seemed to be determined not only by smaller shunts but also by perioperative factors. Strategies to improve survival may include lower postoperative hematocrit and higher pH to promote shunt flow.

Webcast
You can watch a Webcast of this AATS meeting presentation by going to: https://aats.blob.core.windows.net/media/18Apr29/30ABC%20Congenital%20SS/SS2_2.mp4.

Conflict of Interest Statement
Brizard is a member of the Admedus Advisory Board. All other authors have nothing to disclose with regard to commercial support.

References
22. Brink J, in your review of approximately 300 patients over a 10-year period, you demonstrated that among the 1 of 5 patients who died before reaching the superior cavo-pulmonary anastomosis stage, the majority of the deaths

Discussion
Dr M. Jacobs (Baltimore, Md). Dr Brink and colleagues in Melbourne have reported on associations among acute postoperative events, shunt failure associated with acute events, and mortality after palliative shunt procedures in patients with univentricular cardiac anomalies. This is certainly an important topic. The spectrum of functionally univentricular hearts represents a large and challenging segment of the overall congenital heart disease population, and their management involves arguably the largest fraction of human and institutional resources.

Shunt procedures are technically demanding, and the resultant physiology is potentially labile, being prone to maldistribution of blood flow from the single ventricle to the systemic and the pulmonary circulations. Although we may think of a particular shunt as being “too large” or “too small,” we should realize that the same polytetra-fluoroethylene tube graft may carry excessive flow to the lungs at one point in time, diminished flow to the lungs at another point in time, and we must also appreciate that these circumstances may be separated only by minutes. Dr Brink, in your review of approximately 300 patients over a 10-year period, you demonstrated that among the 1 of 5 patients who died before reaching the superior cavo-pulmonary anastomosis stage, the majority of the deaths

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were associated with an acute event after surgery. Of the 79 patients who had an acute event, 40 required shunt re-intervention, either immediately or thereafter. Your multivariable analysis revealed that the following were risk factors for acute events: higher preoperative platelet count and higher postoperative activated partial thromboplastin time. The risk factors for shunt thrombosis associated with an acute event were 3-mm shunt size and higher postoperative hematocrit. Of the 293 patients, 186 received platelets during surgery. In addition, 245 received additional clotting factors intraoperatively: fresh-frozen plasma in 212 patients and cryoprecipitate in 102 patients. Most patients received aprotinin or tranexamic acid.

All of this conveys a picture in which the delicate balance of pulmonary and systemic blood flow may fall victim to an unnatural imbalance between the goals of surgical hemostasis and avoidance of a thrombophilic state. Apart from the theoretical risk of shunt thrombosis, there is the inevitable effect of administration of products such as platelet concentrates on the ratio of systemic and pulmonary vascular resistances.

Have you considered that the situation might be favorably altered by a strategy based on the use of fresh whole blood as the principal agent to restore clotting function and to achieve surgical hemostasis, thereby maintaining a relatively physiologic balance of red cell and non–red cell components, and of natural procoagulant and anticoagulant factors? It is possible that such a strategy could minimize the requirement for transfusion of platelet concentrates and other clotting factors.

**Dr Brink (Melbourne, Australia).** We have not considered fresh whole blood.

**Dr Jacobs.** Would fresh whole blood be available to you? I know it is sometimes a difficult resource to obtain.

**Dr Brink.** I am not sure. We used to have whole blood specially prepared, fresh heparinized blood, in the past, but that was discontinued about 6 years ago.

**Dr Jacobs.** I ask this question simply because it appears that the intraoperative use of these many clotting factors may contribute to a prothrombotic state, which may in turn contribute to the occurrence of these early postoperative events.

**Dr Brink.** I think the reason for using these clotting agents is if you take the whole group into account, approximately 200 patients were in Norwood/DKS status, so it’s not just shunt in isolation. So to require extra clotting factors is somewhat reasonable.

**Dr Jacobs.** Of course. In that regard, we recently reported on an analysis of factors associated with early shunt failure among 9000 patients in the Society of Thoracic Surgeons Congenital Heart Surgery Database. We observed that the overall rate of early shunt failure was 7.3%. For non–HLHS single ventricle patients it was 7.8%, and for non–single ventricle anomalies it was virtually the same, at 7.7%. Among patients undergoing the Norwood procedure, the rate of shunt failure was slightly lower, at 6.6%. In our study that was reflective of a relatively lower incidence of shunt failure with the RV-PA shunt (5.2%), relative to the modified BT shunt or other systemic-to-pulmonary artery shunt (9.4%).

You mentioned that 46 patients in your cohort, or about 1 in 6, received RV-PA conduits as their systemic-to-pulmonary shunt. It would be interesting to know whether you undertook a subcohort analysis to see whether the rate of occurrence of acute events, the occurrence of shunt failure, or the risk factors for those events were different in the RV-PA conduit group versus the S-PA shunt group. Did you look specifically at the RV-PA shunt group?

**Dr Brink.** Yes, we had a look at that and could not find any significant difference for shunt type for acute events nor for thrombosis.

**Dr Jacobs.** That’s interesting. My last question goes to your finding that the 3-mm shunt size was among the risk factors for shunt thrombosis associated with an acute event, apparently independent of shunt size-to-body weight ratio. I infer from your article that you are concerned about avoiding the circumstance that you have referred to as “pulmonary over-circulation.” Given the findings of your study, what are your thoughts about the use of polytetrafluoroethylene shunts in the smallest sizes? Should an alternative graft such as cryopreserved saphenous vein be considered?

**Dr Brink.** In our unit, we use the RV-PA conduit in patients weighing 2.5 kg and less. We use a 5-mm polytetrafluoroethylene (Gore-Tex) graft; if less than 2, then we use a 4-mm Gore-Tex graft. So there is a dilemma in a small patient with a high shunt/weight ratio. In our hands, the MBT shunt works well for us, but it is true, it has a risk for thrombosis and we cannot avoid that. That is the dilemma with small-weight patients.

**Dr Jacobs.** On behalf of all of us here, I want to say that over the years, we have learned a great deal from the careful clinical research done in your unit in Melbourne. Thank you so much for that.
a deliberate policy. Has that changed over the last 10 years, or what drove you to so strictly operate at such a young age?

**Dr Brink.** The cohort is represented by univentricular palliation with a large number of patients that constitute Norwood/DKS subgroup. We don’t have analysis of different eras.

**Dr C. Brizard** *(Melbourne, Australia).* We do operate on hypoplasts at approximately 2 to 3 days of age, and approximately half of these patients are hypoplasts.

**Dr S. Sano** *(Okayama, Japan).* I missed your presentation, the first part. Can you explain postoperative management, because with these patients, management is a bit different, especially like single ventricle palliation. The resistance between the systemic and the pulmonary is key. So do you keep sedation, not only like fentanyl for the muscle relaxant for 1 or 2 days or just only the fentanyl? And the other thing is, when do you start an anticoagulant? I believe that you do start heparin, and do you use aspirin or warfarin or other anticoagulant therapies in the intensive care unit and long term?

**Dr Brink.** So the first question regarding the sedation, 72% of these patients have their chest left open after surgery. Our policy in the Norwood and DKS procedure is to have a delayed chest closure. So in these cases, the children will be well sedated and fentanyl is part of the regimen, and eventually, unless you want the heart stabilized, we close the chest.

Regarding the anticoagulation, once these children are admitted to the intensive care unit, we will commence heparin provided they are not bleeding. We start the dose at 10 to 15 U/kg, and lately we have become a bit more aggressive in the case when they are not bleeding to commence the heparin in the operating room to avoid thrombotic effect. Aspirin and clopidogrel are the agents that we use later. We don’t use warfarin. We commence aspirin for all shunts once they are enterally tolerated, and we add clopidogrel for 3- and 3.5-mm wrap shunts because we know that in children there is up to 80% aspirin resistance, especially in neonates, and therefore we add clopidogrel to cover them.

**Dr Sano.** One of the reasons why I have developed the RV-PA shunt and after I was back from Melbourne, I did a classic Norwood with sedation. Quite often, even with sedation, if the intensive care patient had sedation with fentanyl, when the diagnosis is infection, then the babies respond; they definitely change. So I ask to keep them sedated and use the muscle relaxants, so paralyzed completely, and then the resistance is stable and the shunt-related complications are less. I presented a paper at the last meeting of the World Congress, not single ventricle patients, and I have no early or late deaths since I changed the process.

**Dr Brink.** I am glad you made this comment. After reviewing all the data, which are complicated, because there are many variations in the formula determining shunt flow. One of these factors is resistance, which is difficult to monitor and manage. There are many patients for whom, when reviewing the medical records, we have no answer of what actually happened, and my suspicion is that fluctuation in pulmonary resistance is probably part of the issue.

**Unidentified speaker.** Just a quick comment. In the kids in whom we do end up using a 3-mm polytetrafluoroethylene shunt, we would heparinize them and they would go home with enoxaparin sodium, and in the last couple of years we have also performed the Norwood surgery earlier. So the average is probably 2 to 3 days of life, and I think that helps.