THE EARLY SYSTOLIC SOUND IN DILATATION OF THE PULMONARY ARTERY

BY

AUBREY LEATHAM AND LOUIS VOGELPOEL *

From the Institute of Cardiology, National Heart Hospital

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A pulmonary early systolic sound was investigated in 50 patients. It may be mistaken for splitting of the first heart sound but is loudest in the pulmonary area, and it has been helpful in clinical diagnosis.

Extra sounds in systole were known to Cuffer and Barbillon (1887). Potain (1900) described systolic gallop rhythm when the extra sound was loudest in the aortic area in cases of aortic atheroma and in typhoid fever, and he attributed this sound to the sudden checking of aortic expansion. Wolferth and Margolies (1940) came to the conclusion that this description concerned a rare mid-systolic sound. Gallavardin (1913) described late, occasionally mid-systolic, clicking sounds, different in quality from the heart sounds, usually loudest at the apex and varying with the phase of respiration; he considered them of extracardiac origin and found pleuro-pericardial adhesions in three cases. Subsequent authors including Lian and Deparis (1933), Johnston (1938), and Evans (1943) confirmed that mid or late systolic clicks were of no significance except in differential diagnosis. An exception is the systolic clicking sound in cases of mediastinal emphysema and left-sided pneumothorax (Hamman, 1937; Scadding and Wood, 1939). Mid or late systolic clicks or sounds, or "gallops" will not be considered in this paper.

An early systolic sound (claquement protosystolique) was heard and recorded by Lian and his collaborators (1937, 1941, 1951) at the pulmonary area in patients with pulmonary stenosis and with dilatation of the pulmonary artery, and at the aortic area with aortic valve disease. It had been described by Petit (1902). Wolferth and Margolies (1945) stated that they had recently become aware of a sound in early systole which they had either overlooked or mistaken for the second component of a split first sound, and attributed it to the opening of the semilunar valves. Arrilaga and Taquini (1941) had reported a similar sound in one case of pulmonary stenosis and called it the semilunar opening click.

Method of Investigation. Routine diagnostic cardiac catheterization had been performed in all but 4 of 50 consecutive patients in whom an obvious early systolic sound was heard in the pulmonary area. A larger number of cases without this sound on clinical auscultation but showing some or all of the cardiac abnormalities found in the positive group, were used as control cases. The clinical examination in each case included special attention to the first sound and to added systolic sounds in the mitral, tricuspid, pulmonary, and aortic areas. The intensity of the pulmonary sound was graded from one to four in expiration and in inspiration, and in relation to upright and reclining postures. Enlargement of the main pulmonary arc (pulmonary trunk and left branch) and of the right pulmonary artery were separately graded from one to four by means of teloradiograms in anterior and oblique views. Electrocardiographic evidence of ventricular hypertrophy was also graded and, to obtain some indication of the degree of ventricular asynchrony, the width of the QRS was measured in the widest V chest lead. Pressure tracings were obtained from the right ventricle and pulmonary artery with a Sanborn capacitance electromanometer; many records were taken at a speed of 50 mm. a second to permit accurate timing. In four cases the catheter failed to enter

* Nuffield Foundation Dominion Travelling Fellow from the University of Cape Town, South Africa.

the pulmonary artery, but in two of these right ventricular pressure tracings were used to determine the pulmonary arterial systolic pressure.

In every case high-frequency phonocardiograms were taken, the low frequencies being attenuated by approximately the same proportion as by the stethoscope and human hearing mechanism, so that the graphs closely resembled the findings of clinical auscultation (Posener and Trendelenburg, 1929; Rappaport and Sprague, 1942). Synchronous high-frequency phonocardiograms (Leatham, 1949, 1952) taken at high speed from the mitral, tricuspid, and pulmonary areas were needed to separate the first sound, and its components when split, from the added sound. Synchronous carotid traces in ten cases gave a time relation between these sounds and the opening of the aortic valve. A crystal microphone was placed directly on the neck, thus avoiding delay from air conduction, and a suitable time constant was obtained electrically. Delay in pulse wave transmission from the aortic valve to the carotid artery was assumed to be given by the time interval from the onset of the aortic component of the second sound to the nadir of the dicrotic notch of the carotid tracing. This interval was found to be about the same when calculated from the speed of pulse wave transmission of 8–10 metres/sec. given by Wiggers (1949) and averaged 0.03 sec.

The heart sounds were related to the pressure pulses from the pulmonary artery and the right ventricle in 36 patients, and from the left ventricle in 4, by using the same electrocardiogram during catheterization and phonocardiography, noting any differences in rate by measuring the preceding R–R interval. In 12 of these patients we were able to obtain more precise relationships by taking pressure tracings synchronously with phonocardiograms from the pulmonary and mitral areas. The delay in the recording system was estimated by applying sudden pressure changes through a number eight catheter and the usual connector, damper, and electromanometer to a multi-channel photographic recorder. The sound produced by the onset of the pressure change was transmitted instantaneously to another galvanometer on the same recorder. The time interval between the two deflections was measured at varying levels of damping which corresponded approximately to those used during routine catheterization. With critical damping, as used for most of our right ventricular pressure pulses, the delay was found to be from 0.015 to 0.02 sec., and a subtraction of 0.02 sec. has been made from all estimations of the time interval between the onset of QRS and the rise of pressure in the right ventricle. Additional hydraulic damping was used for the pulmonary artery tracings; the delay with damping comparable to that used in the majority of cases was found to be 0.03 sec., and this figure has been subtracted from all estimations of the time interval between the onset of QRS and the rise of pressure in the pulmonary artery. These corrections can only be approximate since the degree of damping was not known in every case.

Sternal angle level has been taken as the reference point for our pressure measurements, and pulmonary hypertension has been defined as a resting pressure exceeding 30/15 mm. Hg. All time intervals have been measured from the onset of an event, whether QRS (lead II) of the electrocardiogram, heart sound, or pressure pulse. The onset of the first heart sound was taken at the beginning of its major high-frequency components, thus excluding inaudible low-frequency auricular or ventricular components.

RESULTS

The pulmonary early systolic sound is high-pitched and often sharp like a click. It is loudest in the second and third left intercostal spaces and only transmitted to the mitral area and tricuspid area (lower left sternal edge) if very loud. It is louder during sustained expiration, as pointed out by Lian and Welti (1937), sometimes almost disappearing in full inspiration (Fig. 1). It is unaffected by posture, and has a constant place in the cardiac cycle. It gives the impression of wide splitting of the first heart sound, but the second component usually has this different sharper quality, suggesting the term pulmonary early systolic click. When the first heart sound is soft or inaudible in the pulmonary area the added sound has sometimes been mistaken for a first sound of unusual snapping quality in the pulmonary area (Fig. 1); listening between the pulmonary and mitral areas both sounds can be heard. Sometimes the extra sound just precedes and is obscured by a pulmonary systolic murmur (Fig. 2 and 3), but can again be discerned more plainly one intercostal space lower down (Fig. 3). Synchronous high-frequency phonocardiograms from the pulmonary, tricuspid, and mitral areas in every case confirmed that the maximum intensity of the extra sound was in the pulmonary area and that it was preceded by the first sound at the apex when the latter was poorly conducted to the pulmonary area (Fig. 1, 2, and 3). The time interval between the onset of the major

FIG. 1.—Pulmonary early systolic sound (x) in idiopathic dilatation of the pulmonary artery (PA enlargement grade 3, PA pressure 10/5, RV (right ventricular) pressure 25/0 mm. Hg). It had been described as a first sound of unusual snapping quality in the pulmonary area where the main first sound was inaudible, and was very loud during expiration (exp.) and soft during inspiration (insp.), as shown in the high-frequency (HF) phonocardiogram from the pulmonary area (PA). A synchronous medium frequency (MF) recording from the mitral area (MA) shows the main first heart sound (1) preceding the pulmonary sound by 0.06 sec. Both sounds could be heard in an intermediate area. The time intervals in this and subsequent records are 0.2 and 0.04 sec.

high-frequency components of the first heart sound and the onset of the extra pulmonary sound in the 50 patients averaged 0.07 sec., ranging from 0.05 to 0.14 sec. except two measurements of 0.02 to 0.03 sec. in cases without pulmonary hypertension to be discussed later.

An auricular sound followed by the main first heart sound has sometimes been mistaken for the first sound and extra pulmonary sound, but the auricular sound is very low-pitched and both these sounds are loudest at the mitral and the tricuspid areas. It may be more difficult to distinguish the pulmonary sound from the second component of a split first heart sound (Table I). The time interval between the onset of the two components of the first heart sound in 9 healthy subjects ranged from 0.03 to 0.05 sec. and was thus less than between the first sound and the added sound, giving the auscultatory impression of a splitting which was narrow rather than wide. Splitting of the first heart sound is most obvious at the mitral area and lower left sternal edge rather than the pulmonary area (Lian and Welti, 1937; Orías and Braun-Menéndez, 1939; Wolferth and Margolies, 1940); either component may be abrupt though not clicking in quality. In expiration, splitting may be more obvious, but there is no striking accentuation of the second component as with the pulmonary early systolic sound. Synchronous phonocardiograms demonstrate the site of maximum intensity of the two components of a split first sound to be at the mitral or tricuspid areas (Fig. 4). Sometimes this technique can show the separate identity of the pulmonary sound by

TABLE I
CLINICAL DIFFERENTIATION OF THE PULMONARY EARLY SYSTOLIC SOUND FROM A SPLIT FIRST SOUND

Characteristics	Pulmonary early systolic sound	Second component of split first sound
Position in early systole	Later (wide "splitting" of first sound)	Earlier (narrow splitting of first sound)
Site of maximum intensity	Pulmonary area	Tricuspid and mitral areas
Quality	High pitched, may be clicking	Abrupt
Expiration	Louder	Clearer

FIG. 2.—Pulmonary early systolic sound immediately preceding a pulmonary systolic murmur (SM) in severe pulmonary hypertension (PA grade 2, PA pressure 90/65, aortic pressure 95/65 mm. Hg) associated with a ventricular septal defect. The onset of the extra sound occurs 0.08 sec. after the onset of the major components of the first heart sound and 0.05 sec. after the onset of rise of pressure in the pulmonary artery and aorta (estimated from indirect carotid tracing, CAR) which in this case were synchronous as expected with equal or balanced pulmonary and aortic pressures.

FIG. 3.—Pulmonary early systolic sound immediately preceding a pulmonary systolic murmur in a similar case of severe pulmonary hypertension (PA grade 4) associated with a ventricular septal defect. The extra sound was difficult to appreciate in the pulmonary area where the first sound was faint and the systolic murmur loud, but both sounds could be easily heard in the third left space (3LS) and were separated by 0.07 sec. The significance of the early systolic sound as an auscultatory sign of pulmonary hypertension was supported in this case by the loudness of the pulmonary component of the second sound (P2) which was louder than the aortic component (A2) in the pulmonary area and transmitted to the apex (MA).

showing splitting of the first heart sound preceding the extra sound (Fig. 8). In bundle branch block the splitting of the first sound may be very wide and in some cases of right bundle branch block the later component may be loud (Fig. 5) and occasionally well transmitted to the pulmonary area making clinical differentiation difficult or impossible. Synchronous phonocardiograms, sometimes even with pressure pulses, may be necessary for differentiation, but only three of our cases had a prolongation of QRS beyond 0.10 sec.

Sounds occurring later in systole must be distinguished from the pulmonary early systolic sound because they are seldom of significance. They may be single or multiple and are usually louder at the mitral area than at the base; their position in systole may vary from cycle to cycle, especially with respiration, occasionally just preceding mid-systole (Fig. 6), but not early enough to be associated with the first sound. Thus, they are unlike the pulmonary sound in site, variability, and timing.

CLINICAL SIGNIFICANCE OF THE PULMONARY EARLY SYSTOLIC SOUND

Of the 50 patients showing this physical sign on clinical auscultation, 44 had pulmonary hypertension which was confirmed by cardiac catheterization in all but two. The pulmonary

FIG. 4.—Physiological splitting of the main components of the first heart sound (11) is most obvious at the tricuspid area or lower left sternal edge (LSE). The time interval between the onset of each of these two components measures 0.03 sec. which is less than half the usual interval between the onset of the main first sound and the pulmonary sound.

FIG. 5.—Splitting of the first sound from right bundle branch block (QRS 0.13 sec.). The separation of the two components of the first sound is wide (0.08 sec.) but the second component is very small in the pulmonary area, and thus unlike the pulmonary sound. Right bundle branch block was suspected clinically from the wide splitting of the second sound.

FIG. 6.—Apical systolic clicking sounds (Y) in a healthy patient.

hypertension was associated with a shunt from congenital heart disease in nearly half of the patients, seven with patent ductus arteriosus, ten with ventricular septal defect, and seven with atrial septal defect. There was one case of transposition, three of primary pulmonary hypertension, and the remaining sixteen had mitral stenosis (Table II). All but three of our patients with pulmonary hypertension had obvious enlargement of the pulmonary artery (Fig. 9). The pressure in the pulmonary artery was much above normal (average PA diastolic pressure 38 mm. Hg; RV end diastolic 0 mm. Hg) and the total pulmonary resistance always more than twice normal, varying from 6–28 units (Wood,

TABLE II
THE DIAGNOSIS AND INCIDENCE OF PULMONARY HYPERTENSION AND PULMONARY ARTERY ENLARGEMENT IN 50 PATIENTS WITH A PULMONARY EARLY SYSTOLIC SOUND

Diagnosis	No. of patients	No. with dilatation of pulmonary artery and pulmonary hypertension *	No. with dilatation of pulmonary artery without pulmonary hypertension
Patent ductus arteriosus	7	7	0
Ventricular septal defect	10	10	0
Atrial septal defect	9	7	2
Transposition	1	1*	0
Primary pulmonary hypertension	3	3	0
Mitral stenosis	16	16	0
Pulmonary valve stenosis (slight)	1	0	1
Idiopathic dilatation of pulmonary artery	1	0	1
Pulmonary incompetence (slight)	1	0	1
Normal	1	0	0
Total	50	44	5

* Pulmonary hypertension was inferred in the patient with transposition: all the 44 patients except this one had dilatation of the pulmonary artery as well as hypertension.

1952) in the 35 patients in whom it was measured. There was clinical and electrocardiographic evidence of right ventricular hypertrophy in all except five, of whom two had complete right bundle branch block.

The diagnoses in our 50 patients are summarized in Table II.

In the 17 patients with patent ductus arteriosus or ventricular septal defect the pulmonary pressure was sufficiently high to balance or reverse the usual left-to-right shunt, thus abolishing or diminishing the typical loud continuous or long systolic murmurs, which were replaced by a soft or moderately loud pulmonary mid-systolic murmur commencing immediately after the added sound. In the three patients without dilatation of the main pulmonary arc, the pulmonary pressure was very high; in two of them some dilatation of the pulmonary arc was probably concealed by rotation but was suggested indirectly by enlargement of the right main branch in both, and confirmed directly by angiocardiology in one of them. The third was found to have transposition of the great vessels at necropsy; pulmonary hypertension was assumed but the pulmonary trunk was no larger than the aorta. Obvious pulmonary artery enlargement accompanying the pulmonary hypertension seems an important factor in production of the sound because it was absent in five control cases of patent ductus arteriosus with equally high pulmonary artery pressures and little or no pulmonary artery enlargement. In a further seven control cases severe pulmonary hypertension was accompanied by moderate or great dilatation of the pulmonary artery but no extra sound could be heard. In all but one there was a loud systolic murmur which might have been expected to obscure the sound, though it could be recorded on the phonocardiogram in half of them. In the remaining one who had a patent ductus arteriosus the pulmonary artery pressure was equal to the aortic and the main pulmonary arc was moderately dilated; the pulmonary systolic murmur was faint yet no early systolic sound could be heard or recorded.

In the 16 cases with mitral disease the stenosis was usually pure, occasionally accompanied by minor degrees of mitral or aortic incompetence. The complicating pulmonary hypertension was moderate or severe considering the small pulmonary flow, and the total pulmonary resistance was high, averaging 14 units and never less than 6 units. The main pulmonary arc was always enlarged, but less than in the previous groups and the pulmonary sound tended to be less loud (Fig. 7). In twelve control cases of mitral stenosis without this sound on auscultation there was pulmonary hypertension and enlargement of the pulmonary artery of about the same degree; in four of them this sound could be recorded (Fig. 8). It has not been heard or recorded by us in cases of mitral

FIG. 7.—Pulmonary early systolic sound in pulmonary hypertension from mitral stenosis with auricular systolic and mitral diastolic murmurs, and an opening snap (OS). PA enlargement was grade two and PA p. 55/20 mm. Hg with pulmonary resistance six units.

FIG. 8.—Pulmonary early systolic vibrations (x) in mitral stenosis not heard as a pulmonary sound because of the proximity of a loud late component of a split first sound (1') presumably due to late closure of the mitral valve. The earlier component (1) is probably due to closure of the tricuspid valve and shows a normal relationship to the pulmonary sound.

stenosis without pulmonary hypertension. Thus, the presence of this added sound in the pulmonary area in mitral stenosis indicates a severe degree of pulmonary hypertension, but its absence cannot be interpreted as evidence of lower pressures. In mitral stenosis the first heart sound is both loud and late (Cossio and Berconsky, 1943) and may approach (Fig. 8) or even coincide with the extra sound, and this may account for the absence of this physical sign in some cases of mitral stenosis with pulmonary hypertension.

In the remaining six patients there was no evidence of pulmonary hypertension but in five of them the pulmonary artery was enlarged. In two the dilatation of the pulmonary artery was secondary to atrial septal defect, in two it was idiopathic or secondary to mild pulmonary valvular stenosis, in one it was slight and associated with a faint pulmonary diastolic murmur, and in the single remaining patient it occurred without any evidence of heart disease. The extra sound was not heard or recorded in patients with simple patent ductus arteriosus or ventricular septal defect and enlargement of the pulmonary artery without pulmonary hypertension. The pulmonary early systolic sound was a particularly striking physical sign in the patient with idiopathic dilatation (Fig. 1 and 13) and the one with slight pulmonary stenosis; we have seen eight such cases since this series was closed. The sound was always loud, especially in expiration, and was particularly obvious since the pulmonary systolic murmur was soft. The first sound was difficult to hear at the pulmonary area but both sounds could be clearly heard lower down. The second sound in the pulmonary area

FIG. 9.—Great enlargement of the pulmonary artery in a patient with severe pulmonary ("balanced") hypertension associated with a patent ductus arteriosus. There was a loud pulmonary early systolic sound and a faint pulmonary diastolic murmur, but no loud systolic or continuous murmurs.

FIG. 10.—Dilatation of the pulmonary artery (grade 2) secondary to mild pulmonary valve stenosis (PA pressure 14/7, RV 40/1 mm. Hg). There was a loud pulmonary early systolic sound.

was widely split into a clear earlier aortic component and a soft later pulmonary component. There was no clinical or electrocardiographic evidence of right ventricular hypertrophy; X-ray showed moderate dilatation of the pulmonary arc but not of its branches, and the heart was small or normal in size (Fig. 10). A diagnosis of mild pulmonary valve stenosis had been made in both these cases by finding an increase of systolic pressure on withdrawing the catheter from the pulmonary artery into the right ventricle. The right ventricular systolic pressure was not raised in one of them (Fig. 1), and it has been suggested that this means idiopathic congenital dilatation of the pulmonary artery without stenosis despite the systolic pressure difference across the valve (Greene *et al.*, 1949). Although recorded in one case, the extra sound was never heard in severe pulmonary stenosis with a dilated pulmonary artery, and this will be discussed later.

The pulmonary sound tended to be earlier in systole in the six patients without pulmonary hypertension averaging 0.04 sec. (range 0.02 to 0.06 sec.) after the onset of the first sound—the figure for 44 patients with pulmonary hypertension averaging more than 0.07 sec. (range 0.05 to 0.14).

An aortic early systolic sound (claquement aortique protosystolique) was described by Lian (1937, 1941, 1951) in cases of aortic stenosis and aortic incompetence. Wolferth and Margolies (1945) described this sound in cases of hypertension and aortic aneurysm as the aortic semilunar opening click because they thought it took place 0.01 to 0.02 sec. before the primary carotid oscillation and about the time when they expected ejection to take place. We have recorded this sound in 10 patients of whom three had coarctation of the aorta (Fig. 11), three aortic stenosis (Fig. 12), two aortic incompetence, and two aortic sclerosis; the ascending aorta was prominent on X-ray in all. The time interval between the onset of the first heart sound and the added sound averaged 0.06 sec., ranging from 0.04 to 0.08 sec., making this sound comparable in timing to the pulmonary sound. Indirect carotid tracings showed that the rise of pressure in the carotid artery preceded the aortic sound (Fig. 11) and, after making the subtraction for delay of pulse wave transmission, suggested that the rise of pressure in the aorta at the opening of the aortic valve preceded the extra

FIG. 11.—Aortic early systolic sound (x) in coarctation of the aorta, occurring after the upstroke of the indirect carotid tracing and 0.03 sec. after the estimated time of aortic ejection.

FIG. 12.—Aortic early systolic vibrations (x, inaudible) preceding the aortic systolic murmur in aortic stenosis.

sound by 0.03 to 0.05 sec. This sound is easily heard (Fig. 11) except when starting a loud systolic murmur as in aortic stenosis (Fig. 12), is not much augmented in the expiratory phase of respiration, and is less sharp in quality than its pulmonary counterpart. It is very widely transmitted to all areas, and is often most obvious at the apex making it more easily confused with splitting of the first heart sound. So far we have found it of much less use as a physical sign.

THE RELATION OF THE PULMONARY EARLY SYSTOLIC SOUND TO OTHER EVENTS IN THE CARDIAC CYCLE

Time intervals obtained from the 44 patients with pulmonary hypertension and a pulmonary early systolic sound are summarized in Table III. Figures for the remaining 6 patients without pulmonary hypertension are not given because the data was insufficient. The interval between Q and the onset of rise of pressure in the right ventricle (Q–RV) averaged 0.06 sec., and was slightly shorter than the intervals found by Coblenz *et al.* (1949) of 0.075 sec. in 30 adults with essentially normal circulations, and of 0.073 sec. in 13 patients with elevated right heart pressures. The interval between Q and the onset of rise of pressure in the left ventricle averaged 0.06 sec. in four patients. The interval between Q and the onset of rise of pressure in the pulmonary artery (Q–PA) averaged 0.11 sec. The Q–PA interval in the 13 cases with pulmonary hypertension studied by Coblenz *et al.* (1949) was 0.96 sec. giving an isometric (RV–PA) time of 0.023 sec. The average isometric time in the present investigation was 0.05 sec., but the right ventricle was working against a higher pressure, averaging 38 mm. Hg instead of 25 mm. Hg. The average time interval between Q and the onset of rise of pressure in the carotid tracing in 10 patients was 0.12 sec., and after subtracting the delay in pulse transmission, the time interval between Q and the onset of rise of pressure in the aorta was estimated to average 0.09 sec. The interval between Q and the onset of the major high-frequency components of the first heart sound averaged 0.07 sec. Thus, the onset of rise of pressure in the ventricles was about synchronous with the main first heart sound (Fig. 13 and 14). The interval between Q and the onset of the pulmonary early systolic sound was 0.14 sec. Since

FIG. 13.—The relation of the right ventricular pressure pulse (RV) to the first heart sound and pulmonary early systolic sound in a patient with idiopathic dilatation of the pulmonary artery (PA grade 3, PA pressure 10/7, RV pressure 16/0 mm. Hg) not in this series. The first heart sound is approximately coincident with the rise of pressure in an under-damped tracing from the right ventricle. The extra sound occurs 0.06 sec. later showing that it has no connection with closing of the tricuspid valve.

FIG. 14.—The relation of the right ventricular pressure pulse to the first heart sound and pulmonary early systolic sound in a case of pulmonary hypertension with atrial septal defect (PA pressure 100/45 mm. Hg, PA enlargement grade 3). The extra sound is 0.09 sec. after the onset of the first heart sound and rise of pressure in the right ventricle.

FIG. 15.—The relation of the pulmonary arterial pressure pulse (PA) to the pulmonary early systolic sound in the same case as Fig. 14. The extra sound is 0.05 sec. after the rise of pressure in the pulmonary artery at the opening of the pulmonary valve (estimated delay of 0.03 sec. has been subtracted).

TABLE III

THE RELATION OF THE PULMONARY EARLY SYSTOLIC SOUND TO OTHER EVENTS IN THE CARDIAC CYCLE IN 44 PATIENTS WITH PULMONARY HYPERTENSION

Interval	Average duration in seconds * (range)	No. of patients
Q-RV	0.06 (0.04-0.10)	35
(QRS 0.10 sec. or less)		
Q-LV	0.06 (0.05-0.08)	4
Q-PA	0.11 (0.08-0.16)	34
RV-PA	0.05	34
(RV isometric time)		
Q-Carotid	0.12 (0.10-0.15)	10
Q-Aorta	0.09 (0.08-0.11)	10
(calculated)		
Q-1st sound	0.07 (0.04-0.10)	44
Q-Pulmonary sound	0.14 (0.10-0.19)	44
1st sound-Pulmonary sound	0.07 (0.05-0.14)	44

* The estimated delay in our pressure pulse recording system has been subtracted.

the Q-PA interval averaged 0.11 sec. the extra sound was estimated to be later than the rise of pressure in the pulmonary artery at the opening of the pulmonary valve (Fig. 15) by an average of 0.03 sec.

DISCUSSION

An early systolic sound, usually of high-pitch and clicking quality, heard in the pulmonary area and louder in expiration, strongly suggests dilatation of the pulmonary artery generally accompanied by pulmonary hypertension.

The basal site and relative lateness of the pulmonary sound made it more likely to be associated with the ejection phase than with the earlier major components of the first heart sound which take place during the isometric contraction phase of the ventricles (Wiggers and Dean, 1917) and are thought to be caused by closure of the tricuspid and mitral valves (Dock, 1933). The results of the present investigation confirm that the major components of the first heart sound occurred almost simultaneously with the onset of the rise of pressure in the right ventricle, and the expected time of closure of the tricuspid valve. Approximate synchrony between the major components of the first sound and the onset of rise of pressure in the left ventricle, and the expected time of closure of the mitral valve, could be shown in four patients, and calculated from the carotid tracings in another eight. The added pulmonary sound occurred 0.07 sec. later and was therefore not related to closure of the tricuspid or mitral valve. This confirmed the clinical impression that the pulmonary early systolic sound was not related to splitting of the first heart sound which is generally thought to be due to slight asynchrony of tricuspid and mitral valve closure.

Pressure pulses from the pulmonary artery showed that the pulmonary early systolic sound occurred during ejection about 0.03 sec. after the opening of the pulmonary valve, and a similar timing has been found for the aortic sound in relation to the carotid pulse. This suggests that these ejection sounds are related to sudden distension of the great vessels. Ejection vibrations have been recorded directly by Wiggers and Dean (1917) from the aorta and pulmonary artery of normal dogs, and indirectly by Oriás and Braun-Menéndez (1939) from the base of the heart in normal subjects. These vibrations, inaudible in the normal subject, might become audible if the vessels approximated to the chest wall, if their intensity was augmented, or if the interval separating them from the major components of the first sound was increased. Conditions were present in the group of cases under investigation that might be expected to render these vibrations audible as an extra sound. Thus, dilatation of the pulmonary artery, present in all patients except two, would bring the vibrations nearer to the chest wall and may have been the major factor in the production of an

added sound in those without pulmonary hypertension. In the patients with pulmonary hypertension increased intensity of ejection vibrations may have been caused by the high pressure and increased vascular resistance; prolonged isometric contraction and delayed ejection would be expected to cause greater separation of the major components of the first sound from the ejection vibrations, and there was a longer time interval between the first sound and the pulmonary sound. Conversely, earlier ejection from shortened isometric contraction due to low pulmonary artery pressure would be expected to make an ejection sound too early to be separated from the main components of the first heart sound. This may explain the fact that we have not heard an extra pulmonary sound in any patient with severe pulmonary stenosis and dilatation of the pulmonary artery, and have only once recorded it. We have often noticed in these cases a loud clicking first heart sound at the base which might be ascribed to superimposition of a pulmonary sound on the major components of the first sound.

Since the first heart sound is related to the onset of rise of pressure in the right ventricle, and the pulmonary sound closely follows the opening of the pulmonary valve, the time interval between these two sounds is a clinical indication of the duration of the isometric contraction of the right ventricle.

SUMMARY AND CONCLUSIONS

An early systolic sound in the pulmonary area has been investigated in 50 patients. This sound had usually been mistaken for the second component of a widely split first heart sound.

In 44 of these patients pulmonary hypertension was present, and was associated with enlargement of the pulmonary artery in all except one. Of the 6 patients without pulmonary hypertension five showed enlargement of the pulmonary artery and one was healthy.

The added sound was louder during expiration, and was high-pitched and sharp in character; often it could be described as a pulmonary early systolic click (*claquement protosystolique*). Unlike splitting of the first heart sound it was louder in the second and third left intercostal spaces than in the mitral and tricuspid areas.

The major high-frequency components of the first heart sound were confirmed to occur at the onset of rise of pressure in the right ventricle while the pulmonary sound occurred during ejection, shortly after the opening of the pulmonary valves. The interval between the first sound and the pulmonary sound was thus a measure of the isometric time of the right ventricle and was prolonged in the patients with pulmonary hypertension.

The pulmonary early systolic sound is a valuable clinical sign of enlargement of the pulmonary artery, usually in association with pulmonary hypertension.

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