Analysis of the Radiocardiogram in Heart Failure

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A scintillation counter permits the use of very low doses of I\(^{131}\)-albumin in the recording of radiocardiograms. In the presence of mild heart failure there is a distinct widening of the interval between the humps derived from the right and left heart. Application of the principle of three-compartment serial dilution suggests that this prolongation may be due in large part to reduced cardiac output; however, cardiac dilatation may contribute significantly. Although radiocardiographic tracings represent time concentration curves, wide angle counting over the heart does not permit a reliable direct estimate of cardiac output. The chief source of error lies in the variable contribution of surrounding extracardiac tissue to the total activity which is recorded.

Although Blumgart and Weiss,\(^1\) in early studies on circulation time, were able to detect the arrival of radioactivity in the heart by direct counting over the precordium, they were unable to demonstrate two peaks arising from the right and left heart in succession. In 1949 Prinzmetal and co-workers\(^2\) using Na\(^{24}\), were able to obtain double-humped curves when a Geiger counter was used in conjunction with a rapidly responding ratemeter and chart recorder. The tracings were designated "radiocardiograms." Wazer and Hunzinger\(^3\) also have succeeded in obtaining satisfactory double-humped curves. Prinzmetal's group has presented tracings illustrative of a variety of cardiac disorders. Alterations were noted in the height and duration of one or both humps in the presence of valvular disease, asymmetric enlargement, or decompensation. Little is as yet known of the physiologic factors or physical variables which may operate to distort the shape of such a tracing in the presence of heart disease, nor have sufficient data been compiled to evaluate the statistical reliability of the method as a clinical tool.

The present study was designed to determine not only whether characteristic changes can regularly be demonstrated in the presence of heart failure, but also whether it is possible to interpret the observed differences in terms of physiologic alterations which commonly accompany cardiac decompensation. Among such variables are pulmonary circulation time, lung blood volume, cardiac output, and cardiac volume.

Method

Isotope. Radiiodine was used as the tracer rather than Na\(^{24}\). The longer half time of I\(^{131}\), its ease of shipment, and its ready availability in most laboratories represent distinct advantages.

Apparatus. The detecting device consists of a recently described sodium iodide scintillation counter\(^4\) used in conjunction with the power supply of an SC-1 Autoscaler coupled to a ratemeter with a time constant of less than 0.5 seconds. A conventional Esterline-Angus recorder with chart speed adjusted to 4 inch per second serves to record the tracing. The response of the apparatus is linear for counting rates up to 100,000 per minute. The point of injection is indicated on the paper by a signal marker connected to a circuit closing clip on the plunger of a 2 ml. syringe.

The scintillation counter is positioned in contact with the chest at the center of the cardiac silhouette which has been previously outlined on the skin with the subject recumbent under a fluoroscope. The viewing characteristics of the counter are illustrated in figure 1. The front end of the lead shield consists of a shoulder 12 mm. thick which surrounds a circular opening 1½ inches in diameter for exposure of the crystal of similar diameter. The decline in count toward the periphery of the field is due partly to collimation by the orifice below the crystal and partly to the inverse square effect. Narrower collimation as provided by the addition of an extra lead ring 1 inch thick with a 1½ inch hole was also
tried. This technic was discarded because of the frequent necessity of searching for optimal positioning of the counter in order to record a second peak. The field of view which was employed thus includes considerable extracardiac tissue.

Procedure of Injection. T1-albumin (Abbott) was used routinely. The usual dose was 4 microcuries contained in a volume of 0.2 to 4.0 ml. A 20 gage needle was adequate for volumes less than 1 ml, but a number 18 was required for larger quantities. This dosage may be repeated at least 15 to 20 times without danger of excessive radiation to the body or to the thyroid gland. Preliminary saturation of the gland with iodide would permit even greater total dosage if the occasion so required.

The procedure whereby test material was delivered rapidly to the right heart was similar to that of Prinzmetal. The arm was warmed for approximately five minutes under a baking cradle. After venipuncture with the patient recumbent and the arm horizontal the solution was injected as rapidly as possible, the needle immediately withdrawn, and the subject's arm raised rapidly to a vertical position to flush the material without delay into the right auricle. Injection was complete within less than one second and the time from its beginning till the onset of a sharp rise in activity over the heart was usually one to two seconds. The longest was five seconds in a patient with severe decompensation.

Experimental Subjects. Normal controls were male patients on the gastroenterologic service who had no clinical or x-ray evidence of heart disease, and who were free from any disorder which might impair cardiac function. Their ages ranged from 25 to 50 years. Patients in heart failure had all shown unequivocal signs of decompensation when admitted to the hospital. They complained of dyspnea on exertion and edema. Examination revealed varying degrees of pulmonary congestion, hepatomegaly and generalized cardiac dilatation. They were males ranging in age from 37 to 67 years. At the time tracings were made digitalization had been completed, and save for one patient, compensation had been very substantially regained. No subject suffered any apparent discomfort or apprehension during the test. Pulses varied from 60 to 104 in normal series, and from 80 to 104 in patients with failure.

Double humped curves were obtained with ease in all normal subjects. In two severely decompensated subjects a second peak could not be demonstrated. Their records were not included.

Results

A record obtained on a normal subject is reproduced in the upper part of figure 2. The tracing reads from right to left. Replotting and smoothing of the tracing as in the lower part of figure 2 provides a curve which is more satisfactory for extrapolation and for the determination of subtended areas by planimetric measurement. The replotted curve now reads from left to right. Approximately half of the tracings were replotted separately by two individuals. Differences arising from variations in the technic of smoothing were minor.

Since the first hump of the tracing is derived from the chambers of the right heart, it will be labeled R.H., while the second hump, arising from the left heart, will be referred to as L.H.

Pulmonary Circulation Time

Peak to Peak Time. The lower curves of figure 3 illustrate a replotted tracing obtained from a normal subject along with another from a patient in decompensation. The simplest measurement which may be made is that of the distance between the peaks of R.H. and L.H. This will be termed uncorrected peak-to-peak time. In the upper part of figure 3, the individual values are recorded for a series of normal subjects and for a group of patients with heart failure. The average for the normals is 5.1 seconds and for the cardiacs 11.0 seconds. There is a single overlap in the vicinity of 6.5 seconds.

A more precise measurement is obtainable by correction of the L.H. peak for possible lateral displacement produced by overlap from the terminal portion of R.H. not recorded in the tracing. As shown in the lower part of figure 4 this correction involves an extrapo-
lation of the descending limb of R.H. and a subtraction of its height from that of the original tracing so as to obtain a derived curve pattern.\textsuperscript{5} Values for the extended limb of R.H. were derived by extrapolation of the recorded portion of its downslope on semilogarithmic paper. It may be seen from the plotted values of corrected peak-to-peak time in the upper part of figure 4 that there is a tendency for the measurements to exceed those of crude peak

Fig. 2. Radiocardiogram from a normal subject. The original tracing (A) at the top reads from right to left. Curve B was replotted from the original tracing and reads from left to right. Small fluctuations have been smoothed. R.H. and L.H. are the peaks due to emanations from the right and left hearts respectively. M represents the final level after mixing, and B corresponds to background count. The latter becomes the adjusted zero line for analysis of the curves.

which represents the pure L.H. component. An exponential decline of R.H. may tentatively be assumed on the basis of previous observations that dye dilution curves follow such a
to peak time. Thus the corrected time for the normals averages 5.8 seconds, and for the cardiaics 12.3 seconds. There is no overlap.

**Mean Pulmonary Circulation Time.** Although the distance between peaks is related to the time required for traversal of the lung pool, a more specific measurement would be that of mean pulmonary circulation time. This would be equivalent to the average time required for passage of a series of small and hypothetically isolated units of blood which move at differing rates because of chance variations in route of travel. When time-concentration curves are employed, the correct reference point for the timing of such a column is a vertical line which divides the area bounded by the curve and its baseline at its “center of gravity.” Although with sharply peaked curves, as obtained after rapid injection of dye into the pulmonary artery, this reference point is nearly identical with that of peak concentration, there is a tendency for it to shift significantly to the right when the downslope of the curve is prolonged. Mean times so determined between the R.H. and derived L.H. curves in normal subjects differed very little from that of corrected peak-to-peak time, being 6.5 seconds. The range was 4 to 9 seconds.

The above procedure was not applied to the curves obtained from decompensated subjects because of the frequent difficulty encountered in constructing the downslope of derived L.H. with any degree of precision. A considerable error arises from the estimation of a difference between two extrapolated curves, both of which are subject to individual errors of construction. Moreover, a long extrapolated projection of a gently falling curve, often encountered in these cases, further increases the error. A simple inspection of the curves obtained in the presence of decompensation suggests that the line corresponding to the center of gravity of the L.H. curve frequently is located further to the right of the peak than the corresponding line for R.H. In such instances mean lung time would be considerably longer than peak-to-peak time.

**Time-Concentration Analysis**

Tracings obtained by direct measurement of radioactive material coursing through the heart actually represent time-concentration curves made up of overlapping components from the right and left chambers. Time-concentration curves obtained by serial sampling...
of arterial blood after the intravenous injection of dye have been studied extensively in Hamilton's laboratory. MacIntyre, Pritchard, and co-workers succeeded in making direct recordings of such curves when 111-albumin was used as the test substance and the arterial blood allowed to pass through a tube placed adjacent to a scintillation counter.

In this formula it is apparent that blood volume (V) is multiplied by a fraction H/A. Thus the following expression may be used:

$$F_I = \frac{H}{A}$$

(3)

where $F_I$ represents output in terms of fraction of total blood volume discharged per unit time.

Equation 3 may tentatively be applied to the radiocardiogram in order to determine how closely the derived values approximate those which have been obtained for cardiac output by accepted methods. The term “fractional discharge index” will be applied to these values. Because the height of the final plateau is derived from the joint contribution of the right and left sides of the heart, the combined area under both humps of the curve was used in the calculation. Results are presented in figure 5, along with illustrative curves showing the origin of the measurements $A$ and $H$. The mean value in the normal subjects was 3.8 per cent. This is considerably higher than the fraction of total blood volume discharged per second by the heart as determined in normal subjects by conventional methods, which is in the range of 2 per cent.

It may be noted from figure 5 that the magnitude of the “fractional discharge index” is usually considerably below the range of normal in the presence of decompensation (mean 1.8).

Slope of Descending Limbs

It is apparent that the slope of the descending limb of R.H. is related to the propulsion of blood by the right ventricle. Difficulties in the determination of the downslope of derived pure L.H have already been mentioned. While it is conceded that overlap from varying extensions of R.H. occurs, the final slope of the second hump as recorded may be measured. It is pertinent to compare the steepness of the two slopes in normal and decompensated subjects.

The values for half time of the logarithmic extrapolation of the descending limbs are presented in the upper part of figure 6. Within the normal group the mean for R.H. was 2.8
seconds as compared to 3.7 seconds for the second hump. One explanation for this difference which immediately comes to mind is the extra dilution of tracer which is contributed by the reservoir of blood in the lungs while the material is in transit from the right to the left heart. Another contributing factor might be recirculation of a certain amount of blood which is moving rapidly via short systemic channels back into the right heart while the main mass is still in the left.

While the mean half-time for the R.H. component is distinctly prolonged in the group with decompensation, four of the hypertensive cases lie completely within the normal range (figure 6). Prolongation of the half-time of the final downslope is more consistently seen in all cases.

Relative Areas

Because of the frequent difficulty in defining the area under the derived L.H. curve in cardiac subjects the ratio of the area R.H. to total area was determined rather than R.H. to L.H.

With normal subjects there is a wide scatter of these ratios (figure 7). The mean is 0.64. If the two sides of the heart were viewed by the counter with identical conditions of geometry and with similar attenuation of count by intervening tissue a ratio of 0.5 would be anticipated. The higher value, which was observed, is compatible with the deeper situation of the left side of the heart as compared with the right, along with its greater shielding by overlying structures. The wide variation in the magnitude of the ratio is probably incidental to a variety of inconstancies in positioning of the counter or in the orientation of the heart within the chest. The scatter within the group of cardicus as a whole likewise is extreme, but there is a discernible tendency to cluster according to the type of heart lesion. The ratio in the two cases of cor pulmonale is higher than that encountered in any of the normals. Hypertensives lie toward the low end of the scale while patients with mitral stenosis are intermediate. This distribution of ratios correlates with right or left sided enlargement as the case may be. In mitral stenosis, of course, the right ventricle and left auricle are both dilated. It therefore would appear that the area under a given hump of the tracing is correlated with the volume of the corresponding chamber.

An increase of area under a hump obviously may be due either to greater height or greater width. Height is affected by the relative proximity of the counter to the chamber in question and by the aim of the collimated window.
Width, on the other hand, is primarily a function of slope. The latter is not theoretically affected by variations in positioning. Therefore, if it be shown that the area of a hump is positively correlated with the half-time of the declining limb, it would suggest that the demonstrated relationship of chamber size to area is not due solely to geometric relationships of the counter and the heart. That area actually is positively correlated with half-time is demonstrated in figure 8. The ratios of area (R.H./total) are plotted against those of half-time (R.H./total). The coefficient of correlation ($r$) is 0.74 St. E. 0.04.

This correlation strongly suggests that both half-time and area may increase preponderantly on one side of the heart in the presence of dilatation. Similar observations were made by Prinzmetal. It is therefore clear that cardiac output is not the sole determinant of either half-time or area. Moreover, a change in output would be shared equally by both sides of the heart.

### DISCUSSION

While it is conceded that a wide-angle detector as here employed picks up radioactivity from intrathoracic channels other than the heart, the sharp peaks manifested in the records permit the deduction that emanations arise chiefly from the two cardiac compartments. Consequently, a critical analysis seems justified. Conclusions must necessarily be somewhat tentative and invite further study.

### Technical Aspects

In previously reported studies radiosodium has been used as the test substance. Although the interstitial tissue of the lungs is small, it might be questioned whether passage of a readily diffusible ion into pulmonary tissue would affect the prominence of the second hump. In five cases Na$^{131}$ was compared to I$^{131}$-albumin to determine whether such might be true. No significant difference in the size or timing of the second hump could be demonstrated, but with Na$^{131}$ the height of the final plateau was appreciably lower even as early as 40 seconds after injection.

The effect of shifting the counter 1 to 2 cm. to either side of the midpoint of the cardiac silhouette was tested in nine normal subjects and in three patients with decompensation. The maximum change in corrected and uncorrected peak-to-peak time was one second in the normal subjects and two seconds in the cardiac patients. The "fractional discharge index" was not altered by more than 20 per cent except in one decompensated subject where the recorded segment of the final downslope was particularly short, and extrapolation was thus attended with considerable uncertainty. In this instance the variation was 40 per cent. Neither the peak-to-peak times nor the values for the index were affected sufficiently to shift any of the values outside of the ranges established for normal and decompensated subjects by the recordings from the mid position made on the series as a whole.

### Pulmonary Circulation Time

The observed value of 6.5 seconds for the mean lung circulation time in normal subjects is identical with that of the pulmonary circulation time as estimated by Blumgart and Weiss. The formula of Stewart would be applicable for the determination of the volume of blood in the lungs if cardiac output were known. The latter might be determined simultaneously with the radioangiogram by employment of simultaneous arterial sampling. The equation may be expressed: $q = T \times F$ where $F$ is cardiac output, and $T$ the mean time for passage of labelled material through a volume $q$. The line marking the mean time for R.H.
defines the beginning of the $q$ compartment at the exit of the right heart. Mean time of L.H. is at the exit of the left heart. The volume of $q$ therefore would include that of the pulmonary vessels and the left heart. Because of the inaccuracies involved in the construction of a pure L.H. curve in the presence of decompensation the present technic would not appear to be suitable for the estimation of lung blood volume in heart failure. It is possible, however, that a more rapid injection of tracer into the right heart by means of a catheter would give longer visible downslopes of the two humps and thus permit a more precise extrapolation.

It has already been stated that peak-to-peak time and mean time are not necessarily identical. It is pertinent to attempt an evaluation of the factors which might account for the prolongation of peak-to-peak time in the presence of heart failure. A formula which may be applicable is similar to that which has been derived by Newman and co-workers:

$$ P_3 = K_1 e^{-(r_1+r_3)} - K_2 e^{-(r_1+r_2)} + K_3 e^{-(r_1+r_3)} $$

The constants are as follows:

$$ K_1 = \frac{MV_1}{(V_1 - V_2)(V_1 - V_3)}; $$

$$ K_2 = \frac{MV_2}{(V_1 - V_2)(V_2 - V_3)}; $$

$$ K_3 = \frac{MV_3}{(V_1 - V_3)(V_2 - V_3)} $$

$P_3$ is the activity within or at the exit of the left heart at time $t$, $F$ the cardiac output, $V_1$, $V_2$ and $V_3$ the volumes of the right heart, lungs and left heart, respectively, and $M$ the size of the dose. It is assumed that successive dilution of a test substance occurs in serial compartments or pools represented by the two sides of the heart and lungs. It is also assumed that mixing is complete in all the pools and that delivery to the right heart is essentially instantaneous.

If the volume of the lungs of a normal subject is considered to be 900 ml., that of each side of the heart approximately 150 ml., and output 100 ml. per second, substitutions of the respective values in the above equation will give a peak of L.H. in the vicinity of five seconds. By reducing the value of $F$ it becomes apparent that the timing of the peak is very sensitive to changes in cardiac output. When flow is reduced to 50 ml. per second the peak shifts to 10 seconds. Upslope and downslope are considerably less steep. Lung volume, on the other hand, may be doubled without delaying the peak more than one second, although slopes again are markedly decreased.

The effect produced by a change in heart volume varies with the degree of enlargement and also depend on whether both sides participate. Doubling of the capacity of either side alone causes a delay of about one second and decreases both the upslope and the early part of the downslope. From the formula it can be seen that, because of the relatively large size of $V_2$ with respect to $V_1$ and $V_3$, the downslope of the terminal segment of $P_3$ approaches the rate determined by the exponential $e^{-(r_1+r_3)}$ (lung pool). $V_1$ and $V_3$ nevertheless modify the slope in the vicinity of the peak.

Tripling the volume of both sides of the heart retards the appearance of the peak to 12 seconds and flattens both slopes quite markedly. $V_1$, $V_2$ and $V_3$, then, are all involved, but are not identical in their effects on the shape of the L.H. curve in regard to upslope, the height and timing of the peak, the rate of change in the vicinity of the peak, and the terminal downslope.

The determination of lung vascular volume by employment of the formula $V_2 = \frac{F}{Slope \, L.H.}$ would not appear to be practical.

The portion of downslope visible in the L.H. hump is so nearly confined to the region of the peak that significant alteration is contributed by $V_1$ and $V_3$. The volume of the right heart might be determined with some precision in those instances where the downslope of R.H. is sufficiently long to allow an accurate extrapolation. Intracardiac injection would undoubtedly increase this likelihood.

If the above theory of serial compartmental dilution is applicable the twofold increase of peak-to-peak time which was seen in the cardiac subjects could be due in large part to
reduced cardiac output. Increased heart volume might contribute to an undetermined extent, but an expanded lung pool would be of very little consequence. It should be noted however that although recent experimental evidence supports the validity of the serial compartment analogy, and in spite of the probability that the complex anastomotic network of the lungs favors complete mixing, nevertheless insofar as the pulmonary compartment deviates from a true pool and approaches a series of parallel tubes which promote columnar flow the timing of the second peak will be proportionally delayed by an increased volume in the lungs. Moreover it should be emphasized that although the two humps of the curve are derived primarily from events occurring within the right and left heart respectively, there is obviously considerable contribution from all of the great vessels and their branches lying adjacent to the heart. The region viewed by the counter is not so selectively localized as when sampling is performed by catheterization.

The consistent prolongation of peak-to-peak time in minimal cardiac decompensation, as here observed, suggests that such a measurement might be a more reliable diagnostic procedure than is the clinical estimation of circulation time. Arm-to-tongue circulation time, as done at the bedside, is subject to unpredictable variations arising from subjective errors on the part of the patient, along with uncertainties in the rate of venous flow in the arm. Although the present results do not exclude the possibility of a lengthened interval occurring in certain disorders other than heart failure, three cases of severe emphysema, not included in the present series, were well within the range of normal.

Fractionate Cardiac Output

The feasibility of determining cardiac output or an index of this function by a time-concentration analysis as here performed merits discussion. It has already been pointed out that application of the formula \( F_T = H/A \), gives values about twice those of the expected range for normal volume output. A most likely explanation of the discrepancy is the presence of radioactive material in extracardiac tissue which adds to the height of the tracing after systemic mixing has occurred, and gives a spuriously high value for \( H \). Sharper collimation, perhaps concentrated as closely as possible on one chamber of the heart with no intent to obtain two humps might markedly reduce this error. Prolongation of the time of mixing before the height of the final plateau is measured would in some instances give a lower value, but in the present tests there was usually little difference between the height at one minute and at five minutes. The maximum decline was 15 per cent.

The effect of heart volume on the formula should be critically examined. It is apparent that in the presence of dilatation, if collimation remains the same, the relative contribution by the extracardiac field will be reduced. Hence the magnitude of the spurious increase of \( H \) as mentioned above will be lessened. Without doubt the low values given by the formula in cardiac decompensation are partially dependent on the relative decrease in contribution from extracardiac tissue.

Because of the unilateral augmentation of area observed to be correlated with dilatation of the corresponding chamber it is pertinent to consider whether this is consistent with expected dynamics of flow, and also to determine its effect on the formula under discussion. If the counter were responding to a portion of heart blood of constant volume, \( A \) would not be affected by enlargement of the chamber because the attendant dilution would reduce the height of the hump in proportion to the widened spread resulting from a relative delay in expulsion of active material. \( H \) likewise would be unaffected. But in the present technic, the counter views essentially all of a chamber, whether large or small, and at least in the right heart, dilution is not perceived. In fact, the effect of the entire dose may be fully registered for an instant in either a large or small cavity. In the case of a large cavity, area may be expected to increase because of slower proportional emptying of tracer irrespective of a constant volume output. But if the counter views the larger chamber in its entirety the final height \( H \) will also
be increased. If it can be shown that a large chamber causes increases in \( A \) and \( H \) which are of identical proportion, the validity of an equation for output will not be affected by this phenomenon. That such is the case may be shown in the following manner.

The assumption will be made that the volume of a chamber is doubled. \( H \) will now be twice as high if total blood volume remains constant. This follows from the fact that the counter is viewing twice as much blood of identical concentration. Now if the formula is to remain valid, that is, \( F \) unaffected by chamber capacity, \( A \) must also be doubled. When the dose is rapidly deposited in the right heart the area under the first hump will be dependent on the slope of the descending limb provided that the dose and various geometric relationships remain constant. The formula for the descending curve is the same as that of exponential decay: \( N = N_0 e^{-rt} \) where \( N \) is the fraction of original activity \( (N_0) \) present after time \( t \), and \( r \) is the slope of the exponential curve. Since the area under such a curve will vary as \( 1/r \), and it may be shown that doubling the chamber volume will cut the value of \( r \) in half the final result is to double the area. The derived calculation is therefore not influenced by increases in \( A \) resulting from unilateral dilatation.

**Asymmetric Dilatation**

Diagnostic application of the radiocardiogram for detection of preponderant dilatation on one side of the heart may be possible, but it would appear to be less specific than x-ray procedures because of the impossibility of differentiating between the contribution of auricle and ventricle.

**SUMMARY**

Satisfactory radiocardiograms were obtained by the use of a scintillation counter and \(^{14} \text{C} \) albumin in doses as low as 4 microcuries. Wide angle counting yielded a double-humped curve which corresponds to the passage of labeled blood through the right and left sides of the heart in sequence.

The interval between the two peaks averaged 5.8 seconds in normal subjects, and 12.3 seconds in a series of decompensated patients. Theoretic considerations would indicate that the prolongation observed in failure is probably due in large part to decreased cardiac output, but bilateral cardiac dilatation, if severe, may contribute significantly. It may be expected that this interval will be relatively less sensitive to changes in lung blood volume than to output and extreme dilatation.

Mean pulmonary circulation time was determined in normal subjects by the present technic and was found similar to peak-to-peak time, but such a measurement frequently cannot be determined accurately in tracings obtained from decompensated subjects.

The curve in its entirety has been analyzed in accordance with the time-concentration principle of cardiac output. Values thereby derived were considerably higher in normal subjects than the expected range of values for true cardiac output. This undoubtedly was due to nonspecific radioactivity arising from extracardiac tissues after systemic mixing has occurred. Values were relatively low in the presence of heart failure. Enlargement alone could be largely responsible for this difference, because of the attendant reduction of the relative contribution of blood in the extracardiac field of view. Wide-angle counting, as here employed, thus does not provide a satisfactory method for the determination of cardiac output by the time-concentration principle.

When dilatation is predominantly confined to one side of the heart, the area under the corresponding hump of the curve is proportionately increased because of the retarded emptying of tracer from the enlarged cavity.

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