The Dynamics of the Heart in Complete A-V Block
An Angiocardiographic Study

By John Lind, M.D., Carl Wegelius, M.D., and Henrik Lichtenstein, M.D.

The conduction disturbances in atrioventricular block are well known from numerous electrophysiological studies, but the effect on the heart work, especially on the individual chambers, could not be recorded before the introduction of visualization of the heart chambers by angiocardiography. Rapid biplane angiocardiography with simultaneous electrocardiographic yields information on cardiac dynamics not available by procedures with a purely static anatomic perception.

In complete atrioventricular block the atria and ventricles contract independently and the relationship of the atrial to the ventricular contractions is always changing. This type of dissociation offers a unique opportunity to study the mode of action of the atria and the ventricles independently and in various combinations.

Interesting clinical and pathophysiologic observations in complete atrioventricular block have been contributed by electrocardiography, phonocardiography and phlebography, and by pressure curves obtained by heart catheterization. However, the contributions of angiocardiography to the study of this condition has not been reported previously. A discussion of the supplementary information on the dynamics of the heart which this method of study may supply is the purpose of this paper.

Angiocardiographic Technic
Used in the Present Investigation

The technic of the angiocardiographic method has been reported elsewhere. Seventy per cent Umbradil was injected as contrast medium. The patients were sedated with a 2.5 per cent solution of Avertin by rectum, the dosage being 0.1 ml. per kilogram of body weight. Children remained quiet with shallow respiration during the examination.

From the Pediatric Clinic of the Caroline Institute and the Wenner-Gren Cardiovascular Research Laboratory at Norrtulla Hospital, Stockholm.

Presented at the Seventh International Congress of Radiology, Copenhagen, Denmark, July 19-24, 1953.

Case Report

Case 1. A female infant weighing 3100 Gm. was born at term on May 6, 1947 after an uneventful pregnancy and delivery. There was no cyanosis but persistent dyspnea and lethargy. Mental development was normal, but the child did not walk until the age of 18 months and was never able to be very active. On May 28, 1950, the infant girl was found unconscious in bed and taken immediately to a hospital where she had about 30 generalized convulsions always preceded by loss of consciousness. The seizures were controlled after three days with barbiturates and ephedrin.

Soon thereafter the child was admitted to this clinic for further investigation. At the age of 3 years and 3 months her weight was only 9.5 Kg. and height 88.5 cm. Exertional dyspnea but no cyanosis was present. Physical examination showed the heart to be enlarged to the left and slightly to the right; the point of maximum impulse was in the sixth left intercostal space in the nipple line. A long, rather soft systolic murmur was heard over the precordium, maximal in the third left intercostal space 1 cm. to the left of the midsternal line. The rhythm was regular, the rate 70 per minute. The second pulmonic sound was accentuated. The femoral pulse was normal. Two days after admission a sudden change in the rhythm of the heart developed when it became completely irregular with a rate of 35 to 45 beats per minute.

Electrocardiogram and phonocardiogram revealed complete A-V block with a ventricular rate of 45 per minute. The QRS complexes were normal and there was no axis deviation. The S-T segments were slightly depressed in leads I and II, and the T waves were positive in the three conventional leads. At the apex the phonocardiogram revealed a systolic murmur in the frequency range of 50 to 1000 vibrations per second with the maximal amplitude in early systole. Over the pulmonary valve was recorded a systolic and diastolic murmur in the same frequency range.
An x-ray film of the chest showed generalized cardiac enlargement with a volume of 490 cc. per square meter of body surface area. 

Angiocardiography was performed on Aug. 26, 1930, with injection of the contrast substance into the right antecubital vein. Left and right anterior oblique projections were taken simultaneously. The superior vena cava was normal and all the heart chambers were dilated. No right-to-left or left-to-right shunt was demonstrated.

No further evidence of the Adam-Stoke's syndrome occurred during the hospital stay. The cardiac rhythm continued to be slow and irregular; it was not affected by ephedrin. Circulation time (fluorescin) from the right antecubital vein to the lips was 10 seconds, a normal value.

The dynamics of the right heart in complete atrioventricular block are illustrated by the accompanying angiocardiograms shown in figure 1.

![Fig. 1.](image)

The time of each exposure after the injection of Umbradil is indicated. These times are also shown on the semischematic electrocardiogram.

**Exposure 5.** The right atrium is in late diastole and right ventricle in early diastole, immediately after the T wave. The contrast medium is rapidly filling the ventricle.

**Exposure 6.** This exposure shows the end of the first of the two atrial systoles preceding the ventricular contraction. The ventricle is still poorly visualized, indicating that it is dilated and already partially filled with blood. Note the complete emptying of the atrium and its appendage.

**Exposure 7.** The heart is again in diastole. Traces of the contrast substance are seen in the inferior vena cava, probably because of the rapid injection of the contrast medium.

**Exposure 8.** Early atrial systole, the second preceding the ventricular systole is demonstrated. Since the ventricle is already practically filled, the atrial emptying is much more difficult. This is indicated by a heavy true reflux into the inferior vena cava, the orifice of which is normally functionally closed during atrial systole. The atrial appendage is already empty.

**Exposure 9.** The ventricle is still poorly visualized after two atrial contractions, and the atrial appendage is filled again.

**Exposure 10.** The heart is in diastole. The right atrium has increased little in size, and the reflux into the inferior vena cava has not disappeared.

**Exposure 11.** As soon as the right ventricle starts to contract, the right atrium increases suddenly in size, and the contrast medium in the superior and inferior vena cava streams into the atrium. In diastole the right atrium can be seen to be moderately enlarged.

**Exposure 12.** Atrial and ventricular systole coincide. The atrioventricular valves do not open, but there are heavy reflexes into the inferior and superior vena cavae and the sinus venosus. The atrium decreases little in size. The pulmonary artery is considerably dilated.

**Exposure 13.** In early atrial diastole the atrium increases somewhat in size, and the contrast medium, forced back into the afferent veins by the reflux, is returning to the atrium.

**Exposure 14.** Immediately at the end of ventricular systole the right ventricle fills rapidly and almost completely before atrial systole occurs. (Compare with the filling effect of the first atrial systole, 6.) The contrast substance in the caval veins and sinus venosus has simultaneously returned to the atrium. (Note there is an interval of 1/2 second between 13 and 14.) The caliber of the inferior vena cava and the hepatic veins has decreased.

**Exposure 15.** The ventricle is well filled up to the pulmonary valves in spite of the fact that no effective atrial systole has occurred since the last ventricular systole. The atrium has diminished in size.

**Exposure 16.** The atrium does not decrease much in size when it does contract. A reflux into the inferior caval vein is seen again. The ventricle is much enlarged in diastole. The fact that the size of the atrium decreased more during the systole visualized in 5 and 6 may be due to the circumstance that this systole occurred earlier in ventricular diastole, before the ventricle was so well filled.
Fig. 1. See legend on facing page.
Angiocardiography performed on the seventeenth day of life in another patient with complete heart block revealed the same findings but less dilatation of the cardiac chambers.

**DISCUSSION**

The atrium empties very effectively when the atrial contraction occurs immediately after the T wave, for then the ventricle is virtually empty and the ventricular pressure is low (exposures 5, 6). The later in ventricular diastole the atrial systole occurs, the less complete is the emptying of the atrium. (Compare exposures 26 and 27 with 5 and 6 and 15 and 16.) When the atrium contracts for the second time during ventricular diastole, it is difficult to force more blood into the already well-filled ventricle, because the atrioventricular pressure difference is small. Since emptying is less effective, the atrium diminishes little in size, and the contrast medium is forced into the inferior vena cava, indicating increased intra-atrial pressure. If the atrium begins to contract during ventricular systole, the atrioventricular valve fails to open, and the resulting increased atrial pressure results in a reflux of blood into all the afferent veins (exposures 12 and 23).

The ventricle fills rapidly as soon as the atrioventricular valves open. The diastolic filling of the ventricles, even without atrial systole, takes much less time than the systolic emptying. At the same time the atrium diminishes markedly in size without having contracted (exposures 13, 14, 15). During the latter part of diastole neither the right ventricle nor the right atrium changes visibly in size.

The sequence of pictures 20, 21, 22 and 23 demonstrates the great systolic dilatation of the pulmonary artery, which results from the

![Fig. 1 (continue)](http://circ.ahajournals.org)

As in figure 1 the time of each exposure after the injection of Umbradil is indicated. These times are also shown on the semiechocardiogram.

**Exposures 17 and 18.** In spite of a prolonged atrial diastole the atrium does not increase markedly in size probably because the distended ventricles nearly fill the pericardial sac and prevent complete atrial dilatation.

**Exposure 19.** Atrial systole, occurring when the ventricle is almost maximally filled, forces a reflux of contrast medium into the inferior vena cava. Since the ventricle is too full to contain more blood, the atrium apparently diminishes in size, particularly the atrial appendage.

**Exposure 20.** The atrial appendage has refilled and the atrium is slightly larger.

**Exposure 21.** Early ventricular systole dilates the pulmonary artery. The right atrium fills simultaneously.

**Exposure 22.** During ventricular systole the pulmonary artery increases markedly in caliber. The right atrium is now larger than it ever was during diastole.

**Exposure 23.** Again an atrial systole occurs during the ventricular contraction with consequent reflux into the venae cavae and the sinus venosus. The atrioventricular valves do not open and the atrium maintains the approximate former size.

**Exposure 24.** The end of ventricular systole demonstrates increased residuum in the ventricle. The pulmonary artery remains dilated in spite of the fact that the ventricle is now in the phase of reduced ejection.

**Exposure 25.** The ventricle has resumed its diastolic size and shape and is already well filled. Now the pulmonary artery decreases in caliber.

**Exposure 26.** The right atrium is now smaller in full diastole.

**Exposure 27.** The atrium is still smaller in systole. The pulmonary artery and its main branches have finally resumed their narrow diastolic caliber.

**Exposure 28.** In atrial and ventricular diastole the right atrium is virtually stationary in size. During ventricular diastole the pulmonary artery slowly assumes a more curved configuration.
Fig. 1 (continued). See legend on facing page.
increased stroke volume with bradycardia. Maximal diameter is reached in picture 22 and is maintained during the entire systole, not only during rapid ejection but even throughout the phase of reduced ejection. The dilatation, however, is functional and disappears gradually during diastole.

In addition to the change in size, there is also a change in shape, with a straightening of the normal curve of the pulmonary artery during ventricular systole. In spite of the fact that the complete heart block has existed for more than three years in this patient, the pulmonary artery is slender in diastole and does not appear to be organically dilated.

The above description of the dynamics of the right heart during complete atrioventricular heart block applies equally well to the left heart. There is comparable reflux into the pulmonary veins when the atrium cannot empty into the ventricle; it is less strikingly demonstrated, however, because of dilution of the contrast medium in the lesser circulation.

**Summary**

The cardiac dynamics in complete atrio-ventricular heart block have been studied by means of rapid biplane angiocardiography with simultaneous electrocardiography. The method permits the study of the mechanism of contraction of the separate heart chambers.

**SUMARIO ESPAÑOL**

La dinámica cardíaca en el bloqueo atrioventricular completo ha sido estudiada por medio de angiocardiografía de biplano rápido con electrocardiografía simultánea. El método permite el estudio del mecanismo de contracción de las cámaras separadas del corazón.

**REFERENCES**

The Dynamics of the Heart in Complete A-V Block: An Angiocardiographic Study
JOHN LIND, CARL WEGELIUS and HENRIK LICHTENSTEIN

Circulation. 1954;10:195-200
doi: 10.1161/01.CIR.10.2.195

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on
the World Wide Web at:
http://circ.ahajournals.org/content/10/2/195

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