Physical Sciences Seminar

The impact of turbulent blood flow on aortic valve calcification

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Calcific aortic valve disease (CAVD) is the most prevalent heart valve disease in western countries. In this disease, valvular interstitial cells (VICs) undergo a phenotype switch towards osteoblasts starting to produce bone tissue in the heart. This leads to heart failure and patient death. The only current treatment option is surgical valve replacement including all risks of heart surgery.
Heart valves are responsible for the unidirectional flow of blood during the cardiac cycle. The semilunar valves prevent the blood stream from retrograde flow into the ventricles during diastole, whereas the atrioventricular valves secure regurgitation from the ventricles into the atria. The pathology starts at the non-coronary sinus, the valve leaflet which is subjected to the highest oscillatory blood flow. In addition, patients with valve malformations resulting in increased turbulent blood flow show early development of calcific aortic valve disease. Laminar blood flow causes the endothelial cells to continuously produce nitric oxide (NO), which is believed to keep the VICs in a quiescent state. However, when turbulent flow occurs, endothelial cells start producing pro-inflammatory cytokines instead of NO leading to subsequent bone formation and cell death.
The field of mechanotransduction is working on identifying the pathways of how the mechanical stimulus is translated into a biological response. Although biological mechanisms behind the pathology are poorly understood, turbulent blood flow and mechanical strain clearly play a major role in the pathogenesis of the disease and provide a main risk factor for the development of CAVD. It remains unknown which patients finally develop CAVD and which do not. Identification of specific flow patterns and receptors sensing the mechanical strain would be of great clinical significance, as they could develop a novel target for the pharmacological inhibition of the development of the disease.

Monday, May 15, 2017 11:00am - 12:00pm
Mondi Seminar Room 1, Central Building

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