Editorial Comment

The Bantamweight Right Ventricle and the Fontan Operation
One-Two-Three and the Right Ventricle is Out . . . Maybe

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In this issue of Circulation, Nakazawa and colleagues\(^1\) examine in a thoughtful and comprehensive way the hemodynamic effects of inclusion of the right ventricle (RV) in the circulation in seven patients after the Fontan operation using methodology previously published from their institution.\(^2,3\) Based on this relatively small number of patients these authors conclude that the inclusion of the right ventricle does not improve overall hemodynamics of the Fontan circulation in the majority of patients.\(^4\) The implication of this study is that there is no hemodynamic advantage to the incorporation of the RV into the delicate post-Fontan circulation. But how does one reconcile these observations with survival data?

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Fontan et al\(^4\) recently addressed the outcome in 334 patients after a “perfect” Fontan operation. Of those incremental risk factors for death after Fontan operation, these authors identified a right atrial–pulmonary artery (RA-PA) (rather than an RV) connection as one identifiable early hazard. Similarly, Coles and his colleagues,\(^5\) also using a multivariate analysis, identified an atriopulmonary connection as a risk factor for early and late death. The conclusions of Nakazawa et al,\(^1\) however, are based on cardiac catheterization data obtained 3–4 weeks after operation. Maybe there is some positive hemodynamic impact of the type of connection in the immediacy of atrial separation and atriopulmonary connection. Because of the small cohort of patients studied, it would be of interest to apply similar methodology to a larger group of patients at 1, 5, and 10 years after the Fontan operation. With advancing age and, presumably, ventricular hypertrophy (and by inference an altered compliance), it might be hemodynamically advantageous to have the RV involved in the post-Fontan circulation, but this is of course only speculative.\(^6-8\) Similarly, it would be interesting to stratify a larger study group by mean PA pressures or PA index and then to ascertain hemodynamic differences by the type of connection: RA-PA or RA-RV. In this institution we have performed a successful Fontan-type operation with a PA pressure of 40/10 (25) mm Hg, but the connection was RA-RV with a valved conduit. There was a clear-cut consensus that this patient with pulmonary artery hypertension could not survive with an RA-PA connection.\(^9\)

But what about the RV in these patients? Not all bantamweights are alike, and neither are the RVs in tricuspid atresia. In some patients with tricuspid atresia, the RV is a mere slit on the surface of the dominant left ventricle. In other patients the RV is well developed, with a rich trabecular zone and an expansive subarterial outlet or infundibulum.\(^10\) In some patients with so-called classic tricuspid atresia, remnants of the ventricular inlet may also be evident.\(^11\) Is one to believe that these morphological differences in the development of the RV cannot influence the volume of blood conducted to the lungs or the calculated ventricular work? Obviously, before the RV is excluded from contention as a participant in the peculiar post-Fontan circulation, such volumetric and morphological analyses must be applied to these patients.\(^10-12\) The necessity for these types of observations becomes clearer when mechanical aspects of the normal RV are examined. What component of the RV contributes to ventricular contraction or work? And is there a disproportionate contribution from one morphological component of the RV compared with the other component(s)? The answers to these questions have obvious ramifications when one considers the contribution of the hypoplastic RV to cardiac output. There is data from the canine RV that regional shortening patterns differ between the RV inflow tract and the outlet or the infundibulum. In the canine RV the outflow tract seems to be a physiologically distinct region that contracts later, and remains contracted longer, than the inflow tract.\(^13\) Data from Raines and colleagues also suggest that septal and free wall movements contribute to these changes. How these observations of the
canine RV translate into functional equivalents in humans with hypoplastic RVs is uncertain, but there can be little doubt that such considerations should be integrated into any surgical algorithm before the RV is counted out.14

References
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