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Systolic Honk in Heart Failure: Its Origin and Mechanism of Production

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Summary: Systolic honks have so far been synonymous with prolapse of the mitral valve leaflet. Evidence of tricuspid valve involvement in two cases is presented. These patients developed systolic honk during the deterioration of their congestive heart failure. Simultaneous recordings of echocardiograms and phonocardiograms showed fluttering of the tricuspid valve, coinciding with the systolic honk. This fluttering was not seen when honk was absent. The mechanism of production of this honk is discussed.

Keywords: echocardiography, heart failure, phonocardiography, systolic honk, tricuspid valve

Introduction

The systolic honk or whoop, an unusual auscultatory phenomenon, has, in most cases, been ascribed to mitral valve prolapse (1, 4) with or without mitral regurgitation, and found in almost every age group from childhood onward. The involvement of the tricuspid valve in the production of these systolic honks is not well recognized in the literature (5, 14). The purpose of this communication is to present the evidence for tricuspid valve origin of the systolic honk and discuss the mechanism of its production.

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Methods

Simultaneous echocardiogram and phonocardiogram were recorded using an Irex multichannel recorder on a photographic paper at a paper speed of 50–75 mm/s. Echocardiograms were obtained according to the techniques already described (3) using a Unirad 100 series ultrasonoscope with a 2.25 MHz transducer of 0.5 inch in diameter. Output of the ultrasonoscope was displayed on the oscilloscope of the multichannel recorder via an interface in conjunction with an electrocardiogram and phonocardiogram. Phonocardiograms were obtained with a frequency range of 200–500 cps.

Case I

The first case is that of a 24-year-old female, who had been followed at D. C. General Hospital since 1973 with the diagnosis of congestive cardiomyopathy, and had multiple admissions for heart failure, for which she had been treated with digoxin, diuretics and anticoagulants. Echocardiograms and phonocardiograms had been taken on many occasions. Cardiac catheterization done in 1975 revealed a mean right atrial pressure of 20 mm Hg, with a = 15 and v = 22 mm Hg. Right ventricular, pulmonary artery and pulmonary capillary wedge pressures were 58/20, 60/23 and 24 mm Hg respectively. Cardiac index was 1.5 l/min/m^2. Right and left ventricular angiograms showed tricuspid and mitral regurgitation with 30% ejection fraction.

She was readmitted in March 1977 for decompensated heart failure. Physical examination revealed an obese female in moderate distress, with a pulse rate of 120/min, blood pressure of 140/90 mm Hg, with distended neck veins up to the angle of the mandible, at a 45° angle, with a predominant V wave. A diffuse apical impulse was palpable in the 6th intercostal space along the anterior axillary line. The first and second heart sounds were normal. Atrial and ventricular gallops were heard at the apex and

along the left sternal border. A Grade II/VI mitral regurgitant murmur was present at the apex, and a tricuspid regurgitant murmur was appreciated along the left sternal border. An intermittent Grade IV/VI systolic honk was heard along the left sternal border, which increased in intensity on expiration, squatting and leg raising, and decreased or disappeared on inspiration and standing (Table I). On chest roentgenogram, massive cardiomegaly was noted. Electrocardiogram (ECG) showed left atrial elargement and nonspecific ST-T changes. Phonocardiogram confirmed the auscultatory findings (Fig. 1).

Echocardiogram of the tricuspid valve demonstrated fine systolic fluttering, which coincided with the timing of the systolic honk (Fig. 2). The systolic fluttering was coarse during expiration and disappeared during inspiration, following the behavior of the honk. Other echocardiographic findings are shown in Table II. No pericardial effusion was noted.

Case II

A 50-year-old male with a history of hypertension, diabetes mellitus and chronic alcohol abuse was admitted to D. C. General Hospital with congestive heart failure and paroxysmal atrial tachycardia. Subsequently, his arrhythmia was electrically cardioverted to sinus rhythm and he was discharged on methyldopa, furosemide, quinidine and digoxin.

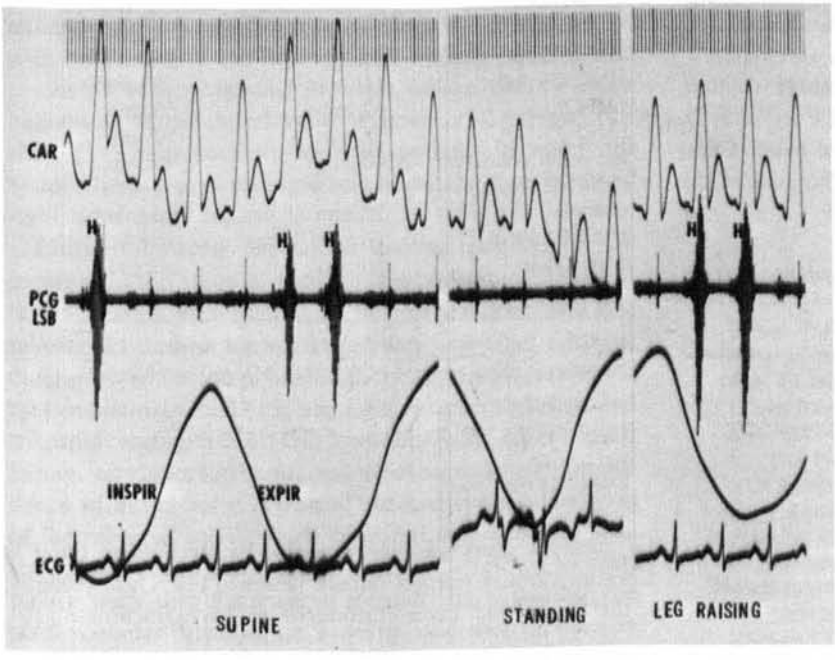

Fig. 1 Phonocardiogram (PCG) from case I showing systolic honk (H), appearing on expiration (Expir.), disappears on inspiration and standing and gets louder on leg raising. CAR = Carotid Pulse, LSB = Left Sternal Border, ECG = Electrocardiogram.

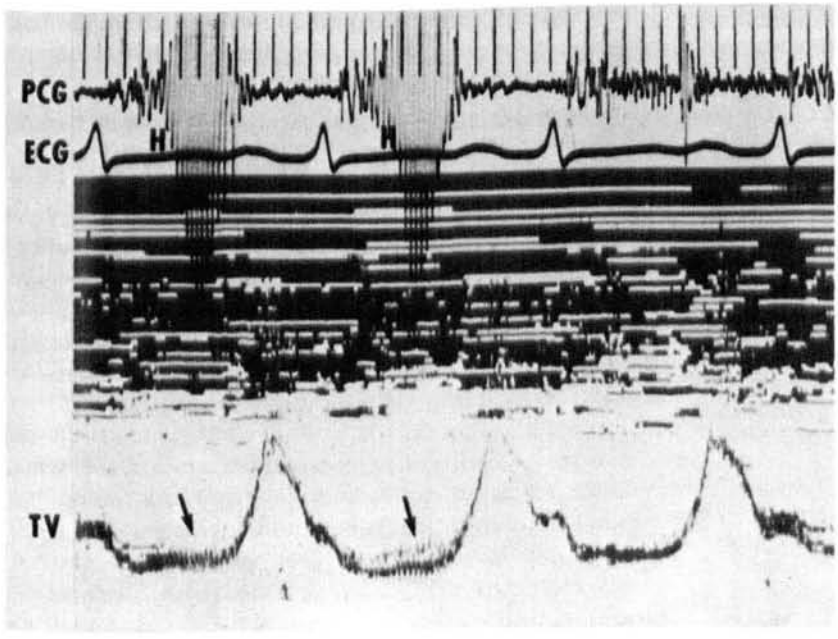

Fig. 2 Simultaneous phonocardiogram (PCG) and echocardiogram of the tricuspid valve (TV) from case I showing systolic honking (H), coincident with the systolic fluttering (arrow) in the first two beats, while honk and fluttering are absent in the third beat.

Nine months later, he was readmitted for decompensated heart failure. Physical examination revealed a well developed black male with a temperature of 98.6° F, pulse 110/min and blood pressure of 150/90 mm Hg. The upper level of jugular venous pulse was noted at 13 cm above the manubriosternal angle. The apex beat was in the 6th left intercostal space in the anterior axillary line. On auscultation, the first heart sound was of normal intensity, the second heart sound was widely split, a ventricular (S3) gallop was audible at the apex and along the left sternal border, and a mitral regurgitant murmur was present. An intermittent systolic honk was audible along the left sternal border when the patient was in severe heart failure. After the treatment, with improvement of cardiac decompensation, the honk disappeared, and a murmur of tricuspid regurgitation appeared. Response of this systolic honk to different maneuvers is shown in Table I.

The electrocardiogram showed normal sinus rhythm, right bundle branch block, abnormal Q wave in leads II, III and AVF, and occasional premature atrial beats. Chest x-ray showed gross cardiomegaly. Echocardiogram at the time the systolic honk was recorded demonstrated fine systolic fluttering of the tricuspid valve, which coincided with honk. The fluttering was absent in beats without the honk (Fig. 3). The mitral valve did not show evidence of prolapse or fluttering. Other echocardiographic features are shown in Table II. Treatment for his congestive failure resulted in a 41 pound weight loss with great improvement. Subsequently, the patient was readmitted twice with heart failure. The systolic honk again followed the pattern described above on both occasions.

Table I Behaviour of systolic honk with different maneuvers

maneuver	case I	case II
inspiration	disappeared	variable
standing	disappeared	decreased
squatting	increased	increased
leg raising	increased	increased
timing	mid-systolic	mid-systolic
amyl nitrite	no change	not done
after treatment of heart failure	disappeared	disappeared
location	left sternal border	left sternal border

Table II Echocardiographic findings in two patients with systolic honk

	case I	case II
MV prolapse pattern	absent	absent
MV E-F slope	140 mm/s	150 mm/s
LVED	62 mm	60 mm
LVES	55 mm	50 mm
left atrial diameter	40 mm	45 mm
right ventricular diameter	25 mm	20 mm
tricuspid valve	systolic flutter	systolic flutter
aortic valve	normal	normal
pulmonary valve	normal	not obtained

LVED = Left Ventricular End – Diastolic Diameter, LVES = Left Ventricular End – Systolic Diameter, MV = Mitral Valve

Discussion

Systolic honk was first reported by *Sir William Osler* in an apparently normal female patient (11). Until recently, the origin had been considered to be extracardiac (10).

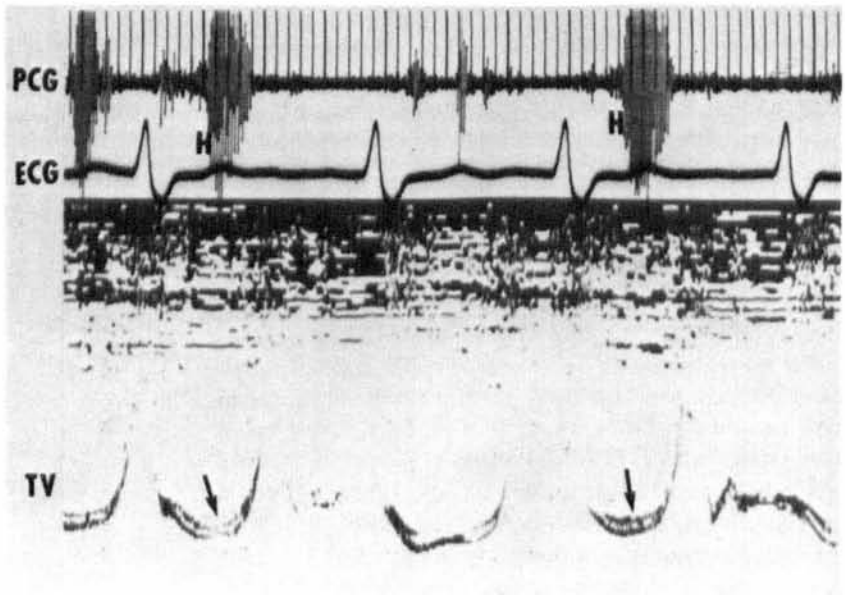

Fig. 3 Echo-phonocardiogram from case II showing that systolic honk (H) coincides with the systolic fluttering (arrow) of the tricuspid valve (TV).

Levine and *Harvey* (9) described two types of systolic murmurs, one a musical murmur, associated with rheumatic mitral valve disease, and the other a systolic whoop, which was present in what is now known as the prolapsed mitral valve syndrome. *Victor Behar* (1) divided the patients with systolic honks into two groups and showed that the 5 patients without symptoms of heart failure had ballooning of the mitral valve. On the other hand, only one of the three patients with symptoms of heart failure had ballooning of the mitral valve. *Rackley* (12) suggested that some of the honks could be intracardiac in origin and *Leon* et al (7) demonstrated by means of intracardiac phonocardiography, that the systolic whoop originated from the mitral valve. *Rizzon* et al (13) had similar experience and found that the late systolic honk, present in three patients without significant heart disease, was associated with ballooning of the mitral valve, while 6 patients who presented with symptoms of heart failure did not show ballooning of the mitral valve. *Keenan* et al (5) and *Upshaw* (14) have recently reported patients in whom the systolic honk was of tricuspid origin. All three patients had right sided heart failure.

Echocardiography has added another noninvasive dimension to the study of these musical murmurs. *Felner* et al (4) recently presented three healthy children with systolic honks and prolapse of the mitral valve on echocardiography, while one patient with tricuspid honk presented by *Venkatarman* et al (15) had right ventricular failure and tricuspid regurgitation. Our patients with biventricular failure, on the other hand, had no echocardiographic evidence of mitral valve prolapse, but had systolic fluttering of the tricuspid valve, which appeared and coincided with the systolic honk. These honks seem to be related to heart failure since they decrease in intensity by improving the cardiovascular function, on standing, and they increase in intensity with worsening of the heart failure, or squatting and leg raising maneuvers, which increases the right ventricular volume. It is rather unusual that a systolic honk related to tricuspid valve should decrease during inspiration and might even be considered paradoxical, however, there are reports in the literature showing that the right sided and tricuspid regurgitation murmurs decrease during inspiration (6, 8).

We believe that in our patient, the inspiratory decrease of systolic honk may be related to the severity of the right sided failure. Review of the literature suggests that most of the systolic honks associated with heart failure are probably not due to prolapse of the mitral valve, but have different mechanisms of production. We postulate that with severe congestive heart failure and dilatation of the right ventricle, there is a development of functional tricuspid insufficiency, and, that further dilatation causes the leaflets to be pulled centrifugally until they are tensed in an almost straight line and may even leave a central space. The whole leaflet apparatus then acts as a single curtain, torn in the middle. It is this separated curtain effect that produces the fluttering of the leaflets (during tricuspid regurgitation) and causing the high frequency sounds of the honk. As the failure improves, the dilated right ventricle decreases in size and the tricuspid leaflets become laxed towards normally coapted position, thereby eliminating the separated curtain effect, and finally losing even the tricuspid insufficiency. The echocardiographic findings in our patients demonstrated that the systolic honk is produced in the right side of the heart due to vibration of the tricuspid valve. We, therefore, agree with *Cobbs* (2) that in some of the patients with heart failure, especially those with right ventricular failure, the systolic honk may be originating from the tricuspid valve.

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