The Calibration of Heart Sound Intensity

By Maurice McGregor, M.D., M.R.C.P., Maurice B. Rappaport, E.E., Howard B. Sprague, M.D., and Allan L. Friedlich, M.D.

There is no standardized method for the calibration of total energy conducted to the surface of the chest caused by the cardiac sounds. Loudness is a subjective term which is dependent upon total energy, frequency of the main components and the specific sensitivity of the human hearing mechanism. A method is described in which the amplitude of the chief component of the heart sounds is compared to that produced by a standard sound signal at 80 decibels at 500 cycles per second and an intensity ratio calculated. The method has been applied to normal individuals of different ages and to those with mitral valve disease and those with pulmonary hypertension. The findings confirm the clinical observation that mitral stenosis with persisting mobility of the valve leaflets is associated with a loud first heart sound. However, the measurement of intensity of the second heart sound did not show so reliable a correlation between the loudness of the sound and the pulmonary blood pressure. The relationship is somewhat obscured by the wide range of normal values in the intensity of the second heart sound.

The clinical subjective assessment of the intensity of heart sounds is generally accepted as valuable, but few attempts at objective calibration have been made. Since heart sounds consist of superimposed vibrations of various frequencies and intensities, most workers have approached the problem by separating the various components of a sound into frequency bands by means of filters and comparing the signal thus received with a signal of known frequency and energy. By this means, the intensity of each frequency band can be recorded in absolute units of energy. While the intrinsic value of this procedure may be considerable, the technic is too complicated for widespread clinical application and the results are not easy to correlate with the subjective impression of loudness.

An alternative approach would be to employ a microphone constructed to attenuate low frequency vibrations to the same extent as the average human ear and to compare the resultant heart sound deflections with those produced by a standard sound signal. Methods for the registration of a standard sound signal have been reported by Wells and co-workers in 1949 and Sloan and Greer in 1955. There is obviously some variation in the audiograms of different individuals so that no microphone can be constructed with response characteristics which will exactly reproduce the auditory impression of each individual physician. However a microphone constructed to the “average audiogram” will produce deflections which are closely comparable to the impression of loudness obtained on auscultation by all with normal hearing. This method of calibrating heart sounds would not give a result in terms of absolute units of intensity. It would rather supply a method of measuring the intensity of sound phenomena in an arbitrary unit, thus enabling comparison of all records obtained by instruments which have the same frequency response characteristics, a chest piece of the same size and shape, and the same standard sound signal. So far as we are aware, no clinical application of this method of calibration has been made.

The intensity of the first and second heart sounds of a series of normal and abnormal cases was measured, and the method was found to be both practical and valuable in interpretation of records. Because of variations in the phonocardiographs in general use, however, these results are only applicable to a limited number of centers using identical equipment. This report is made in the hope that it will stimulate
measures to secure some standardization in the manufacture of phonocardiographs for clinical use.

**METHOD**

"Logarithmic recordings" were made with a Sanborn Tribeam Stethocardieter while the patient was at rest in the supine position. To minimize the attenuation due to pressure of the chest piece on the skin, a large open bell of 4 cm. diameter was applied with the minimum amount of pressure necessary to produce skin contact over the whole diameter of the bell. A standard sound signal (80 decibels at 500 cycles per second) was recorded after each tracing at the same intensity setting of the instrument, as described by Wells and associates. The maximum deflection produced by a heart sound divided by the deflection produced by the standard signal gave a value which will be referred to as the intensity ratio (I.R.).

The normal variability of the first sound was ascertained by measuring the intensity ratio from apical records of 67 subjects without cardiovascular or respiratory disease. Comparison was made with two abnormal groups (table 1). The first consisted of 34 asymptomatic individuals with "mild or doubtful mitral valve disease," who had previously had rheumatic fever and who showed evidence of mild or minimum cardiac involvement. The only evidence of cardiac involvement was a soft basal diastolic murmur in four cases, an apical systolic murmur in seven, and a systolic murmur and faint or doubtful mid-diastolic in five. In the remainder, apical mid-diastolic murmurs of more than grade 1 intensity were heard and recorded. The second group with which comparison of the first sound was made consisted of 44 patients with well developed mitral valve disease and varying but definite disability. Of these, 39 were thought to have moderate or severe stenosis, and five to have predominant regurgitation.

The normal variability of the second sound was estimated from records obtained in the pulmonary area from 63 subjects in whom there was no evidence of, or cause to suspect, pulmonary hypertension, ab-

| Table 2.—The Age and Sex Distribution of 90 Subjects in whom the Intensity of the Second Sound Was Measured |
|-------------------------------------------------|-----|-----|
| Cases  | Sex | Age |
| Normal | M | F | Mean | Range |
| 63 | 23 | 40 | 24 | 9-58 |
| Pulmonary hypertension | 27 | 14 | 13 | 34 | 11-60 |

normality of the semilunar valves, or chest disease, and was compared with the second sound of 27 subjects with pulmonary hypertension (table 2) proved by cardiac catheterization.

**RESULTS**

The mean intensity ratio (I.R.) of the first sound of normal subjects (after excluding one case with a P-R interval of less than 0.13 second) was 4.2 (standard deviation 2.6). Comparison of males and females of equivalent ages showed no significant difference while analysis by age groups suggested slightly higher values in the younger ranges (table 3). For the purpose of this study, an intensity ratio of 9.4 was considered to be the upper limit of normality (mean + 2 S.D.).

The first sound of 44 cases of well developed mitral valve disease exceeded this limit in 39 instances (fig. 1), all of which, except one, represented cases of significant stenosis. This exception was considered on clinical grounds to have predominant regurgitation. Of the five with normal intensity ratios, one presented as predominant stenosis (with calcification of the mitral valve), one was found at autopsy to have pure mitral regurgitation, one was found at surgery to have predominant regurgi-

| Table 3.—The Intensity Ratio of the First Sound in Normal Subjects Divided into Age Groups |
|----------------------------------------------------------|-----|-----|
| Subjects | Age (yrs.) | Intensity Ratio |
| Mean | S.D. |
| 10 | 9-19 | 6.3 | 3.3 |
| 30 | 20-29 | 4.2 | 2.2 |
| 11 | 30-39 | 3.3 | 2.5 |
| 16 | 40-76 | 3.4 | 3.5 |
| 67 | 9-76 | 4.2 | 2.6 |

M.V.D. = Mitral Valve Disease.
The mean intensity ratio of the pulmonary second sound in the 63 cases without pulmonary hypertension was 5.9 and the standard deviation, 4.5. This extremely wide scatter was partly due to the fact that the intensity of the second sound apparently diminished

Table 4.—The Intensity Ratio of the Second Sound in "Normal" Subjects Divided into Age Groups

<table>
<thead>
<tr>
<th>Subjects</th>
<th>Age (yrs.)</th>
<th>Intensity Ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Mean</td>
</tr>
<tr>
<td>14</td>
<td>9-19</td>
<td>8.6</td>
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<tr>
<td>36</td>
<td>20-29</td>
<td>5.9</td>
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<tr>
<td>13</td>
<td>30-58</td>
<td>2.7</td>
</tr>
<tr>
<td>63</td>
<td>9-58</td>
<td>5.9</td>
</tr>
</tbody>
</table>

Fig. 2. Comparison of the intensity ratio of the second sound in the pulmonary area in normal and pulmonary hypertensive subjects. The pulmonary hypertensive subjects in the three right hand columns are divided into age groups. The mean pulmonary artery pressure at cardiac catheterization is represented with each case. The horizontal intermittent lines represent the upper limit of normality (mean plus twice standard deviation) for each age group.

Fig. 1. The intensity ratio of the apical first sound of normal subjects, subjects with mild or doubtful mitral disease (column 1), and subjects with well developed mitral disease (column 2). Open circles represent cases with predominant mitral regurgitation.

tation, and in the remaining two, clinical evidence of combined regurgitation and stenosis was strong.

The intensity ratio of the first sound in the 34 cases of mild or doubtful mitral valve disease was above normal in 14 instances. Of these, in only nine, could an apical diastolic murmur be heard or recorded phonocardiographically, and in one a basal diastolic murmur was the only evidence of rheumatic involvement.

The correlation between high intensity ratio and clinical assessment of loudness was moderately good. Of those first sounds with an abnormally large intensity ratio, 79 per cent were reported as clinically loud, and of those with an intensity ratio within normal limits, 89 per cent were judged to be normal or reduced on auscultation.
strikingly with increasing age (table 4). No difference was apparent between the two sexes. Although the limits of normality could not be accurately assessed without a larger normal series of more representative ages, a rough approximation could be made for age groups 0 to 19 years, 20 to 29 years and 30 to 60 years (table 4). Using the values of the mean plus twice the standard deviation as the upper limit of normal, 12 of the 44 pulmonary hypertensive subjects had abnormally loud second heart sounds in the pulmonary area. It is evident from figure 2, in which the mean pulmonary artery pressure at cardiac catheterization is represented against each plot of intensity ratio, that the latter measurement is of relatively little value in determining the presence and degree of pulmonary hypertension.

**Discussion**

Anatomic differences, such as chest wall thickness, influence the intensity of the heart sounds recorded on the surface to a large extent and, as has been pointed out by Luisada and Ganna, the standardization signal should be made ideally to arise within the heart. In this manner the true intensity of heart sounds could be estimated. However, this is impracticable, and it would seem that the application of a standard signal to the left anterior axillary line as practiced by these workers is no solution. As demonstrated by their results, the facility with which sound is transmitted from the axilla to a microphone at the apex is not always an index of its transmission from the heart to the apex. Therefore, the method described above does no more than quantitate the intensity of sound received at the surface of the chest without attempting to ascertain the intensity generated in the heart.

"Logarithmic" recordings attenuate low frequency vibrations in a similar fashion to the human hearing mechanism so that heart sounds are registered as they are perceived on auscultation. An abnormally high intensity ratio may consequently be the result either of abnormally large vibrations of low intensity or of unusually high frequency components or both. The loud first sound of mitral stenosis appears to be due to both these factors and, in fact, the presence of high frequency components is perceived by the ear as a "sharp" or "ringing" or "snapping" quality.

The fact that there was not an even closer correlation between subjective impressions of loudness and the intensity as measured in this manner may be due partly to observer error. However, two other reasons for a discrepancy exist. One is the fact that subjective evaluation of the intensity of a sound is inevitably influenced by the presence and intensity of other sounds. Thus a first sound which is preceded or followed by loud murmurs will be somewhat "masked" and will be assessed as softer than it would in the absence of such a murmur. Secondly, it has been observed that subjective evaluation of intensity is influenced to some extent by the duration of a sound, a factor which does not influence the assessment of intensity ratio recorded by this method.

The results of this study serve to illustrate the reported observation that a loud first sound is an almost invariable feature of mitral stenosis sufficient to produce disturbance of function, and that its absence in mitral disease implies either considerable regurgitation or extensive calcification of the mitral valve. In the present series there was only one exception to this rule.

The results also demonstrate the less well known fact that a loud first sound may be the only abnormality in very mild or early cases. This finding occurring alone should cause the presence of mitral stenosis to be suspected in the absence of the other well known causes of a loud first sound (e.g. thyrotoxicosis, hypertension, pregnancy and rapid atrioventricular conduction).

The second sound in the pulmonary area shows great variability in normal subjects. However, it is evident that it has some diagnostic value when normal variation associated with age is taken into consideration. Although this is accomplished subjectively by the experienced auscultator, it may be difficult, and some workers have found auscultation of the second sound practically valueless in the detection of pulmonary hypertension. Objective measurement provides a cross check on subjective
impression and statistical backing for the probable significance of the results observed.

**Summary**

A simple method of objective measurement of heart sound intensity has been described and applied to a sufficient number of cases to demonstrate its practicability and potential value.

It has been demonstrated again that a loud first sound is an almost invariable accompaniment of well developed mitral stenosis, that its absence in mitral disease implies the presence of significant regurgitation or extensive calcification of the mitral valve and that it may be the only abnormal feature of early and mild cases.

The value of measurement of intensity of the second pulmonic sound as an index of pulmonary hypertension is limited by the wide range of normality. It was demonstrated, however, that with proper correction for age differences, it is of some value.

**Summario in Interlingua**

Es descrivite un simple metodo pro le mesuration del intenitate del sonos cardiac. Le metodo esseva applicate a un sufficiente numero de casos pro demonstrar su practicabilite e su valor potential.

Esseva demonstrate de novo que un forte prime sono es le quasi invariable accompanimento de ben-disveloppate stenosis mitral, que su absentia in morbo mitral indica le presentia de significative regurgitation o de extense calcification in le valvula mitral, e que illo pote esser le sol characteristica anormal de casos inceptive o leve.

Le valor de mesurationes del intenitate del seconde sono pulmonic como indice del hypertension pulmonari es limitate per le extense spectro de normalitate. Nonobstante, nos ha potite demonstrar que caute correcturas pro differentias de etate augmenta su utilitate.

**References**

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