Cavity Potentials of the Human Ventricles

By Henry A. Zimmerman, M.D., and Herman K. Hellerstein, M.D.

Intracavitary potentials of the human left ventricle were studied in 8 patients by the technic of retrograde ulnar artery or, in one case, pulmonary vein catheterization. The intracavitary potentials from the left ventricle were of the QS variety and confirm Wilson’s concept of the earlier depolarization of the intraventricular septum from left to right.

In recent years the genesis of the human electrocardiogram has been clarified as a result of catheterization of the right side of the heart. The fundamental concepts of Wilson and others, based upon dog experiments, have been amply substantiated in practically all respects. However, in man, proof of the earlier depolarization of the left side of the upper part of the interventricular septum has been based upon the demonstration of an R wave in the right ventricular cavity, and upon the assumption that an R wave would be absent in the corresponding region of the left ventricular cavity. The answer to this problem is now afforded by electrocardiograms obtained from the cavity of the human left ventricle by the technic of left heart catheterization. The first left ventricle catheterization was done on October 8, 1948.

Methods

The intracavitary potentials of the left ventricle of 9 male patients were obtained, 8 by the technic of retrograde ulnar arterial catheterization, and one by passing a catheter electrode through a pulmonary vein into the cavity of the left ventricle at the time of operation for pneumonectomy. In 5 of 8 patients, the right ventricle was also catheterized in the usual fashion at the same time and intracavitary electrocardiograms recorded. The Sanborn Viso-Cardilette and Stetho-Cardilette were used to record the electrocardiograms.

To avoid damaging the aortic valve leaflets, patients with functional and organic insufficiency of the aortic valve were selected for retrograde ulnar arterial catheterization. Earlier attempts to bypass the normal aortic valve were uniformly unsuccessful. In 6 patients, the aortic insufficiency was of syphilitic origin, and in 2 hypertensive patients there was functional aortic valvular insufficiency presumably due to dilatation of the aortic ring.

There was an average pulse pressure of 98 mm. Hg, and the usual auscultatory and dynamic signs of aortic insufficiency were present. The patients ranged in age from 48 to 74 years. Only 2 were on digitalis therapy. Safety precautions included ample sedation and continuous observation of the cardiac mechanism by means of a direct writing electrocardiograph during the procedure. The patients withstood the procedure well and suffered no immediate or delayed ill-effects from the arterial catheterization.

In most patients, the electrocardiographic data were supplemented by determination of intracavitary pressures by means of Fathman strain gages. A No. 6 Courmand single lumen catheter was advanced to the desired position. A continuous stream of heparinized saline was forced through the catheter at approximately 180 mm. Hg pressure. Intracardiac tracings were obtained with the column of blood or saline acting as a linear conductor. In several patients, a single lumen catheter with an indwelling spring steel stylet 3 to 5 mm. from the end of the catheter was used. The potential variations recorded in this fashion were definitely those taking place at the tip of the catheter, and resembled tracings obtained from the arm end of the catheter lumen when the stylet was removed. In the latter instance, there was some distortion due to stray 60 cycle current and the amplitude of the deflections was about one-half as great as when the stylet was close to the distal orifice of the catheter.

In 6 of 8 patients, the catheter entered the left ventricle with ease; however, in 2 patients, the tip was obstructed by the deformed aortic valve leaflets. Tracings obtained in the proximal part of the arch of the aorta, just above the valve leaflets, resembled those taken with the tip of the catheter in the cavity of the left ventricle.

The position of the tip of the catheter was determined in several ways: (a) by fluoroscopy, (b) from the form of the pressure curve, and (c) by the marked increase of voltage when the catheter tip entered the cavity of the ventricle. Numerous records were obtained in various parts of both ventricles; particular interest was focused on the region of the upper interventricular septum. A technic originally employed by one of us (HKH) in dog experiments was successfully employed in this study.
Fig. 1. Three limb leads, three augmented leads and the cavity potentials from the right and left ventricles of a white male with syphilitic aortic insufficiency without evidence of congestive heart failure. The left precordial leads (not shown here) exhibited negative ST-T complexes. Note that the cavity leads are negative and the right arm and left arm leads have net positive values which indicates that the right arm "unipolar" lead does not measurably reflect true cavity potentials in this case. The magnified curves in the last column show more clearly the detail of the complexes.

Fig. 2. Routine twelve lead electrocardiogram taken precatheterization from C. S., a Negro male with syphilitic aortic insufficiency in congestive failure at the time of study.
the catheter was withdrawn from the tip of the left ventricle to the left arm, and from the pulmonary artery to the right arm, continuous electrocardiographic tracings were recorded. In this way changes in form and amplitude of the electrocardiogram were noted and correlated with the position of the tip of the catheter as ascertained by continuous fluoroscopic observation. In several patients, roentgenograms of the chest were obtained with catheters in place in both ventricles. In addition to intracavitary electrocardiograms, standard, “unipolar” limb, and precordial leads were obtained.

The 8 patients studied by arterial catheterization had marked left ventricular hypertrophy, and the other patient, studied during pneumonectomy, had both right and left ventricular hypertrophy. In one patient intraventricular conduction was prolonged (0.12 second), on the basis of left ventricular hypertrophy, and in all others, the duration of the QRS complexes was 0.11 second or less.

**Results**

*Intraventricular Electrocardiograms.* In the right ventricle in the region of the upper part of the interventricular septum, the intracavitary electrocardiogram showed a small positive deflection followed by a large negative wave, i.e., an rS complex. Tracings of the rS variety were obtained when the catheter tip entered the right ventricle from the right atrium and when the catheter tip was withdrawn from the pulmonary artery into the outflow tract of the right ventricle. This initial positivity has been observed by others and has been attributed to the earlier activation of the upper portion of the interventricular septum from left to right.1, 7, 8

In the corresponding region of the left ventricle, the intracavitary tracing was in each instance of the QS variety, with an initial slurring on the downward limb, apparently corresponding to the R wave of the right ventricular lead (figs. 1, 4, 6 and 11). In no case was a positive deflection recorded within the left ventricle in this region. These observations confirm Wilson’s concept of the earlier activation of the uppermost part of the left side of the septum.

However, in one case, a positive deflection was recorded in the tip of the apex of the cavity of the left ventricle (fig. 4, no. 1, 5). The complex was of the rS variety, and the positive deflection was most conspicuous when there was S-T elevation due to endocardial pressure injury. When the catheter was withdrawn about 1 cm, a small negative deflection appeared, which preceded the R wave, and the ventricular complex was of the QRS variety. Several minutes later, the complex reverted to the rS form, with persistence of the positive deflection, although injury effects had disappeared. The positive deflection in this isolated instance may have been due to (1) depolarization of the lower septum from right to left, (2) manifestation of injury current of depolarization, or (3) orientation of the tip of the catheter electrode so that it would be “facing” the wave of depolarization passing down the Purkinje system. The possibility of the latter was suggested by earlier dog experiments. Hellerstein and Liebow9 have noted that when an intracavitary electrode was pressed gently against the endocardium of the left ventricle, a positive deflection occurred, without manifestations of injury in the S-T segment. When pressure was released, this small positive deflection disap-
FIG. 4. Fifteen curves taken at the levels shown in figure 3.

Curve 1 is from the apex of the left ventricle at N/15 sensitivity recorded from position 1, figure 3. The complex is unusual being of the rS variety and is discussed in the text.

Curve 2 was taken from the upper left ventricle just below the aortic valve ring at N/15 sensitivity from position 2, figure 3. Note the absence of the r wave and slurring of the downward limb of the QS complex.

Curve 3 was taken from the ascending aorta just above the aortic valve at N/15 sensitivity from position 3, figure 3. Note the broad negative P wave, QS complex, and positive T wave. The record resembles the tracing within the left ventricle, and not that of the right ventricle. The right arm unipolar lead differs from the left cavity and aortic leads in that the T wave is oppositely directed.

Curve 4 was taken from the right pulmonary artery at N/3 sensitivity from position 4 in figure 3.

Curve 5 was taken from the right pulmonary artery at N/3 sensitivity from position 5 in figure 3. The position fluoroscopically corresponded to the same level as curves 3 and 12. Note the similarity of the complexes.

Curve 6 was taken from the main pulmonary artery, near the bifurcation at N/3 sensitivity from position 6, figure 3. Note the increase in amplitude of the complexes.

Curve 7 was taken from the pulmonary artery just above the pulmonic valve at N/3 sensitivity from position 7, figure 3. A positive deflection now is more conspicuous (rS complex) and is found in all tracings in the right ventricle. Presumably tracings from positions 4 and 5 resemble left cavity potentials closer than those from the right arm and right atrium.

Curve 8 was taken from the outflow tract of the right ventricle below the pulmonic valve at N/3 sensitivity from position 8, figure 3. Note the increase in amplitude of the complexes.

Curve 9 was taken from the mid right ventricle at N/6 sensitivity from position 9, figure 3.

Curve 10 was taken from the right ventricle below the tricuspid valve at N/3 sensitivity from position 10, figure 3.

Curve 11 was taken from the mid right atrium at N/3 sensitivity from position 11, figure 3.

Curve 12 was taken from the lower superior vena cava at sensitivity N/3, from position 12, figure 3.

Curve 13 was taken from the mid superior vena cava at N/3 sensitivity from position 13, figure 3.

Curve 14 was taken from the right axillary vein at N/3 sensitivity from position 14, figure 3.

Curve 15 was taken from the brachial vein at the level of the mid-upper arm at N/3 sensitivity from position 15, figure 3.
peared. Presumably in these experiments, the altered spatial orientation of the exploring electrode to the oncoming excitation wave spreading down the Purkinje system was responsible for the positive deflection.

FIG. 5. An injury current produced by pressure of the tip of the catheter against the endocardium of the apex of the left ventricle. Curves A, B, C, D, and E are portions of a continuous record taken as the catheter was withdrawn one centimeter. Note the return of the S-T segment toward the isoelectric line. The origin of the R wave is obscure and is discussed in the text.

**Endocardial Pressure Injury Effects.** Typical monophasic curves of injury were recorded when the tip of the catheter came in contact with the endocardium of either the right ventricle or the left ventricle (fig. 5). The muscle region affected was minute, since distant leads were uninfluenced. Upon withdrawal of the catheter for 1 to 2 cm., the injury effect subsided promptly, similar to the findings in animals.6

_Electrocardiograms from the Root of the Aorta._ Tracings obtained in the aorta above the aortic ring and from the ascending portion of the arch of the aorta reflected the left intraventricular potentials. The complexes were of the QS type in the arch, and in the root of the aorta of the QS or Qr form (fig. 8). The delayed positive deflection probably reflected the later activation of different portions of the base of the left ventricle. The direction of the ST-T complex (S-T segment displacement, or T wave) was opposite in sign to that found in the lateral precordial leads; elevated in aortic and left intraventricular leads, and depressed in leads $V_4$, $V_5$ and $V_6$. The potentials within the left ventricle were greater than those from

![Fig. 6](image1.png)

Fig. 6. Curves from the right and left ventricular cavities. The curves were synchronized by the use of heart sound tracings recorded simultaneously.

![Fig. 7](image2.png)

Fig. 7. The similarity of premature ventricular beats when recorded locally. Curve A is a right ventricle premature beat recorded by a right intraventricular lead. Curve B is a left ventricular premature beat recorded by a left intraventricular lead. Note the similarity of the QS complexes and the positive ST-T areas.
within either the right ventricle, the aorta, or from the anterior chest wall as registered by the catheter in the ventricle has been noted above.

Fig. 8. Tracing taken from J. L., a white male with malignant hypertension, and functional aortic valvular insufficiency. The patient was not in congestive cardiac failure nor had he been taking digitalis. Shown here are the conventional twelve leads, a lead from the right ventricular cavity and a lead from the lower portion of the ascending aorta. The aortic valve in this patient could not be bypassed. Tracings obtained from the ascending aorta shows S-T elevation and Qr complexes. The right intraventricular tracings show an initial positive deflection, the S-T elevation may be due to pressure injury.

the V leads. There was a marked decrease in the amplitude of the complexes when the catheter was withdrawn from the cavity of the left ventricle (fig. 9). The value of this change in potential in the accurate localization of the tip

Sensitivity of the Ventricular Endocardium. The endocardium of the ventricles is more sensitive to mechanical stimulation than that of the atria. Clinically, premature beats and ectopic rhythms occur more frequently when the
Ventricular endocardium is disturbed during catheterization. In dog experiments, the atrial endocardium can be abraded without the appearance of arrhythmias. In studies of the effect of endocardial injury in dogs, Hellerstein and Katz noted the sensitivity of the ventricular endocardium and also noted that the upper part of the ventricular septum and the tips of the papillary muscles are extremely irritable, giving rise to runs of ventricular beats, and occasionally to ventricular tachycardia.

In our experience with right heart catheterization, premature ventricular beats were noted at one time or another during the procedure in all patients. Ordinarily slight movement of the catheter will eliminate the disorder. Premature beats were most frequently produced when the catheter first entered the left ventricle. The number of premature beats spontaneously decreased without moving the catheter. This apparent acclimatization of the endocardium to an indwelling catheter has been noted also in the right side. It is possible that the catheter may shift from an irritable focus due to respiration or to the heart beat.

**Fig. 9.** Tracing taken from L. J., a white male with syphilitic aortic insufficiency without congestive failure. The curves shown here represent portions of a continuous strip recorded from the tip of the catheter as it was withdrawn from the apex of the left ventricle to the left subclavian artery. The upper row, A, represents a control curve. Row B is the curve recorded as the catheter was withdrawn. The middle row is continuous with the upper row. Note the transient ventricular tachycardia (rate 250) which lasted 4.4 seconds, and was produced when the catheter tip was situated near the upper septum. Also note the marked diminution of voltage when the catheter tip enters the root of the aorta. Curve A on the bottom row was taken from the ascending aorta, curve B from the arch of the aorta, and curve C was taken from the left subclavian artery. This series of tracings demonstrates the irritability of the upper septum and the change in voltage as the catheter is withdrawn from the ventricle. All curves were taken at N/3 sensitivity.

The left ventricle. The number of premature beats spontaneously decreased without moving the catheter. This apparent acclimatization of the endocardium to an indwelling catheter has been noted also in the right side. It is possible that the catheter may shift from an irritable focus due to respiration or to the heart beat.

Form of the Premature Ventricular Beats. In the intraventricular tracings, premature beats were of the QS variety, when recorded in the
ventricle from which they arose. The ST-T area was positive (fig. 7). When the premature beats originated in the right ventricle, the form of the QRS complexes in the limb leads resembled that of left bundle branch block and when they arose in the left ventricle, right bundle branch block (fig. 10).

Relation of "Unipolar" Right Arm Lead to Intraventricular Leads. It has frequently been assumed that because the right arm exploring electrode is situated above the atrioventricular orifices, tracings recorded here reflect intracavitary potentials. Generally, aVR tracings are negative, as are the intracavitary leads.

However, this relationship may be affected by changes in the electrical and anatomic position of the heart. For example, in figure 1, the heart is rotated to the left, electrically. The net value of the right arm lead is positive, although the cavity leads are negative. In this

![Figure 10](image10.png)

**Fig. 10.** Tracings taken from C. W., a Negro man with syphilitic aortic insufficiency and congestive heart failure. Shown under A is a control record taken shortly before catheterization of the left ventricle. Curves under B are augmented limb leads and show multiple premature ventricular beats when the catheter was inserted into the apex of the left ventricle. Curves under C, taken 5 minutes later than those in B, show the spontaneous disappearance of the premature beats. Discussed in the text.

![Figure 11](image11.png)

**Fig. 11.** Curves taken during pneumonectomy on K. W., a white male. The intraventricular curve was secured by passing a catheter through a left pulmonary vein. The intraventricular tracing taken at N/5 sensitivity shows a QS complex with elevated S-T segment.
particular patient the duration of the QRS complex is prolonged (0.12 second). The tall delayed R wave in lead aVR resembles that found in right bundle branch block. However, the configuration of the precordial leads and the absence of a large R wave in the right cavity tracing makes the diagnosis of right bundle branch unlikely.

At the present time, therefore, it is probably safe to state that the right arm lead reflects intracavitary potentials only when it is in a negative field and when the intraventricular conduction is normal. A net positive value of a right arm lead may occur in abnormal intraventricular conduction, or with rotation of the heart. In the latter instance, any lead which is in a negative field would reflect the net electrical potentials of the ventricles better than the right arm lead. In our cases, the right arm lead resembled the intracavitary leads only when the right arm lead had a net negative value. Therefore one cannot assume that the right arm lead invariably reflects intracavitary potentials.

Relation between Precordial Leads V5 and V6 and Leads from within the Left Ventricle. When a deflection in one direction occurs in a precordial lead (or epicardial lead) simultaneously with a deflection in the opposite direction in the cavity lead, both deflections may be attributed to forces across a boundary between active and resting muscle in the free wall of the ventricle. In all our cases, the net value of the QRS complexes of the left cavity leads was negative, and of the left precordial leads, positive. In 6 of 9 patients, in V5 and V6 there was a small negative deflection preceding the major positive deflection, i.e., qR complexes. This negative wave is due to depolarization of the upper septum from left to right. In the other 3 patients, the complexes were of the R or Rs variety. In 8 of 9 patients, the net value of the ST-T complex of the left cavity leads was opposite in sign to that of the left precordial leads, positive and negative, respectively. The vector force producing the ST-T deviations is such that any electrode "facing" the endocardium of the free wall of the left ventricle is in a positive field (in left ventricular cavity, root of aorta) and those exploring electrodes on the other side (epicardial) will be in a negative field (V4, V5 and V6). This again demonstrates the importance of the spatial orientation of the exploring electrode to an altered area of repolarization.

Relation between Right Precordial Leads (V1 and V2) and Intraventricular Leads. In 8 of 9 patients, the right precordial leads had ST-T complexes which had positive net values and showed S-T segment elevation, similar to that in the left intracavitary leads. Furthermore, in leads from the right ventricular cavity and right precordium, the ST-T complexes had net positive values in 3 cases, and in 2, they were positive in the precordial lead, and negative in the right intraventricular lead. The similarity of the RS-T complexes in the right precordial and left intracavitary leads, the concordant displacement of the S-T segment in both right precordial and right intraventricular leads indicated that the form of the RS-T complex was determined to a great extent by forces produced by repolarization of the septum and the free wall of the hypertrophied left ventricle. In the 2 patients with oppositely directed RS-T complexes, one may assume an earlier repolarization of the subepicardial lamina of the free wall of the right ventricle.

Summary and Conclusions

1. The intracavity potentials of the human left ventricle have been studied in 8 patients by the technic of retrograde ulnar arterial catheterization, and in one patient by passing an electrode into the left ventricle through a pulmonary vein at the time of operation for pneumonectomy.

2. In the region of the upper part of the interventricular septum, curves from the right ventricle showed a small R wave and a deep S wave; in the corresponding region of the left ventricle, the complex was of the QS variety. This consistent finding is interpreted to be confirmatory of Wilson's concept of the earlier depolarization of the upper interventricular septum from left to right.

3. In one patient, a tracing from the apex of the left ventricle showed a definite positive deflection, and a deep S wave (rS). The origin of this positive deflection is obscure.
4. In 8 of 9 cases, the direction of the RS-T complex of the cavity leads of the left ventricle was opposite to that in the lateral left precordial leads, indicating that the RS-T deviation was produced by forces in the free wall of the hypertrophied left ventricle.

5. That the right arm "unipolar" limb lead may not reflect cavity potentials is demonstrated in one case which had negative intracavitary leads, and a positive right arm lead.

ACKNOWLEDGMENTS

We are indebted to Dr. Franklin D. Johnston for valuable suggestions in the preparation of this paper and to Gladys Heckman, R.N., and Hanna Janouskovec, R.N., for their technical assistance.

REFERENCES

4 Personal experience of authors.
7 WILSON, F. N., JOHNSTON, F. D., and HILL, I. G. W.: The interpretation of the galvanometric curves obtained when one electrode is distant from the heart and the other near or in contact with the ventricular surface. Am. Heart J. 10: 196, 1934.
Cavity Potentials of the Human Ventricles
HENRY A. ZIMMERMAN and HERMAN K. HELLERSTEIN

Circulation. 1951;3:95-104
doi: 10.1161/01.CIR.3.1.95

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1951 American Heart Association, Inc. All rights reserved.
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/3/1/95

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/