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Higher ventricular rate during atrial fibrillation relates to increased cerebral hypoperfusion and hypertensive events

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Background. The association between atrial fibrillation (AF) with cognitive impairment/dementia, independently of clinical cerebrovascular events (stroke/TIA), has been recently discovered. AF-induced transient critical hemodynamic events are plausible mechanisms of this association; however, the impact of ventricular rate during AF on cerebral hemodynamics is presently unknown.

Purpose. Aim of the present study was to compare cerebral hemodynamics during AF at different ventricular rates by a computational approach.

Methods. We simulate AF at different ventricular rates (50, 70, 90, 110, 130 bpm) by two coupled lumped parameter validated models of the systemic and cerebral circulation, respectively, and compared to corresponding control sinus rhythm (SR) simulations. Hemodynamic outcomes and occurrence of critical events (hypoperfusions and hypertensive events) were assessed along the internal carotid artery-middle cerebral artery pathway up to cerebral capillary-venous bed.

Results. Increasing ventricular rates relate to a reduced heart rate-related dampening of hemodynamic signals compared to SR (p=0.003 and 0.002 for flow rate and pressure, respectively) at the distal cerebral circle level (downstream middle cerebral artery). This response causes a significant progressive increase in hypertensive events and hypoperfusions in the distal cerebral circle (p<0.001) at progressively faster ventricular rates (see Figure, panel a, for arteriolar hypoperfusions, and Figure, panel b, for hypertensive events in the cerebral capillary districts). However, at the lowest ventricular response rates (50 bpm), a significant risk of transient hypoperfusions at the systemic-proximal cerebral circle level (up to middle cerebral artery) was demonstrated (p<0.001).

Conclusions. Higher ventricular rates relate to a progressive increase in critical cerebral hemodynamic events at the distal cerebral circle, while low ventricular response during AF can induce rare but potentially dangerous proximal cerebral hypoperfusions. The extent of rate control strategy could influence cognitive outcomes in patients with permanent/persistent AF.

Figure. Absolute frequency of hypoperfusions and hypertensive events during AF in the distal circle (downstream MCA) over 5000 computed heartbeats: (a) cerebral arteriolar level, (b) cerebral capillary level.
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