The Opening Snap of the Tricuspid Valve: A Physical Sign of Tricuspid Stenosis

By Charles E. Kossman, M.D.

In two patients with rheumatic heart disease a short, loud, high pitched, early diastolic, snapping sound was heard at or below and to the right of the xiphisternum. Except for the different area of audibility it was similar on auscultation to the opening snap of the mitral valve. Both patients showed clinical evidence of tricuspid as well as mitral and aortic valvular disease. There was hemodynamic evidence of tricuspid disease in one, and the other displayed tricuspid stenosis at necropsy. The abnormal sound was regarded as the opening snap of the tricuspid valve.

The precise clinical localization of structural valvular defects in the heart, and an exact estimation of the dynamic alterations caused by them have assumed considerable practical importance with the advent of surgical procedures for the correction of such defects. Among the more difficult of these to diagnose clinically is tricuspid stenosis. Since surgical treatment of it can be, and has been, successfully achieved,1, 2, 3 any additional aid in its preoperative detection should be of more than academic interest.

Neither the normal mitral valve or the normal tricuspid valve make any sound on opening at the termination of isometric relaxation of the ventricles. Luisada4, 5 has recorded a single vibration in normal subjects in early diastole which he regards as originating from the opening of the atrioventricular valves, but unlike the sound arising from pathological valves it is of low frequency. Disturbing, too, is that it may occur before the peak of the V wave in the jugular phlebogram. By contrast, the structurally deformed mitral valve was believed as early as 18726 to make a sound which has come to be known as the opening snap of the mitral valve.7 It is short, of snapping quality, and occurs anywhere from 0.03 to 0.014 second after the beginning of the second heart sound.8, 9 It is usually heard best in the region of the fourth rib at the left sternal border, but this may vary to include the pulmonic and left nipple regions of the precordium. Occasionally it may be heard elsewhere but never louder than in the regions noted. It is believed to be created by the opening of stiffened, not rigid, mitral leaflets. It has come to be regarded as being as pathognomonic of mitral stenosis as the apical diastolic rumbling murmur associated with this lesion.8

So far as could be determined, the first reference* to the creation of a sound by the diseased tricuspid valve was made by Rivero-Carvallo10 who heard or recorded the "chasquido de apertura de la tricúspide" in 10 of 50 cases with tricuspid stenosis. No details were given although he pointed out that it was less intense, shorter, and sharper than the corresponding snap of the mitral valve, and could easily be confused with it if the latter were propagated toward the tricuspid region of auscultation. He published one example in which the opening snap was recorded only during a period of postinspirational apnea.

It is the purpose of this paper to describe two additional patients with this new physical

---

From the Cardiovascular Service, Lenox Hill Hospital; the Pathological Laboratories, Bellevue Hospital; and the Department of Medicine, New York University College of Medicine, New York.

Presented at the Second World Congress of Cardiology held in Washington, D. C., Sept. 16, 1954.

Aided in part by a grant of the Mona Bronfman Scheckman Foundation to Lenox Hill Hospital.

* The present author's initial observations were made late in 1952 and early in 1953 without knowledge of Rivero-Carvallo's paper. The latter was called to his attention by Dr. A. A. Luisada at the recent Second World Congress of Cardiology. He takes this opportunity to thank Dr. Luisada for pointing out the unintentional omission of this reference in the paper as presented.

Circulation, Volume XI, March, 1955
sign, in both of whom the existence of tricuspid disease was clinically obvious, and in one of whom the lesion of tricuspid stenosis was proven at necropsy.

Case 1. E. S., a 34-year-old, single, white man, developed acute rheumatic polyarthritis after pneumonia at the age of 11 years. While still being observed in another hospital during the acute attack, evidence of mitral stenosis and mitral insufficiency developed.

He first came under our observation at the age of 15 years when the diagnosis of mitral valvular deformity was confirmed. He was seen regularly at the Cardiac Clinic. At 18 the basal diastolic murmur of aortic insufficiency appeared. In 1941, at the age of 20, the first symptoms of diminished cardiac reserve occurred. Active rheumatic carditis was suspected, and he was admitted to the hospital for the first time. However, the suspicion was not confirmed by the laboratory data accumulated.

At 21 years of age the first bout of atrial fibrillation occurred which was treated with quinidine. Between the ages of 21 and 26 years there were repeated attacks of palpitation documented on three additional hospitalizations as atrial fibrillation, and on several clinic visits as either this arrhythmia or normal sinus rhythm with atrial and ventricular premature systoles. Control was restored by manipulation of the dosage of quinidine, on one occasion preceded by temporary digitalization.

In the summer of 1946, when the patient was 26 years old, he was admitted for the fifth time to the hospital for his first bout of congestive heart failure. He was critically ill and the course was complicated by development of an atonic bladder and a small cerebral embolus with partial and transient paralysis of the right arm and the right side of the face. Rather large doses of digitalis were required to bring the ventricular rate under control. The fibrillation of the atria became permanent. At the end of five months he was well enough to return to the clinic on digitalis.

Except for the development of a renal infarct in October 1947, the patient was fairly well for the next four years, sometimes requiring as little digitalis as 0.05 Gm. five days per week.

In 1950 he began to have four to seven bowel movements a day. For this symptom he was studied on his seventh, eighth, and ninth admissions to the hospital between April 1951 and June 1952. Gastrointestinal reviews revealed only an enlarged liver which on biopsy revealed increased fibrosis suggestive of cirrhosis, and an elevated serum bilirubin (3.3 mg. per 100 cc.). Soon thereafter pulsation of the liver was noted, and tricuspid insufficiency was added to the diagnosis.

Late in 1952, when the patient was 32 years old, an early diastolic snapping sound was heard in the fourth right intercostal space 3 cm. lateral to the right sternal border. This tenth admission to the hospital was for palpitation and dyspnea as were the next three admissions, the last in February 1954. In each instance the patient was found to have a rapid ventricular rate, was quite ill, and was given additional digitalis either as ouabain or Digoxin intravenously despite the fact that he had been faithful in taking his "maintenance" dose. In each instance the ventricular rate was reduced quite promptly to acceptable levels at which palpitation disappeared. The fourteenth and fifteenth admissions were for special studies.

The physical findings varied over the years. At the time of most of the stethographic studies in 1953 and 1954 the cardiac examination revealed the following: There were three distinct impulses visible and palpable on the anterior thorax. The easiest to see was a systolic retraction in the right fifth intercostal space just medial to the nipple. The apex beat in the fifth intercostal space just beyond the left midclavicular line was also a retraction. Below this, the lower and outer point in the sixth intercostal space near the left anterior axillary line was palpable as a retraction but could not be seen. The third impulse was of the xiphoid itself which was thrust forward with systole in a visible, forceful manner. A long diastolic thrill was felt at the apex with the patient in the left lateral supine position. The first sound at the apex was poor; at the base the pulmonic second sound was louder than the aortic second but neither was unusually loud. A moderately loud blowing, transmitted systolic murmur and a long, holo-diastolic, decrescendo, rumbling diastolic murmur were audible at the apex. A soft systolic blow and a soft diastolic blow were heard along the lower left sternal border but no sound simulating the opening snap of the mitral valve could be heard in this area. In the aortic region there was a soft systolic blow also. At the xiphoid there was a loud, harsh, rasping systolic murmur transmitted upward and to the right and followed by an early diastolic rumble of different quality than the one at the apex. Upward and farther to the right near the fifth rib in the right parasternal line this murmur was blowing rather than rumbling in character. On the lower right anterior thorax in a roughly triangular area with its apex at the fifth rib in the right anterior axillary line and its base extending along the costal margin from the fifth rib in the midsternal line to the eighth rib in the anterior axillary line (figure 1, E. S.) there was a loud, snapping sound occurring immediately after the second sound and ascribed to the opening of a deformed tricuspid valve. The area of audibility indicated was that found in 1954. A year earlier it was farther to the left and included the xiphoid but did not extend beyond the left sternal border. The ventricular rate was approximately 50 per minute, and premature systoles with coupling were common. The blood pressure averaged 120/80,
and for a good many years had varied between 90 and 130 systolic, and 60 to 90 diastolic. There were none of the usual peripheral phenomena of aortic insufficiency. However, both the large liver and the dilated jugular veins pulsed with systole. Although the spleen was enlarged it could not be felt to pulsate.

Roentgenograms. The change in the x-ray appearance of the heart over the years is demonstrated in figure 2. Fluoroscopy on many occasions disclosed evidence of marked enlargement of both atria, and of the right ventricle, less of the left ventricle. The esophagus was displaced posteriorly and to the right. The hilar shadows were prominent.

Electrocardiograms were recorded frequently for 19 years. The changes which occurred may be seen in a record taken in 1940 and another in 1954 (figure 3). During this interval, in addition to the changes in rhythm, there was a gradual widening of the QRS interval to 0.11 second, and development of a form and axis of QRS deviated to the right ascribed to incomplete block of the right bundle-branch. There was on occasion a rapid ventricular rate at which time the QRS interval would usually be longer. In the record of 1940 the left deviation of the electrical axis of the P wave is to be noted.}

Stethograms were recorded on several occasions in 1953 and 1954. An example of one made on May 18, 1953 is shown in figure 4.* At this time, the opening snap could be heard farther to the left.

* The stethograph of the Cambridge Instrument Co. was used to make this and the record in figure 7. Its characteristics were not tested but its galvanometric response to sound waves is reported by the company as being approximately stethographic. The frequency response curve falls below 50 and above 600 cycles per second, and shows a peak at 400 cycles per second. Overall recording with amplifier and microphone in circuit causes some modifications of the curve described.
near the xiphoid than was the case in 1954 (figure 1). The simultaneous mechanical record is a right jugular phlebogram with the bifid appearance characteristic of tricuspid insufficiency when the atria are fibrillating. The records clearly show a second sound which begins 0.40 second after the beginning of the QRS interval (Q-2), a similar value being found in the basal record (0.41 second). On the other hand, the interval from the beginning of QRS to the beginning of the opening snap (Q-O.S.) is 0.53 second. Other phonocardiograms were made from other areas both by stethographic and logarithmic recording. One of these was calibrated, and revealed an intensity of the snap of approximately 86 decibels. A feature not revealed by auscultation was a split second sound in the pulmonic area. An opening snap of the mitral valve was not recorded.

The interval between the beginning of the second sound and the beginning of the opening tricuspid snap varied from 0.097 second to 0.129 second with a mean of 0.111 second (10 observations). It showed a general tendency to elongate when the preceding cycle was longer, but the relationship in this patient was not definite. It was a little better when the interval between the highest peaks of the two sounds were used as reference points for the interval. Because of frequent premature systoles with coupling it was difficult to get many cycles uncomplicated by the arrhythmia.

Cardiac catheterization* was attempted on April 7, 1954 and again nine days later. In each instance the voluminous right atrium was entered easily but with no amount of manipulation within the safe limits of the necessary fluoroscopic radiation was it possible to get the catheter's tip through the presumed button-hole tricuspid orifice. Success in this regard was undoubtedly thwarted further by a vigorous systolic jet, which during one set of observations carried the regurgitant pressure in the right atrium to 40 mm. Hg, with a diastolic pressure of 21 mm. Hg and a mean of 25 mm. Hg.

Atrial pressure pulses on another occasion were made simultaneously with an electrocardiographic lead (aV2) and with a stethogram. The latter was recorded linearly after passing the sound through a filter with a band pass of 400 to 800 cycles per second. The atrial pressure recorded by strain-gauge transduction, electronic amplification, and cathode-ray oscilloscopic visualization‡ showed a bifid systolic curve with a rapid fall in early diastole. A v wave could not be recognized but of possible significance in interpretation was that the opening

---

* The author had the indispensable help of Drs. Alfred Kaltman, Rejâne M. Harvey, and George Thomson in doing these studies.

† Filter on the Cambridge Instrument Company’s Tape Recorder.

‡ Manufactured by Electronics in Medicine, New York.

---

Fig. 3. Case 1 (E. S.). Bipolar leads I, II and III (top line) made on Oct. 23, 1940 when the patient was taking digitalis. The P waves are broad and the axis of P deviated well to the left, but the record is not otherwise beyond normal limits. The lower two lines show electrocardiograms made on April 8, 1954 when the patient was taking digitalis. In each frame two electrocardiograms are recorded simultaneously. The symbols have the usual meaning.27

In the interval between records the electrical axis of QRS has deviated to the right, the QRS interval has increased to 0.11 second, and the form of the ventricular deflections in leads I, V4R, V3R, and V1 suggest the presence of some block in the right bundle branch, and hypertrophy of the right ventricle. Records from the left side of the precordium show relatively high R waves. A ventricular premature systole occurred while recording leads V2 and V3.

Leads V1 through V6 (last three frames, lowest line) were made with the galvanometer at half normal gain (1 mv = 0.5 cm). Time lines, 0.04 second.

snap occurred approximately 0.03 second after the pressure curve began rapid descent (dashed vertical lines in fig. 5) and 0.05 second after initial slower descent. Although much distortion of the pressure pulse probably exists, recorded as it was through a cardiac catheter,13 nevertheless the abnormal sound occurred at an appreciable interval after a rapid fall in pressure began, and actually at a time when the steepest part of the curve had passed (see Discussion).

The similarity of the atrial pressure pulse to the jugular phlebogram (figure 4) and the hepatogram (figure 6) recorded by means of a piezo-electric microphone with linear displacement placed on the abdomen over the liver is to be noted.
Fig. 4. Case 1 (E. S.). Stethograms made with the microphone in the pulmonic area simultaneous with the right jugular phlebogram and lead II (upper record), and with the microphone just to the right of the xiphisternum simultaneous with the right jugular phlebogram and lead III (lower record) on May 18, 1953. S1, first sound; S2, second sound; SM, systolic murmur; DM, diastolic murmur; O.S., opening snap. Q-2 and Q-OS. are the intervals between the beginning of QRS and the beginning of the second sound (Q-2) and the beginning of the opening snap (Q-O.S.) respectively. The stethograms were made with a stethoscopic bell 5 cm. in diameter and equipment of the Cambridge Instrument Company. Volume controls were at six and eight respectively for the two records.

Limitation of the snap to the area of the xiphoid, and the bifid nature of the jugular phlebogram are to be noted.

Diagnosis. The patient was regarded as having chronically or recurrently active rheumatic heart disease with enlarged heart, mitral stenosis, mitral insufficiency, tricuspid stenosis, tricuspid insufficiency, aortic insufficiency, intracardiac thrombi, atrial fibrillation with frequent ventricular premature systoles, and congestive heart failure. Cerebral and renal infarction secondary to emboli had occurred in the past, and there was cardiac cirrhosis of the liver.

Case 2. E. C., a 45 year old white married housewife, did not know of rheumatic fever but knew of rheumatic heart disease since the age of 12 years. She had an appendectomy at 18, a successful caesarean section for her only pregnancy at 35, and a partial hysterectomy at 42.

Although she was under the care of a physician for the last 10 years of her life, during which time her physical activity was limited, nevertheless she had no symptoms other than mild dyspnea on effort until the last year. In the spring of 1952, at the age of 44, she complained of increasing dyspnea, ascribable in part to atrial fibrillation first noted then. Digitalis was begun and soon thereafter mercurial diuretics as well. Because her personality was characterized by extreme anxiety and emotional instability, hyperthyroidism as a complicating
etiology was considered as a possibility. Several studies of the basal metabolic rate and determination of the radioactive iodine uptake failed to substantiate the suspicion.

She was admitted to Lenox Hill Hospital on May 18, 1953 for a period of two weeks; again on July 1, 1953 for a period of five weeks; transferred to the Psychiatric Hospital at Bellevue Hospital on August 7, 1953; and died there on August 11, 1953. These hospitalizations were really for a continuing and rapidly progressive illness.

When first admitted to the hospital the ventricular rate was 144, there was hepatosplenomegaly, the venous pressure was 250 mm. water, and the Decholin and ether circulation times were 50 seconds and 18 seconds, respectively. The venous pressure fell to 160 mm. water in five days after 1.5 mg of Gitaligen daily. She lost 5 pounds in weight with the aid of mercurials, after which she was discharged to her home with instructions to continue digitalis.

She returned one month later with pain in the right lower chest and right shoulder, hemoptysis, fever, nausea, extreme apprehension and further loss of weight. Examination revealed effusion in the right base and presumptive roentgenographic evidence of infarction in the right lower lobe. Edema occurred when an attempt was made to mobilize the patient. She eventually became hysterical and noisy to a degree which required transfer to a psychiatric hospital. There her condition remained essentially unchanged until a few hours before death when a shock-like state developed.

The physical findings differed only slightly from time to time. The patient was always loquacious, dyspneic, apprehensive, and showed variable degrees of hepatosplenomegaly, venous congestion, and in the last few weeks of life, pendent edema and right pleural effusion.

Except for the rate, the findings in the heart were quite constant. The point of maximum impulse was initially in the fifth intercostal space in the midclavicular line, later in the sixth intercostal space beyond this line. There was no thrill. The first sound at the apex was valvular; at the base, largely obscured by murmurs. At the apex there was a loud, long blowing systolic murmur. There was also a short, loud, rumbling diastolic murmur in the same area. At the base, but loudest in the aortic area, was a long, harsh systolic murmur. It was followed by a short, soft diastolic blow transmitted downward and to the left toward the apex. At the xiphisternum and in a circular area several centimeters in diameter below it (figure 1, E. C.) there was a high pitched, short, loud, diastolic snapping sound. This occurred just after the second sound which in this area was of greatly diminished intensity. A systolic murmur but no distinctive diastolic murmur could be heard in this area although one was recorded (figure 7). The ventricular rate varied from 144 to 80 at various times, and was always irregular. The blood pressure was usually in the range of 130 mm. Hg systolic and 85 diastolic.

Roentgenograms made on the initial admission (figure 8) disclosed a greatly enlarged heart involv-
Fig. 7. Case 2 (E. C.). Stethograms made with a microphone to the right of the xiphoid on May 26, 1953 (upper record), and at the fourth rib at the left sternal border on July 14, 1953 (lower record). The records simultaneously made with the former are the right carotid arteriogram (Rt. Car) and lead II; with the latter a somewhat changed lead II. Instrumentation, symbols, and time lines as for figure 4.

The presence of the snap (O.S.) at the xiphoid, its absence at the left sternal border, but the presence of a later, low pitched, third sound (S₃) in the latter area are to be noted.

Electrocardiograms repeated at weekly intervals did not differ greatly one from the other (figure 9). All displayed atrial fibrillation, left deviation of the electrical axis of QRS, and depression of the S-T junction with inversion of the T waves in leads I, aV₅, and V₆. Intrinsicoïd (RS) deflections in leads from either side of the precordium were not delayed in onset. Some later records disclosed ventricular premature systoles, and on one occasion coupling ascribed to excessive digitalis.

Stethograms were recorded on several occasions, two of these being shown in figures 7 and 10. In figure 7 the upper record was made simultaneously with the right carotid sphygmogram and electrocardiographic lead II on May 26, 1953 when the microphone with a shallow bell 5 cm. in diameter was placed to the right of the xiphoid. In addition to an opening snap in this region (O.S.), the stethogram shows systolic (SM) and early diastolic (DM) murmurs. The second heart sound (S₂) shows poor definition. It was difficult to hear, and one observer apparently mistook the opening snap for the second sound in the area of the xiphisternum. The carotid sphygmogram, with a latency of 0.06 to 0.08 second due to inertia of the mechanical recording system, identified the opening snap as occurring well after the incisura.

The lower stethogram in figure 7 was made sometime later, on July 14, 1953, simultaneously with lead II, with the microphone at the fourth rib and left sternal border, the usual site of greatest audibility of the opening snap of the mitral valve. None could be heard in the area, and none was recorded. However, the record does show a low frequency (approximately 30 cycles per second) vibration (S₃) of approximately 1.5 cycles in length occurring 0.16 to 0.17 second after the beginning
FIG. 8. Case (E. C.). Posteroanterior (P-A) and right anterior oblique (RAO) teleroentgenograms made on June 19, 1953. The barium filled esophagus of the latter (barely visible) is displaced backward. The right atrial curvature is not as prominent as in case 1 (fig. 2), and the left ventricle more so.

of the second sound. This could be heard close to the lower sternal. It was neither heard nor recorded elsewhere. It is believed to be a physical sign to which infrequent reference is made,16 namely gallop rhythm limited to the right ventricle.

On the same day that this latter stethogram was made, three additional ones were recorded from the xiphoid with another instrument* (figure 10) both by stethographic and logarithmic recording at the same gain (upper records) and by stethographic recording at higher gain (lower record). Logarithmic recording did not alter the high frequency snap (O.S.) very much, while high gain recording greatly exaggerated its single, high frequency component.

The interval between the beginning of the second sound and the beginning of the opening snap was correlated with the length of the preceding cardiac cycle on several records. On one of these there was a direct relationship between the two (figure 11) as in the case of the opening snap of the mitral valve.9, 18 Breath holding, required when recording heart sounds, apparently had no bearing on this relationship (figure 11). In another record, made at the same sitting but after the patient had been "resting" for a time, the average interval was fairly constant (0.084 to 0.097, mean of 0.092 second), and did not vary in any recognizable manner with the preceding cycle length. The word "resting" is placed in quotes because during the entire time of recording the patient was apprehensive and complaining. The fact that the second sound-opening snap interval became shorter (mean of 0.092 second compared with mean of 0.100 of values plotted in figure 11) and relatively fixed may mean that venous pressure toward the end of the recording was rising

* Sanborn Instrument Company's Twin Beam Cardiette, Model 62.
and the gradient of pressure across the tricuspid valve was very high regardless of length of preceding cycle. On all of the records of this patient the 2-O.S. interval had a mean value of 0.097 second, and a range of 0.068 second to 0.120 second (25 cycles measured).

**Diagnosis.** On the basis of the clinical and graphic data, the patient was regarded as having rheumatic heart disease with enlarged heart, mitral stenosis, mitral insufficiency, tricuspid stenosis, tricuspid insufficiency, aortic insufficiency, intracardiac thrombi, atrial fibrillation with ventricular premature systoles, and congestive heart failure. Pulmonary infarction was a complication. Although laboratory evidence was lacking, the fairly rapid course suggested possible activity of the rheumatic process in the myocardium.

**Necropsy.** *Heart:*—The enlarged heart weighed 550 Gm. All chambers except the right ventricle were dilated. The dilated right auricle (appendage) disclosed some punctate hemorrhages and fibrinous exudate externally, a recent organizing thrombus, 3 cm. by 4 cm., internally. The right atrium showed a fine Chiari's network near the ostium of the coronary sinus. Both ventricles were hypertrophied (thickness of right 0.5 cm., of left 1.5 cm.). The patent coronary arteries showed only minimal lipid deposits.

The tricuspid valve was moderately stenotic, admitting only the tip of the index finger. The leaf-

---

*Necropsy was performed two hours post mortem by Drs. Irene Gleason, Resident Pathologist, and Cyril Solomon, Assistant Pathologist, Bellevue Hospital, New York.*
Fig. 11. Case 2 (E. C.). A graph to show the direct, linear relationship between the length of the preceding cycle (R-R interval) and the interval between the beginning of the second heart sound and the opening snap of the tricuspid valve (2-O.S. interval) in one record made on May 23, 1933. In order to demonstrate that holding the breath, as is required when making stethograms, had little effect on the relationship, the values obtained when the breath had been held for less than 7.6 seconds and the values obtained when the breath had been held more than 13.0 seconds are shown in open circles and squares, respectively. Intermediate times are shown as solid circles. The regression line was calculated \( y = 12.13, x - 0.389 \). The overall mean for all plotted values of 2-O.S. interval is 0.100 second.

Leaves were scarred, contracted, fused, and the free margins were rolled. The chordae tendineae were also moderately thickened and shortened. The circumference of the closing margin was 6.5 cm. The pulmonic valve was normal. The mitral valve displayed a "fish-mouth" deformity with a stenotic orifice which just admitted the tip of an index finger. The leaflets were fused, rigid, thickened and calcified, and the line of closure was retracted upward. The attached chordae were moderately thickened and slightly shortened. The circumference of the orifice at the line of closure was 6.5 cm.

The aortic valve was stenotic to a degree which admitted a probe of only 1 cm. in diameter. The leaflets were thick and calcified and there were calcific excrescences not only on the ventricular aspects but also on the walls of the sinuses of Valsalva. Two cm. below one cusp there was a longitudinal raised area of the endocardium on the interventricular septum which measured 1 cm. by 0.3 cm.

Blood Vessels:—There was mild atherosclerosis of the aorta, most marked in the arch. The large pulmonary arteries displayed slight to moderate amounts of intimal atheroma and the smaller arteries disclosed distinct intimal thickening. The superior vena cava, the right and left innominate veins, the right and left subclavian veins, and the internal jugular veins were occluded by a fresh thrombus. Parts of this in the superior vena cava were organizing.

Other Findings:—In addition to bilateral pleural effusion (right 1.0 liter, left 0.3 liter), the somewhat smaller than normal though congested lungs revealed an organizing infarct in the right lower lobe of 5.0 cm. in diameter. Small branches of the pulmonary artery leading to the area were occluded by organizing thrombi. Microscopic sections showed irregular thickening and fibrosis of the alveolar walls in general. The liver was normal in size (1350 Gm.) and revealed only congestion. The right kidney showed an old infarct at its inferior pole which measured 1 cm. in diameter. The spleen and pancreas were congested. There was no microscopic evidence of active rheumatic infection in the heart.

Discussion

The data presented in these two patients favor the belief that the diseased tricuspid valve, and especially the stenotic valve, is capable of making a sound on opening. This sound has been designated as the opening snap of the tricuspid valve.10

It is heard best in the "tricuspid area" but allowances must be made for displacements of this auscultatory area principally to the right and downward produced by hypertrophy and dilatation of the right side chambers of the heart secondary in part to the usually associated mitral disease. Its duration is brief (approximately 0.02 second), and its intensity greater than that of the second sound occurring in the same area. In one patient (case 1) the intensity of the highest vibration had a value of approximately 86 decibels above the average threshold of hearing. No analysis of frequencies was made but some appeared to be in excess of 400 cycles per second, and the sound was distinctly high pitched and snapping. On the average (in both patients) it occurred 0.10 second after the beginning of the second sound. This interval was longer the longer the preceding cycle length but the correlation, with one exception (figure 11), was not definite. In the one case in which the measurement was made, the sound occurred not at the beginning but 0.05 second after the beginning of the rapid fall in atrial pressure as...
measured on the right atrial pressure pulse. Much speculation on the reason can be made, including the possibility that the sound in this one case (E. S.) was not due to opening of the tricuspid valve. Its resemblance in timing and other characteristics, however, to the similar sound encountered in the other case (E. C.) with proven tricuspid stenosis makes some other explanation likely. Clearly an insufficient and stenotic tricuspid valve will remain open during isometric relaxation of the right ventricle. It is likely that atrial pressure, under these circumstances, will fall at the beginning of isometric ventricular relaxation. Flow, on the other hand, ordinarily signalled in venous sphygmosgrams by the peak of the v wave, probably cannot become maximal until isometric relaxation of the ventricles terminates. Only simultaneous records of pressures in the right atrium and right ventricle could give irrefutable evidence on the significance of a gradient in pressure between the two chambers in the creation of the tricuspid snap.

The right ventricular pressure was not recorded in case 1 and therefore a gradient in pressure between the right atrial mean pressure or Z-point pressure and the end diastolic right ventricular pressure as shown by Ferrer and her associates in tricuspid stenosis could not be demonstrated. However, the mean pressure in the right atrium was unusually high (19 mm. Hg) and the possibility that the gradient under consideration existed is good. The fact that clinically and hemodynamically

---

**Fig. 12.** Case 2 (E. C.). Two views of the stenotic tricuspid and mitral valves. *Above left,* the tricuspid valve viewed from the right atrium; *below left,* the incised tricuspid valve viewed from the right, with the right ventricle below, and the right atrium above. *Above right,* the mitral valve viewed from the left atrium; and *below right,* the incised valve viewed from the left with the left atrium above and the left ventricle below.

The orifice in each case admitted the tip of the index finger, but the mitral valve was rigid and calcified and incapable of independent movement. The stenotic tricuspid leaflets, though thickened, were pliable and movable. The dark material at the apex of the right ventricle and in the right atrium is postmortem clot (The photographs were made by Dr. Cyril Solomon).
the patient displayed dominantly insufficiency rather than stenosis of the valve does not militate against the diagnosis of the latter defect.

The opening snap of the tricuspid valve must be differentiated from other sounds in early diastole. Usually no difficulty will be encountered by virtue of the relatively peripheral precordial area in which this sign is heard. In some patients with mitral stenosis, the opening snap of the mitral valve can be heard well to the right of the sternum and as low as the xiphoid cartilage but the intensity in these regions is less than at the lower left sternal border. Other sounds, usually of low pitch, have been heard in a few patients at or near the right lower border of the sternum and xiphisternum which may give difficulty but the mechanism of their creation was not apparent. The observations accentuate the need for further auscultatory scrutiny of the region in patients with heart disease.

The data are too meager and the dynamics too complicated by associated valvular and myocardial lesions to attempt an assessment of the degree of stenosis of the tricuspid valve from the phonocardiogram such as has been done by Wells in cases of mitral stenosis.19

SUMMARY
A short, loud, snapping sound early in diastole was heard and recorded near the xiphisternum in two patients with rheumatic heart disease. The sound was similar in almost all respects to the opening snap of the mitral valve except for its area of audibility. In one of the patients the presence of tricuspid stenosis was demonstrated at necropsy; in the other there was convincing clinical and hemodynamic evidence, at least, of organic tricuspid valvular disease. This physical sign, the opening snap of the tricuspid valve, has practical diagnostic value.

SUMMARIO IN INTERLINGUA
In duo patientes con morbo rheumatic del corde, un clac esseva audite al xiphistern o infra e al dextere de illo. Iste clac esseva breve, forte, e alte e occurreva al comienzamento del diastole. A parte le differente area de su audibilitate, le clac resimilava in le auscultation le clac de apertura del valvula mitral.

REFERENCES
1. Cossio, P.: Personal communication that the first valvulotomy for isolated tricuspid stenosis was done in Santiago, Chile, 1952.
OPENING SNAP OF TRICUSPID VALVE


The Opening Snap of the Tricuspid Valve: A Physical Sign of Tricuspid Stenosis
CHARLES E. KOSSMANN

Circulation. 1955;11:378-390
doi: 10.1161/01.CIR.11.3.378
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1955 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on
the World Wide Web at:
http://circ.ahajournals.org/content/11/3/378

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally
published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not
the Editorial Office. Once the online version of the published article for which permission is being
requested is located, click Request Permissions in the middle column of the Web page under Services.
Further information about this process is available in the Permissions and Rights Question and Answer
document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/