# [Long-term clinical outcomes of valsartan in patients with a systemic right ventricle: Follow-up of a multicenter randomized controlled trial.](https://www.ncbi.nlm.nih.gov/pubmed/30449692)

van Dissel AC, Winter MM, van der Bom T, Vliegen HW, van Dijk APJ, Pieper PG, Sieswerda GT, Roos-Hesselink JW, Zwinderman AH, Mulder BJM, Bouma BJ.

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**Take Home Points:**

* Systemic RV failure remains a leading cause for mortality in patients with transposition of the great arteries and an atrial switch or ccTGA.
* Valsartan therapy, in the long term (8 year follow-up) did not demonstrate an overall clinical or survival benefit.
* It was associated however with a reduction in morbidity in symptomatic patients.



***Commentary by Dr. Blanche Cupido (Cape Town), section editor of ACHD Journal Watch:*** Failure of the systemic right ventricle remains a major source of morbidity and mortality in patients with an atrial switch correction for complete transposition of the great arteries (TGA) or in congenitally corrected transposition of the great arteries (ccTGA). There is limited data on the use of renin-angiotensin-aldosterone-system (RAAS) inhibition in this group of patients.

In the VAL-SERVE trial (Valsartan in the Systemic Right Ventricle), there was no significant overall effect of valsartan on RV function at 3 years – in symptomatic patients, however, there was a signal to positive remodeling. Given that in a similar study of LV function and Enalapril, benefit in asymptomatic patients was only evident after >10 years, this study aimed to assess the longer term (8 year) clinical outcomes of patients from the VAL-SERVE cohort.

88 Patients from 6 Dutch university medical centers (CONCOR Registry) with TGA-atrial switch and ccTGA were enrolled into the VAL-SERVE trial between 2006 and 2009. This was a double blind, randomized control placebo study. Patients were assigned 1:1 to either placebo or control groups (44 valsartan, 44 placebo) and followed up for 3 years. The events recorded included supraventricular arrhythmias, worsening heart failure, tricuspid valve surgery and death. NYHA I was deemed asymptomatic.

Both the valsartan and placebo groups had similar clinical characteristics at baseline. The average age at enrollment was 33 years and one third was symptomatic in each group. By the end of the 3 year trial, 17 patients continued valsartan (10 from valsartan group and 7 from placebo group); 11 (from both groups) patients switched to ARB’s / ACE-I. There was no difference in the proportions of the patients taking RAAS inhibitors during the extended 8.3 year follow-up.

Mortality was similar for the randomized groups (3 deaths in each). Twenty-three patients in the treatment (valsartan) group (52%) and 30 in the placebo group (60%) had a primary clinical event. There was a non-significant reduction in the Valsartan treatment group for the combined primary endpoint of mortality and events (HR 0.65 95% CI 0.38-1.12).

For the individual clinical endpoints, there was no difference between the Valsartan and the placebo groups:

* Supraventricular arrhythmias (41% vs 50%)
* Ventricular arrhythmias (27% vs 27%)
* Worsening heart failure (23% vs 23%)
* Tricuspid valve surgery (6% vs 6%)

In a third of patients, more than one event was recorded.

Overall, Valsartan did not improve survival of the cohort but survival rate was noted to be lower in those patients who were symptomatic at the time of presentation (81% vs 98%, p=0,010).



Valsartan reduced the risk of events in the symptomatic group (HR 0.37 95%CI 0.17-0.92) but not in the asymptomatic patients (HR 0.84 95%CI 0.42-1.69). There was however no significant treatment benefit among symptomatic compared to asymptomatic patients (p=0.146 for interaction)