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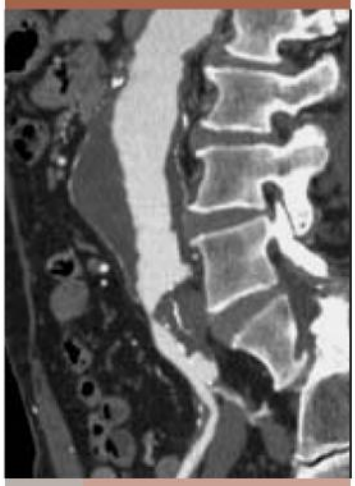


CARDIATIS—TEHMED

Appropriate Flow Modulator Sizing
Cardiatis/TehMED

# Flow Modulator Sizing

Thoraco-abodminal flow modulators sizing is almost always performed on the static CTA or MRA data sets. The fact is though that the static 2D data sets are taken during different points of the cardiac cycle and can therefore lead to problems in adequate modulator sizing. Dynamic imaging (e.g. ECG gated CTA) would be more appropriate and would subsequently lead to better clinical outcome. Dynamic imaging can also provide insights on the modulator behavior after being deployed.

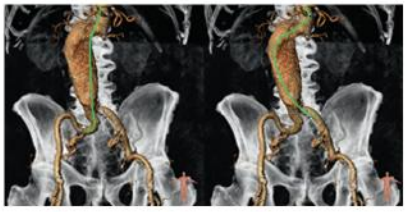
Cardiac-dependent aortic distensions are different at different aortic levels. Heerwarden in 2006 measured average 2mm of aortic distension (based upon MRA imaging) 3 cm above the renal arteries (0.7-4.2mm in range). Teutelink in 2007 used CTA imaging and measured an average aortic distension of 2mm at two different levels (2cm above and 1 cm below the renals). It is expected that the thoracic aorta would have even higher distentibility rate, given the fact that it expands more than the abdominal aorta and is bigger in diameter.

Keulen in 2009 reported inter-observer variability for abdominal aortic diameter of 1.6mm and Vos in 2010 an inter-observer variability of 0.81mm (based upon MRA). Other authors reported similar results of 0.27mm, 1mm and 1,8mm in MRA.

Adding the inter-observer variability on top of the aortic distentibility rate, it is expected that it may sum up to more than 5mm. This has to be taken into considerations when performing flow modulator sizing ahead of the intervention.

Another important issue in determination of an optimal modulator diameter is the proximal aortic neck. Length, diameter and angulation of the neck are the most important morphologic features to be accurately determined. It is known that the patients with angulated necks have more adverse outcomes, hence the neck should be investigated for its shape, length, diameter as well as the presence of thrombus, calcification and bulging.

It is important to estimate the straightening possibilities during the intervention, as it may optimize the sealing zone. Periprocedural aneurysm straightening increases the functional neck, which is desirable during the deployment. Calcification of the aneurysm neck, presence and morphology of the lumbar arteries in the AAA neck and the angle between the AAA neck and the iliac arteries will also determine the possibility of the aneurysm straightening. Another important factor is the stiffness of the guidewire that also causes straightening of the aneurysm tortuosity.



**Inside this issue:**



**Special points of interest:**

- *Flow modulator sizing is based upon the dynamic aortic imaging assessment and accurate determination of the aortic geometric properties.*
- *The aortic wall matrix proteins are being destructed by the enzymatic activity of the MMP family. Plasmin is the major activator of the MMP activity.*

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*TehMED, certified distributor of Cardiatis products for Slovenia*

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## *TehMED— Who Are We?*

***TehMED is a young and dynamic company based in Ljubljana - Slovenia. TehMED is the certified distributor of the Cardiatis (Isnes, Belgium) products for the Slovenian market. The company is primarily focused at the distribution of the medical products, used in the interventional radiology and minimally invasive surgery. Presentation of the Cardiatis product range can be done upon request to the Commercial and Marketing Department of the TehMED company .(see the contact data to the left).***

# The Enzymatic Basis of Matrix Destruction

Aortic vessel wall destruction starts with loss of elastin in the media and adventitia. The destruction effectors are the enzymes that are capable of the connective tissue degradation: elastin, collagen, fibronectin and laminin and proteogylcans. Most of these proteinases are released by the macrophages and mesenchymal cells, including the family of matrix metalloproteinases or matrixins (MMP). The major enzyme is a metalloproteinase.

As collagen failure is considered to be the most important event in the aneurysm formation, significant efforts were spent to investigate collagenolytic activity in the AAA wall. Insterstitial collagenase (MMP-1) is present in the AAA tissue (adventitia) and is produced by both macrophages and cells of mesenchymal origin.

MMP-3 (Stromelysin-1) is an activator of other proteinases (MMP-1 and MMP-9) is also present in the vessel wall.

Plasmin, a serine protease, is the major activator of the MMP family. Plasminogen binds to the extracellular matrix, where it is accessible to cell-surface receptors (e.g. urokinase-type plasminogen activator). This is an importanta step for activation of plasminogen in tissue.

Reilly at all. reported that tissue plasminogen activator (tPA) is abundant in AAA tissue by comparison with normal or occlusive disease aorta and that tPA is detectable in resident macrophages in the adventitia of AAA. The increase in the plasminogen activators without a concomitant rise in plasminogen activator inhibitors may play the delicate balance towards proteolysis of the matrix.

| | Other Names | Molecular Mass (kDa) |
|---|---|---|
| MMP-1 | Interstitial Collagenase<br>Vertebrate Collagenase | 42 |
| MMP-2 | Human 72-kDa Gelatinase<br>Type IV Collagenase | 66 |
| MMP-3 | Stromelysin<br>Proteoglycanase | 48 |
| MMP-7 | PUMP-1 | 19 |
| MMP-8 | Neutrophil Collagenase | 65 |
| MMP-9 | Human 92 kDa Gelatinase<br>Type IV Collagenase | 84 |
| MMP 10 | Human Stromelysin 2 | 47 |