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Key Words: cardiac magnetic resonance imaging, superior vena cava, bidirectional cavopulmonary shunt, takedown

Discussion

Presenter: Dr Shuhua Luo



Dr Neil Cambronerero (*San Francisco, Calif*). Thank you, Dr Luo. My name is Neil Cambronerero. I'm a surgeon at UCSF. I apologize that Dr Mohan Reddy couldn't be here; he sent me in his place. Dr Luo, you are to be commended for these works, which contribute to the armamentarium by

which we as surgeons, can use to better risk-stratify our patients, inform families, and improve decision-making in this challenging group of patients. The current study was undertaken to investigate how SVC flow at the time of BCPS affects clinical outcomes, and is a continuation of work initially presented by your group at the STS Annual Meeting in 2012.

In that initial study, 19 SV patients who had undergone BCPS over a 2-year period from 2009 to 2011 were evaluated. Patient characteristics and pre-BCPS flows were in line and very close to what you found in your study. In that initial series, there were 2 patients with indexed flows of less than 1 L/min/m², who either died or had BCPS takedown for hypoxia. There was 1 patient who had near-equal pre-shunt flows, however, and this patient was found to have significant aortopulmonary collateral flow at the time of the BCPS. The investigators concluded that the SVC flow might be a novel marker for post-BCPS failure or death.

The current study evaluates 65 patients who underwent BCPS over a 5-year period, from 2012 to 2017, and confirms the findings of your original work, that indeed, pre-BCPS flow, measured using cardiac MR, can be used as a marker to predict post-BCPS arterial oxygen saturation and clinical outcome. The mean age at BCPS placement was 6.5 months and weight was 6.9 kg. Each patient had a cardiac MR, cath, and TTE as a pre-BCPS evaluation. The mean indexed flow was 1.7 L/min/m², and total flow was 0.5 L/min. The median follow-up was 17 months. Pre-operative SVC flow was positively correlated with post-BCPS saturations, and low SVC flow was shown by your group to be a significant risk factor for BCPS takedown and/or death.

In your series, there were 7 patients who underwent takedown of the shunt. Of those 7, 5 eventually died. Additionally, there were 4 deaths after BCPS placement in patients who did not have their BCPS taken down, with a total of 9 mortalities, two-thirds of these dying with severe hypoxia.

You have concluded that SVC flow calculated using cardiac MR can be used as a predictor of postoperative saturations, and although you could not determine an exact cutoff value of SVC flow, for BCPS failure you have identified with absolute flows of less than or equal to 0.5 L/min to be associated with BCPS takedown and death. Again, I commend you for this work. I have 2 comments, each followed by a question.

I don't see in your data anything regarding pulmonary vein oxygen saturations obtained during the pre-BCPS cardiac catheterization. In our experience, when we perform a pre-BCPS cath in these patients, we see a fair number of individuals who have low pulmonary venous saturations, but who respond to oxygen. These patients have some form of lung disease, and we often will find chronic aspiration, subclinical infection, veno-venous collaterals, or parenchymal disease. Can you comment on these patients who had either BCPS takedown or death and their pre-shunt pulmonary venous saturations? Obviously, regardless of the amount of flow after BCPS placement, low pre-shunt pulmonary venous saturations will result in low post-BCPS saturations.

For my second comment and question, in your cohort there were 65 patients and 9 deaths. Five of the deaths were hypoplasts, and there were 23 hypoplasts in the group.

Can you comment on the hypoplast mortalities and the type of stage 1 palliation these patients who died had undergone?



Dr Shuhua Luo (*Toronto, Ontario, Canada*).

Those are very good questions. So, I think the postoperative desaturation is multifactorial, and of course the pulmonary vein desaturation is a very important factor. Unfortunately, we cannot collect these data in the time of the patient getting desaturated.

The time of cath is 1.5 months before the BCPS placement, obviously, the pulmonary vein saturation collected during the cath is not related to postoperative desaturation. In the future, pulmonary vein desaturation will be further investigated in patients who had desaturation after BCPS placement.

For the HLHS patients, we have a relatively higher number of deaths among these patients. Three patients had low saturation needing BCPS takedown. One patient had AV valve regurgitation, and 1 patient had massive GI bleeding. One factor to consider is that this was a relatively small cohort. We have a higher number of AV valve regurgitation in this cohort. This is probably one contributing factor for higher number of deaths in HLHS patients.