Mitral regurgitation and characteristic changes in impedance cardiogram*

JAMES N KARNEGIS, JEANNE HEINZ, WILLIAM G KUBICEK

From the Departments of Medicine, United Hospitals—Miller Division, St Paul, Minnesota and the University of Minnesota Medical School, Minneapolis, Minnesota, and the Department of Physical Medicine and Rehabilitation, University of Minnesota Medical School, Minneapolis, Minnesota, USA

SUMMARY We have noted that the impedance cardiographic waveform of patients with mitral regurgitation may show typical abnormalities not found in other forms of cardiac disease, either valvular or congenital. In order to investigate this we reviewed the impedance cardiograms of all our patients who had also undergone cardiac catheterisation, and selected two groups both of which were inclusive and complete: (1) 22 patients with the diagnosis of normal heart, and (2) 36 patients with the diagnosis of isolated mitral regurgitation.

An index was calculated algebraically from the change of impedance (ΔZ) tracing by adding together the height of the C wave and the height of the nadir of the X descent and subtracting the height of the V wave, that is ΔZ index (in units of ohms) = $C + X - V$. The mean ΔZ index for the normal group was 1.64 and for the mitral regurgitation group 0.96 ohms. Similarly, an index was calculated from the first time derivative of the change of impedance (dZ/dt) tracing, that is dZ/dt index (in units of ohms/s) = $C' + X' - V'$. The mean dZ/dt index for the normal group was 1.32 and for the mitral regurgitation group 0.48 ohms/s. Though there was some overlap of individual points between the two groups, the mean values for both the ΔZ index and the dZ/dt index separated the group with mitral regurgitation from the normal group with a high level of statistical significance.

We concluded that mitral regurgitation might be associated with a characteristic abnormality of the impedance cardiographic waveform. In addition, an index can easily be calculated from the tracings which may be useful in identifying patients with mitral regurgitation.

The thoracic impedance cardiogram is a non-invasive method of studying cardiac function. Recent work has shown that the impedance cardiogram can be used for investigating the left atrial ejection fraction,¹ measuring the cardiac output,²⁻⁸ identifying the mechanical contraction of the atria and ventricles,⁹⁻¹¹ assessing aortic valvular regurgitation,¹² measuring left ventricular ejection,¹³ and monitoring systolic time intervals.^{14 15} We have observed that patients with mitral regurgitation may have a grossly abnormal impedance cardiographic waveform. The purpose of this paper is to report the changes in the impedance cardiogram which appear to be characteristically associated with regurgitation of the mitral valve, and to describe an index which can easily be obtained from the tracing and which may be useful in identifying patients with mitral regurgitation.

Materials and methods

The Minnesota impedance cardiographic equipment and the technique of recording the impedance cardiographic waveform have been previously described.² In brief, two electrodes were placed around the base of the neck and two around the upper abdomen. A 100 kHz 4 ma sinusoidal constant current was passed through the outer two electrodes, and the thoracic electrical impedance was monitored from the inner two electrodes. The change in impedance and its first time derivative were recorded on an Electronics for Medicine electron beam recorder. All recordings were made with the patient's breath held at end-expiration. Calibration was by 0.1 ohm and 1 ohm/s signals produced by the electronic circuitry.

The records of all of our patients with impedance cardiograms who had also undergone cardiac catheterisation were reviewed, and two groups were selected. One group consisted of

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patients with mitral regurgitation, but not mitral stenosis or other valvular or congenital cardiovascular defects. There were 38 such patients with mitral regurgitation, but the impedance cardiograms of two of them could not be used in this study: in one patient, the tracings were distorted by the presence of a sinus tachycardia of 120 beats per minute; in the second patient, all of the calibration signals were partly offscale. The study group with isolated mitral regurgitation thus consisted of 36 patients, 13 men and 23 women, ranging in age from 16 to 70 years, with an average of 49 years.

The aetiology of the mitral regurgitation was recorded as follows: rheumatic heart disease eight, chordal rupture seven, myxomatous degeneration four, prolapsed mitral valve two, ruptured papillary muscle one, and bacterial endocarditis one. In 13 cases the aetiology was not specified.

The second group consisted of 22 patients in whom catheterisation had excluded cardiovascular disease. This group included 13 men and nine

Fig. 1 Thoracic impedance waveforms recorded from a normal patient. ΔZ index = 1.54. dZ/dt index = 1.50.

Fig. 2 Thoracic impedance waveforms recorded from a patient without mitral regurgitation.

women, ranging in age from 12 to 47 years, with an average of 25.0 years.

Postoperative impedance cardiograms were available for seven of the patients who had had open-heart surgery for replacement of the mitral valve.

Fig. 1 illustrates the normal impedance cardiogram. From the top of the figure down is shown lead II of the electrocardiogram, the change of thoracic impedance (ΔZ or ΔZ) which is the impedance cardiogram, and below it the first time derivative of the change in impedance (dZ/dt). The curves were recorded such that a decreasing impedance was represented by an upward deflection.

The ΔZ waveform normally is composed of three major deflections.³ Fig. 1 shows that a downward deflection, designated the "A" wave, was associated with the P wave of the electrocardiogram and atrial contraction. This was followed by two upward deflections, the "C" wave and the "V" wave. The "C" wave was the larger of the two and was associated with the electrocardiographic QRS complex and ventricular contraction. After the peak of the C wave was recorded, the impedance increased rapidly (the "X" descent) and then de-

creased again as it moved upward to inscribe the V wave. The V wave was the smaller of the upward deflections and was recorded during ventricular diastole.

The zero reference indicates the lowest point reached just before the beginning of the upstroke of the C wave. Fig. 2 shows that the rapid vibrations of the first cardiac sound also occur at this time. In this patient the V and V' deflections were diminutive. The values for C, X, and V were measured as illustrated in Fig. 1.

It can be seen in Fig. 1 and 2 that the A, C, X, and V components noted in the ΔZ waveform had comparable deflections in the dZ/dt tracing. To be consistent with mathematical convention, these deflections in the first derivative tracing were designated A', C', X', and V'.

The zero reference point for dZ/dt temporally coincides with the zero point for ΔZ . The values for C', X', and V' were measured as illustrated in Fig. 1.

Fig. 3a shows the ΔZ and dZ/dt recordings from

a patient with severe mitral regurgitation and atrial fibrillation. In contrast to the normal tracing reproduced in Fig. 1, there was a smaller C wave, a deeper X descent, and a larger V wave. Parallel changes were evidenced for dZ/dt . Impedance tracings from the same patient taken postoperatively resembled the normal and are shown in Fig. 3b.

Three patients with mitral regurgitation had V and V' waves of even greater amplitude than their respective C and C' waves. An example of this is shown in Fig. 4a. The patient's postoperative impedance tracings resembled the normal and are shown in Fig. 4b.

Five to eight consecutive beats were measured from the ΔZ and the dZ/dt tracings and the averages were tabulated. For each patient an algebraic index was derived from the height of the C, X, and V deflections as follows, all values being expressed in ohms: ΔZ index = $C + X - V$. Similarly, using the dZ/dt tracing an algebraic index was calculated as follows, all values being expressed in ohms/s: dZ/dt index = $C' + X' - V'$.

Fig. 3 Thoracic impedance waveforms recorded from a patient with mitral regurgitation. (a) Before operation: ΔZ index = 0.45; dZ/dt index = -0.31. (b) After operation: ΔZ index = 2.42; dZ/dt index = 3.33.

Fig. 4 Thoracic impedance waveforms recorded from a patient with mitral regurgitation. (a) Before operation: ΔZ index = -0.68; dZ/dt index = -0.12. (b) After operation. ΔZ index = 0.96. dZ/dt index = 0.79.

Results

The Table summarises the data for both the normal and the mitral regurgitation patients. Left ventriculograms had been done in each patient and were used to judge semiquantitatively the severity of the regurgitant mitral flow. In one patient the regurgitation was mild, while in the remainder it was severe.

Fig. 5 shows a plot of ΔZ indices for the normal subjects compared with patients with mitral regurgitation. Four patients from the latter group were excluded because the ΔZ tracing was incomplete. The mean for the normal group of 22 patients was 1.64 with a standard deviation of 0.36. The mean for the mitral regurgitation group of 32 patients was 0.96 with a standard deviation of 0.59. Statistical analysis testing for the difference between means of the two groups gave a *t* value of

4.727, which was beyond the 0.001 level of significance.

Fig. 5 also shows the ΔZ indices for the seven patients with mitral regurgitation who had undergone mitral valve replacement and for whom we had postoperative impedance cardiograms. The preoperative ΔZ tracing of one of the seven patients was incomplete. Compared with their preoperative

Table Summary of data

	Group	No.	Mean	Standard deviation	<i>t</i> test
ΔZ index: (ohms)	Normal	22	1.64	0.36	<i>p</i> < 0.001
	Mitral regurgitation	32	0.96	0.59	
dZ/dt index: (ohms/s)	Normal	22	1.32	0.55	<i>p</i> < 0.001
	Mitral regurgitation	35	0.48	0.69	

values, five of the remaining six patients postoperatively showed an increase of the ΔZ indices toward the normal range.

Fig. 6 shows a plot of dZ/dt indices for the normal compared with the patients with mitral regurgitation. One patient from the latter group was excluded because the dZ/dt tracing was incomplete. The mean of the normal group of 22 patients was 1.32 with a standard deviation of 0.55. The mean for the mitral regurgitation group of 35 patients was 0.48 with a standard deviation of 0.69. Statistical analysis testing the difference between the means of the two groups gave a t value of 4.755, which was beyond the 0.001 level of significance.

Fig. 6 also shows the dZ/dt indices for seven patients with mitral regurgitation after surgical replacement of the mitral valve. It can be seen that in six of the patients the dZ/dt indices increased toward the normal range. The increase in the ΔZ

Fig. 5 Plot of ΔZ indices for normal subjects and for patients with mitral regurgitation.

Fig. 6 Plot of dZ/dt indices for normal subjects and for patients with mitral regurgitation.

and the dZ/dt indices, after surgical correction of the mitral regurgitation, resulted from the waveforms returning toward normal. Examples of this are shown in Fig. 3b and 4b.

One of the surgical patients was an exception and continued to show an abnormal waveform postoperatively in that a broad saddle-shaped plateau separated the C wave from a relatively prominent V wave. There was clinically no obvious evidence of persistent mitral regurgitation, and no other explanation was at hand. The patient died at home a few months after operation, but, since no necropsy was done, the condition of the heart and of the mitral prosthesis remained unknown.

It should be noted that the A and A' waves disappear when there is atrial fibrillation since during

this arrhythmia atrial contraction is absent. This is illustrated in Fig. 3. Fig. 3 also shows that atrial fibrillation does not affect those characteristic changes in the impedance cardiogram which are associated with mitral regurgitation, nor does it affect the waveform returning toward normal post-operatively.

Discussion

The thoracic impedance cardiogram is a convenient and safe way of studying the circulation. It has been shown that a variety of important information about mechanical and physiological cardiac function can be readily extracted from the tracings.¹⁻¹⁵

The impedance cardiogram also appears to manifest characteristic changes in patients with mitral regurgitation with the abnormal waveform returning toward a normal contour after surgical correction of the valvular lesion (Fig. 3).

In our series even the most flagrantly distorted tracings reverted toward normal after operation, as illustrated in Fig. 4. In contrast to the patients with mitral regurgitation, we have not observed this abnormal pattern in patients with mitral stenosis, aortic stenosis, aortic regurgitation, or in patients with various congenital cardiac malformations.

The recognition of these abnormalities in the impedance cardiogram suggested the possibility of devising a formula that might differentiate patients with mitral regurgitation from normal patients. We tried several different approaches in an attempt to quantify the changes and found that an index could easily be obtained from the height of the major deflections of the impedance recordings using either the ΔZ or the dZ/dt tracing. It can be seen in Fig. 5 and 6 that the group of normal patients showed some variability in their indices, and as might be expected there was a greater variability within the mitral regurgitation group. The indices did not perfectly separate the normal patients from the patients with mitral regurgitation, since there was some overlap between the individual measurements. Nevertheless, Fig. 5 shows that the ΔZ index discriminated between the two groups with a very high level of statistical significance ($p < 0.001$). Fig. 6 shows that the same was true for the dZ/dt index, the difference between the means of the normal and the mitral regurgitation group also being beyond the 0.001 level of significance.

In order that the findings would not be confounded by complicating factors, we selected for this study patients with isolated mitral regurgitation. Those who in addition to mitral regurgitation had any mitral stenosis, aortic stenosis, aortic regurgitation, or other cardiovascular lesions were excluded.

Since all but one of our mitral patients had severe mitral regurgitation, our data were not sufficient to determine whether the mitral regurgitation indices derived from the impedance tracings could be used to estimate varying degrees of mitral regurgitation. It was of interest, however, that the single patient who had only a mild valvular leak had one of the highest (that is, closer to the mean of the normal patients) ΔZ and dZ/dt indices found in the mitral regurgitation group, measuring 1.69 ohms and 1.25 ohms/s, respectively.

It is clear that the calculation of these indices is not applicable in those instances where the waveforms are distorted and no definite V and V' waves are discernible. One patient who had a sinus tachycardia of 120 beats per minute was an example of this and therefore was excluded from this study. Her ΔZ tracing was essentially monophasic, the X descent not being interrupted by a definite V wave. This was presumably caused by the rapid heart rate shortening ventricular diastole to such an extent that either the V wave was not clearly inscribed, or was obscured by the earlier falling A wave, or both.

In this study the ΔZ index and the dZ/dt index appeared to be of about equal value in distinguishing patients with mitral regurgitation from normal patients. Since the dZ/dt tracing tends to wander less with changes in respiration than does the ΔZ tracing, the former may be easier to use when studying infants and subjects who have difficulty controlling their breathing.

The pathogenesis of the changes in the waveform of the impedance cardiogram associated with mitral regurgitation is not known. Since the height of the C' wave is highly correlated with stroke volume,² the smaller C' and C waves in mitral regurgitation might result from a reduction in ventricular stroke output, and/or from interference with the normal forward flow of pulmonary venous blood during ventricular systole.⁹ If the latter possibility were operative, the deeper nadir of the X and X' descents might reflect persistent engorgement of the pulmonary venous system and continued slowing of pulmonary venous flow. The onset of the ensuing phase of ventricular diastole would then be expected suddenly to release the surfeit of blood pent up proximal to the mitral valve. The resultant forward rush of pulmonary venous blood might thereby play a role in producing prominent or giant V and V' waves.

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Requests for reprints to Dr James N Karnegis, 5604 Valley Lane, Minneapolis, Minnesota 55435, USA.