"Left Heart" Radiopotassium Dilution Curves in Patients with Rheumatic Mitral Valvular Disease

By Hadley L. Conn, Jr., M.D., Donald F. Heiman, M.D., William S. Blakemore, M.D., Peter T. Kuo, M.D., and Stephen B. Langfeld, M.D.

"Left heart" radiopotassium-dilution curves were carried out in 9 patients with rheumatic mitral valvular disease. From the curves, blood flow, circulation times, and "left heart" and aortic volumes were calculated. When this technic is used along with "left heart" pressure measurements, it appears to have a satisfactory reliability in demonstrating abnormalities of flow, volume, and mitral valve function. In theory, its proper application permits complete quantitation of these parameters.

In some patients having long standing rheumatic heart disease routine clinical studies are inadequate either in establishing the existence or in evaluating the severity of stenosis or insufficiency of the mitral valve. Yet corrective surgical treatment makes evaluation of mitral valve function a matter of considerable importance. The surgical procedure, the surgical mortality, and the prognosis covering postoperative improvement are all influenced to some extent by the nature of the lesion present.

A number of specialized diagnostic procedures have been employed to improve diagnosis and quantify approximately the degree of the abnormalities present. Among these are angiocardiography, measurement of pulmonary arterial "wedge" pressure, ballistocardiography, and direct measurement of "left heart" pressure. Yet, mistaken evaluations are still occasionally made or corroborative evidence is desired, particularly with respect to the amount of insufficiency present. It was considered that indicator-dilution curves made by injections of an indicator into the left heart and aortic root could be used to provide additional and perhaps better information on blood flow, valve function, and also on left heart volume.

The data so far available from radiopotassium-dilution curves in 9 patients with valvular disease limited to the mitral valve have led us to hope that this technic, with the addition of left atrial sampling technics as recently advocated by Wood's group, and combined with presently used left heart pressure measurements, will permit a rather complete and quantitative description of mitral valve orifice area (both in systole and diastole), and of the pressure-volume-blood flow relationships in the left side of the heart.

Method

Patients with known or suspected rheumatic valvular heart disease, candidates for corrective surgical measures, were studied. Nine had valvular disease limited to the mitral valve, and they are included in this report. Only patient 9 had any appreciable signs or symptoms of myocardial failure.

Small plastic catheters were inserted into the left atrium and ventricle and an indwelling Courmand needle was introduced into the femoral or brachial artery. A small plastic catheter was passed in retrograde fashion under fluoroscopic guidance from the other femoral artery until its tip lay in the root of the aorta. After pressure measurements had been recorded in the various chambers, radiopotassium-dilution curves were made. Consecutive injections of 15 to 20 μ of radiopotassium (K°) in a volume of about 0.2 ml. were made into the left atrium, left ventricle, and root of the aorta. Femoral arterial blood was then sampled and its radioactivity counted with respect to time for the next 30 to 60 seconds. For this purpose the blood was drawn by a mercury gravity vacuum pump at the rate of approximately 0.5 ml/sec., through a cuvette (0.3 ml. volume) contained within a well-type scintillation counter. The dead space from blood vessel to cuvette was about 0.8 ml. The scintillation counter was linked to a specially built logarithmic counting rate.
meter,* which in turn fed the counts to a Brown Recorder (0.5 second full scale response) on which the log of concentration was recorded with respect to time.

Calculations

Cardiac output was calculated from the curves according to the Hamilton method. The cardiac index was then determined using standard height-weight body surface tables. The mean circulation time from injection to sampling site was measured as described by Hetzell,7 and the total volume of blood between the sites calculated as mean circulation time times cardiac output.

For purposes of evaluating chamber mixing volumes the part of the circulatory system under study was considered as a system consisting of 3 compartments, left atrium, left ventricle, and aorta, that are connected in series and have no back flow or feed back. The ventricular and at least part of the aortic blood were considered under normal circumstances to undergo complete mixing within 1 cardiac cycle, with exponential washout of indicator in a discontinuous fashion from the ventricle, and in a continuous fashion (obviously an approximation) from the aorta. In practice, the nature of the blood sampling imparted the general appearance of a continuous curve to all parts of the experimental curves. Atrial blood was considered to undergo essentially no mixing except in the case of insufficiency wherein there is a third mixing compartment in the system and regurgitant flow from ventricle to atrium. Unfortunately it was not initially appreciated that with mitral insufficiency a more complicated model system analysis was indicated and that our experimental data were insufficient to permit complete characterization of flow-volume parameters in the more complicated system. The addition of a left atrial sampling curve following left ventricular injection, if representative, would have allowed a complete analysis. In the absence of this information and in the presence of mitral insufficiency the data of left heart residual volume must be thought of as indicating the approximate sum of left atrial and ventricular mixing volumes minus stroke volume, and the amount of mitral regurgitation can be evaluated only indirectly.

Other assumptions and requirements tacitly accepted by users of the indicator-dilution principle have been enumerated and discussed by Newman and associates8 and by Meier and Zierler.9 Mathematical analysis of the model system used here, following the exponential washout equations developed by Newman,* leads to the results that the concentration ($P_1$) of indicator at any time, as sampled in the femoral artery subsequent to left atrial or ventricular injection, has the following relationships:

\[ P_1 = \frac{P_0}{k_2 + \ln \left( \frac{V_R}{V_R + V_E} \right)^{P_0}} \]

where $k_1$ and $k_2$ are exponential washout rate constants from left heart and aorta respectively; $V_R$ = residual volume (left heart); $V_E$ = stroke volume; $F$ = cardiac output.

The time-concentration curve is thus composed of 2 exponential terms whose rate constants ($k_1$ and $k_2$) have the relationships

\[ k_1 = \frac{F}{V} \ln \frac{V_R}{V_R + V_E} \] (2)

and

\[ k_2 = \frac{F}{V} \] (aortic). (3)

The appropriate rate constants ($k$'s) are determined by graphic analysis of the experimental curves. The $k_2$ representing the aortic washout is obtained from injection in the root of the aorta as well, so as to show which of the 2 $k$'s obtained from left heart injection curves represents left heart washout. For the aortic injections the concentration relationships with time are

\[ P_2 = P_0 e^{-k_2} \] (4)

and the relationship of $k_2$ to flow and volume the same as in equation (3). With blood flow already known from the dilution curves, and heart rate obtained, left heart mixing volumes and aortic mixing volumes were calculated.

Evaluation of Patients

An independent estimation of the amount of mitral stenosis and of the amount of mitral insufficiency was made in order to evaluate the radiopotassium-dilution curve as a measure of valve function. The independent estimation was based on information gathered by history and physical examination, fluoroscopy (9 patients), angiocardiography (6 patients), wedge-pressure measurements (4 patients), left heart pressure measurements (9 patients), surgical exploration (4 patients), postmortem examination (1 patient), and ballistocardiography. It was fully realized that the only true test of our technics would be their demonstration of the correct diagnosis in patients in which all other tests were in disagreement, inaccurate, or not informative. This demonstration would require in turn an infallible index of reference, an index that may not be found even in the surgeon's palpating finger or in a postmortem examination. The nearest substitute for an ideal standard of ref-
erence seemed to be the grading of patients into categories of 0 to 4 degrees of mitral stenosis or insufficiency on the basis of the sum of the above studies with emphasis particularly on the findings at operation and necropsy.

**RESULTS**

All the results except the aortic mixing volumes are shown in table 1. The diagnoses determined by other studies are shown in column 1 of that table. The general shape of the curves obtained from left heart injections is shown in figure 1. The contour of the curve in the patient with mitral stenosis was similar to normal but the terminal slope was frequently modestly flattened \((k = 0.15\) to 0.25 per second) whereas with mitral insufficiency the terminal slope was definitely abnormal, being very much flatter, or less steep, than normal \((k = .03\) to .07 per second).

**Cardiac Output and Stroke Volume**

In all 9 patients the cardiac output was subnormal, averaging 2.1 \(L./M.\frac{2}{min.}\), and ranging from a high of 2.6 to a low of 0.9 in patient 9 with “pure” mitral stenosis and cardiac failure. Most of these patients had atrial fibrillation so that only an average stroke volume could be determined. It was decreased in all patients in much the same manner as was cardiac output, ranging from 18 to 66 ml. and averaging 40 ml. per beat.

**TABLE 1.—Hemodynamic Results**

<table>
<thead>
<tr>
<th>Patient no. and diagnosis*</th>
<th>Indicator injection site</th>
<th>Cardiac index ((L./M.\frac{2}{min.}))</th>
<th>Mean stroke volume (ml.)</th>
<th>Onset of curve (sec.)</th>
<th>Peak of curve (sec.)</th>
<th>Mean circulation time (sec.)</th>
<th>Left heart mixing volume ((V_T)) (ml.)</th>
<th>Total volume, inj. to sample site ((V_R)) (ml.)</th>
<th>(\frac{V_R}{V_T})</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>L. At.</td>
<td>2.3</td>
<td>41</td>
<td>5.0</td>
<td>9.5</td>
<td>16.5</td>
<td>890</td>
<td>1020</td>
<td>0.88</td>
</tr>
<tr>
<td>MI 4</td>
<td>L. V.</td>
<td>2.3</td>
<td>41</td>
<td>5.0</td>
<td>9.5</td>
<td>16.0</td>
<td>880</td>
<td>1000</td>
<td>0.88</td>
</tr>
<tr>
<td>2</td>
<td>L. At.</td>
<td>2.2</td>
<td>45</td>
<td>8.0</td>
<td>13.0</td>
<td>22.0</td>
<td>1000</td>
<td>1530</td>
<td>0.65</td>
</tr>
<tr>
<td>MI 4</td>
<td>L. V.</td>
<td>2.2</td>
<td>45</td>
<td>8.0</td>
<td>13.0</td>
<td>19.0</td>
<td>830</td>
<td>1300</td>
<td>0.64</td>
</tr>
<tr>
<td>3</td>
<td>L. At.</td>
<td>2.1</td>
<td>38</td>
<td>7.0</td>
<td>12.0</td>
<td>21.5</td>
<td>1300</td>
<td>1200</td>
<td>1.08</td>
</tr>
<tr>
<td>MI 4</td>
<td>L. V.</td>
<td>2.3</td>
<td>41</td>
<td>6.0</td>
<td>11.0</td>
<td>20.5</td>
<td>1400</td>
<td>1200</td>
<td>1.17</td>
</tr>
<tr>
<td>4</td>
<td>L. At.</td>
<td>2.1</td>
<td>39</td>
<td>10.0</td>
<td>15.0</td>
<td>26.0</td>
<td>1200</td>
<td>1520</td>
<td>0.79</td>
</tr>
<tr>
<td>MI 3 MS 0-1</td>
<td>L. V.</td>
<td>2.1</td>
<td>39</td>
<td>8.0</td>
<td>12.5</td>
<td>23.0</td>
<td>1040</td>
<td>1340</td>
<td>0.78</td>
</tr>
<tr>
<td>5</td>
<td>L. At.</td>
<td>2.1</td>
<td>28</td>
<td>9.5</td>
<td>19.0</td>
<td>19.5</td>
<td>440</td>
<td>940</td>
<td>0.47</td>
</tr>
<tr>
<td>MI 2 MS 1</td>
<td>L. V.</td>
<td>2.1</td>
<td>28</td>
<td>8.5</td>
<td>17.0</td>
<td>18.5</td>
<td>530</td>
<td>870</td>
<td>0.61</td>
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<tr>
<td>6</td>
<td>L. At.</td>
<td>1.8</td>
<td>40</td>
<td>2.5</td>
<td>5.0</td>
<td>8.5</td>
<td>300</td>
<td>460</td>
<td>0.65</td>
</tr>
<tr>
<td>MI 2 MS 2</td>
<td>L. V.</td>
<td>1.9</td>
<td>41</td>
<td>2.0</td>
<td>4.0</td>
<td>6.0</td>
<td>250</td>
<td>330</td>
<td>0.75</td>
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<tr>
<td>7</td>
<td>L. At.</td>
<td>2.3</td>
<td>45</td>
<td>5.0</td>
<td>9.5</td>
<td>11.5</td>
<td>355</td>
<td>690</td>
<td>0.52</td>
</tr>
<tr>
<td>MI 2-3 MS 1</td>
<td>L. V.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>L. At.</td>
<td>2.6</td>
<td>66</td>
<td>7.0</td>
<td>15.5</td>
<td>17.0</td>
<td>500</td>
<td>1420</td>
<td>0.42</td>
</tr>
<tr>
<td>MI 2 MS 2</td>
<td>L. V.</td>
<td>2.5</td>
<td>62</td>
<td>5.0</td>
<td>13.0</td>
<td>15.0</td>
<td>480</td>
<td>1250</td>
<td>0.38</td>
</tr>
<tr>
<td>9</td>
<td>L. At.</td>
<td>1.0</td>
<td>18</td>
<td>16.0</td>
<td>22.0</td>
<td>24.0</td>
<td>90</td>
<td>720</td>
<td>0.13</td>
</tr>
<tr>
<td>MS 4 Failure</td>
<td>L. V.</td>
<td>0.9</td>
<td>18</td>
<td>5.0</td>
<td>8.0</td>
<td>10.0</td>
<td>90</td>
<td>300</td>
<td>0.30</td>
</tr>
<tr>
<td>Normal Values</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* See text for diagnostic evaluation methods; MS = mitral stenosis, MI = mitral insufficiency.
† Most of the patients had atrial fibrillation.
In patients 1 to 4 with essentially pure mitral insufficiency the onset of activity time (beginning of dilution curve) was normal or only slightly delayed, whereas the peak of activity was considerably delayed and mean circulation time prolonged up to 3 times normal. These circulation times in these patients were nearly alike, irrespective of whether the left atrium or left ventricle was the site of injection for indicator, the "left ventricular" times being about 90 to 100 per cent of the "left atrial" times. In patients 5 to 8 having mixed stenosis and insufficiency the same general pattern was seen with regard to the circulation times except that the mean circulation times in general were more nearly normal, and in each patient, all the atrial and ventricular circulation times showed a consistent difference. The "ventricular" times were consistently shorter than the "atrial," averaging about 85 per cent of the latter times. In the 1 patient with essentially "pure" mitral stenosis and cardiac failure this tendency was greatly exaggerated. All atrial injection circulation times in this patient were appreciably prolonged, whereas those from the ventricular injection were only slightly prolonged. Thus there was a considerable time differential, the ventricular times averaging about 35 per cent of the atrial times.

**Volumes**

The total volume of blood between the left atrial injection site and the femoral arterial sampling site was increased in all except patient 6. The increases were most marked in the patients presumed to have the most insufficiency, averaging 1.37 L. in patients 1 to 4. As would be expected from the circulation times, the total blood volume from left ventricular injection site to femoral artery sampling site was also increased appreciably to 1.21 L. in patients with insufficiency. The 2 volumes approximated each other in each of these patients. Total volume increases in the other patients with less insufficiency and more stenosis were generally less marked, total volume in these averaging only 0.85 L. with left atrial injections and only 0.78 L. with left ventricular injections. In the patient with stenosis alone, only the indicated volume of blood between atrium and femoral artery was moderately increased, whereas volume between ventricle and artery was within normal limits. This would be expected with left atrial enlargement alone. The left heart mixing volume (the residual left heart volume after subtraction of ejection volume) was increased in all patients with the exception of patient 9 with pure stenosis. The increases resulted in volumes up to about 1.5 L., or values nearly 20 times normal. The increases correlated reasonably well with the amount of mitral insufficiency as estimated by other procedures and correlated inversely nearly as well with the amount of stenosis (table 1). The ratio of mixing to total volumes \( \frac{V_R}{V_T} \) correlated even better with the functional abnormality. High ratios, averaging 0.85, were found with "pure" mitral insufficiency; lower ratios, averaging 0.53, were found...
with mixed lesions; and an essentially normal or even subnormal ratio was found with "pure" stenosis and cardiac failure.

The aortic mixing volumes ranged from 100 to 160 ml. in the women and 150 to 220 ml. in the men. Total blood volume from root of aorta to femoral artery averaged 310 ml. and ranged from 250 to 350 ml.

**Discussion**

These left heart indicator-dilution curves and their analysis provide evidence bearing on the following matters:

**Diagnosis of Functional State of Mitral Valve.** The contour of the curves obtained from left heart injection in subjects with mitral insufficiency can be easily differentiated from the curves associated with mitral stenosis (fig. 1). The former has a longer ascending limb, a lower peak concentration, and a much flatter terminal exponential slope than do the latter or do normal curves. The curves in "mitral stenosis" differ very little from normal curves we have obtained in dogs. The only consistent difference is a slightly flatter than normal terminal slope, which is presumably due to the subnormal cardiac output, since subjects with normal mitral valves and low cardiac output also have curves with a similar flattening of slope, as would be predicted by the "washout" theory of Newman.8 Thus, differentiation of a normal valve from a stenotic valve on the basis of curve contour alone does not appear feasible, while the presence of insufficiency can be diagnosed merely by casual inspection of the contour of the dilution curve unless blood flow is exceptionally low. In this case quantitation must be employed.

**Stroke Volume and Cardiac Output in Rheumatic Mitral Valvular Disease.** As has been generally recognized in patients with long-standing, marked rheumatic mitral valvular disease, cardiac output and stroke volume are consistently and definitely subnormal; about 3/4 normal, when the subjects are at rest.

**Left Heart Volumes in Mitral Rheumatic Heart Disease.** Also as generally appreciated, left heart total blood volumes (volume of left atrium to femoral artery minus volume of root of aorta to femoral artery) are consistently increased in all patients with mitral disease. In general the largest increases were associated with the most mitral insufficiency. Left heart volumes as high as 1.2 L. were recorded in our patients with marked insufficiency by means of the calculation of the mean circulation time times cardiac output. With regard to the calculated mixing volumes, the curves are not sufficient for making a separate evaluation of ventricular residual mixing (presumably total ventricular residual) volume and of atrial mixing volume when turbulence is occurring in both chambers as in the patient with regurgitation. However, when a maximum value of 300 to 400 ml., well above the normal value of 80 to 90 ml., was assigned to ventricular residual volume, we not only found this increase in ventricular volume but also a large atrial mixing volume attributable to a strong regurgitant jet that would produce atrial turbulence. In fact, this calculation indicated atrial mixing volumes as high as 1.0 L. associated with grade 4 insufficiency in patient 3. This mixing volume is approximately equal to, and in one case greater than, the calculated total atrial volume. Stated otherwise, the high ratios of mixing to total blood volume, averaging near unity in the combined left heart and aortic segments of patients with marked insufficiency indicate that nearly the entire blood flow in the left heart is turbulent. Indeed these ratios, along with the nearly identical, prolonged circulation times resulting from atrial and ventricular K42 injections, suggest that, as far as filling and mixing are concerned, patients with "pure" insufficiency have a functionally single left heart chamber composed of an enlarged left atrium and ventricle that are not separated at any time in the cardiac cycle.

With increasing mitral stenosis and decreasing insufficiency, total and mixing volumes were still elevated, though less markedly so, and the figures of 400 to 600 ml. for mixing volume are in agreement with both a moderate increase in ventricular residual volume and an appreciable atrial mixing volume. However, that the increased atrial volume was not entirely
involved in turbulent mixing, and that the regurgitant jet effect was decreased (com-
pared to "pure" insufficiency) was indicated by the intermediate, only moderately elevated mixing to total volume ratios of 0.40 to 0.80.

In patient 9, the only one with "pure" stenosis, the only abnormality in volume was an increased left atrial volume to about 400 ml. The calculated left heart residual volume (mixing volume) was almost normal, compatible with an essentially normal left ventricular size and mixing and no atrial mixing (volume); thus a regurgitant flow was excluded indirectly. These data are consistent with the view that in "pure" stenosis the only appreciable derangement in left heart volume is an increased atrial volume in which there is no turbulence of flow. It seems prudent to point out, however, that these views are based on the findings in 1 patient, who had a very low cardiac output. Evidence of appreciable mixing in the atrium may be discovered under conditions of more normal blood flow.

Left Heart Circulation Times in Rheumatic Mitral Valve Disease. The prolonged, nearly identical circulation times from left ventricle to artery and left atrium to artery in mitral insufficiency are also in line with the concept that indicator is washed back and forth between 2 enlarged heart chambers, which, except for their out-of-phase activities, could be functionally considered as a unit. The differential in these times with stenosis and the apparent increased differential with increasing stenosis is in line with the concept of delay of indicator, whatever its explanation, on the atrial side of the stenotic valve.¹

Quantitative Diagnosis of Left Chamber Volumes, Forward and Regurgitant Flow, and Valve Orifice Areas. The present analytic method and experimental curves are, as far as we know, adequate to describe the above parameters in subjects with no mitral regurgitant flow, in whom simultaneous pressures are recorded. Thus, it appears "pure" mitral stenosis can be recognized and the degree partially quantified by the difference in circulation times from atrium and ventricle to artery and by the subnormal ventricular residual to total left heart volume ratio as well as by calculation with Gorlin's formulas.¹⁰,¹¹ These parameters are not all properly quantitated in the presence of insufficiency. However, the presently observed good correlations, particularly of the "equalization" and prolongation of left heart mean circulation times and the high ratios of left heart mixing volume to total volume, with the degree of insufficiency diagnosed by other procedures, convince us that we can determine the left atrial mixing volume for practical purposes and thus, indirectly, the amount of insufficiency present. The differentiation of the case with insufficiency with no stenosis from insufficiency with some stenosis does not seem on very firm grounds except from inference based on the knowledge that in rheumatic disease the 2 lesions usually coexist when 1 is of moderate degree.

It is likely, however, that the present inadequacies are based more on the experimental design used than on the methods employed. If a curve from another sampling site is obtained, the entire model system can be characterized with regard to forward and backward flow and chamber volumes. As stated previously, Wood's group has been aware of this problem also and have started drawing left atrial samples following left ventricular injections of indicator. If the blood sample can be shown to be truly representative of mixed atrial blood and the model analysis is essentially valid, the indicator-dilution curves will permit evaluation of all flows and volumes and the simultaneously obtained pressure curves along with Gorlin's formulas will permit an estimation of the functional area of the mitral valve during both systole and diastole. Conversely, if the surgeon can accurately determine orifice area, measurements of indicator-dilution flows and appropriate left heart pressures can be used conjointly to provide an in vivo test of the mitral valve formulas.

Summary

Nine patients having long standing rheumatic valvular heart disease involving only the mitral valve were studied by a left heart radio-kalium-dilution curve technic during left
heart catheterization. Arterial dilution curves were obtained following left atrial, left ventricular, and aortic root injections.

Results showed (a) gross differences between the left heart injection curves in mitral stenosis and those in insufficiency; (b) low cardiac output and stroke volume in all patients; (c) left heart mixing and total volume changes, the ratios of which were high with mitral insufficiency and normal or low with stenosis; (d) variations in circulation times from left atrial and ventricular injections, with similar times in mitral insufficiency, appreciably different times in mitral stenosis, and intermediate values with combined lesions.

The results are consistent with the concept that in mitral stenosis the only important change in volume or blood flow in the left heart is an increased total atrial volume, whereas in mitral insufficiency not only are both atrial and ventricular total volumes increased but also turbulence of flow then occurs, because of the regurgitant jet, in most or all of the atrium, in addition to the ventricle.

Evaluation, by the dilution curves, of mitral valve dysfunction and amount of regurgitant blood flow, although apparently quite satisfactory, is indirect. With the use of an appropriate additional curve and simultaneously measured left heart pressures, it is possible, in all patients, to calculate the volumes of the separate chambers, the volume of regurgitant flow, and the area of the mitral valve orifice, both during systole and diastole.

**Summario in Interlingua**

Esseva studiate 9 patientes con prolongate rheumatic morbo cardiovalvular, afficiente solmente le valvula mitral. Le studio esseva executate per medio de un technica a curvas de dilution de radiokalium in le corde sinistre durante catheterisation sinistro-cardio. Curvas de dilution arterial esseva obtenite post injectiones in le atrio sinistre, le ventriculo sinistre, e le radice aortica.

Le resultatos indicava (a) grande differentias inter le curvas de injection sinistro-cardio in stenosis mitral e in insufficientia mitral; (b) basse valores pro le rendimento cardiac e le volumine per pulso in omne patientes; (c) alterationes de mixtion sinistro-cardio e de volumine total con proportiones che esseva alte in insufficientia mitral e normal o basse in stenosis; (d) variationes del tempores circulatori post injectiones sinistro-atrial e sinistro-ventricular, con simile tempores in casos de insufficientia mitral, satis differente tempores in stenosis mitral, e valores intermediari in casos de lesions combine.

Le resultatos es de accordo con le concepto que in stenosis mitral le sol importante alteratione de volumine o de fluxo sanguine in le corde sinistre es un augmento del total volumine atrial, durante que in insufficientia mitral il ha non solmente un augmento del volumines total tanto atrial como etiam ventricular sed in plus un occurrantia de fluxo turbulente che se manifesta, a causa del regurgitation explosive, in le plus grande parte del atrio o mesmo in le atrio integre e non solmente in le ventriculo.

Le evaluation, per medio de curvas de dilution, de dysfunction del valvula mitral e del quantitate del regurgitante fluxo sanguine es certo satis adequate, sed illo es nonostante indirecte. Per usar un appropriate curva additional e per mesurar simultaneemente le pression sinistro-cardio, il es possibile calcular le volumine del cameras individual in omne patientes e etiam le volumine del fluxo regurgitante e le area del orificio del valvula mitral, tanto durante le systole como etiam durante le diastole.

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Cheyne-Stokes Respiration. But there is a symptom which appears to belong to a weakened state of the heart, and which, therefore, may be looked for in many cases of the fatty degeneration. I have never seen it except in examples of that disease. The symptom in question was observed by Dr. Cheyne, although he did not connect it with the special lesion of the heart. It consists in the occurrence of a series of inspirations, increasing to a maximum, and then declining in force and length, until a state of apparent apnoea is established. In this condition the patient may remain for such a length of time as to make his attendants believe that he is dead, when a low inspiration, followed by one more decided, marks the commencement of a new ascending and then descending series of inspirations. This symptom, as occurring in its highest degree, I have only seen during a few weeks previous to the death of the patient. I do not know any more remarkable or characteristic phenomena than those presented in this condition, whether we view the long-continued cessation of breathing, yet without any suffering on the part of the patient, or the maximum point of the series of inspirations, when the head is thrown back, the shoulders raised, and every muscle of inspiration thrown into the most violent action; yet all this without rale or any sign of mechanical obstruction. The vesicular murmur becomes gradually louder, and at the height of the paroxysm is intensely puerile.

The decline in the length and force of the respirations is as regular and remarkable as their progressive increase. The inspirations become each one less deep than the preceding, until they are all but imperceptible, and then the state of apparent apnoea occurs. This is at last broken by the faintest possible inspiration; the next effort is a little stronger, until, so to speak, the paroxysm of breathing is at its height, again to subside by a descending scale.—William Stokes. The Diseases of the Heart and the Aorta. Dublin, 1854.
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Circulation. 1957;15:532-539
doi: 10.1161/01.CIR.15.4.532

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

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