The praecordial honk

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The 'praecordial honk' or 'systolic whoop' is an intermittent, loud, musical, systolic sound which has been considered until a few years ago an extracardiac noise, probably pleuropericardial in origin, and therefore of no clinical significance. Only recently has it been suggested that this sound can represent a sign of mitral valve involvement.

The pertinent findings of 9 cases are presented. Three were young women with minor congenital skeletal abnormalities, no history of acute rheumatic fever, and no abnormal auscultatory findings except a late systolic apical murmur; left ventricular cineangiocardiograms showed a systolic mitral valve ballooning and mild late systolic mitral insufficiency. The other 6 cases had evident heart disease of known aetiology: rheumatic in 3 cases and ischaemic in 3. All of them presented mitral insufficiency. No definite ballooning of the mitral valve was seen.

Some different features of the 'praecordial honk' in the two groups are pointed out.

The 'praecordial honk' or 'systolic whoop' is an intermittent, loud, musical, systolic sound which can occur with or without evidence of significant heart disease.

The first description was probably given by Osler (1880). Until a few years ago the 'praecordial honk' or 'systolic whoop' had been considered an extracardiac noise, probably pleuropericardial in origin, and therefore of no clinical significance (McKusick, 1958;

Levine and Harvey, 1959). Only recently Rackley *et al.* (1966) suggested that in some cases the 'praecordial honk' could be intracardiac in origin and caused by mitral regurgitation. The same year, Leon *et al.* (1966) showed by means of intracardiac phonocardiography that the late systolic murmurs and systolic 'whoops' arose from the mitral valve and represented a sign of mitral regurgitation. One year later, Behar, Whalen, and McIntosh (1967) presented 2 new cases and a re-evaluation of the cineangiocardiograms of 6 cases of the previous group of Rackley *et al.* (1966).

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FIG. 1 Phonocardiogram of Case 1 (A), Case 2 (B), and Case 3 (C) showing a crescendo-decrescendo late systolic murmur.

TABLE Auscultatory and phonocardiographic features of ‘praecordial honk’

Case No.	Age (yr)	Sex	Diagnosis	Point of maximum intensity and transmission	Timing	Intensity
1	18	F	Ballooning mitral valve; mild mitral insufficiency	Apex and midpraecordium, radiating all over chest	Constantly late systolic	Conspicuously variable (grade 3-6/6), absent for long periods (days or weeks)
2	23	F	Ballooning mitral valve; mild mitral insufficiency	Apex and midpraecordium, radiating all over chest	Constantly late systolic	Conspicuously variable (grade 3-6/6), absent for long periods
3	18	F	Ballooning mitral valve; mild mitral insufficiency	Apex and midpraecordium, radiating all over chest	Constantly late systolic	Conspicuously variable (grade 3-6/6), absent for long periods
4	37	F	Severe mitral stenosis; mild mitral insufficiency	Midpraecordium	Early, mid-, or late systolic	Grade 3/6, absent for long periods
5	66	M	Severe aortic stenosis; mild mitral insufficiency	Apex, radiating into left axilla	Mid- or late systolic	Slightly variable (grade 3-4/6), absent only for short periods (minutes or hours)
6	24	M	Severe aortic stenosis and insufficiency; mild mitral insufficiency	Apex and midpraecordium, radiating into left axilla and back	Early or mid-systolic	Slightly variable (grade 4-5/6), absent only for short periods
7	62	M	Ischaemic heart disease; moderate mitral insufficiency	Apex, radiating into left axilla	Early or mid-systolic	Slightly variable (grade 3-4/6), absent only for short periods
8	54	F	Ischaemic heart disease; mild mitral insufficiency	Apex, and midpraecordium, radiating into left axilla	Early, mid-, or late systolic	Slightly variable (grade 3-4/6), absent only for short periods
9	70	M	Ischaemic heart disease; moderate mitral insufficiency	Apex, radiating into left axilla	Early, mid-, or late systolic	Slightly variable (grade 3-4/6), absent only for short periods

FIG. 2 Phonocardiogram of Case 2 (A) and Case 3 (B) showing the midsystolic clicks.

Body position and respiratory changes	Müller's manoeuvre	Valsalva's manoeuvre	Effort	Amyl nitrite
Constantly initiated or accentuated in upright position and by expiration	Constantly decreased or eliminated	Constantly decreased or eliminated	Constantly increased	Constantly eliminated
Constantly initiated or accentuated in upright position and by expiration	Constantly decreased or eliminated	Constantly decreased or eliminated	Constantly increased	Decreased
Constantly initiated or accentuated in upright position and by expiration	Constantly decreased or eliminated	Constantly decreased or eliminated	Constantly increased	Decreased
No change in upright position, initiated or accentuated by inspiration	Increased	Eliminated	No evident changes	Modified but slightly and unpredictably
Initiated or moderately accentuated in upright position and by expiration	Decreased	Decreased	Slightly increased	Slightly decreased
Initiated or accentuated in upright position; moderately accentuated by expiration	Eliminated or no evident change	Slightly decreased	Conspicuously increased	Slightly increased
Initiated or accentuated in upright position; moderately accentuated by expiration	Moderately increased	Decreased	No evident changes	Modified but slightly and unpredictably
No evident changes	Decreased	Slightly decreased	No evident changes	Modified but slightly and unpredictably
Initiated or accentuated in upright position; moderately accentuated by expiration	No evident changes	Slightly decreased	No evident changes	No evident changes

FIG. 3 Phonocardiogram of Case 1 showing a loud 'honk' which occupies the second half of the systole and extends into the second heart sound.

Three of them had obvious heart disease. The other 5 had no significant heart disease, but presented cineangiographic evidence of a ballooning of the mitral valve leaflets into the left atrium during early ventricular systole. In 4 of them this mitral valve dysfunction was associated with late systolic mitral insufficiency.

Nine cases with 'praecordial honk' will be the subject of this report. Three of them had ballooning of the mitral valve and 6 an obvious heart disease of known aetiology. The clinical and phonocardiographic features of these two groups will be discussed.

Patients and methods

Nine patients were seen in our department between April 1969 and April 1970. Each had a complete history, physical examination, electrocardiogram, chest x-ray, and right heart catheterization. The left ventricle was reached through retrograde aortic catheterization in all but 2 (Cases 8 and 9). Left ventricular cineangiograms were performed by using an Arriflex 35 mm camera at a speed of 34 frames/min. The contrast medium (Angio Conray 80%) was injected by means of a Gidlund apparatus at a pressure of 7 kg/cm². Phonocardiograms were obtained on a four channel Md. R. 108 r. Galileo Direct Recorder with a frequency range of 60 to 600 Hz at a speed of 50 mm/sec in recumbent and upright position, during and after the Müller and Valsalva manoeuvres, amyl nitrite inhalation, effort,

FIG. 4 Phonocardiograms of Case 1 showing a 'honk' that becomes louder when the patient assumes the upright position.

and in some patients (Cases 1-3) after intravenous administration of isoprenaline (0.05 mg) and propranolol (5 mg). All patients had been followed up clinically and phonocardiographically for a period varying from 3 to 12 months.

Results

The cases were divided into two groups according to the presence of either 'ballooning mitral valve syndrome' (Group 1), or a significant heart disease of known aetiology (Group 2).

Group 1 This includes 3 young women, aged 18 (Case 1), 23 (Case 2), and 18 (Case 3) years. None of them had an illness which could have been pleuritis or pericarditis; in Case 3 an atrial septal defect had been previously diagnosed by means of cardiac catheterization.

They were referred to our department because of the following symptoms: occasional palpitation and irregular heart beat, especially when assuming the upright position, after effort or excitement; mild exertional dyspnoea; vague pain over the anterior chest, not typical of angina; an intermittent, squeaking noise in the chest (Case 1) on assuming the upright position, after effort or excitement.

Physical examination revealed red hair and fair complexion (unusual findings in Southern Italy); minor skeletal abnormalities (high arched palate, short antero-posterior diameter of the chest, moderate kyphoscoliosis, and hollow foot in Case 1; incurving little fingers in Case 2; moderate scoliosis and hollow foot in Case 3); extrasystoles; slight (Cases 1 and 3) or no (Case 2) enlargement of the heart; an

intermittent, prominent systolic thrill over the praecordium simultaneously with the 'honk' (Cases 1 and 2); a soft apical late systolic murmur (Fig. 1) accompanied (Cases 2 and 3) by a midsystolic click (Fig. 2); a late systolic 'honk'.

The electrocardiograms showed in all cases ventricular ectopic beats, which were much increased by the slow intravenous injection of a small amount of isoprenaline (0.05 mg), by

FIG. 5 Phonocardiogram of Case 1 showing a 'honk' that develops during expiration from late systole, masking the late systolic murmur.

physical effort, and when in the upright position; an incomplete right bundle-branch block was present in Case 3; the QT interval and the T and U waves were normal. The x-rays showed no left atrial enlargement; in Case 1 a slight prominence of the pulmonary artery and left ventricle was seen; Case 3 presented slight enlargement of the right ventricle and a prominent pulmonary artery. At cardiac catheterization the pressures were found to be normal in the right heart, pulmonary tree, and left heart; the pulmonary capillary pulse showed no abnormalities; in Case 3 the previous diagnosis of atrial septal defect was confirmed. Left ventricular cineangiograms showed in all cases a systolic ballooning of the mitral valve into the left atrium and a mild late systolic regurgitation.

At surgical correction of the atrial septal defect in Case 3 no abnormalities were observed, except for conspicuously redundant mitral leaflets. Mitral incompetence could not be assessed at palpation. No pleuro-pericardial adhesions were found.

The main clinical and phonocardiographic characteristics of the 'honk' (Table and Fig. 3-7) were the following: constantly situated in the second half of the systole; at times ending just before the second sound, at times extending into it; intermittency with long periods of absence; conspicuous spontaneous changes in intensity; at times extremely loud (grade 6/6) and associated with a prominent apical thrill; on these occasions it could be heard by the physician all over the chest; almost constantly initiated or accentuated on expiration, in upright position, by effort, and

FIG. 6 Phonocardiogram of Case 1 showing a 'honk' that becomes smaller on inspiration.

isoprenaline; almost constantly reduced or eliminated for a few seconds by the Müller and Valsalva manoeuvres, by amyl nitrite inhalation, and for longer periods by propranolol.

Group 2 This includes 6 patients (Cases 4-9) with heart disease of known aetiology. In the 4 patients (Cases 4-7) who underwent left ventricular cineangiography, mild (Cases 4-6) or moderate (Case 7) mitral in-

FIG. 7 Phonocardiogram of Case 2 (A) and Case 3 (B) showing a loud, late systolic 'honk' which envelops the second heart sound.

competence, but no definite ballooning of the mitral leaflets was shown. Cases 4, 5, and 6 had a history of rheumatic fever; mitral insufficiency was associated in Case 4 (a woman aged 37) with tight mitral stenosis, in Case 5 (a man aged 66) with severe aortic stenosis, and in Case 6 (a man aged 24) with severe aortic stenosis and insufficiency. All of them presented with symptoms of cardiac failure: classes I, III, and IV, respectively. Case 4 underwent closed mitral valvotomy. At operation no pleuropericardial or pericardial adhesions were found; mitral incompetence could not be assessed at palpation. The cusps were mobile and free from calcium deposits. Both commissures could be easily split with the finger. No relevant abnormalities of the chordae tendineae or papillary muscles were found. Ten days after the operation a decrease of the apical regurgitant murmur from grade 2/6 to 1/6 was observed. The 'honk' disappeared and has not been seen afterwards during a three-months' follow-up. Case 6 underwent aortic valve replacement with a Starr-Edwards prosthesis. At operation the mitral valve was found to be normal, except for the rupture of a small group of chordae tendineae, attached to the posterior leaflet. The patient died a few hours after operation. Permission for necropsy was not obtained.

Case 7 (a man aged 62), Case 8 (a woman aged 54), and Case 9 (a man aged 70) had no history of rheumatic fever. The history and the electrocardiographic changes were typical of ischaemic heart disease; mitral insufficiency was considered of moderate degree in Cases 7 and 9, of mild degree in Case 8, and attributed to papillary muscle dysfunction. There was no cardiac failure.

While in 4 patients in this group (Cases 4, and 7-9), the diagnosis of mitral regurgitation could be based on the typical auscultatory findings, in the 2 with aortic stenosis (Cases 5 and 6) mitral insufficiency could be suspected only by the presence of the 'praecordial honk'.

The main features of the 'praecordial honk' (Table and Fig. 8-15) in this group were the following: variable in timing, moving in the same patient from the early to the middle or late systole; at times absent, but only for short periods; generally of moderate and slightly variable intensity; often difficult to diagnose because of its association with the murmurs of mitral insufficiency or of aortic stenosis (in some cases its presence could be suspected only by an intermittent change in the quality of the murmur which acquired a vibratory, honking quality), not associated with a thrill; inconstantly and less evidently

FIG. 8 Phonocardiogram of Case 5 showing an ejection murmur and a late systolic 'honk'.

accentuated in the upright position and by effort; less evidently and unpredictably modified by respiration, the Müller and Valsalva manoeuvres, and by amyl nitrite inhalation.

Discussion

The sex incidence, history, symptoms, and the clinical findings of Group I closely resemble those of the syndrome of 'mid-late

FIG. 9 Phonocardiogram of Case 5 showing the variations in the intensity of the 'honk' from beat to beat; the 'honk' disappears in the third systole.

FIG. 10 Phonocardiogram of Case 5 showing the variable intensity of the 'honk'; the association with a loud ejection murmur makes the identification of the 'honk' difficult in the first systole, almost impossible in the second one.

has often been found to be familial (Barlow and Bosman, 1966; Linhart and Taylor, 1966; Hancock and Cohn, 1966; Stannard *et al.*, 1967; Barlow *et al.*, 1968; Shell *et al.*, 1969), as well as in our cases of Group 1, a peculiar mitral valve dysfunction characterized by the systolic ballooning of the mitral leaflets into the left atrium, often associated with mild late-systolic mitral insufficiency, has been observed by cineangiocardigraphic studies (Barlow and Bosman, 1966; Linhart and Taylor, 1966; Criley *et al.*, 1966; Hancock and Cohn, 1966; Stannard *et al.*, 1967; Kesteloot and Van Houte, 1965).

In a group of patients with this syndrome studied by Hancock and Cohn (1966) 'several patients stated that their own murmur had been audible to them on occasion'. It can be presumed that on these occasions a

FIG. 11 Phonocardiogram of Case 6 showing: (A) decreased heart sounds, a loud ejection murmur, and a soft decrescendo diastolic murmur; (B) an early systolic 'honk' in the first beat and two 'honks' in the second beat: the first occupies early systole, the second late systole.

FIG. 12 Phonocardiogram of Case 7 showing a systolic murmur and a midsystolic 'honk'; the intensity of the 'honk' varies from beat to beat

systolic click and late systolic murmur' (Barlow and Pocock, 1963; Barlow *et al.*, 1963; Barlow, 1965; Barlow and Bosman, 1966; Linhart and Taylor, 1966; Criley *et al.*, 1966; Hancock and Cohn, 1966; Stannard *et al.*, 1967; Barlow *et al.*, 1968; Shell *et al.*, 1969; Leachman, De Francheschi, and Zamalloa, 1969), also described as 'late systolic dysfunction of the mitral apparatus' (Engle, 1969).

In many cases with this syndrome, which

FIG. 13 Phonocardiogram of Case 7 showing an early and midsystolic 'honk' that presents a changing shape; sometimes more than one 'honk' seems to be present

physical examination would very likely have detected a praecordial honk.

Mid-late systolic clicks and/or late apical systolic murmurs have recently been seen in patients with mitral insufficiency associated with the Marfan syndrome or other connective tissue heritable disorders (Hancock and Cohn, 1966; Barlow *et al.*, 1968; Segal, Kasparian, and Likoff, 1962). Though none of these diseases was present in our cases, they all presented some minor congenital deformities of the skeleton. According to Shell *et al.* (1969), 'there would appear to be some relationship between the mitral valve abnormality of the Marfan syndrome, the floppy mitral valve and mid-late systolic click and late-systolic murmur syndrome'.

The nature of this valve dysfunction is still obscure. A voluminous posterior mitral valve leaflet and thin elongated chordae tendineae have been found at necropsy in one case of Barlow *et al.*'s (1968) series with mid-late systolic click and late systolic murmur. Voluminous mitral valve leaflets were also seen in our patient who underwent operation for the correction of an atrial septal defect.

In the patients of Group 2 with significant heart disease, the most important finding was the constant association of the 'praecordial honk' with mitral valve incompetence of different degree (mild to moderate) and different aetiology (rheumatic or ischaemic). A ballooning of the mitral valve was not present in any of these patients who underwent a cineangiographic study.

In conclusion, our observations indicate: (1) the 'praecordial honk' is not an innocent

sound, being constantly associated with heart disease; (2) it seems to present some distinctive features according to the nature of the heart disease: the still poorly recognized 'ballooning mitral valve syndrome' on one side, the acquired valvular diseases (rheumatic or ischaemic) on the other side. The first type of lesions produces a 'praecordial honk' which is constantly late systolic, intermittent (when absent a midsystolic click and/or an apical late systolic murmur can always be detected), at times very loud, presenting conspicuous spontaneous changes, often associ-

FIG. 14 Phonocardiogram of Case 7 showing a 'honk' that occupies the last part of the systole and masks the second heart sound.

FIG. 15 Phonocardiograms of Case 9 showing a holosystolic apical murmur (A), an early systolic 'honk' that partially envelopes the first heart sound (B), and the intermittent character of the 'honk' (C).

ated with a prominent apical thrill, almost constantly initiated or modified in a predictable way by a certain number of manoeuvres or drugs. The second type of lesions produces a 'honk' which is variable in timing in the same patient (moving from the early systole to the middle or late systole on different occasions), not associated with a thrill, more constant, with less evident spontaneous changes, difficult to recognize because of its association with a systolic murmur, often scarcely or unpredictably modified by the already mentioned manoeuvres; sometimes more than one 'honk' is present in one systole.

References

- Barlow, J. B. (1965). Conjoint clinic on the clinical significance of late systolic murmurs and non-ejection systolic clicks. *Journal of Chronic Diseases*, **18**, 665.
- Barlow, J. B., and Bosman, C. K. (1966). Aneurysmal protrusion of the posterior leaflet of the mitral valve. *American Heart Journal*, **71**, 166.
- Barlow, J. B., Bosman, C. K., Pocock, W. A., and Marchand, P. (1968). Late systolic murmurs and non-ejection ('mid-late') systolic clicks. An analysis of 90 patients. *British Heart Journal*, **30**, 203.
- Barlow, J. B., and Pocock, W. A. (1963). The significance of late systolic murmurs and mid-late systolic clicks. *Maryland State Medical Journal*, **12**, 76.
- Barlow, J. B., Pocock, W. A., Marchand, P., and Denny, M. (1963). The significance of late systolic murmurs. *American Heart Journal*, **66**, 443.
- Behar, V. S., Whalen, R. E., and McIntosh, H. D. (1967). The ballooning mitral valve in patients with the 'precordial honk' or 'whoop'. *American Journal of Cardiology*, **20**, 789.
- Criley, J. M., Lewis, K. B., Humphries, J. O'N., and Ross, R. S. (1966). Prolapse of the mitral valve: Clinical and cine-angiographic findings. *British Heart Journal*, **28**, 488.
- Engle, M. E. (1969). Editorial. The syndrome of apical systolic click, late systolic murmur, and abnormal T waves. *Circulation*, **39**, 1.
- Hancock, E. W., and Cohn, K. (1966). The syndrome associated with midsystolic click and late systolic murmur. *American Journal of Medicine*, **41**, 183.
- Kesteloot, H., and Van Houte, O. (1965). On the origin of the telesystolic murmur preceded by a click. *Acta Cardiologica*, **20**, 197.
- Leachman, R. D., De Francheschi, A., and Zamalloa, O. (1969). Late systolic murmurs and clicks associated with abnormal mitral valve ring. *American Journal of Cardiology*, **23**, 679.
- Leon, D. F., Leonard, J. J., Kroetz, F. W., Page, W. L., Shaver, J. A., and Lancaster, J. F. (1966). Late systolic murmurs, clicks, and whoops arising from the mitral valve. A transseptal intracardiac phonocardiographic analysis. *American Heart Journal*, **72**, 325.
- Levine, S. A., and Harvey, W. P. (1959). *Clinical Auscultation of the Heart*, 2nd ed. Saunders, Philadelphia.
- Linhart, J. W., and Taylor, W. J. (1966). The late apical systolic murmur: clinical, hemodynamic and angiographic observations. *American Journal of Cardiology*, **18**, 164.
- McKusick, V. A. (1958). *Cardiovascular Sound in Health and Disease*. Williams and Wilkins, Baltimore.
- Osler, W. (1880). On a remarkable heart-murmur, heard at a distance from chest-wall. *Medical Times and Gazette*, **2**, 432.
- Rackley, C. E., Whalen, R. E., Floyd, W. L., Orgain, E. S., and McIntosh, H. D. (1966). The precordial honk. *American Journal of Cardiology*, **17**, 509.
- Segal, B., Kasparian, H., and Likoff, W. (1962). Mitral regurgitation in a patient with the Marfan syndrome. *Diseases of the Chest*, **41**, 457.
- Shell, W. E., Walton, J. A., Clifford, M. E., and Willis, P. W. (1969). The familial occurrence of the syndrome of mid-late systolic click and late systolic murmur. *Circulation*, **39**, 327.
- Stannard, M., Sloman, J. G., Hare, W. S. C., and Goble, A. J. (1967). Prolapse of the posterior leaflet of the mitral valve: a clinical, familial, and cine-angiographic study. *British Medical Journal*, **3**, 71.