Splitting of Second Heart Sound in Ventricular Ectopic Beats

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Doubt has frequently been expressed as to whether electrical asynchrony of the ventricles (e.g. right or left bundle-branch block, right or left ventricular ectopics) causes an equivalent mechanical counterpart. Samet *et al.* (1959b), using intra-ventricular pressure records, found evidence of mechanical asynchrony in only 28 per cent of ventricular extrasystoles.

The two components of the second sound produced by closure of the sigmoid valves mark the end of systole in each ventricle. Changes in their usual relationship thus indicate changes in the time relationship of mechanical events of both ventricles. Haber and Leatham (1965), using simultaneous recording of electrocardiogram, phonocardiogram, and carotid arteriogram, found evidence of mechanical asynchrony in all cases of ventricular ectopic beats studied. Though these authors provided us with a sensitive method of detecting changes in the relation of the two components of the second sound, they did not study all the different possibilities under which a ventricular extrasystole may arise, nor did they study a large number of examples.

When right or left ventricular extrasystoles occur during either normal conduction or right or left bundle-branch block, it is possible, based upon previous experimental data (Wiggers, 1925; Bisteni *et al.*, 1957), to predict the effect of 6 different combinations of changes in electrical conduction on the two components of the second sound. These are summarized in Table I. The aim of the present communication is to test how far these theoretical predictions are met in clinical situations.

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SUBJECTS AND METHODS

A total of 99 recordings of ventricular extrasystoles obtained from 25 patients was studied. To be certain of including only true ventricular extrasystoles, those with a QRS duration of less than 0.1 sec. and those that occurred within the P-R interval were rejected (Sodi Pallares, Bisteni, and Medrano, 1955; Bisteni *et al.*, 1957). The ectopics were diagnosed as right or left ventricular in origin by widely accepted criteria (Castex, Battro, and González, 1941; Bisteni *et al.*, 1957). In 10 patients there was normal ventricular conduction, in 9 there was complete right bundle-branch block, and in 6 complete left bundle-branch block. The cause of the extrasystoles was not established, though digitalis toxicity and/or ischaemic or rheumatic heart diseases were the most likely causes in most cases. There was in addition 1 case of cardiomyopathy (non-obstructive), 1 of a femoral arteriovenous fistula (traumatic in origin), 2 of atrial septal defect, 1 of patent ductus arteriosus, and 1 with coarctation of the aorta and aortic regurgitation.

Phonocardiograms, together with an electrocardiographic lead and an external carotid tracing using an

TABLE I

PREDICTION OF TYPE OF SPLITTING OF SECOND HEART SOUND WITH DIFFERENT COMBINATIONS OF BASIC AND EXTRASYSTOLIC ELECTROCARDIOGRAPHIC PATTERN

Basic Pattern		Extrasystolic pattern	
Electrocardiographic pattern	Type of splitting	Electrocardiographic pattern	Type of splitting predicted
Normal	Normal	Comp. LBBB*	Reversed
Normal	Normal	Comp. RBBB†	Wide
Comp. RBBB	Wide	Comp. LBBB*	Reversed
Comp. RBBB	Wide	Comp. RBBB†	Wide
Comp. LBBB	Reversed	Comp. LBBB*	Reversed
Comp. LBBB	Reversed	Comp. RBBB†	Normal

* Extrasystoles from the right ventricle.

† Extrasystoles from the left ventricle.

TABLE II

ELECTROCARDIOGRAPHIC PATTERN AND SPLITTING OF SECOND SOUND DURING BASIC RHYTHM AND PREMATURE BEATS IN 25 PATIENTS

Case No.	Age (yr.), sex, and diagnosis	Basic pattern				Extrasystolic pattern			
		Conduction	QRS	Type of splitting	Interval (sec.)	Conduction	QRS	Type of splitting	Interval (sec.)
1	13, M, PDA	Normal	0.065*	A2-P2	0.060	LBBB	0.105	P2-A2	0.050
		"	"	"	0.055	"	0.120	"	0.065
		"	"	"	0.050	"	0.110	"	0.060
		"	"	"	0.050	"	0.120	"	0.070
		"	"	"	0.050	"	0.120	"	0.070
		"	"	"	0.055	"	0.100	"	0.020
		"	"	"	0.035	"	0.100	"	0.020
		"	"	"	0.050	"	0.130	"	0.040
		"	"	"	0.040	"	0.140	"	0.065
2	49, M, IHD	RBBB (RV-pacing)	0.100	A2-P2	0.090	RBBB (escaped beat)	0.120	A2-P2	0.115
3	51, M, IHD	RBBB (unpaced)	"	"	0.060	LBBB	0.100	"	0.020
		RBBB	0.125	A2-P2	0.055	RBBB	0.130	A2-P2	0.045
		"	0.135	"	0.050	"	0.125	"	0.050
		"	0.130	"	0.050	"	0.130	"	0.050
4	56, M, RHD	Normal	0.080	A2-P2	0.020	RBBB	0.130	A2-P2	0.060
		"	0.075	"	0.025	"	0.150	"	0.050
5	63, M, IHD	LBBB	0.160	P2-A2	0.050	RBBB	0.110	A2-P2	0.090
		"	0.160	"	0.050	RBBB	0.130	A2-P2	0.040
		"	0.140	"	0.040	LBBB	0.160	P2-A2	0.050
		"	0.150	"	0.060	RBBB	0.150	A2-P2	0.100
6	68, F, IHD	RBBB	0.130	A2-P2	0.085	LBBB	0.160	P2-A2	0.090
		"	0.140	"	0.080	"	0.170	"	0.095
7	79, F, IHD	Normal	0.080	A2-P2	0.050	LBBB	0.140	A2/P2	0.000
		"	"	"	0.060	"	0.160	A2/P2	0.000
		"	"	"	0.060	"	0.180	P2-A2	0.020
		"	"	"	0.060	"	0.190	P2-A2	0.020
		"	"	"	0.050	"	0.150	A2/P2	0.000
		"	"	"	0.060	"	0.140	P2-A2	0.010
		"	"	"	0.060	"	0.150	A2-P2	0.010
8	85, M, IHD	Normal	0.070	A2-P2	0.040	LBBB	0.130	A2-P2	0.020
		"	"	"	0.050	"	0.140	"	0.040
		"	"	"	0.050	"	0.150	"	0.030
		"	"	"	0.050	"	0.160	"	0.020
		"	"	"	0.060	"	0.140	"	0.040
		"	"	"	0.070	"	0.130	"	0.030
		"	"	"	0.050	"	0.130	"	0.030
9	56, M, RHD	Normal (LVH)	0.100	P2-A2	0.020	LBBB	0.160	P2-A2	0.040
10	53, M, Myopathy	LBBB	0.165	P2-A2	0.050	LBBB	0.140	A2/P2	0.000
		"	0.150	"	0.040	"	0.125	P2-A2	0.020
		"	0.150	"	0.030	"	0.130	"	0.020
		"	0.155	"	0.040	"	0.130	"	0.015
		"	0.160	"	0.050	"	0.125	"	0.020
		"	0.170	"	0.030	"	0.140	"	0.010
11	32, M, RHD	Normal	0.080	A2-P2	0.020	LBBB	0.140	P2-A2	0.020
		"	"	"	0.035	"	0.130	"	0.090
12	53, M, AVF	Normal (LVH)	0.110	P2-A2	0.050	RBBB	0.200	A2-P2	0.060
		"	"	"	0.045	"	0.170	"	0.050
		"	"	"	0.040	"	0.180	"	0.055
13	60, M, IHD	LBBB	0.140	P2-A2	0.035	RBBB	0.145	A2-P2	0.110
		"	0.140	"	0.035	"	0.145	"	0.100
		"	0.150	"	0.050	"	0.140	"	0.075
		"	0.150	"	0.040	"	0.180	"	0.095
		"	0.150	"	0.035	"	0.180	"	0.090
14	50, M, IHD	RBBB (LVH)	0.180	A2-P2	0.050	RBBB	0.195	A2-P2	0.070
		"	"	"	0.035	"	0.190	"	0.035
		"	"	"	0.050	"	0.180	"	0.040
15	21, M, ASD	RBBB	0.090	A2-P2	0.060	LBBB	0.140	A2-P2	0.025
16	76, F, IHD	RBBB	0.165	A2-P2	0.080	LBBB	0.170	A2-P2	0.020
		"	0.170	"	0.090	"	0.150	"	0.020
		"	0.180	"	0.090	"	0.120	"	0.030
		"	0.175	"	0.110	"	0.140	"	0.025
17	23, M, ASD	RBBB	0.120	A2-P2	0.045	LBBB	0.160	P2-A2	0.050
18	78, F, IHD	RBBB	0.120	A2-P2	0.050	LBBB	0.160	P2-A2	0.040
		"	"	"	"	LBBB	0.120	P2-A2	0.030
		"	"	"	"	RBBB	0.150	A2-P2	0.050
19	47, F, RHD	Normal (RVH)	0.070	A2-P2	0.085	LBBB	0.110	P2-A2	0.020
		"	0.075	"	0.095	"	0.150	A2-P2	0.040
		"	0.065	"	0.095	"	0.155	"	0.040
		"	0.075	"	0.075	RBBB	0.145	"	0.090
		"	0.070	"	0.065	"	0.135	"	0.110
		"	0.070	"	0.065	"	0.120	"	0.115
20	59, M, RHD	Normal	0.065	A2-P2	0.040	RBBB	0.140	A2-P2	0.065
21	27, M, RHD	Normal (LVH)	0.100	P2-A2	0.035	LBBB	0.140	P2-A2	0.060
		"	"	"	0.030	LBBB	0.140	"	0.060
		"	"	"	0.040	RBBB	0.190	"	0.010
		"	"	"	0.030	LBBB	0.170	"	0.065
		"	"	"	0.035	RBBB	0.160	A2-P2	0.030

TABLE II—continued

Case No.	Age (yr.), sex, and diagnosis	Basic pattern				Extrasystolic pattern			
		Conduction	QRS	Type of splitting	Interval (sec.)	Conduction	QRS	Type of splitting	Interval (sec.)
22	45, M, RHD	LBBB	0.140	P2-A2	0.060	LBBB	0.150	P2-A2	0.090
23	12, M, RHD	RBBB	0.090	A2-P2	0.075	RBBB	0.150	A2-P2	0.095
		"	"	"	0.025	"	"	"	0.070
		"	"	"	0.030	"	"	"	0.055
		"	"	"	0.040	"	"	"	0.060
24	36, M, CoAo	LBBB	0.160	P2-A2	0.030	RBBB	0.190	A2-P2	0.060
		"	0.160	"	0.045	"	0.210	"	0.010
		"	0.150	"	0.040	LBBB	0.165	P2-A2	0.015
		"	0.150	"	0.070	"	0.180	"	0.035
		"	0.155	"	0.035	"	0.180	"	0.055
		"	0.155	"	0.045	"	0.180	"	0.040
		"	0.155	"	0.050	"	0.185	"	0.030
		"	0.160	"	0.060	"	0.210	"	0.060
25	47, M, RHD	LBBB	0.130	P2-A2	0.080	LBBB	0.110	P2/A2	0.075
		"	0.130	"	0.090	"	0.110	"	0.000
		"	0.130	"	0.085	"	0.110	P2-A2	0.040
		"	0.125	"	0.070	"	0.110	"	0.050
		"	0.125	"	0.070	"	0.120	"	0.060

IHD, ischaemic heart disease; RHD, rheumatic heart disease; PDA, patent ductus arteriosus; ASD, atrial septal defect; CoAo, coarctation of aorta; AVF, arterio-venous fistula.
Note: LVH and RVH indicate left or right ventricular hypertrophy coinciding with a normal basic conduction pattern and thus explaining an unusually wide QRS.

"Infraton", Boucke-Brecht type pick-up, were recorded at 100 or 50 mm./sec. paper speed on a four-channel Mingograph B-12 recorder. All tracings were obtained in the supine position during normal breathing or in held mid-expiration, with the microphone over the area where both components of the second sound could best be distinguished. The dicrotic notch in the carotid tracing was used to identify the second aortic sound (Duchosal and Moret, 1960; Hartman, 1961). A complete electrocardiogram was obtained at the time of the study in each case, to ensure the proper diagnosis of the basic rhythm and that of the extrasystoles.

RESULTS AND DISCUSSION

The results, summarized in Table II, are presented in two groups, those from extrasystoles originating in the right ventricle and those from extrasystoles originating in the left ventricle. In each group an extrasystole may follow one of three basic ventricular conduction patterns, normal, complete right bundle-branch block, or complete left bundle-branch block (Table I). In each case the splitting of the second sound of the ectopic beat is compared with that of the beat of the basic pattern.

Group I: Right Ventricular Extrasystoles. In the presence of normal ventricular conduction a right ventricular extrasystole causes reversed splitting of the second sound (Fig. 1), while the QRS has the appearance of complete left bundle-branch block. Likewise, when there is complete right bundle-branch block causing wide splitting of the second sound, right ventricular extrasystole again causes reversed splitting of the second heart sound (Fig. 2).

Finally, when the right ventricular extrasystole appears in the presence of a complete left bundle-branch block, the splitting of the second sound that is already reversed is unaltered (Fig. 3). Thus, the changes of the second sound indicate that mechanical asynchronism of the ventricles invariably follows the changes in the pathway of activation.

Previous workers have reported (Samet *et al.*,

FIG. 1.—Basic normal ventricular conduction followed by an extrasystole originating in the right ventricle, showing reversed splitting of the second sound due to delayed aortic component (Case 1). Carot., external carotid pulse; Med., medium frequency range, from 120 c.p.s. to 240 c.p.s.; Log., logarithmic, frequency range from 240 c.p.s. to 500 c.p.s.; Pulm., pulmonary area.

FIG. 2.—Right bundle-branch block pattern followed by an extrasystole originating in the right ventricle. Reversed split of the second sound due to early P2 and delay in A2 (Case 6). Both phonocardiograms were taken at high frequency, range from 500 c.p.s. to 1000 c.p.s.

FIG. 3.—Left bundle-branch block pattern followed by an extrasystole originating in the right ventricle, showing no change in the splitting of the second sound (Case 5). Abbreviations as in Fig. 1.

1959b) that the correlation between electrical and mechanical events is by no means invariable. This discrepancy may be the result of incorrect diagnosis of some ventricular ectopics (Sodi Pallares *et al.*, 1955; Bisteni *et al.*, 1957). Furthermore, when extrasystoles come from different foci they produce a variable degree of asynchrony of ventricular contraction. Extrasystoles that arise from high in the

septum might show a lesser degree of asynchrony than those that originate in the free wall of the ventricle, and variations in the length of the electrical pathway must cause differences in electrical and mechanical delay. This could be observed in some of our cases in which multifocal extrasystoles caused variable degrees of movement of the second sound (Fig. 4).

FIG. 4.—Left bundle-branch block pattern with extrasystoles originating in two different foci in the left ventricle (Case 5). Carotid and phonocardiographic tracings as in Fig. 3. A dotted line unites in every instance A_2 and the dicotic notch of the carotid pulse wave. Notice the wide variation in the splitting of the second sound of the ectopic beats.

Group II: Left Ventricular Extrasystoles. When the ventricular conduction is normal an extrasystole arising from the left ventricle increases the duration of the split (Fig. 5). If there is already complete right bundle-branch block a left ventricular extrasystole does not change the already increased splitting of the second sound (Fig. 6). Finally, when

there is a basic pattern of complete left bundle-branch block, left ventricular extrasystole causes the reversed split of the second sound to become normal (Fig. 7). The degree of movement of the affected sound varies, as might be expected, with the site of origin of the extrasystole.

Our results are consistent with several previous

FIG. 5.—Basic normal ventricular conduction followed by an extrasystole originating in the left ventricle, showing widening of the splitting of the second sound (Case 4). Abbreviations as in Fig. 1.

FIG. 6.—Right bundle-branch block pattern followed by an extrasystole originating in the left ventricle. The splitting of the second sound is unaltered (Case 3). Abbreviations as in Fig. 1.

reports dealing with ventricular asynchrony (Eppinger and Rothberger, 1910a, b; Wolferth, Margolies, and Bellet, 1933; Wolferth and Margolies, 1935; Nichol, 1933; Richards *et al.*, 1947; Ellinger *et al.*, 1948; Coblentz *et al.*, 1949; Haber and Leatham, 1965). However, the subject is controversial, and others have not observed good correlation between

electrical and mechanical events (Katz, Landt, and Bohning, 1935; Schwedel, Samet, and Mednick, 1950; Samet, Mednick, and Schwedel, 1950; Braunwald and Morrow, 1957; Samet, Mednick, and Schwedel, 1959a; Samet *et al.*, 1959b). Most of these latter studies were based upon chronic disturbances, such as complete bundle-branch block

FIG. 7.—Left bundle-branch block pattern followed by an extrasystole originating in the left ventricle. The reversed split becomes normal (Case 5). Abbreviations as in Fig. 1.

or Wolff-Parkinson-White syndrome, where other variables have to be taken into consideration. Bilateral lesions of the bundle branches, ventricular hypertrophy, or altered haemodynamics such as increased volume load or increased pressure load, will tend to alter the mechanical response of the muscle. These variables do not affect our observations since we only look at the "acute" relative changes that occur in the extrasystolic beat when compared with the preceding one.

Another probable reason for the failure of many authors to observe correlation between electrical and mechanical events is the fact that most have studied the mechanical events by looking at the beginning of the ventricular pressure curve. Delayed activation may occur, however, at any time during electrical systole, and the chance of detecting mechanical delay should be greater if we observe the end of the contraction, as in this study. Furthermore, in our experience timing of the onset of contraction of a ventricle from its pressure pulse may be difficult because of transmitted mechanical artefacts from the contraction of the opposite ventricle.

In every instance, the changes in the second sound were those which would have been predicted if mechanical delay had accompanied electrical delay. We have found that changes in electrical sequence always have a counterpart in mechanical systole. Furthermore, when multifocal extrasystoles were present in the same patient, the degree of movement of both components of the second sound varied from one extrasystole to another. This was presumably because differences in the pathway of activation produced different degrees of intraventricular conduction delay and correspondingly different degrees of mechanical delay.

SUMMARY

Patients with ventricular extrasystoles superimposed on different ventricular conduction patterns (normal, right, and left bundle-branch block) were studied with simultaneous electrocardiogram, phonocardiogram, and carotid arteriogram. Changes in the splitting of the second sound were correlated with the site of origin of the extrasystole for each basic pattern of ventricular conduction. Right ventricular extrasystoles delayed the second aortic sound and/or moved the second pulmonary sound towards the first sound. Left ventricular extrasystoles delayed the second pulmonary sound and/or made the second aortic sound appear earlier. With multifocal extrasystoles, the degree of displacement of both second sounds varied from one extrasystole to another, presumably because differences in the pathway of activation produced different

degrees of intraventricular conduction delay and thus different degrees of mechanical delay.

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