Biomechanical studies on type B aortic dissection

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The Role of Branch Vessels in Aortic Type B Dissection An In-vitro Study

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ABSTRACT

Objective

In Acute type B Aortic Dissection (ABAD) patent false lumen portends a poor outcome. Patent branch vessels originating from the false lumen in an aortic dissection type B are assumed to contribute to persistent blood flow and patent false lumen. Therefore we studied the morphologic changes of the false lumen generated by different outflow rates in an in-vitro model.

Materials and Methods

An artificial dissection was created in two ex-vivo porcine aortas. A thin cannula was placed in the false lumen simulating a branch vessel originating from the false lumen. The aorta was positioned in a validated in-vitro circulatory system with physiological pulsatile flow (1500-2700ml/min) and pressure characteristics (130/70mmHg). The cannula was attached to a small silicone tube with an adjustable valve mechanism. Three different valve settings were used for creating outflow of the false lumen (fully closed, opened at 50% and fully opened at 100%). With time-resolved Magnetic Resonance Imaging measurements of lumen areas and flow rates were assessed. The experiment was performed twice in two different porcine aortas with a similar morphology in order to study reproducibility.

Results

Increasing antegrade outflow through the branch vessel of the false resulted in a significant ($p < 0.01$) increase of the mean false lumen area at the proximal and distal location in both models. The distal false lumen expanded up to 107% in case of high outflow of the false lumen through the branch vessel.

Conclusion

In this in-vitro study, we showed that increasing antegrade outflow through branch vessel originating from the false lumen when no distal re-entry tear is present results in an expansion of the cross-sectional false lumen area.

INTRODUCTION

Uncomplicated Acute type B Aortic Dissection (ABAD) is still associated with a 30-day mortality of approximately 10%.[1] Although the introduction of thoracic endovascular aneurysm repair (TEVAR) showed promising results in complicated ABAD, best medical treatment (BMT) is still todays treatment for uncomplicated ABAD. The acutely dissected aorta is fragile and TEVAR can result in malperfusion, ischemia, retrograde dissection, rupture and finally even perioperative mortality. The results of the ADSORB trial, as the first randomised comparison between acute (< 14 days) endovascular surgery and BMT for uncomplicated ABAD, showed only that aortic remodelling after one year was in favour of endograft placement. The ADSORB trial did not show an improved 1 year survival rate (although not powered for survival).[2]. The INSTEAD trial, as the first randomized comparison between elective endovascular surgery and BMT, justified medical management in the early phase for uncomplicated chronic ABAD from 2 to 52 weeks of onset.[1] For stable survivors of acute type B dissection, benefits of TEVAR begin to show after 2 years of follow-up.[3, 4] Although the preliminary results of the uncompleted ABSORB trial did not show any beneficial effect of early TEVAR, theoretically early TEVAR might save the lives of around 10% of patients, minus the induced perioperative mortality by TEVAR, with initially uncomplicated ABAD treated with BMT.[2] Therefore, identification of clinical and imaging predictors of poor prognosis in uncomplicated ABAD seems mandatory to select patients who will benefit from early TEVAR.

In the acute ABAD the false lumen may remain patent, thrombose, recommunicate with the true lumen through fenestrations, or rupture. Complete false lumen thrombosis is a major predictor of prognosis because it excludes the false lumen from the circulation and is thought to be a prerequisite for complete healing in the long run.[5,6] Incomplete thrombosis or patent false lumen portends a poor outcome.[7-9] The occurrence of thrombosis in the false lumen depends on coagulability, endothelial injury/dysfunction and blood flow. The blood flow in the false lumen is highly variable due to morphological differences between various types of dissections.[10] It is conceivable that patent branch vessels originating from the false lumen in an aortic dissection type B may contribute to persistent blood flow and patent false lumen, and thus to prognosis. Therefore, an in-vitro study was performed using two ex-vivo porcine aortas, both with a surgically-constructed false lumen and an adjustable outflow branch. We studied the morphologic changes of the false lumen generated by different outflow rates and hypothesize that increased outflow through the branch vessel originating from the false lumen will result in an increase of the false lumen.

MATERIALS AND METHODS

Aortic dissection model

Two fresh porcine aortas were obtained from the abattoir and within 4 hours prepared as follows: from the aortic arch to the iliac bifurcation all side branches were ligated with prolene 5.0. At the descending aorta a transverse semi-circular incision was made though all vessel layers and a dissection was surgically created in the media layer resulting in a false and true lumen with a dissection flap. The surgically created false lumen in the media may be considered comparable to a human aortic dissection (Figure 1). A thin plastic cannula (diameter of 2 mm) was placed in the false lumen simulating a branch vessel originating from the false lumen. The morphology of both models is representative for an aortic dissection type B with no distal tear or partial thrombosis occluding distal tears, impending outflow resulting in a blind sac from where a single branch vessel originates (Figure 2). The models were stored in a refrigerator for approximately 48 hours and subsequently defrozen before the start of the experiments.

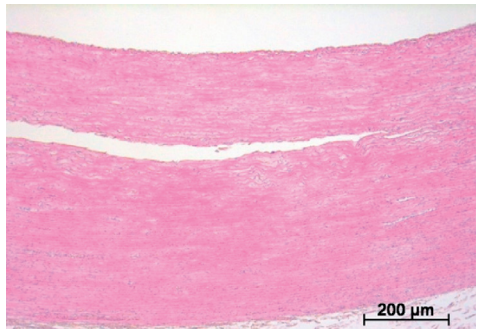

Figure 1. Microscopy after hematoxylin and eosin staining for the histological evaluation of the artificially created false lumen. The false lumen is created between intima with partial thickness media and partial thickness media with adventitia.

The cannula simulating a branch vessel was attached to a small silicone tube with an adjustable valve mechanism enabling a setting of variable outflow rates. Three different valve settings were used: fully closed, opened at 50% and fully opened at 100% for creating antegrade outflow of the false lumen. Magnetic Resonance Imaging (MRI) measurements were performed (details are given below) at four equidistantly spaced locations (32 mm apart) and perpendicular to the aortic model: proximally to the dissection, at the beginning of the dissection, half way of the dissection and distally to the dissection. A schematic representation of the models and measurement locations is presented in Figure 2**.**

In Model 1 data were acquired first with the valve setting fully closed, next opened at 50% and then fully opened at 100%. For Model 2, data was acquired with valve settings in inverse order, preventing a potential bias due to the duration of the experiment.

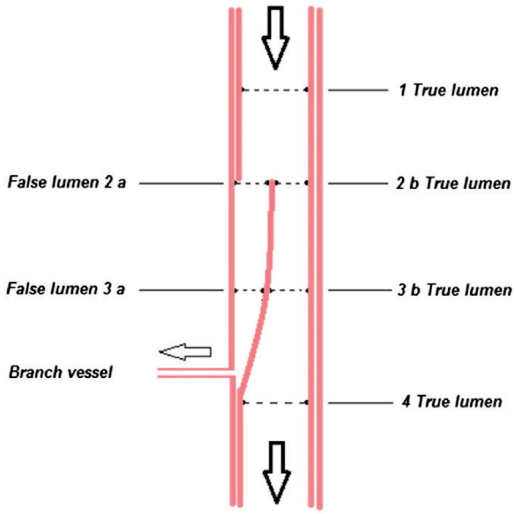

Figure 2. A schematic representation of the model. The four imaging planes are indicated and the arrows represent the flow direction.

In-vitro circulatory system

A validated in-vitro circulatory system with physiological flow and pressure characteristics was used to simulate the human circulatory system.[11-13] The main components of this circulatory system are: a pneumatically-**driven** pulsatile pump with periodic triggering connecting to the MRI system for synchronization, simulating ECG-triggering, a compliance chamber and a watertight synthetic box with the aortic dissection model (Figure 3). All components are connected by a silicone tubing system. The flow depends on the resistance in the in-vitro model and varies from 1500-2700ml/min. Water doped with a low concentration of gadolinium-based contrast (Dotarem, Guerbet) to shorten T1 relaxation time was used as circulating fluid. During the experiments the pneumatically-**driven** pulsatile pump was set on a fixed frequency of 68 beats/minute and at the start of each beat, a trigger was produced by the triggering unit connected to the MRI system. The pressure of the circulated water was 130/70mmHg and was calibrated several times.

The synthetic box with the aortic dissection model was placed inside the MRI gantry and filled with water, resulting in a submerged aortic dissection model. The antegrade outflow through the simulated branch originating from the false lumen vessel was redirected to the main reservoir.

Imaging

Time-resolved multi-slice two-dimensional imaging was performed with a 1.5T MRI system (Gyroscan, Philips Medical Systems, Best, The Netherlands) at four equidistantly spaced locations perpendicularly positioned to the aorta model as indicated in Figure 2. The spatial resolution used in the MRI protocol was $1.2 \times 1.2 mm^2$. Phase-contrast velocity-encoding was performed, resulting in both anatomical images and velocity images of the through-plane flow. Specific imaging parameters

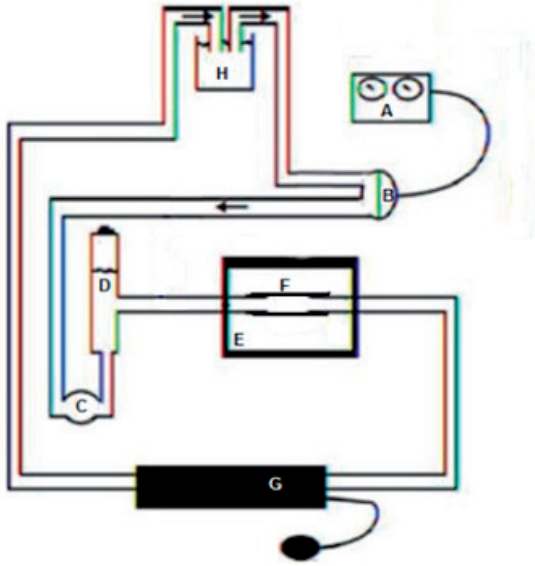

Figure 3. Circulation set up. A schematic representation of the circulation set up, which consisted of (A) an artificial pneumatic heart driver, (B) a tube to left ventricle, (C) a left ventricle, (D) a silicon tube, (E) a ball valve, (F) an air chamber, (G) the aortic dissection model (see Fig. 2), (H) a watertight synthetic box, (I) a blood pressure cuff, and (J) an open reservoir.

were: echo time 3.0ms, repetition time 5.0ms, flip angle 20°, slice thickness 8mm, field-of-view 300×150mm^2, two signal averages and velocity-encoding with sensitivity of 120 cm/s. Retrospective gating was used with a total of 160 phases reconstructed. Image analysis was performed using in-house developed and validated software and manual contour segmentation. [15] Cross-sectional area distention of the aorta was determined from the anatomical images acquired at each location. Therefore, the lumen area of both the true and false lumen were manually segmented in each phase of the cardiac cycle for a total of 160 phases. Lumen area (in mm^2) versus time-graphs were determined.

Statistical analysis

The cross-sectional lumen area (in mm^2) at all four locations in the aortic dissection model (Figure 2) were determined for all three valve settings, creating different false luminal outflow. Paired t-tests were used to determine statistical significance in lumen area change at each location in the aortic dissection model during different false luminal outflow. Statistical significance was assumed at $P < .05$.

RESULTS

The general flow characteristics of the in-vitro circulation in both models are presented in Table 1. Both models had a similar morphology but differed in lumen diameter resulting in a different vascular resistance and thereby resulting in different general flow characteristics (Table 1 and 2). The mean cross-sectional lumen area proximal to the dissection increased when the outflow settings were adjusted in model 1 but not in model 2 (Table 2).

Table 1. General flow characteristics for models 1 and 2.

In vitro circulatory system flow characteristics			
		Model 1	Model 2
Heart beat/min		68	68
Mean stroke volume/beat		40 mL	21.5 mL
Stroke volume/min		2,720 mL	1,460 mL
False lumen outflow/min	Valve setting Closed		
	50%	31 mL	24 mL
	100%	86 mL	58 mL

Table 2. Data summary of mean ± SD lumen area for the three different outflow conditions in models 1 and 2

Outflow by branch vessel vs. false lumen area							
Scan plane area	Outflow by branch vessel	Model 1			Model 2		
		0	0.5 mL/beat	1.3 mL/beat	0	0.5 mL/beat	0.9 mL/beat
True lumen 1		249.4 ± 38.0	274.1 ± 15.4	274.0 ± 15.3	159.6 ± 3.8	165.3 ± 16.6	150.3 ± 18.6
False lumen 2 A (mm^2)		70.3 ± 32.2	72.9 ± 31.7	75.3 ± 25.6	11.2 ± 8.9	14.8 ± 10.3	22.9 ± 11.9
True lumen 2 B (mm^2)		198.3 ±11.2	187.6 ± 7.3	179.2 ± 13.7	157.2 ± 13.9	159.3 ± 14.1	151.9 ± 21.9
False lumen 3 A (mm^2)		46.5 ± 8.2	52.2 ± 5.8	56.7 ± 5.7	10.2 ± 6.2	13.4 ± 8.8	21.1 ± 14.8
True lumen 3 B (mm^2)		180.1 ± 21.4	173.3 ± 18.7	165.8 ± 18.6	137.7 ± 15.1	143.2 ± 11.6	137.8 ± 12.2
True lumen 4 (mm^2)		165.7 ± 41.5	190.8 ± 38.6	186.9 ± 40.4	125.4 ± 14.9	128.3 ± 15.5	120.9 ± 13.9

The proximal imaging plane was positioned at the opening of the dissection at location 2 (Figure 2). At this location, the cross-sectional area of the false lumen in model 1 expanded with more than 7% (70.3mm^2 to 75.3mm^2; Figure 4) when the outflow of the false lumen increased to 3.3% of the mean stroke volume (1.3ml/beat versus 40ml mean stroke volume). In model 2 the cross-sectional area of the false lumen at the distal location (i.e., location 3) expanded with more than 104% (11.2mm^2 to 22.9mm^2; Figure 5 and 6) when the outflow of the false lumen was increased to 4.2% of the mean stroke volume (0.9ml/beat versus 21.5ml stroke volume).

Increasing the outflow through the branch vessel of the false lumen resulted in an increase of the mean cross-sectional false lumen area in both models (Figure 4 and 5), which was statistically significant ($P < .05$) for the three outflow settings. The range in lumen area changed during the three different outflow conditions with the largest range observed in model 2 (Figure 4 and 5).

In the proximal acquisition plane of the true lumen, the mean cross-sectional lumen area changed for the three different outflow conditions (2 B, Figure 2), however no correlation was found (Table 2.).

Distally in the false lumen (3A, Figure 2), an increase in mean lumen area was measured when the outflow of the false lumen was adjusted (Figure 4 and 5). This increase is statistically significant ($P < .05$) for the three outflow settings in both models. The distal false lumen area in model 1 expanded with more than 21% (46.5mm^2 to 56.7mm^2; Figure 4) when the outflow of the false lumen was increased to 3.3% of the mean stroke volume (1.3ml/beat versus 40ml mean stroke volume). In model 2 the distal false lumen area expanded with more than 107% (10.2mm^2 to 21.1mm^2; Figure 5) when the outflow of the false lumen was increased to 4.2% of the mean stroke volume (0.9ml/beat versus 21.5ml stroke volume). The range of the lumen area remains consistent for the three different outflow conditions in model 1 but showed an increase in model 2 (Figure 4 and 5).

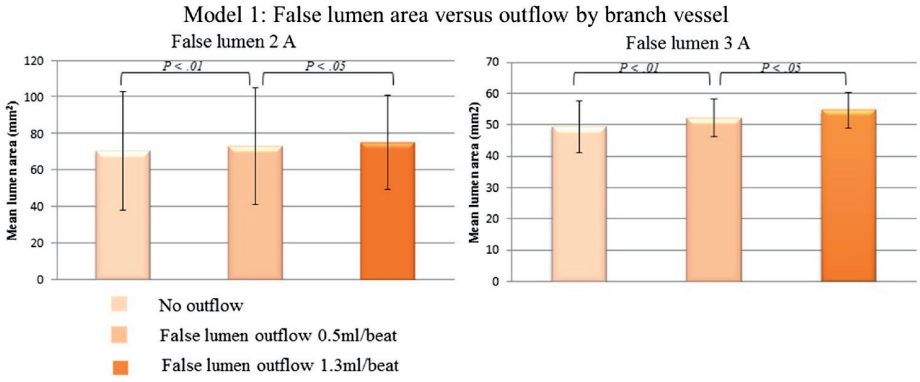

Figure 4. Model 1. The mean ± SD area of the proximal and distal plane in the false lumen for the three different outflow conditions. False lumen outflow result in significant area increase of the false lumen (2A and 3A).

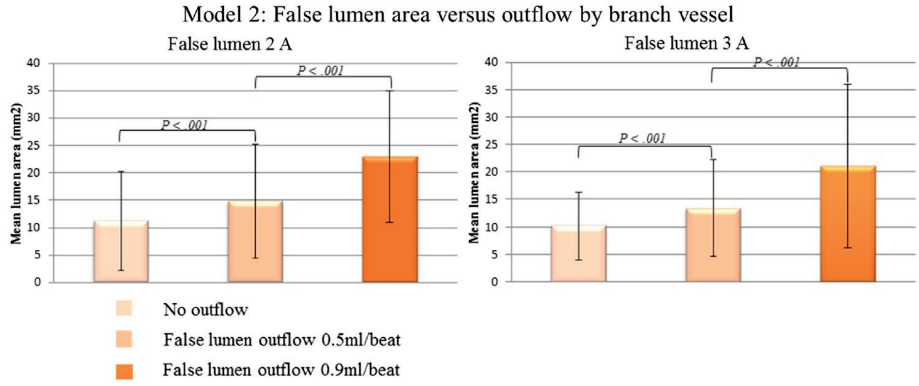

Figure 5. Model 2. The mean ± SD area of the proximal and distal plane in the false lumen for the three different outflow conditions. False lumen outflow result in significant area increase of the false lumen (2A and 3A).

For the distal acquisition plane (3 B, Figure 2), a statistically significant decrease ($p < 0.01$) of the mean lumen area of the true lumen was measured in model 1 when the outflow of the false lumen was increased (Table 2.). In model 2, the measured true lumen area remained stable at the highest false luminal outflow (Table 2.).

For both models, the mean lumen area measured in the acquisition plane 4 distalto the dissection (4, Figure 2) changed during the three different outflow conditions, however no correlation was found.

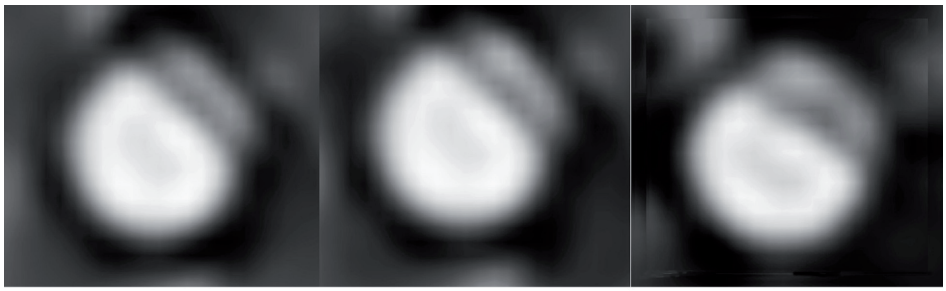

Figure 6. Magnetic resonance angiography images at the same cardiac phase of the distal false lumen in model 2 visualizing the expansion of the false lumen. (A) No outflow; (B) outflow 0.5 mL/second; (C) outflow 0.9 mL/second.

DISCUSSION

Patients with uncomplicated ABAD have an overall in-hospital mortality of around 10% despite best medical treatment. [7, 16, 17] Once complications occur the prognosis declines, with hospital mortality greater than 50%.[18] Early TEVAR might save theoretically the lives of around 10% of patients with initially uncomplicated ABAD although the preliminary results of the uncompleted ABSORB trial did not show any beneficial effect of early TEVAR. [2 19]

Therefore, identification of clinical and imaging predictors of poor prognosis in uncomplicated ABAD seems mandatory to select patients who will benefit from early TEVAR. Although theoretically saving lives the opposite effect of TEVAR is the procedure induced complications like malperfusion, ischemia, retrograde dissection, rupture and finally even perioperative mortality.

Complete false lumen thrombosis in ABAD excludes the false lumen from the circulation and is a major predictor of prognosis.[5, 6] However, ABAD with patent false lumen including partial thrombosis presents a high risk of complications. [9, 20, 21] The occurrence of incomplete thrombosis will depend on the presence of flow in the false lumen which is maintained by outflow of patent branch vessel(s) and / or fenestrations including re-entries of the dissection flap. Outflow by patent branch vessel(s) results in continuing flow in the false lumen and may contribute to adverse events. We hypothesized that increased outflow by branch vessels originating from the false lumen

results in a larger false lumen. To study this hypothesis an in-vitro study with circulatory flow was performed in which ex-vivo porcine aortas were included with a surgically-constructed false lumen and to which a branch vessel was added originating from the false lumen with an adjustable outflow, representing an uncomplicated ABAD (Figure 2). To validate reproducibility of our results, the experiment was performed in two different porcine aortas with similar morphology.

Ex-vivo models are useful for studying haemodynamics of aortic pathologies, as individual factors in a complicated circulation system can be isolated and analysed. In this study, we applied a porcine aorta as 'modelled' aorta, replacing the synthetic polymer or silicon tubing used in previous in-vitro studies. [22, 23] Additionally we used MRI (including simulating ECG-triggering synchronization) for time-resolved imaging, which was not described previously in in-vitro studies. [22-25] The presented porcine aorta model morphology is representative for dissections with no distal tear or with partial thrombosis occluding distal tears, impending outflow resulting in a blind sac.[20]

Our model confirmed that the cross-sectional area of the false lumen expanded when outflow of the false lumen was present through a branch vessel originating from the false lumen. The expansion of the false lumen was observed in both models and in both acquisition planes of the false lumen, proximal and distal in the dissection. The largest expansion was observed in the distal location of the false lumen in model 2, with an area expansion of more than 107%.

Translation of the results from our in-vitro model to a physiological situation is of course limited and therefore, evaluation of patients should be performed to confirm our findings. Additionally, in part due to limitations inherent to replicating in vivo conditions, preclinical testing has a limited ability in reproducing clinical settings.[25] Therefore, following limitations of the pulsatile flow model need to be acknowledged: water was used as a circulatory medium instead of blood, which is a thrombotic medium. Spontaneous thrombosis could either block the tubing system or disturb the function of the pulsatile pump.[24] The applied pulsatile flow was not equal to an aortic flow. Also, the aortas were prepared, frozen and after two days de-frozen. This might have altered the elastic properties of the arterial wall. Furthermore, the absence of dissected lamella might have influenced the outflow vessel. Implementation of lamella in a model would be too complex and would result in many variables. Next, the aortic model was submerged in water without support, which is not be representative for the connective tissue normally surrounding the aorta. Next, only two porcine aortic models were used in the experiments and more aortic models would result in more accurate data. Finally, we studied a small spectrum of outflow by branch vessel originating from the false lumen although these outflow settings already resulted in a significant increase of the false lumen area.

In conclusion, our in-vitro study showed that outflow through a branch vessel originating from the false lumen in an aortic dissection results in expansion of cross-sectional false lumen area. False lumen expansion might result in higher stress in the aortic wall, increasing the risk of dilatation which contributes to the conversion of uncomplicated into complicated ABAD. Our findings suggest that initially uncomplicated ABADs with no distal entry tear, but patent branch vessels

originating from the false lumen, have a higher risk for complications and that in such cases, early TEVAR may be considered.

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