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Letter to the editor

Response to: 'Early worsening of cardiac function in Fontan patients'

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We thank the correspondents for their interest in our recently published paper about cardiac magnetic resonance (CMR) feature tracking in patients with a Fontan circulation [5]. Our goal was to assess ventricular strain in the Fontan circulation and to investigate the relationship between ventricular strain and cardiac systolic function over time.

We stipulate that to date, no 'gold standard' has been established for measuring functional decline in Fontan patients. There is increasing evidence that traditional cardiac function parameters such as ejection fraction (EF) and cardiac index (CI) do not correlate with attrition in the morphologically heterogeneous group of functionally univentricular hearts. As opposed to EF, a volumetric marker that does not correct for ventricular dilation, strain directly and independently measures deformation of the myocardium and has shown superiority by being able to detect changes in ventricular function before a fall in EF [1]. This study therefore adds valuable insight supporting the paradigm shift away from traditional cardiac function parameters towards the use of strain as a sensitive marker of intrinsic myocardial (dys)function in the Fontan population [2,3].

The correspondents correctly state that cardiac function is negatively affected by arrhythmias. Arrhythmias are indeed a common

occurrence in patients with a Fontan circulation [4]. However, due to the nature of our study, patients with severe arrhythmias requiring pacemaker or ICD therapy were excluded. Furthermore, in our original article, we did state that 6 of the 51 (11%) participants had a history of arrhythmias. All of those were atrial arrhythmias. Patients with a history of arrhythmias did have slightly lower, albeit not significant levels of strain. When comparing the overall change (Δ) of ventricular strain over the period of two years between patients with a history of arrhythmias and those without, no significant differences were found (Δ GLS no history of arrhythmia: -1.9 vs. Δ GLS history of arrhythmia: -1.3 , $P = 0.421$; Δ GCS no history of arrhythmia: -1.4 vs. Δ GCS history of arrhythmia: -1.0 , $P = 0.617$). Therefore, with due respect to the correspondents, we posit that we minimized the bias of arrhythmias negatively impacting our study results.

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None.

Declaration of competing interest

The University Medical Center Groningen contracts with Actelion and Lilly, for consultancy activities of Professor R.M.F. Berger, outside the submitted work. All other authors have nothing to disclose.

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¹ All above mentioned authors take responsibility for all aspects of the reliability and freedom from bias of the data presented and their discussed interpretation.