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Analysis of flow dynamics in right ventricular outflow tract

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ARTICLE INFO

Article history:
Received 6 October 2014
Received in revised form
19 January 2015
Accepted 3 March 2015
Available online 14 March 2015

Keywords:
Pulmonary artery
Right ventricular outflow tract
Computational fluid dynamics modeling

ABSTRACT

Background: The mechanism behind early graft failure after right ventricular outflow tract (RVOT) reconstruction is not fully understood. Our aim was to establish a three-dimensional computational fluid dynamics (CFD) model of RVOT to investigate the hemodynamic conditions that may trigger the development of intimal hyperplasia and arteriosclerosis.

Methods: Pressure, flow, and diameter at the RVOT, pulmonary artery (PA), bifurcation of the PA, and left and right PAs were measured in 10 normal pigs with a mean weight of 24.8 ± 0.78 kg. Data obtained from the experimental scenario were used for CFD simulation of pressure, flow, and shear stress profile from the RVOT to the left and right PAs.

Results: Using experimental data, a CFD model was obtained for 2.0 and 2.5-L/min pulsatile inflow profiles. In both velocity profiles, time and space averaged in the low-shear stress profile range from 0–6.0 Pa at the pulmonary trunk, its bifurcation, and at the openings of both PAs. These low-shear stress areas were accompanied to high-pressure regions 14.0–20.0 mm Hg (1866.2–2666 Pa). Flow analysis revealed a turbulent flow at the PA bifurcation and ostia of both PAs.

Conclusions: Identified local low-shear stress, high pressure, and turbulent flow correspond to a well-defined trigger pattern for the development of intimal hyperplasia and arteriosclerosis. As such, this real-time three-dimensional CFD model may in the future serve as a tool for the planning of RVOT reconstruction, its analysis, and prediction of outcome.

1. Introduction

Structural deterioration of the right ventricular outflow tract (RVOT) conduit is a well-known phenomenon. Reintervention rates because of intimal hyperplasia and arteriosclerosis of the implanted valved conduit are between 20% and 25% [1,2] in the short term and in the mid term as high as 75%. This is independent on the type of graft used and its biological preparation [3,4]. In recent literature, there are numerous reports that deal with the mechanism of early and late failure of the valved RVOT conduit. To date, no clear explanation for this relatively common phenomenon was found. Young age, small body surface area, and small graft size have been detected as independent risk factors for graft failure [5,6]. Graft size and patient age are in reverse relation, somatic growth may explain graft undersizing with time. However, independently

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http://dx.doi.org/10.1016/j.jss.2015.03.001

on the impact of somatic growth, it is common in almost all failed conduits to have well-localized intimal hyperplasia resulting in stenosis at a distal anastomosis site, pulmonary artery (PA) bifurcation, or entry of the PAs [3,6]. It is well known that local hemodynamic conditions may influence the endothelial function and as thus maybe considered as one of the key factors for induction of intimal hyperplasia.

However, to date, the role of hemodynamic elements in intimal hyperplasia in RVOT has not been investigated in complete detail. In our previous report, alteration of the local shear stress and pressure profiles in RVOT was shown [7]; however, this study was conducted under nonphysiological steady-state flow conditions [7]. Therefore, our aim was to consider physiological conditions in a pediatric heart by establishing a real-time three-dimensional computational fluid dynamics (CFD) model of the RVOT and PAs. This new insight into the RVOT physiology may facilitate the evaluation of local hemodynamic conditions recognized as a trigger of local intimal hyperplasia and also serve us as a tool for analysis and planning of surgical procedures at RVOT and prediction outcome after valved conduit implantation.

2. Material and methods

2.1. Animal care and protocol

The protocol was approved by the Committee on Animal Care, Office Vétérinaire Cantonal, Lausanne. All animals received care in compliance with the Principles of Laboratory Animal Care formulated by the National Society for Medical Research and the *Guide for the Care and Use of Laboratory Animals* prepared by the National Academy of Sciences and published by the National Institutes of Health (NIH Publication No. 80–23, revised 1985).

Ten domestic pigs with a mean body weight of 24.9 ± 0.7 kg were used. After general anesthesia with volatile anesthetics, the animals were equipped with a jugular central venous catheter and a femoral arterial catheter for hemodynamic monitoring and five lead electrocardiography.

2.2. Steady-state measurements

After the median sternotomy was completed, the pulmonary root, PA, and both PAs were dissected. Flow measurement under normal hemodynamic conditions (heart rate of 90 per min and arterial pressure 120 per 75 mm Hg) were performed with 14-mm PeriVascular flow probes (Medistim ASA, Oslo, Norway) at the pulmonary trunk and with 12 -mm probes at the left and right PAs. Invasive pressure measurement was carried out in the pulmonary root at the infravalvular and supravalvular positions; at the PA; at the bifurcation of the pulmonary trunk; and at the left and right PAs, as described previously [7].

2.3. Cardiopulmonary bypass and perfusion protocol

Cardiopulmonary bypass (CPB) was established using 16 and 18F venous cannulas (DLP; Medtronic Inc, Minneapolis, MN) for superior and inferior vena cava cannulation and 14F

Table – Repartition of the mean velocity and shear stress values in the RVOT, PA, and both its branches. Note that the mean velocity values are almost identical in three different inflow areas along the pulmonary tract. The same may be stated for the shear stress; however, shear stress is augmented at bifurcation and both PAs.

Area	2.0-L/min inflow profile						2.5-L/min inflow profile					
	Velocity (m/s)		Shear stress (Pa)		Pressure (Pa)		Velocity (m/s)		Shear stress (Pa)		Pressure (Pa)	
	Mean	Max/min	Mean	Max/min	Mean	Max/min (%)	Mean	Max/min	Mean	Max/min	Mean	Max/min (%)
RVOT	0.30	0.96/0.05	0.33	1.68/0.17	2554.03	1692.9/3045.9 (55.6)	0.37	1.18/0.07	0.42	2.33/0.11	3143.21	1734.23/3925.68 (44.2)
Infravalvular area	0.41	1.68/0.09	1.47	2.29/0.17	2506.07	1571.6/3045.9 (51.6)	0.51	2.11/0.08	2.03	2.92/0.17	3084.56	1546.28/3925.68 (39.4)
Supravalvular area	0.37	1.77/0.028	0.35	2.44/0.01	1475.63	1466.3/1617.29 (90.6)	0.48	2.15/0.09	0.56	4.12/0.11	1483.63	1468.96/1679.58 (87.4)
Pulmonary trunk	0.38	1.73/0.02	0.69	3.84/0.01	1479.63	1466.3/1652.9 (88.7)	0.49	2.06/0.09	1.13	4.90/0.14	1483.63	1468.96/1744.89 (84.2)
Bifurcation	0.38	1.85/0.02	2.13	6.13/0.02	1483.63	1466.3/1572.94 (93.2)	0.50	2.21/0.15	3.02	7.93/0.38	1498.29	1468.96/1652.92 (88.8)
Right PA	0.37	2.20/0.05	1.17	5.17/0.04	1474.29	1452.97/1502.29 (96.7)	0.54	2.64/0.15	1.71	6.38/0.16	1479.63	1466.3/1511.6 (97)
Left PA	0.38	2.18/0.03	2.68	6.85/0.01	1464.97	1452.97/1479.63 (98.2)	0.40	2.63/0.13	3.87	8.50/0.05	1467.63	1466.3/1511.6 (97)