How to Predict Aortic Wall & Damage in Aortic Disease. The Role of Turbulence & How to Measure it!

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Declar. Conflict of Interest

...I do not have any disclosure to this subject & lecture.

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Abstract

Type A and B aortic dissections and aneurysms (TAAD) are potentially lethal diseases and clinical typically silent before they progressively expand until rupture or dissection occurs. Our understanding of the genetics of TAAD has increased exponentially in the last 20 years. 29 genes have been shown to be associated with TAAD development, although they are only the weakening factors of the aortic wall (AOW) in the extracellular matrix, in the smooth cell contraction and in their metabolism. Moreover genotype-phenotype correlations have been established for both syndromic and non-syndromic meaning.

We want to demonstrate here by our description and our hypothesis that the intimal and medial injuries of the AOW of TAAD are caused primarily only from ONE Type of Kinetic Force, which appears on the AOW in THREE different forms of dissection, such as: 1. Local (Pouch), 2. with Entry and Re-Entry or 3. without Entry. (Wall Haematom). It depends also from the severity of the above mentioned AOW weakening genetic factors!!!

(Wall Shear Stress and Wall Force are not significant factors in aortic dissections.)

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Aortic Valve Vorticis by Leonardo

Source: https://twitter.com/TetyanaMk/status/904750636427829249

The Aortic „Laminal Flow“ is in the Reality a Parabolic, Rotating Pressure & Blood Mass Wave Sliding on the Endothelium

The Endothelium is Multilayer Cell-, Non-Thrombotic-, & in the Sagital View – A Non Flat - Wave Form Surface -

By the Change of Aortic Geometry Changes Flow Turbulence-Core & Flow Kinetics Either, There is a Significant Side-Vortex Development !!!

The Aortic „Laminal Flow” is in The Reality a Rotating, Parabolic Pressure & Mass Wave Which is Sliding on the Endothelial Surface

Most Frequent Entry Line & Main Vortex Force Location in Typ „A”Aortic Dissection

Human Aortic Dissections and Aneurysmas are Developing Mainly Behind Side-Branches and Valves – Behind Turbulences & Vorticis -

Genetic Factors are Associated with Type A and Type B Aortic Dissections and Aneurysmas
Genetics Diseases are Important Associative-Weakening Factors of the Aortic & Vessel Wall in Type A and Type B Dissections. The Real Initial Cause is Always Kinetic Force.

Endothelial Structure Changes

Endothelial Surface Changes

Endothelial Migration in Static, Disturbed & "Laminal Flow"

Different Velocity & Turning Point of Turbulence after Ao. Root Replacement
Higher Flow Velocity Turn-Point & Side Vortex Formation (Sinus Valsalva) in the Dilated Ascending Aorta with BAV Valve

Different Velocity & Vortex Turning Point at Tricuspid Aortic Valve

Maximal Wall Stress Points in BAV

Maximal Wall Stress Point in TAV

Different LV–Asc. Aorta Angulation
Different Flow Velocity & Wall Forces

Different Location of Max. Pressure and Max. WSS in the Asc. Aorta
The Flow and Turbulance-Vorticis Shows Why?

The Numbers of Possible Re-Entries Are Dependent from the Size & the Location of the Main Entries

Different Aortic Arch Type, Form, Flow, Means Different Entry-Re-Entry Size & Location


Bare Stent Movement at Systolic and Dyastolic Flow

Inaccurate Stent Position Elevates Significantly Turbulences & Side-Vortex Formation
Different Phases of Vortex Merge

Energy Transfer at Vortex Merge
/Light, Heat, Velocity Changes, Kinetic Force/

At Vortex Merge: The Filaments Going to Create a Double-Helix

Visual & Kinetic Effects/Explosion/ at Vortex Collision in Fluids

Conclusion & Take Home Message

1. We need to develop in the future New Imaging Programs for NMR or CONTRAST TEE to transform visualise / these information into KINETIC VECTORS.!!!

2. We need : A PROGNOSTIC CALENDAR for Time Related Aortic Wall Changes based on Genetics and Patients Symptomatic.!!!

3. Device Needed : for IN VIVO ONLINE AORTIC & WESSEL WALL Resistance Measurement

We conclude here, that the Gradient between this significant Turbulence-Vortex Merge or Collision Generated Kinetic Force / Wall Index-Resistance is the REAL "Deciding Factor" about an endothelial injury or a real potentially fatal AOW dissection!!!

Vortex Generated Kinetic Force = Current Aortic Wall Resistance + Wall Index Gradient (+/-)

It will give us a realistic prognosis & can determine the severity of the aortic wall injury, from simple endothelium injury to the life threatening type A or type B aortic dissection.
Surface & Energy Changes /+&-/ at Vortex Merge-Collision (LIGO)

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