# [Safety and Efficacy of Vasopressin After Fontan Completion: A Randomized Pilot Study.](https://www.ncbi.nlm.nih.gov/pubmed/31400337)

Bigelow AM, Ghanayem NS, Thompson NE, Scott JP, Cassidy LD, Woods KJ, Woods RK, Mitchell ME, Hraŝka V, Hoffman GM.

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**Take Home Points:**

* Vasopressin infusion after Fontan completion was associated with reduced transpulmonary gradient and chest tube drainage in the early postoperative peri.od
* A larger multi-institutional study will be necessary to confirm safety and to see if this impacts length of stay and is cost-effective.



**Commentary from Dr. Jared Hershenson (Greater Washington DC), section editor of Pediatric Cardiology Journal Watch:** While surgical mortality following Fontan palliation is low, postoperative morbidity is common, most often from prolonged pleural fluid drainage. This occurs in 15-40% of patients and is thought to be related to the specific physiologic changes present in the single ventricle population. Many strategies have been attempted to try to reduce chest tube (CT) output with nearly all being unsuccessful on a consistent basis. The authors hypothesized that vasopressin, through its effect on increasing systemic vascular resistance, decreasing capillary leakage, and increasing coronary and pulmonary vasodilation, may be quite suitable in this patient population. This was a small pilot study primarily done to evaluate the safety and efficacy of reducing CT output in a small prospective group of Fontan patients.

This was a randomized, double blinded, placebo controlled trial that included all planned Fontan completions except for those with a history of Fontan takedown, planned AV valvuloplasty, or arch reconstruction, or history of renal replacement therapy. All patients underwent non-fenestrated extracardiac conduit, with standard monitoring and use of inotropes (epinephrine and milrinone) post-operatively. The study drug was initiated at 0.3 mU/kg/min in the OR and continued for 21 hours. It was weaned 0.1 mU/kg/min each hour until discontinued at 24 hours. Additional fluids and titration of inotropes (including open label vasopressin) were at the discretion of the cardiac intensivist. Chest tubes were removed when draining less than 2 ml/kg for 24 hours. 20 patients were studied after a few were excluded or did not consent, with 10 in each group.

There were no differences in the vasopressin and placebo groups in terms of patient characteristics, ventricular morphology, pre-Fontan O2 saturation or hemodynamics, or surgical technique and CPB factors. CT drainage was significantly lower in the vasopressin group during the first postoperative night, POD 1, and at 48 hours after surgery. Median CT duration was 92 hours in the vasopressin group vs 114 hours in the control group. Urine output and fluid balance was similar in both groups. The vasopressin group received significantly higher milrinone dose and had a lower transpulmonary gradient on the first postoperative night. Two patients (one in each arm) received open label vasopressin due to hypotension. Median duration of vasoactive support was the same in both groups. Median hospital stay was 180 hours in the vasopressin group compared to 203 hours in the control group, but this did not meet statistical significance. See table 2, Figure 1, and Table 4.

This was an interesting small single-center RCT that suggests improved transpulmonary gradient and reduced CT output using vasopressin, with no significant safety concerns or deleterious hemodynamic consequences. The vasopressin group did receive a higher dose of milrinone likely due to increased BP; however, the authors think this is likely not a significant confounding variable based on prior studies of milrinone. As a pilot study and as mentioned by the authors, this study was not powered to detect many important clinical outcomes. While CT duration is predicated on CT output, and is likely a main factor in length of hospital stay, statistical significance in this study was not reached. A larger multicenter trial would be recommended.





