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Int J Cardiovas Res 2017, 6:5
DOI: 10.4172/2324-8602-C1-003

19TH ANNUAL CARDIOLOGY CONFERENCE

August 31-September 01, 2017 Philadelphia, USA

Mechanism of torsades de pointes elucidated in human AV block

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Two successive mechanisms may explain Torsades de Pointes (TdP) in patients with complete AV block. A phase 2 reentrant phenomena in adjacent side-to-side myocardial fibers based on dispersion of action potential duration to explain the first TdP beat this phenomenon is followed by a fast circus movement reentry in agreement with the leading circle concept with a speed only limited by the ventricular refractory period. The initiation of multiple rotors may result in VF because long episodes may lead to myocardial ischemia. However, intraventricular myocardial conduction blocks may explain that most TdPs stop spontaneously. Because this phenomenon needs a thin myocardial structure as demonstrated by optical mapping we suspect that its origin is located in the crista supraventricularis. Two exit sites of the circus movement can take place along the antero-superior and postero-inferior sulcia explaining the opposite orientation of the initial vectors observed at the beginning of most of the torsades. This is followed by a Wenkebach phenomenon on at least one of these two pathways. This may explain the feature of the twisting of the QRS tips around the isoelectric line. This theory is comforted by the abrupt change in the direction of activation suggesting a Mobitz type 2 blocks occurring on one of these two preferential pathways. The same mechanism can be observed on a reentrant loop around an anatomical obstacle producing a TdP-like arrhythmia in case of two exit sites of opposite directions.

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