascular computer model that incorporates pulsatile flow, arterial and venous dynamics, the baroreflex response, and vascular refilling in order to simulate the effects of dialysis treatments. We use this model to investigate the mechanisms that may contribute to intradialytic instability.

Methods:

We have developed an in-silico model of the cardiovascular system, which acts as a 'virtual' patient receiving dialysis using the MathWorks MATLAB and Simulink packages. The model[1] can be qualitatively described as a lumped-component model, driven by a pulsatile cardiac output that pressurises both pulmonary and systemic vascular beds, and controlled by aortic and carotid baroreceptors through sympathetic and vagal pathways. We have developed an adjustable ultrafiltration and vascular refilling profile that is introduced to the system to emulate dialysis. Physiological properties were parameterised to represent patients with impaired cardiovascular functions such as reduced autonomic function, decreased arterial compliance and dampened baroreflex response. Patients aged >18 years were recruited from our prevalent dialysis population as part of the iTREND (Intelligent Technologies for Renal Dialysis) project, and had continuous non-invasive monitoring of haemodynamics using pulse wave analysis (Finapres NOVA) during dialysis treatment. The reconstructed central aortic waveform allows continuous calculation of a full range of haemodynamic variables that were compared to predictions from our model.

Results:

Our model produces similar intradialytic instabilities when compared to patient data collected during the iTREND study. Outputs of the model include flows, pressures, and volumes throughout the circulatory system, nervous pathway frequencies, and heart rate [figure 1a].
Our extended model has the ability to simulate the general haemodynamic responses of a virtual-patient undergoing simulated dialysis and vascular refill. We have further confirmed that this in-silico model is able to replicate similar pressure waveforms observed in haemodialysis patients as well as haemodynamic changes that would be expected of patients with impaired cardiovascular function [figure 1b].

Conclusions:

The model allows for the control and variation of a large quantity of discrete patient variables to facilitate the investigation of mechanisms that contribute to IDH. We aim to develop the model further in accuracy so

that it can be used to closely replicate a patient's physiological profile, allowing potential future treatments to be trialled first on the patient's 'digital twin'.