Mechanism behind sudden cardiac deaths in sports uncovered

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Researchers have worked out the mechanism behind sudden cardiac deaths that follow a hard blow to the chest.

Cardiac arrhythmias caused by a short, hard impact without visible lesions of heart muscle are called Commotio cordis, which can be responsible for sudden cardiac death after chest impact of baseballs and

ice hockey pucks in professional sports.

Commotio cordis is believed to be rare, but is one of the most frequent causes of sudden cardiac death among young, otherwise healthy athletes. The risk increases, the smaller and harder the contact is to the chest covering the heart. Previous studies on animals have shown that the window of time during the normal cycle of a beating heart in which Commotio cordis can lead to sudden death is very short indeed: only about 20 milliseconds, or 2% of the normal cardiac cycle.

The rare condition was responsible for the recent death of French blogger Rebecca Burger, who died after part of a compressed air powered cream dispenser bounced against her chest.

In a collaborative BHF-funded study, the cellular mechanism of Commotio cordis has now been elucidated. This involves activation of so-called ion channels - membrane proteins in heart muscle cells that trigger a new 'stray' wave when physically stimulated.

If, and only if, this occurs exactly at the boundary with a preceding wave edge, circular currents form, which prevent coordinated heart beats.

The results of the study have been published in the journal journal Circulation: Arrhythmia and Electrophysiology. The paper's senior author, Visiting Professor Peter Kohl at the Department of Physiology, Anatomy and Genetics at Oxford University, and Director of the Institute for Experimental Cardiovascular Medicine at the Universitätsklinikum Freiburg, comments: 'Our work shows that there is a critical window in which Commotio cordis can interfere with the heart's rhythm, which is limited by time as well as a defined space.

'Since the position of the trailing wave end of the preceding normal beat moves across the heart surface, a blow to the chest in everyday life is

generally inconsequential.'

The researchers also identified two types of excitation patterns, only one of which is susceptible to Commotio cordis.

First author, Professor Alex Quinn of the Department of Computer Science at Oxford and Dalhousie University, explains: 'To use the analogy of waves on a beach, water can either flow visibly back to the sea, or seep away into the sand. Only in the first case, i.e. only if there is a clearly defined boundary of the receding excitation wave, can sudden cardiac death be triggered, if the most unfavourable factors are combined. This combination of constraints involving time, location and critical wave pattern explains why the nature of Commotio cordis has remained obscure for such a long time.'

The research also identifies ways in which personal risk can be assessed for participants in sports such as ice hockey, baseball, lacrosse and cricket.

The full paper, 'Mechanically Induced Ectopy via Stretch-Activated Cation-Nonselective Channels Is Caused by Local Tissue Deformation and Results in Ventricular Fibrillation if Triggered on the Repolarization Wave Edge (Commotio cordis)' can be read in the journal *Circulation: Arrhythmia and Electrophysiology*.

More information: T. Alexander Quinn et al. Mechanically Induced Ectopy via Stretch-Activated Cation-Nonselective Channels Is Caused by Local Tissue Deformation and Results in Ventricular Fibrillation if Triggered on the Repolarization Wave Edge (Commotio Cordis), *Circulation: Arrhythmia and Electrophysiology* (2017). DOI: 10.1161/CIRCEP.116.004777