The Tricuspid Component of the First Heart Sound in Mitral Stenosis

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In mitral stenosis the pressure in the left ventricle during the initial portion of systole does not exceed the elevated pressure in the left atrium until 0.08 to 0.12 second after the onset of the QRS complex of the electrocardiogram. This is probably the most important factor responsible for the prolongation of the interval from the QRS onset to the sound of mitral closure in mitral valve disease, although the stiffness and reduced mobility of the valve may also be contributory. Since the sound of tricuspid closure is not delayed in the absence of tricuspid disease and occurs 0.05 to 0.07 second after the QRS onset, one would expect splitting of the first sound to be a characteristic of mitral stenosis. This is not apparent by auscultation because the intense sound of mitral closure obscures the faint preceding sound of tricuspid closure. Kelly has stated that the sound of tricuspid closure contributes little to the first sound in mitral stenosis and Dock has suggested that tricuspid closure is faint because of the frequent presence of associated pulmonary hypertension which causes the tricuspid leaflets to move together just before closure, thus reducing the intensity of the sound. However, Leatham has published a phonocardiogram of a patient with mitral stenosis with a prominently split first sound and Wells has cautioned against overlooking a prominent tricuspid component in making measurements of the Q-1st sound interval.

While it may be correct to use the term "first heart sound" to describe auscultatory findings in mitral stenosis, phonocardiographers should be more precise and should refer to the tricuspid and mitral components of the first sound as discrete and separate events when they can be identified. This is particularly important when studying the interval from the onset of the QRS complex to the sound of mitral closure in mitral stenosis and other conditions. It is the purpose of this study to demonstrate that the sound of tricuspid closure can be identified in the phonocardiograms of the majority of patients with mitral valve disease and that in many of these patients, it produces a significant, audible component of the first heart sound at the apex.

Phonocardiograms were recorded in 40 patients with surgically proven mitral valve disease. Twenty patients had tight mitral stenosis without mitral insufficiency and the majority were improved by valvulotomy. Twenty patients had combined mitral stenosis and mitral insufficiency with a fixed mitral valve orifice greater than 1.0 cm², and a prominent regurgitant jet palpable in the left atrium at surgery. In 12 of these patients valvulotomy with or without an attempt to reduce the degree of mitral insufficiency was performed. No clinical improvement occurred. In 8 patients no valvulotomy was performed. Phonocardiograms were recorded in recumbency, at paper speeds of 75 mm. per second using a Sanborn Twin Beam oscillograph. Simultaneous electrocardiograms, apex impulses, and carotid pulses were recorded for timing purposes. The peak of the largest deflection of the identified components of the first sound was used in all measurements. The peak was found to occur from 0.01 to 0.02 second after the onset of the sound.

The sound of tricuspid closure was identified in 17 of 20 patients with tight mitral stenosis and 18 of 20 patients with combined mitral stenosis and insufficiency. The sound is usually of low frequency, faint and is usually inaudible. It is most consistently recorded over the tricuspid area (lateral to the

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left sternal margin in the fifth intercostal space) or between this area and the cardiac apex. It occurs from 0.05 to 0.07 second (mean 0.062 second) after the QRS onset. It occurs either simultaneously with or up to 0.02 second after the onset of precordial motion due to ventricular systole. When recorded with simultaneous tracings of right ventricular pressure, the tricuspid sound coincides with the onset of pressure rise in the right ventricle.3

An important characteristic of the tricuspid sound is its variation of intensity in relation to the length of the preceding diastole, when the ventricular rhythm is irregular due to atrial fibrillation. The leaflets of the tricuspid and mitral valves are thrust most widely apart during the rapid filling period in early diastole. If ventricular systole occurs during this period a louder sound will be produced than would occur if ventricular systole began later when the valve leaflets have floated back toward a position of partial closure. In patients with atrial fibrillation who do not have mitral valve disease, a graphic analysis of the intensity of the first heart sound and the length of diastole preceding the sound will reveal a predictable inverse relationship—the sound is loud following short diastoles and faint following long diastolic periods.4 The sound of tricuspid closure should behave in a similar manner even in the presence of mitral valve disease, providing the tricuspid valve is normal. This feature was examined. Atrial fibrillation with sufficient variation in the duration of diastole to alter the intensity of the sound of tricuspid closure was present in 6 patients. After short diastolic periods the sound of tricuspid closure is invariably accentuated. In addition, the sound of mitral closure is further delayed because of the higher left atrial

Fig. 1. Apex phonocardiograms from 2 patients with combined mitral stenosis and mitral insufficiency with a simultaneously recorded electrocardiogram (lead II). Note accentuation of the tricuspid component (√) of the first sound after short diastolic periods. In each record a third heart sound (∆) is present. Note: In the upper tracing the first sound (M) and the QRS complex of the initial cardiac cycle are not visible but the T wave and the second sound can be seen.
pressure. Under these circumstances the sound of tricuspid closure may be identified as the initial component of a split first sound after short diastolic periods. Examination of the upper tracing in figure 1 demonstrates these features. In the first cycle there is accentuation of the sound of tricuspid (x) and and mitral (M) closure when compared to the second cycle. The interval from the second sound to the sound of mitral closure is 0.31 second for the first cycle and 0.54 second for the second cycle. The interval from the onset of the QRS complex to the onset of the sound of tricuspid closure is 0.05 second in each cycle, while the sound of mitral closure begins 0.12 second (first cycle) and 0.10 second (second cycle) after the QRS onset. The lower tracing in figure 1 also demonstrates that the sound of tricuspid closure is accentuated when ventricular systole begins at the time of rapid ventricular filling (third and fourth cycles), and the sound is faint or absent when ventricular systole begins after a long diastolic period (second, fifth, and sixth cycles). The relationship between the intensity of the sound of tricuspid valve closure and the length of the preceding diastolic period is graphically illustrated in figure 2. Despite variations in intensity of the sound of tricuspid closure no consistent variation is observed in the Q to tricuspid sound interval. Loud sounds of tricuspid closure after short diastoles do not appear to occur later than faint sounds. It is possible that more precise measurements of a larger number of records may reveal that the loud sounds occur slightly later than the faint sounds in a manner similar to the sound of mitral closure in the absence of mitral valve disease.

The predictable variation of the intensity of the sound of tricuspid closure in relation to the length of the preceding diastole removes any possibility that the sound is due to any mechanism other than A-V valve tension. In most records with irregular ventricular rhythm, it is possible to observe a gradual transition from the faint, low frequency sounds preceding mitral closure to the loud, audible, accentuated sounds after very short diastoles. In addition, the sound occurs at the time of the onset of right ventricular pressure rise and is loudest at the tricuspid area. The sound is not due to impact of the heart against the chest wall, since it has been recorded directly from the surface of the exposed heart.

It appears likely that similar low frequency sounds recorded by other workers in the past are sounds of tricuspid valve closure. In two papers by Cossio’s illustrations which demonstrates a probable tricuspid sound (o) preceding the louder sound of mitral closure (1). A similar phenomenon appears in a paper by Groedel and Kisch, who interpreted the sound as a pre-systolic murmur occurring in the presence of atrial fibrillation. Battro suggested that the sound may be due to mitral reflux occurring during isometric contraction. This concept has been recently revived, curiously enough in an era in which hemodynamic measurements have shown that in mitral stenosis mitral closure does not occur until the left
ventricular pressure exceeds the left atrial pressure, which makes the concept of mitral reflux occurring before this event untenable.

Faint, low frequency sounds preceding the loudest component of the first sound at the apex have been previously ascribed\textsuperscript{12,13} to sounds of muscular contraction. It is apparent from this study that similar sounds can be produced by tricuspid valve closure in mitral valve disease and it is quite possible that all such sounds ascribed to muscular contraction are due to A-V valve closure.

It is quite apparent from the magnitude of the tricuspid sound after short diastoles in atrial fibrillation that Q to first sound measurements made to the onset of the first sound could be erroneously short since the measurement would actually be a Q to tricuspid sound interval (fig. 1). It is possible that the short Q to first sound intervals recently reported by Proctor\textsuperscript{15} were due to such an error in measurement.

The presence of the sound of tricuspid closure in both tight mitral stenosis and mitral stenosis with significant insufficiency indicates that its identification offers no diagnostic information except that there is a delay of mitral closure.

**Summary**

The sound of tricuspid valve closure has been identified in 35 of 40 phonocardiograms of patients with mitral valve disease. Identification of the sound is facilitated by the delay of closure of the mitral valve thus separating these 2 components of the first heart sound.

When the ventricular rhythm is irregular due to atrial fibrillation, the intensity of the sound is inversely proportional to the length of the preceding diastolic interval. When the sound of tricuspid closure is accentuated, it may be identified by auscultation as the initial component of a split first sound mesial to the apex. Measurement of the interval from the onset of the QRS complex of the electrocardiogram to the ‘‘first heart sound’’ should be made to the mitral component of the first sound when this can be identified on the phonocardiogram.

**Summario in Interlingua**

Le sono del clausión del valvula tricuspídel eseva identificáte in 35 ex 40 phonocardiogrammas de patientes con morbo del valvula mitral. Le identification del sono es facite plus facile per le retardo del clausión del valvula mitral, con le resultato de un separation del 2 componentes del prime sono cardiac. Quando le rhythmio ventricular es irregular in consequentia de fibrillation atrial, le intensitate del sono es inversee proportional al longor del precedente intervallo diastolic. Quando le sono del clausión tricuspid es accentuan, illo pote esser identificare per auscultation como le componentee initial de un findite prime sono, medial con respecto al apice. Le measuretion del intervallo ab le declaration del complexo QRS del electrocardiogramma usque al ‘‘prime sono cardia’’ deberea terminar se al componente mitral del prime sono si iste componente pote esser identificare in le phonocardiogramma.
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