Right and Left Ventricular Volume Characteristics in Common Atrioventricular Canal

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RIGHT AND LEFT (LV) ventricular volume characteristics were determined from biplane cineangiography in 29 patients with atrioventricular canal (AVC). The patients were classified into two groups: group I (N = 19), uncomplicated AVC; group II (N = 10), AVC associated with RV obstruction. In group I, LV end-diastolic volume (EDV) [177 ± 9 (SEM)%] of normal and RVEDV (125 ± 9%) both were greater than normal (P < 0.001 and < 0.01, respectively). LV ejection fraction (EF) was decreased (0.59 ± 0.02, P < 0.001) but RVEF was normal (0.58 ± 0.03). LV stroke volume index (SVI) was increased (48 ± 3 ml/m², P < 0.005), and RSVI was normal (34 ± 3 ml/m²). One patient had a markedly small RVEDV (45%). In group II, LVEDV and RVEDV were not different from normal (119 ± 11% and 97 ± 15%, respectively). LVEF was depressed (0.52 ± 0.04, P < 0.001) and RVEF was normal (0.55 ± 0.05). LVSVI was normal (38 ± 5 ml/m²) and RVSVI was slightly decreased (29 ± 4 ml/m², P < 0.025). Two patients had a markedly small RVEDV (31%, 55%). EDV correlated with the pulmonary-to-systemic flow ratio (LV, r = 0.71; RV, r = 0.68).

The data show that in most patients with AVC, LV and RV are enlarged in the uncomplicated form but not in the form with RV obstruction. LV function is more compromised than RV in both groups. RV hypoplasia is rare but was documented in both uncomplicated forms and forms with RV obstruction.

Bharati and Lev, in an autopsy study of common atrioventricular canal (CAVC), described hearts with small right (RV) or left (LV) ventricles. The authors suggested that a small ventricle might be a contraindication for surgery. With this point in mind, we have studied angiographic ventricular volumes in CAVC to determine the incidence of ventricular hypoplasia in this lesion in vivo.

Methods

Twenty-nine patients with common CAVC who underwent diagnostic cardiac catheterization and cineangiography at the University of Illinois Hospital were studied. Left ventricular volume was determined in all patients. Right ventricular volume could be determined in only 23 due to poor visualization of RV borders in the other six. The patients were classified into two groups. Group I included 19 cases of uncomplicated CAVC. Group II consisted of 10 patients, seven with RV outflow obstruction (pulmonary stenosis [PS] in four, pulmonary artery banding [PAB] in three) and three with pulmonary vascular obstructive disease (PVO) (table 1).

Right and left ventricular volumes were calculated from biplane cineangiograms following the injection of contrast medium (sodium iothalamate injection USP 66%, 1–2 ml/kg) into the inferior vena cava, RV or LV. The first frames allowing adequate identification of ventricular borders were used. The effects of contrast medium on ventricular function were therefore minimized. Angiograms with arrhythmias were excluded. Due to the goose-neck deformity of the LV, the usually assumed elliptical shape of that chamber was felt to be invalid for volume determination. In order to test that premise, left ventricular volumes were determined by Simpson’s rule as well as by the area-length method. Right ventricular volumes were determined by Simpson’s rule method described previously. Volumes were corrected by regression equations derived from ventricular cast studies in our laboratory. For LV: \( V' = 0.88 \ V - 0.29 \) for Simpson’s rule, and \( V' = 0.85V \) for the area-length method; for RV: \( V' = 0.71 \ V - 1.15 \); where \( V' = \) corrected volume and \( V = \) calculated volume. Correction of measurements for linear X-ray magnification was made by use of a grid system. End-diastolic volume (EDV) was expressed as a % of the predicted normal value for body surface area (BSA). Stroke volume index (SVI) was expressed as ml/m² BSA. Regression equations derived from volume studies in 70 and 77 children with normal LV or RV, respectively, were used for calculating normal expected EDV from BSA. These equations were: log LVEDV = 1.775 + 1.188 log BSA; and log RVEDV = 1.821 + 1.236 log BSA for patients with BSA greater than 0.3 m². As our normal population does not include a significant number of cases with BSA less than 0.3 m², the UCLA regression equations for RV (RVEDV = 75.1 \times [BSA]^{0.40}) and LV (LVEDV = 72.5 \times [BSA]^{0.40}) were used for calculation of normal expected EDV for patients below that size. Volume data from the study groups were compared with normal and with each other, using the Student’s t-test for unpaired and paired data. The normal ranges for LV and RV EDV were considered to be the mean ± 1 SD (table 2).

Results

The hemodynamic findings for both groups are given in table 1. Cardiothoracic ratios averaged 0.62 in group I and 0.53 in group II.

LVEDV calculated by Simpson’s rule method and by the area-length method correlated well (\( r = 0.98 \)). Volumes calculated by Simpson’s rule method, however, were slightly larger than those calculated by the area-length method [36 ± 5 (SEM) vs 34 ± 4 ml]. All subsequent volume data presented were determined by Simpson’s rule method.

Group I

RVEDV was significantly (\( P < 0.01 \)) increased, averaging 125 ± 9% of normal (fig. 1). RV ejection fraction (EF) and
RVS VI averaged 0.58 ± 0.03 and 34 ± 3 ml/m², respectively, and were within normal limits. Fifteen patients in this group had RV volume determined. EDV was above normal range in eight and below normal range (45%) in one (no. 6).

LVEDV was significantly (P < 0.001) increased, averaging 177 ± 9. LVEF was less than normal (P < 0.001), with a mean of 0.59 ± 0.02. RVS VI was greater than normal (P < 0.005), averaging 45 ± 3 ml/m². Eighteen patients had EDV above normal range and none had EDV below normal range.

Group II

RVEDV and RVEF were not significantly different from normal, averaging 97 ± 15% and 0.55 ± 0.05, respectively. RVS VI was slightly decreased (P < 0.025), averaging 29 ± 4 ml/m². Eight patients in this group had RV volume determined. EDV was above normal range in one and below normal range (31, 55%) in two (nos. 25, 29).

LVEDV and LS VI were not different from normal, averaging 119 ± 11% and 38 ± 5 ml/m², respectively. LVEF was less than normal (P < 0.001) with a mean of 0.52 ± 0.04. Four patients had EDV above normal range and the other six were within the normal range.

When RV and LV volume data were compared, the only significant differences found were in group I, where LVEDV and S VI averaged higher than RV values (P < 0.001, < 0.025, respectively). LVEDV and S VI were significantly (P < 0.001, < 0.025, respectively) higher in group I than in group II.

LVEDV correlated well (r = 0.71) with the pulmonary-to-systemic flow ratio (Qp/Qs) (fig. 2). Similar correlation (r = 0.68) was found for RVEDV and Qp/Qs (fig. 3), when case 6 was excluded from the analysis. This case falls far outside the 95% confidence limits for the other cases. It was

VENTRICULAR VOLUMES IN CAVC

![Figure 1. Right and left ventricular end-diastolic volumes in common atrioventricular canal. Black horizontal lines indicate the respective means. 1 = pulmonary stenosis, 2 = pulmonary vascular obstruction, 3 = pulmonary arterial banding.](http://circ.ahajournals.org/doi/abs/10.1161/01.CIR.57.5.992)
excluded because we feel this patient may represent an anatomic entity different from the other 28 cases and not capable of responding to volume overloading as the majority do. To include this case yields an r value of 0.44 and gives an erroneous impression of the relationship of RV volume to Qp/Qs in CAVC.

Two of the patients with decreased RVEDV underwent surgery. One (no. 6) had PAB and is doing well. The other (no. 25) had a total repair and died during surgery. Autopsy findings were consistent with a relatively small RV and enlarged left ventricle. The common atrioventricular valve was equally related to both ventricles.

Discussion

Bharati and Lev, in an autopsy study, classified CAVC according to ventricular size into balanced, dominant right or dominant left forms. In the balanced form, the RV is enlarged and the LV normal or enlarged. In the dominant right or dominant left forms, the dominant ventricle is enlarged while the other is smaller than normal. This classification is a functional one, stressing the surgical aspects. The possible cause of ventricular hypoplasia was not considered essential to the classification. Therefore, dominant right and left forms were found in both uncomplicated and complicated CAVC. Our autopsy data correspond in general with this classification. Thus, 14 patients in group I and six in group II had both ventricles normal or enlarged and can be classified as the balanced form. One patient in group I and two in group II had a small RV and normal or enlarged LV, and can be classified as the dominant left form. No patient had the dominant right form. Four patients of group I and two of group II in whom RV
volume could not be determined were not classified. The LV in these patients was normal or enlarged. 

The causes of right or left dominance in CAVC have not been explained. Bharati and Lev⁴ recognized that hemodynamic factors may account for dominance of a ventricle in CAVC. The present study lends support to this view. Both right and left ventricular volumes correlated with the Qp/Qs. Thus, as expected, in most patients with uncomplicated CAVC (and increased pulmonary blood flow) the LV, and to a lesser extent RV, are enlarged. These findings are similar to those reported by Graham⁷ in children with large ventricular septal defects. Despite the large atrial communication present in patients with CAVC, the right ventricle is less enlarged than the left, perhaps due to the contribution of left-sided A-V valve insufficiency to left heart volume overloading. As expected, in patients with CAVC and RV obstruction, where the Qp/Qs is close to or less than 1, the ventricles in the majority of cases are normal in size.

There is some pathological evidence supporting the role of developmental or embryological factors in some patients. Bharati and Lev⁴ felt the relationship of the common atrioventricular (A-V) valve to the ventricles could explain some cases. In their study many cases with the dominant left form had an A-V valve with only a single attachment to the RV, reminiscent of straddling tricuspid valve. Goor⁶ described one case in which the A-V valve was entirely related to the RV and the LV was hypoplastic, a dominant right form. In four other cases, he found the A-V valve exclusively related to the LV, but did not mention the size of the RV. In the present study only one patient with a small RV came to autopsy. The A-V valve was equally related to both ventricles, suggesting that hemodynamic rather than developmental factors had resulted in RV hypoplasia.

In our study, the RV was small in three patients (nos. 6, 25, 29). The RV hypoplasia in the two patients in group II may be secondary to a decrease in pulmonary blood flow.

The group I patient with a small RV had a very large Qp/Qs and a markedly enlarged LV. The hemodynamics apparently do not explain the RV hypoplasia, and therefore developmental factors may have played a major role in this case.

In this study no patient had a small LV. Mair and McGoon⁷ and Hagler⁸ mention patients with an angiographically small LV, but neither author presents volume data. Hagler's patient had a high Qp/Qs. The Qp/Qs in the case of Mair and McGoon was not reported, but other hemodynamic data do not suggest the presence of decreased pulmonary blood flow in that patient.

Several authors have suggested that the presence of a small ventricle may increase surgical risk in CAVC,¹ ⁷ ⁹ ¹¹ ¹² but the reported surgical experience in such patients is scanty. Mair and McGoon⁷ reported congestive heart failure and death in a patient with CAVC and small LV following intracardiac repair.

Further surgical experience is needed in patients with CAVC with hypoplastic ventricle before definitive recommendations for the surgical approach can be made. If this experience confirms the impression that ventricular hypoplasia does increase surgical risk, further volume data will be needed to answer the question, "How small is too small?"

We believe the data in the present study support the following conclusion regarding surgery in CAVC: 1) In most patients with the uncomplicated form or the form with RV obstruction, ventricular size is not a limiting factor in intracardiac repair. 2) RV obstruction may result in or contribute to RV hypoplasia. Consideration should therefore be given to early intracardiac repair⁶ rather than PAB in patients with increased pulmonary blood flow. 3) In patients with pulmonary stenosis, early repair or a two-stage approach with initial systemic-pulmonary anastomosis followed by later intracardiac repair should be considered. Although a two-stage approach may increase surgical risk, in patients with severe ventricular hypoplasia the combined risk may prove to be less if the hypoplastic ventricle increases significantly in volume in response to increased pulmonary blood flow.

The dominant left form without RV obstruction, such as our group I patient, presents a difficult surgical problem. Pulmonary artery banding may be a satisfactory short-term approach, but will result in decreased pulmonary blood flow.
and may contribute further to lack of development of the RV as the patient grows. The absence of a hemodynamic explanation for ventricular hypoplasia makes the presence of a developmental factor such as markedly unequal relationship of the A-V valve to the ventricles probable and it may be that such a heart cannot be repaired by conventional techniques.

In conclusion, our volumetric studies show that the LV, and to a lesser extent the RV, are enlarged in most cases of uncomplicated CAVC but normal in most cases with right ventricular outflow obstruction or PVO. LV dysfunction is more frequent than RV in both groups. RV hypoplasia is rare in CAVC but does occur in both uncomplicated forms and forms with RV obstruction. Further ventricular volume studies are needed in CAVC to confirm these findings and document the occurrence of LV hypoplasia, which was not found in this study. Although data concerning surgery in CAVC with ventricular hypoplasia are scanty, there is reason to believe that the surgical approach to these patients may need to be modified.

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References


Total Effective Compliance,
Cardiac Output and Fluid Volumes
in Essential Hypertension

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SUMMARY Total effective compliance, hemodynamic parameters, extracellular fluid volume, cardiopulmonary (CPBV) and total blood (TBV) volumes were determined in 32 men, including 14 normotensive controls and 18 sustained essential hypertensive patients. The effective compliance was calculated from the changes in central venous pressure recorded simultaneously with the changes in blood volume obtained after a rapid Dextran infusion. In normotensive controls, compliance was 2.08 ± 0.09 ml/mm Hg/kg and was positively correlated with plasma (r = 0.79) and extracellular fluid (r = 0.84) volumes. In hypertensives, compliance was significantly reduced (1.49 ± 0.66 ml/mm Hg/kg; P < 0.001) and was correlated negatively with the CPBV/TBV ratio (r = -0.75) and positively with the plasma volume/interstitial fluid volume ratio (r = 0.84). These results suggest that in normotensives, there is a regulatory mechanism between volume and compliance and that this contributes to maintaining filling pressure and cardiac output within normal ranges. In hypertensives, the reduced compliance could participate in the maintenance of normal values of cardiac output and extracellular fluid volume by influencing the partition of intravascular and extracellular fluid volumes.

and originate a neuro-humoral control of extracellular fluid volume and its partition.2-4

In experimental hypertension5-7 and in spontaneous hypertension in rats,7, 8 decreased venous distensibility has been demonstrated. Thus, the consequences of a changed vascular compliance on the overall circulation have been investigated. No comparable results have been obtained in human hypertension, since venous compliance has only been studied in the skin or in forearm vascular beds.8-11 In human hypertension, blood volume is often found to be lower than

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