— O r i g i n a l —

*From the 3rd Medical Clinic, Kyoto University Hospital, Sakyo-Ku, Kyoto
(Director: Prof. M. Maekawa, M. D.)*

Studies on the Genesis of Late R Waves in Right Chest and Intracardiac Leads

ETSU HASHIDA

(Received for Publication, Aug. 28, 1961)

The significance of late or secondary R waves is studied on ninety subjects with miscellaneous electrocardiographic patterns including the normal by means of intracardiac electrocardiography. In classical right ventricular hypertrophy and complete right bundle branch block, late R waves can be attributed to a delay in activation of the right ventricular free wall. In the other patterns including a part of incomplete right bundle branch block, activation of the left ventricular posterior base constitutes these late R waves.

CONCERNING the origin of the late R waves recorded in the right chest leads and lead aVR, and that of right ventricular hypertrophy pattern of clinical electrocardiogram, there have been many reports and studies on the basis of correlation with autopsy materials and direct-epicardial or intracardiac leads, but any authorized theory has not yet been established.

Goldberger¹⁾ believed that late R waves appeared in lead aVR when clockwise rotation of the heart around its longitudinal axis occurred and then the posterior base of the left ventricle faced the right shoulder, and thus he adopted this pattern in lead aVR as a criterion of clockwise rotation of the heart. With intracardiac leads Kossmann et al²⁾ demonstrated the fact that almost the same pattern and amplitude were obtained both in hypertrophied right ventricular cavity and at the corresponding precordial site, and concluded that electrocardiographic pattern of right ventricular hypertrophy has nothing to do with the thickness of right ventricular free wall.

Moreover, they deduced like Goldberger

that late R waves recorded from the upper right precordium were due to the inversion of the heart electrical field through clockwise rotation of the heart around its longitudinal axis. Later on, they³⁾ analysed precisely the intracardiac electrocardiograms of fourteen normal subjects, and frequently obtained large late R waves just above the pulmonic valve and in the lower right atrium. Because these late R waves (QR, qR etc) abruptly changed to an rS pattern when the catheter was withdrawn from either portion into the right ventricle, they concluded that these late R waves were derived from a certain particular portion near both the tricuspid and the pulmonic valves, and were due to the activation of the crista supraventricularis in the right ventricle, whose depolarization caused right precordial R waves in right ventricular hypertrophy. Walker et al⁴⁾ also agreed with this crista supraventricularis theory by correlating electrocardiographic with hemodynamic data in more than ninety subjects with atrial septal defect, and using the autopsy materials of Kjellberg et al⁵⁾ who discovered hypertrophy of the crista supraventricularis in atrial septal

defect, they concluded that a late R wave in an rsR' pattern was derived from the hypertrophied crista supraventricularis and that as pulmonary artery pressure became greater, ventricular complex in lead VI changed from an rsR' to an rR' or R pattern because the free wall of the right ventricle hypertrophied in conformity with the degree of right ventricular hypertrophy. Sodi-Pallares et al⁶⁾, on the other hand, performed extensively animal experiments with dogs and demonstrated that activation wave of the interventricular septum started from the portion for the left branch of His bundle to ramify in the lower one-third of the left septal surface, penetrated from that area the septum from left to right and then ascends the both septal surfaces. They⁷⁾ further proved that in experimental right bundle branch block an early R wave could be attributed to the propagation of the excitation wave from the left interventricular surface to the "physiologic barrier" which existed between the borders of the right and left septums, and a late R wave to the activation wave which covered this barrier and ascended the right septal surface. Recently Dickens and Goldberg^{8,9)} recorded intracardiac electrocardiograms of fifty subjects, some of whom had complete right bundle branch block, simultaneously with lead V4R and/or standard limb lead II, showed the peaks of intracardiac late R waves synchronous with that of lead V4R and/or the nadir of S wave in lead II, and concluded according to Sodi-Pallares et al that late R waves in the right precordial leads were due to the anomalous activation of the interventricular septum. Also Emslie-Smith¹⁰⁾ observed frequent occurrence of intracardiac late R waves in fifty subjects whose lesions were mainly apt to incur right ventricular hypertrophy, but the origin of these late R waves was not mentioned. Camerini and Davies¹¹⁾, Morris and Whitaker¹²⁾, Rosenmann¹³⁾ and Blount et al¹⁴⁾ mostly used precordial leads and at times recorded intracardiac and/or direct-epicardial leads, and concluded that late R waves recorded from the right precordium were due to a delay in activation of the outflow tract of the right ventricle. With

direct-epicardial leads, formerly Lewis¹⁵⁾ and fairly recently Wilson et al¹⁶⁾ performed animal experiments with dogs and both observed that the excitation wave arrived at the outflow tract of the right and the posterior base of the left ventricle in the last phase of the QRS complex. On the other hand, however, studies of Barbato et al¹⁷⁾ with normal clinical cases showed that an rS pattern was obtained from the outflow tract of the right ventricle and an rsR' pattern appeared mostly in cases with congenital heart anomalies¹⁸⁾. The results of Wasserburger et al¹⁹⁾ are identical with those of Barbato et al. Therefore, the results of Lewis and Wilson et al with animal experiments do not appear to be applied to clinical cases. McGregor²⁰⁾, on the other hand, recorded in cases of Fallot's tetralogy an rS pattern from the right and a qR pattern from the left ventricle with direct-epicardial leads, and thus attributed the genesis of right ventricular hypertrophy pattern to the clockwise rotation of the heart around its longitudinal axis and therefore to the inversion of the heart electrical field like Goldberger and Kossman et al. Latour and Puech²¹⁾ showed that intracardiac late R waves which were often recorded from the pulmonary artery in cases with congenital heart disease were transmitted to the right precordium and thus these late R waves engendered those in lead aVR and right chest leads, and they further pointed out that the peaks of these intracardiac late R waves occurred earlier than those of late R waves in leads V1 and V2 in both cases with right bundle branch block and right ventricular hypertrophy and therefore activation of the free wall of the right ventricle contributes to the height of the R waves in the right precordial leads.

The summary of the foregoing with regard to the origin of these late R waves is as follows:

1. the posterior base of the left ventricle.
2. the free wall of the right ventricle, especially its outflow tract.
3. the crista supraventricularis of the right ventricle.
4. the upper portion of the right ventricle.

5. the outflow tract of the right ventricle and the upper portion of the right inter-ventricular septum.

Furthermore, concerning the genesis of electrocardiographic right ventricular hypertrophy pattern, almost the same mechanism as that causing the above-mentioned late R waves is considered to be operative.

During right heart catheterization of ninety subjects with and/or without heart disease, simultaneous recordings were made of leads V1 and V5, leads from the right intracardiac cavities and their neighboring vessels, and intracardiac pressure curves. We dealt with thirteen cases in which coronary vein catheterization was performed, and forty-six cases in which electrocardiograms were recorded from the right pulmonary capillary, especially cautiously. Thus the origin of late R waves, and that of electrocardiographic right ventricular hypertrophy and right bundle branch block patterns will be discussed. In addition, how the intracardiac late R waves are transmitted to standard limb, unipolar limb and unipolar right chest leads will be elucidated.

MATERIALS AND METHODS

The diagnoses are shown in Table I. There is almost every kind of heart disease, both congenital and acquired, and some of these cases are normal. The real number of subjects is eighty-five, of whom five were repeatedly catheterized before and after operation. As shown in this table, only a few cases with congenital heart disease are included in this series. About half of the cases are occupied by those with mitral valvular lesion, and more than one-third of all the cases have pure or almost pure mitral stenosis.

The diagnoses were decided in all cases by history, physical examination, chest X-ray, electrocardiogram and cardiac catheterization. Criteria of Sokolow and Lyon²²⁾ for ventricular hypertrophy and those of Barker and Valencia²³⁾ for right bundle branch block were employed. Criteria for biventricular hypertrophy are those modified from Sokolow and Lyon's criteria in which each criterion for isolated ventricular hypertrophy is applied to leads V1 and V5, respectively.

The technique, using an electrode-tipped catheter is that of Cournand. Tracings were recorded from the right pulmonary capillary, right

TABLE I INCIDENCE OF LATE INTRACARDIAC R IN 90 CASES

Diagnosis	Number of cases	Number of cases with late intra-cardiac R
Normal	7	6
Mitral stenosis	37	31
Mitral insufficiency (predominant form)	8	7
Constrictive pericarditis	2	2
Aortic valv. disease	10	8
Bronchial asthma	2	2
Pulmonary stenosis (including Fallot's tetralogy)	6	6
Patent ductus arteriosus	6	3
Ventricular septal defect (including Roger's disease)	3	3
Atrial septal defect	1	1
Congenital heart disease not fully diagnosed	3	2
Pulmonary thrombosis	1	1
Essential hypertension	3	2
Myxedema	1	1
Total	90	75

TABLE II INCIDENCE OF LATE R IN VARIOUS INTRACARDIAC SITES

Site	Number of cases	Number of with late intra-cardiac R	%
Great cardiac vein or coronary sinus	13	13	100
Right pulmonary capillary	46	38	82.6
Left pulmonary capillary	6	6	100
Right pulmonary artery	48	33	68.8
Main pulmonary artery	88	62	70.4
Outflow	83	15	18.1
Inflow	85	3	3.5
Low right atrium	75	39	52.0
Superior vena cava	61	44	72.1

and main pulmonary arteries, right ventricular outflow and inflow tracts, right atrium, and superior

vena cava, and furthermore, continuous recordings were made as a rule during slow withdrawal of the catheter-tip from the right pulmonary capillary to the superior vena cava. In the early period of this study, we employed two-element photographic electrocardiograph (manufactured by SAN-YEI MFG. CO., LTD. OSAKA) and recorded simultaneously lead II and intracardiac leads. But recently simultaneous records were made of leads V1, V5, intracardiac leads and intracardiac pressure curves with sixelement photographic electrocardiograph (manufactured by the same maker). In the present study, the expression that peaks of late R waves occur synchronously or simultaneously in several portions will be frequently used. This means for example that the peaks of late R waves in several portions occur synchronously with the nadir of S wave in lead V5. That in some portion late R wave is recorded earlier is indicated by the fact that this late R wave occurs earlier than the nadir of S wave in lead V5.

The morphologies of QRS complexes recorded from the right intracardiac cavities and their neighboring vessels are described, and when late R waves

are recorded, the time-intervals from the onset of QRS in lead V1 or V2 to the peak of that late R wave are measured. Then, following abbreviations are employed: Cor. S., Cor. V., PC, PA, MPA, Outf., Inf., RA, SVC. mean coronary sinus, coronary vein, pulmonary capillary, pulmonary artery, main pulmonary artery, outflow and inflow of the right ventricle, right atrium and superior vena cava, respectively. The "electrocardiographically normal" or simply "normal" cases are those whose electrocardiograms are normal after Sokolow and Lyon's criteria irrespective of the existence of cardiac diseases. Cases of right and/or left ventricular hypertrophy are those in which ventricular hypertrophy is present after the above criteria. Cases of right bundle branch block pattern belong to those by which Barker and Valencia's criteria are satisfied. The underlying diseases, morphologies of QRS complexes, time-lags from the onset of QRS complex in minter-lead, namely lead V1 or V2 to the peak of the late R wave and those from the same onset to the nadir of S wave in lead V5, and intracardiac pressures are respectively shown in Tables III, IV, V, VI, VII and VIII. Time-

TABLE III ELECTROCARDIOGRAPHIC DATA IN 13 PATIENTS WHOSE ELECTROCARDIOGRAMS ARE TAKEN IN THE CORONARY SINUS OR THE PROXIMAL PORTION OF THE GREAT CARDIAC VEIN

Patient	Age	Diag.	Cor. S. or Great Card. V	RPC	RPA	MPA	Outf	Inf	LRA	SVC	V ₁ or CR ₁	V ₅ or CR ₅
S. K.	27	MS	Qr (.062)	Qr (.062)	Qr (.064)	rS	rS	rS	Qr (.064)	Qr (.062)	Rs	qRs (.062)
K. S.	59	NCA	Qr (.053)	qs	qR (.056)	qR (.056)	rS	RS	rS	Qr (.056)	rS	qRs (.056)
H. S.	21	NCA	QR (.058)	r	Qr (.058)	rSr' (.056)	rS	rS	rsR's' (.058)	Qr (.058)	rS	Rs (.058)
S. Y.	41	MS	QR (.064)	Qr (.066)	Qr (.066)	rSr's' (.066)	rsR's'	rS	rSr' (.067)	Qr (.066)	rsR's' (.066)	qRs (.066)
K. K.	24	MS	qR	0	Qr	rSr'	rSr'	rS	rsr's'	0	rSr'	qRs
R. K.	26	MS	qr	qr	0	rS	QS	0	Qr	QS	rS	Rs
G. I.	20	PS	Qr	0	0	rSr'	Rsr'	rS	Qr	0	rS	qRs
G. N.	60	AI	qR	qr	0	rSr'	rS	rsR'S'	Qr	QS	rS	qRs
H. M.	21	NCA	qR	0	0	rsR'S'	rS	rS	Rs	0	rS	qRs
K. O.	42	MS	qR	0	0	rsr's'	rS	rS	QS	0	rS	Rs
F. Y.	35	MS	qR	0	0	rS	rS	rS	rS	0	rS	qRs
H. N.	19	AI	QS	0	0	RsR's'	Rs	Rs	Qr	Qr	rS	qRs
H. Y.	41	MYX	qr	0	0	rSr'	rS	rS	qR	0	rS	qRs

Abbreviations: Cor. S., coronary sinus; Great card. v., great cardiac vein; RPC, right pulmonary capillary; RPA, right pulmonary artery; MPA, main pulmonary artery; Outf. and Inf., outflow and Inflow of the right ventricle, respectively; LRA, low right atrium; SVC, superior vena cava; MS, mitral stenosis; AI, aortic insufficiency; PS, pulmonary stenosis; NCA, neurocirculatory asthenia; MYX, myxedema.

Note: Figures in the bracket indicate the time interval in seconds of the peak of late R or the nadir of s in V₅ from the onset of the QRS complex of V₁ or V₂.

TABLE IV ELECTROCARDIOGRAPHIC AND HEMODYNAMIC DATA IN 39 PATIENTS WITH NORMAL ELECTROCARDIOGRAM

Patient	Age	Diag.	RPC	RPA	MPA	Outf	Inf	LRA	SVC	V ₁ or CR ₁	V ₅ or CR ₅	RVP (mmHg)
R. K.	26	MS	qr	0	rS	QS	0	Qr	QS	rS	Rs	32/2
T. Y.	27	VSD	low volt.	0	qR	rS	rS	qrs	qr	rS	Rs	28/3
R. F.	17	MS	qrs (qR)	0	0	rS	0	QS	Qrs	rS	Rs	27/0
M. M.	23	BA	qrs (qs)	0	rSr'	rS	rS	qRs	QR	rS	Rs	28/-2
K. N.	42	MS	0	0	Rsr's'	RS	rS	rS	0	rS	qRs	48/1
T. K.	39	MS	0	0	rs	rS	rS	0	0	rS	Rs	65/-2
H. T.	25	CP	0	0	rS	rS	rS	qrs	0	rS	Rs	56/8
H. Y.	41	MYX	0	0	rSr'	rS	rS	qR	0	rS	qRs	21/1
G. I.	20	PS	0	0	rSr'	Rsr'	rS	Qr	0	rS	qRs	66/-6
Y. S.	35	MS	0	0	rsr'S'	rS	rS	rS	qr	rS	Rs	20/0
I. F.	51	CP	0	0	rS	rS	rS	Qrs	0	rS	Rs	50/9
T. I.	39	AI	0	0	Qrs	RS	RS	qR	0	rS	Rs	48/-3
H. M.	21	NCA	0	0	rSR'S'	rS	rS	Rs	0	rS	qRs	25/-3
H. T.	21	PDA	0	0	rsr'S'	rsr'S'	rS	rS	0	rS	Rs	31/0
T. F.	44	NCA	0	0	rsr'S'	rS	rS	rS	0	rS	qRs	35/-5
K. O.	42	MS	0	0	rsR'S'	rS	rS	QS	0	rS	Rs	54/6
T. O.	15	BA	qr	rS	RS	rS	RS	Qr	QS	rS	qRs	24/-2
S. K.	31	NCA	0	Qr	rSr's'	rS	rS	Qr (.053)	Qr	rS	qRs (.053)	25/-3
H. T.	21	PDA	qr (.051)	QS	rS	rS	rS	rSr' (.055)	rSr' (.055)	rS	qRs (.055)	23/0
F. Y.	35	MS	0	0	rS	rS	rS	rS	0	rS	pRs	48/-5
H. S.	21	NCA	r	Qr (.058)	rSr' (.056)	rS	rS	rsR's' (.058)	Qr (.058)	rS	Rs (.058)	27/-2
K. O.	56	MS	0	QS	rS	rS	rS	0	0	rS	qRs	29/-3
T. I.	29	MI	rSr' (.075) (qR) (.058)	Qr (.075) (Qr) (.058)	rSR's'	rS	rS	rS	rSr' (.075)	RS	qRs	46/5
K. S.	59	NCA	qr	qR (.056)	qR (.056)	rS	RS	rS	Qr (.056)	rS	qRs (.056)	25/-3
E. M.	33	MS	QS	0	rS	rS	rS	QS	QS	rS	Rs	56/-4
A. F.	34	MS	Qr (.073)	rSr' (.073)	rsr' (.080)	rsR's' (.057)	rS	rS	Qr (.073)	rS	qRs (.073)	41/0
K. T.	16	MS	qr (.040)	Qrs (.056)	rSr's' (.058)	rSR'S' (.058)	rS	QR (.056)	Qr (.056)	rS	qRs (.058)	32/-2
K. T.	17	MI	qr	Qr (.071)	rS	rS	rS	QS	QS	rS	R	30/-2
S. O.	24	MS	QS	Qr (.071)	rSr' (.071)	rS	rS	rS	Qr (.071)	rS	Rs (.071)	29/-3
K. K.	31	MS	rsr's' (.058) (qr) (.058)	Qr (.062)	Qr (.058)	rS	rS	Qrs (.060)	Qr (.058)	rS	Rs (.060)	57/-3

T. T.	48	MI	Qr (.060)	Qr (.060)	QR (.060)	rS	rS	Qr (.060)	Qr (.060)	rS	Rs (.060)	38/-1
Y. K.	28	MS	qr (.056)	Qr (.056)	rSr' (.056)	rS	0	0	qr	r	Rs (.056)	44/-3
M. N.	17	MS	rSr' (.069)	rSr' (.069)	rSr's' (.069)	rS	rS	rS	rSr'	RS	qRs	76/-7
M. N.	17	MS	Qr (.071)	Qr (.071)	rSR's' (.071)	0	0	rSr' (.071)	Qr (.071)	RS	qRs (.071)	60/-7
K. I.	22	NCA	0	Qr (.056)	rSR' (.056)	rS	rS	Qrs (.056)	Qr (.056)	rS	Rs (.056)	33/3
S. T.	46	NCA	qr (.056)	rSr' (.056)	rSR' (.058)	rS	rS	QRs (.058)	Qr (.056)	rS	qRs (.056)	20/-2
T. K.	23	MS	rsr' (.055)	Qrs (.054)	Sr's' (.054)	rS	0	Qrs (.054)	rsr's' (.054)	rS	qRs (.056)	68/-2
K. K.	31	MS	Qr (.053)	Qr (.055)	Qr (.056)	rSr' (.087)	QS	0	Qr (.055)	rS	Rs (.058)	48/-2
T. O.	51	MS	0	0	RsR's'	rS	rS	0	Qr	rS	Rs	55/1

Abbreviations which appear here for the first time: RVP, right ventricular pressure (systolic/diastolic); MI, mitral insufficiency; PDA, patent ductus arteriosus; BS, bronchial asthma; CP, constrictive pericarditis; VSD, ventricular septal defect.

These and previous abbreviations will be used in the following tables together with other abbreviations.: EH, essential hypertension; ASD, atrial septal defect; CHD, congenital heart disease not fully diagnosed; PT, pulmonary thrombosis; FT, Fallot's tetralogy; AS, aortic stenosis.

Note: Letters in the bracket of RPC recording indicate ventricular complex pattern of LPC in the same patient.

TABLE V ELECTROCARDIOGRAPHIC AND HEMODYNAMIC DATA IN 17 PATIENTS WITH ELECTROCARDIOGRAM INDICATIVE OF LEFT VENTRICULAR HYPERTROPHY

Patient	Age	Diag.	RPC	RPA	MPA	Outf	Inf	LRA	SVC	V ₁ or CR ₁	V ₅ or CR ₅	RVP (mmHg)
M. S.	25	AI	rsr'	0	Qr	rS	rS	Qr	Qr	rS	qRs	20/4
G. N.	60	AI	qr	0	rSr'	rS	rsR'S'	Qr	QS	rS	qRs	35/3
T. H.	20	AI	qr	0	rS	rS	rS	0	0	rS	qR	29/1
M. S.	48	EH MI	0	0	rS	QS	rS	0	0	rS	Rs	79/2
Y. N.	31	MI	0	0	rS	rS	rS	0	0	rS	qR	35/-1
A. S.	43	AI	0	0	rS	rS	rS	QS	Qr	rS	qRs	73/-4
H. N.	19	AI	0	0	RsR's'	RS	Rs	Qr	Qr	rS	qRs	21/-2
H. H.	45	EH VSD	0	0	rsr'S'	rS	rsR'S'	QS	0	rS	Rs	38/-4
S. S.	24	MI	0	0	rsR'S'	rS	RS	0	0	rS	Rs	43/4
T. H.	28	AS	0	0	rsr'	rSr's'	rS	0	0	rS	qRs	38/2
S. O.	57	EH MS	qr	QS	Qr	rS	rS	QS	0	QS	Rs	50/-4
T. H.	29	AS	0	rS	0	rS	rS	QS	0	rS	qR	29/-2
S. K.	58	AI	0	QS	rS	RS	rs	Qr	QS	rS	qRs	53/4
K. T.	33	MI	Qr (.062)	Qr (.063)	Qr (.063)	rS	rS	QR (.063)	Qr (.063)	rS	qRs (.062)	44/-1
K. S.	58	AI	Qr (.081)	QS	rS	rS	rS	rS	Qr (.080)	rS	qRs (.080)	46/2
K. T.	21	MI	Qr (.058)	Qr (.060)	rsR' (.060)	rS	rS	rS	Qr (.058)	rs	Rs (.060)	36/3
T. M.	17	CHD	Qr (.056)	Qr (.056)	Qrs (.056)	QRs (.056)	QS	Qr (.056)	QS	rS	R	50/-4

TABLE VI ELECTROCARDIOGRAPHIC AND HEMODYNAMIC DATA IN 13 PATIENTS WITH ELECTROCARDIOGRAM INDICATIVE OF RIGHT VENTRICULAR HYPERTROPHY

Patient	Age	Diag.	RPC	RPA	MPA	Outf	Inf	LRA	SVC	V ₁ or CR ₁	V ₅ or CR ₅	RVP (mmHg)
K. H.	39	MS	qr	0	QS	rS	rS	Qr	QS	R	qRs	37/3
K. I.	33	MS	0	0	qrs	rS	rS	QR	Qr	Rs	RS	32/0
S. K.	40	MS	0	0	rS	rS	QS	0	QS	Rs	Rs	72/-16
Y. O.	38	MS	0	0	rsR'S'	RS	rS	QS	0	Rs	Rs	61/2
T. S.	29	MS	0	0	rsR'	rsr'S'	0	0	0	Rs	Rs	38/1
T. U.	22	MS	0	rS	rS	rS	rS	QS	QS	Rs	RS	83/1
T. N.	15	FT	0	0	rsr'S'	rsR'S'	0	rS	rSr'	R	RS	137/-9
J. K.	11	FT	0	0	qRS	rS	rS	qRs	qR	R	rS	109/-2
T. T.	25	PT	r	qr	rsr's'	rsR's'	rS	qR	qr	Rs	Rs	54/-3
S. M.	12	FT	qR	Qr	rS	rS	rS	Qr	Qr	Rs	rS	76/3
H. T.	8	FT	0	Rs	rSr'	0	0	0	R	R	rS	125/-4
S. K.	27	MS	QR (.055)	Qr (.054)	rS	rS	rS	Qr (.054)	Qr (.054)	Rs	qRs (.056)	89/-4
N. Y.	15	PS	0	0	Rs	rS	rS	Qr	0	Rs	RS	20/3

TABLE VII ELECTROCARDIOGRAPHIC AND HEMODYNAMIC DATA IN 5 PATIENTS WITH ELECTROCARDIOGRAM INDICATIVE OF BIVENTRICULAR HYPERTROPHY

Patient	Age	Diag.	RPC	RPA	MPA	Outf	Inf	LRA	SVC	V ₁ or CR ₁	V ₅ or CR ₅	RVP (mmHg)
S. Y.	22	VSD PS	0	rS	rS	rS	rS	rS	Qr (.074)	Rs	qRs (.071)	165/-24
K. H.	15	PDA	rS	QS	rS	0	rS	rS	QS	RS	qR	33/-5
Y. A.	18	PDA	(QR)	(QR) (.064)	rS	RS	rS	rS	rS	RS	qRs (.080)	88/-1
Y. O.	17	PDA	0	0	RS	RS	rS	rS	0	RS	qR	46/-3
Y. O.	17	PDA	rs (qR)	QS	QS	rS	rS	rS	QS	RS	qR	14/-3

measurements are performed in about the half cases in which unipolar right chest leads are employed as monitor-lead and alternate current waves do not mix into the recordings. The peaks of R waves and nadir of S waves can be clearly identified even when photographic development is light or alternate current is mixed. For this reason the expressions such as the peak of R wave or the nadir of S wave are used in this article.

RESULTS

1. Electrocardiograms recorded from the great cardiac vein and coronary sinus: Normal electrocardiograms were recorded in seven cases irrespective of underlying disease. Right bundle branch block, right and left ventri-

cular hypertrophy patterns were obtained in two cases, respectively. The total number is thirteen. The patterns recorded from the extremity of the great cardiac vein are an rS or a qrS pattern. Withdrawal of the catheter these patterns shift to a Qrs or a QS pattern, in the coronary sinus a QR pattern, and ultimately in the right atrium an rSr', an rS or a Qr pattern. The peaks of these late R waves of a Qr or a QR pattern recorded from the coronary sinus or the portion of the great cardiac vein near the coronary sinus occur simultaneously with those of the late R waves recorded from the superior vena cava, pulmonary artery, right atrium and lead aVR

TABLE VIII ELECTROCARDIOGRAPHIC AND HEMODYNAMIC DATA IN 16 PATIENTS WITH ELECTROCARDIOGRAM INDICATIVE OF SO-CALLED RIGHT BUNDLE BRANCH BLOCK

Patient	Age	Diag.	RPC	RPA	MPA	Outf	Inf	LRA	SVC	V ₁ or CR ₁	V ₅ or CR ₅	QRS Intenal	RVP (mmHg)
K. Y.	32	MS	0	0	rS	rS	rS	R	0	rR' (.076)	qRs (.054)	.107	62/-3
R. K.	38	MS	0	0	rsr's' (.053)	rS	rS	QS	Qr (.056)	RSr's' (.056)	Rs (.050)	.075	57/-1
K. K.	24	MS	0	Qr	rSr'	rSr'	rS	rsr's'	0	rSr'	qRs	.085	36/6
H. N.	30	ASD	qr (.071)	Qrsr' (.071)	qrsr's' (.069)	rS	rsr's'	qrsr' (.071)	qr	rsR' (.075)	rS (.069)	.110	44/-8
T. K.	49	MS	qr (.058)	Qr (.058)	qr (.048)	rS	QS	QS	qr (.058)	qr	rS (.058)	.084	78/6
S. Y.	41	MS	Qr (.066)	Qr (.066)	rsr's' (.066)	rsR's'	rS	rSr' (.067)	Qr (.066)	rsR's' (.066)	qRs (.066)	.093	70/-7
H. A.	22	MI	Qr (.078)	rSr' (.078)	rSr' (.078)	rS	rS	rsr's' (.076)	rSr' (.078)	rSr' (.078)	qRs (.078)	.100	59/-5
Y. N.	39	CHD	0	QS	rS	rS	rS	QR (.077)	Qr (.077)	rsR' (.077)	Rs (.077)	.100	129/-2
K. T.	28	MS	qR (.080)	rsr's' (.062)	rsr's' (.062)	rS	0	qR (.080)	rsr's' (.062)	rsR' (.080)	RS	.105	71/7
T. S.	28	MS	qrs (.056)	0	rsr's' (.058)	rsr's' (.053)	RS	Rs	qr (.056)	rsR' (.093)	qRs (.058)	.136	52/2
H. O.	32	CHD	0	0	rsr's' (.079)	rsR's' (.082)	Rs	qrs (.066)	0	rsR' (.079)	qRs (.066)	.134	59/-1
S. Y.	23	MS	Qr (.064)	Qr (.064)	rsr's' (.063)	rS	0	QS	Qr (.064)	rsr's' (.062)	Rs (.064)	.078	43/-8
T. T.	51	MS	qr (.053)	rsr's' (.053)	rsr's' (.054)	rS	rS	rS	rsr's' (.053)	rsr's' (.054)	qRs (.056)	.075	27/-1
K. S.	49	MS	rs	Qr (.049)	Qr (.049)	rS	rS	0	Qr (.050)	rsr's' (.050)	Rs (.049)	.077	30/-3
K. K.	33	MS	qr	Qr (.053)	Qr (.047)	rS	rS	Qr	Qr	rsR' (.082)	qRs (.053)	.120	24/4
K. S.	14	VSD	rsr's'	QS	QS	rS	rS	Qr (.093)	Qr (.082)	rsR' (.093)	qRs (.060)	.127	39/-4

Note. In cases of plateau-formation of the peak of late R, time-measurment are not performed.

Fig. 1. Excerpts from a continuous tracing during withdrawal of the catheter from the great cardiac vein into the right atrium. Note that tracings from the great cardiac vein or the coronary sinus show distinctly the left atrial intrinsicoid deflection, while tracings from the right atrium show the right atrial intrinsicoid deflection and that the peaks of late R of the tracings from both coronary sinus and right atrium occur all simultaneously with the returning portion of descending limb of the monitor lead II to the base line. Lead II (above) and leads from the coronary venous system (below). A, B, Patient H. Y. with myxedema. C, D, E, Patient R. K. with mitral stenosis. Neither of both patients shows abnormalities of the form of QRS complex. Time lines=0.05 sec.; paper speed=40 mm./sec.

with the exception that in these intravascular regions patterns such as a QrS or Qrs are rarely recorded. This is shown in Fig. 1.

2. Electrocardiograms recorded from the pulmonary capillary, especially from the right pulmonary capillary: In forty-six cases, electrocardiograms were recorded from the right pulmonary capillary, in six cases from the left pulmonary capillary and in five cases from the both pulmonary capillaries. Right pulmonary capillary recordings were mostly taken in the lower lobe just above the diaphragm several centimeters from the cardiac silhouette. Left pulmonary capillary recordings were also taken in the lower lobe and were frequently concealed by the cardiac silhouette. The depth of the catheter-tip was not measured but was fairly great because almost all recordings had left atrial intrinsicoid deflection in the atrial complex. Thirty-eight of these forty-six cases with right pulmonary capillary recordings and all cases with

left pulmonary capillary recordings have late R waves. Irrespective of the location of the catheter-tip and/or the degree of ventricular hypertrophy, a QRS complex of a Qr a qr pattern was most frequently obtained. An rsr' or an rSr' pattern was also frequently observed but their occurrences were relatively small. The peaks of these late R waves were all simultaneous with those recorded from the pulmonary artery and right atrium. Furthermore these late R waves occurred simultaneously with the nadir of S wave in lead V5. When there were observed early R waves, their peaks occurred simultaneously with those recorded from the right ventricle or pulmonary artery. Electrocardiographic patterns and their time-relationships were the same in both the right pulmonary capillary and right pulmonary artery, but during withdrawal of the catheter from the former to the latter position, the amplitude increased in height progressively, which was chiefly due to the growth in S wave in the same lead (Fig. 2). The case whose electrocardiogram was recorded from both pulmonary arteries and capillaries (Case T. I. of Fig. 3) had a qR pattern in the left pulmonary capillary. During withdrawal of the catheter from the left pulmonary capillary, the increase in the depth of a q wave and the decrease in the height of an r wave were observed, and ultimately a Qr pattern appeared in the left pulmonary artery. The time-lags from the onset of lead V2 to the peaks of these late R waves recorded from the left pulmonary capillary and artery were both 0.058 sec.. On the contrary, the late R waves recorded from the right pulmonary capillary whose morphologies were an rSr' type and those recorded from the right pulmonary artery, right atrium and superior vena cava whose patterns were a Qr type occurred all synchronously (0.075 sec.). The late R waves recorded from these sites occurred later than those from the left pulmonary capillary and artery. In this case recording simultaneous with lead V5 was not made, but the peak of late R wave in the pulmonary capillary occurred synchronously with R wave in lead V5. For this reason, it seemed that

Fig. 2. Excerpts from a continuous tracing during withdrawal of the catheter from the RPC to the MPA. In each recoring, from above downward intracavity lead, lead V2, lead V5 and pressure except for the MPA tracing where are shown from above to below intracavity lead, lead V2, pressure, lead V5 and respiration. A, B, C, Patient K. T. with mitral stenosis and normal ventricular complex. D, E, F, Patient S. Y. with mitral stenosis and RSR' pattern at VI. Time lines=0.05 sec.; paper speed=40 mm./sec.

electrocardiograms recorded from the left pulmonary capillary were taken from the lateral surface rather than from the posterior surface of the left ventricle. In the case whose electrocardiograms were recorded from the right pulmonary capillary and artery, and left pulmonary capillary. (Case K. K. of Fig. 3), the peaks of late R waves recorded from the both pulmonary capillaries, right and main pulmonary arteries, and right atrium occurred simultaneously with the nadir of S wave in lead V5. In short, atrial complexes recorded from the either side of both pulmonary capillaries exhibit left atrial intrinsicoid deflection in most cases, and late R waves were also frequently recorded from these sites. The peaks of these late R waves occurred simul-

taneously with those recorded from the other sites of the intracardiac cavities and the nadir of S wave in lead V5. These time-relationships are shown in Tables IV, V, VI, VII, VIII.

3. Electrocardiograms recorded from the right and main pulmonary arteries: In thirty-two of the thirty-eight electrocardiographic normal cases, early R waves were recorded from the main pulmonary artery, and twenty-eight of these cases had late R waves. An rS or an rSr' pattern was most frequently observed, and their occurrences were eight and seven, respectively. An rsr'S' and an rSr's' pattern were obtained fairly frequently in four cases, respectively. An RS, a QR and an rSR' pattern were rarely observed. The heights of these late R waves were in general small and in only seven cases the amplitude surpassed that of the negative wave in the same lead. The electrocardiographic morphologies recorded from the right pulmonary artery were almost identical with those from the main pulmonary artery, but the occurrence of these late R waves was much greater in the right than in the main pulmonary artery. In fifteen of nineteen cases with recordings from the right pulmonary artery, a late R wave was obtained whose morphology was most frequently a Qr pattern. The tendency to the disappearance of an early R wave was generally observed. In only five cases, an early R wave was observed. Instead, the depth of a Q wave increased and a Qr pattern was usually observed in these cases.

Electrocardiograms of the cases exhibiting left ventricular hypertrophy recorded from the main pulmonary artery. An rS and a Qr pattern were observed in four and three cases, respectively. In the other ten cases, at any rate, there were observed late R waves. The amplitudes of these late R waves were in general smaller than the depth of the negative waves in the same lead. There was an early R wave in thirteen cases.

Electrocardiograms of the cases exhibiting right ventricular hypertrophy recorded from

Fig. 3. Ventricular complex patterns from both the right and the left PC. A, B, Patient K. K. with mitral stenosis and normal ventricular complex. From above downward intracavitary lead, lead V2 lead V5 and pressure. In the tracing from the LPC an artefact is found before the second atrial complex. Note that the peak of the late R wave in both positions are synchronous with the nadir of the s wave of lead V5. C, D, Patient T. M. with mitral insufficiency and normal ventricular complex. From above downward intracavitary lead, lead V2 and pressure. In this case the peak of the late R wave in RPC occurs simultaneously with the peak of the late R waves in RPA, RA and SVC, while the peak of the late R wave in LPC is synchronous with the peak of lead V5 (not simultaneously recorded). Time lines=0.05 sec.; paper speed=40 mm./ec.

the main pulmonary artery. Early R waves were recorded in ten cases in which an rS pattern was most frequently obtained. Late R waves were observed in seven cases. As regards the amplitudes of these late R waves, there was no uniform tendency.

Electrocardiograms of the cases exhibiting biventricular hypertrophy recorded from the main pulmonary artery. Late R waves were not recorded in any case. An rS, an RS and a QS pattern were obtained in three, one and one cases, respectively.

Electrocardiograms of the cases exhibiting right bundle branch block. Late R waves were observed in thirteen cases. Early R waves were obtained in eleven cases.

In summary, in electrocardiographically normal cases, early R waves generally disappeared in the right pulmonary artery and the depth of a Q wave increased. A Qr pattern was most often obtained. Aside from the cases with right ventricular hypertrophy and right bundle branch block with QRS interval over 0.10 sec., the peaks of late R waves occurred simultaneously with those recorded from the other sites of intracardiac cavities and the nadir of S wave in lead V5. These relations were shown in Tables IV, V, VI, VII, VIII and Fig. 2.

4. Electrocardiograms recorded from the outflow and inflow tracts of the right ventricle: Electrocardiograms of the cases exhibiting normal electrocardiogram recorded from the outflow tract of the right ventricle. Late R waves were observed in five of thirty-eight cases. Their heights were almost the same as the depths of the corresponding S or S' wave. In two cases, heights of these late R waves surpassed the depths of S waves in the same lead. An rS, an RS and a QS pattern were observed in thirty, three and one cases, respectively.

Electrocardiograms of the cases exhibiting normal electrocardiogram recorded from the inflow tract of the right ventricle. An rS, an RS and a QS pattern were recorded in thirty, three and one cases, respectively. No late R wave was recorded in any case.

Electrocardiograms of the cases exhibiting left ventricular hypertrophy recorded from the outflow tract of the right ventricle. An rS, a QS and a QRs pattern were recorded in eleven, one and one cases, respectively. Late R waves were observed in three cases. There were early R waves in fourteen cases. From the inflow tract of the right ventricle, an rS, a QS and an RS pattern were observed in eleven, one and two of seventeen cases, respectively. Late and early R waves were each obtained in two and five cases.

Electrocardiograms of the cases exhibiting right ventricular hypertrophy recorded from the outflow and inflow tracts of the right ventricle. From the outflow tract, an rS and an RS pattern were observed in seven and one of these twelve cases, respectively. Late R waves were observed in three and early R waves in all the cases. From the inflow tract, an rS and a QS pattern were recorded in nine and one of ten cases, respectively. Early R waves were observed in nine cases, but late R waves were not recorded in any case.

Electrocardiograms of the cases exhibiting biventricular hypertrophy recorded from the right ventricle. From the outflow tract, all five cases showed an rS pattern. Late R waves were not recorded from either site.

Electrocardiograms of the cases exhibiting right bundle branch block recorded from the right ventricle. From the outflow tract, an rS pattern was recorded in twelve of sixteen cases. Early R waves were observed in all cases, and late R waves in four cases. From the inflow tract, an rS, an RS and a QS pattern were recorded in ten, two and one of fourteen cases, respectively. There was a late R wave in only one case.

In summary, irrespective of the location of the catheter-tip in the right ventricle, outflow or inflow tract, and whether electrocardiographic patterns were normal or not, late R waves were relatively rarely observed. As shown in Tables V, VI, VII, VIII, there were much or less deep S' or S'' wave following the late R wave in many cases and these late R waves seemed to occur earlier than those

recorded from another locations concerning time-relationships.

5. Electrocardiograms recorded from the right atrium: In thirty-four cases exhibiting normal electrocardiograms, an rS pattern was recorded in nine, and a QS, a Qr and a Qrs pattern in each four cases, respectively. Early R waves were observed in thirteen and late R waves in eighteen cases, respectively.

In twelve cases exhibiting left ventricular hypertrophy, a Qr, a QS, an rS and a QR pattern were recorded in five, four, three and one cases, respectively.

In nine cases exhibiting right ventricular hypertrophy, a Qr and a QS pattern, late and early R waves were observed in three, two, six and one cases, respectively.

Fig. 4. Intracavitary tracings (above) in the RA (left) and the SVC (right), respectively. From above downward intracavitary tracing, lead V2, lead V5, pressure and respiration, the last one or two of which are not taken in some recordings. A, B, Patient H. A. with mitral insufficiency and RSR' pattern at VI. C, D, Patient M. N. with mitral stenosis and normal QRS complex. Note that in both cases the peaks of the late intracardiac R are simultaneous with the nadir of the s of lead V5. In case H. A. the peak of the late R at VI (not recorded simultaneously) occurs synchronously with the peaks of the intracavitary late R waves. Time lines=0.05 sec.; paper speed=40 mm./sec.

In five cases exhibiting biventricular hypertrophy, all showed an rS pattern.

In fifteen cases exhibiting right bundle branch block, a QS and a Qr pattern were recorded in each three, and early and late R waves in nine and five cases, respectively.

In summary, the peaks of all the late R waves in cases other than those exhibiting right bundle branch block whose QRS interval was more than 0.10 sec. and classical right ventricular hypertrophy occur simultaneously with those of the other intracardiac leads and the nadir of S wave in lead V5 as shown in Tables V, VI, VII, VIII and in Fig. 4.

6. Electrocardiograms recorded from the superior vena cava: In twenty-four normal cases, a Qr and a QS pattern were recorded in eleven and four cases, an rSr' and a qr pattern in each three cases, respectively. Late and early R waves were obtained in eighteen and four cases, respectively.

In nine cases exhibiting left ventricular hypertrophy, a Qr and a QS pattern were recorded in six and three cases, respectively.

In eleven cases exhibiting right ventricular hypertrophy, a QS and a Qr pattern were recorded in four and three cases, and late and early R waves in six and two cases, respectively.

In four cases exhibiting biventricular hypertrophy, a QS, a Qr and an rs pattern were recorded in two, one and one cases, respectively.

In thirteen cases exhibiting right bundle branch block, a Qr and a qr pattern were recorded in seven and three cases, respectively. All cases exhibited late R waves, but early R waves were observed in only three cases.

In summary, the peaks of the late R waves recorded from the superior vena cava occur simultaneously with the nadir of S wave in lead V5 and those of late R waves in the other intracardiac leads with the exception of the cases of right ventricular hypertrophy and a part of the cases of right bundle branch block as shown in Tables V, VI, VII, VIII and in Fig. 4.

The résumé of the foregoing results is as

follows: In all cases, irrespective of the underlying diseases and/or electrocardiographic abnormalities, early R waves occur synchronously in any site of the intracardiac cavities and late R waves appear very frequently in any group, electrocardiographically normal and abnormal (Table I), and these late R waves are registered in all locations of the right ventricle other than the outflow and inflow tracts (Table II). The peaks of these late R waves occur simultaneously with the nadir of S wave in lead V5 and the same point of lead II or the returning point of R wave to the base line in the absence of S wave when monitor-lead is lead II with the exception of the cases of right bundle branch block whose QRS interval is over 0.10 sec., and those with classical right ventricular hypertrophy.

DISCUSSION

1. Whether cardiac electromotive force can be represented as a fixed single dipole or not has been a controversial problem since Einthoven. Schmitt et al²⁴⁾²⁵⁾²⁶⁾ used normal seventeen subjects and thirty-seven cardiac patients as experimental materials, and connected through Wilson central terminal arbitrary two points over the body surface symmetric to the heart in about one hundred couplings. In ninety per cent of the cases, they obtained good cancellation of potential at every point over the body surface including the precordial area, and thus concluded that cardiac electromotive force could be ascribed to a fixed single dipole. With four-electrode cancellation method, Frank²⁷⁾ demonstrated that cardiac electromotive force can be attributed to a fixed single dipole. Further he²⁸⁾ used torso models of normal subjects, made image surface and conversely calculated potentials of the fixed single dipole by measuring potentials over the image surface. Thus he showed the close resemblance of potential variations between over the image surface and over the actual body surface in each corresponding two points. He²⁹⁾ proved that as much as ninety-five per cent of potential variations over the body surface was contributed to by those due to such a fixed single

dipole. If this force can be represented as a fixed single dipole in this manner, the terminal vector of normal cardiac forces must be derived from excitation of the posterior basal portion of the left ventricle as indicated by Barbato et al¹⁷⁾. It is this vector that produces late R waves in the coronary sinus, right atrium, pulmonary artery, superior vena cava and others, and a S wave in lead V5, not the vector derived from activation of the outflow tract of the right ventricle. Except for cases with extreme right ventricular hypertrophy and right bundle branch block whose QRS interval is over 0.10 sec., the peaks of late R waves in coronary sinus, right atrium, pulmonary artery, superior vena cava etc. occur all simultaneously with the nadir of S wave in lead V5 though there are small time differences of 0.01 sec. to 0.02 sec. as shown in Tables III, IV, V, VII, VIII. Though we did not perform cancellation method (mirror pattern study) of Schmitt et al, it is clear that both the above-mentioned intracardiac late R waves and a S wave in lead V5 exhibit good cancellation as indicated by the many reproduced multilead electrocardiograms and measured time-relationships. With the exception of the foregoing two instances, this late R wave may be thought to be due to the vector derived from activation of the posterior basal region of the left ventricle.

2. Both the proximal extremity of the great cardiac vein and the coronary sinus lie near the left atrium and ventricle (left cardiac cavities)³⁰⁾, and the main trunk of the pulmonary artery, right atrium and superior vena cava are all in the position to peep into both ventricular cavities. The above-mentioned locations have all relatively small solid angles extended by the surface of the free walls of both ventricles. When they face the free wall of the right ventricle, pulmonary conus contributes to the solid angle, and when they face that of the left ventricle, its posterior basal region plays a chief rôle. The results of Macleod et al³¹⁾ and Barbato et al¹⁷⁾, both of whom used direct-epicardial leads, showed that electrocardiographic pattern of direct-epicardial leads

over the right ventricle including its outflow tract was an rS type and no late R wave was observed in any case. Also, as pointed out by Rosenmann¹³⁾, a late R wave in lead V1 is observed in only five per cent of normal cases. For these reasons, the outflow tract of the right ventricle does not seem to be the last portion of the ventricular activation process with the exception of the cases of right ventricular hypertrophy, chiefly due to congenital heart disease¹⁸⁾¹⁹⁾.

On the other hand, as indicated by many authorities³²⁾³³⁾, morphologies of the esophageal electrocardiography at the locations corresponding to the posterior basal region of the left ventricle are always a qR and a QR pattern concerning QRS complex, and according to the results of Levine and Goodale²⁴⁾, Kert and Hoobler³⁵⁾, Latour and Puech²¹⁾ and ours, those of the coronary venous system electrocardiography are the same as at the corresponding positions of the esophagus facing the posterior basal region of the left ventricle. At any rate, in the absence of extreme right ventricular hypertrophy, this portion may be regarded as the last region of the ventricle to be depolarized. Ventricular complexes with late R waves occurring in the same time-relationships as those from the coronary venous system are recorded quite frequently from the right atrium, superior vena cava, pulmonary artery and capillary as shown in Tables III, IV, V, VII, VIII. Most recordings from the pulmonary capillary show late R waves whose atrial complex has in general left atrial intrinsicoid deflection. Both left atrium and ventricle form the posterior surface of the heart, normal and abnormal, and according to Grant⁷⁶⁾, the left ventricle should be called preferably posterior ventricle. Thus the presence of late R waves recorded from the pulmonary capillary seems to be rather possible, but the same late R waves are observed at the right phrenico-costal angle whose location is fairly remote from the left ventricle through the air-contained tissue. For this reason it seems that the late R waves in the latter position can not be considered as due to solely the left ventricular activation.

3. At times from the right pulmonary capillary, more frequently from the right pulmonary artery, almost always from the main pulmonary artery, always from the right ventricle, and fairly frequently from the right atrium and superior vena cava, early R waves are recorded and these have the same onsets and peaks concerning time-relationships. The peaks of those late R waves which are recorded almost always from the right pulmonary capillary occur simultaneously with those from the coronary sinus, right atrium, superior vena cava and pulmonary artery as shown in Tables III, IV, V, VII, VIII. The electrocardiographic patterns over the body surface corresponding to the right pulmonary capillary from which electrocardiograms are recorded are chiefly an rS, rs or low voltage polyphasic form. Morphologies from both lead points are quite dissimilar. As mentioned before, atrial complexes with ventricular morphologies of a Qr or a qr pattern recorded from the right pulmonary capillary show left atrial intrinsicoid deflection and morphologies over the corresponding body surface, on the other hand, exhibit right atrial intrinsicoid deflection. This is due to the fact that the exploring points in the right pulmonary capillary are fairly deeply located from the chest wall and lie near the posterior surface of the left ventricle rather than the anterior aspect of the right ventricle. As the exploring points in the left pulmonary capillary are shadowed by the cardiac silhouette, these locations are clearly behind the heart and lie near the left ventricle. That both late and early R waves have the same time-relationships in the right pulmonary capillary, right and main pulmonary arteries, right atrium, superior vena cava and coronary sinus is clarified from the fact that these locations except for the right pulmonary capillary have almost the same solid angles extended by the heart and the enclosed region of the heart by these solid angles is practically the same. On the contrary, exploring points in the right pulmonary capillary do not peep into the cardiac cavities, but face the right posterior surface of the heart. Morphologies of the electrocardiograms recorded from these locations are similar to those from the right

pulmonary artery though early R waves are frequently lost and their amplitudes become smaller (Fig. 2). This observation seems to suggest that pulmonary arteries enclosed by air-contained tissues such as lungs operating as insulator act like "linear conductor". Recently, Luisada and Liu^{37,38} introduced the catheter into the cardiac cavities by the conventional venous approach or punctured the left atrium directly with a punctured-needle insulated except for the tip, thus recorded left intraatrial electrocardiograms from the root of the needle and further inserting a polyethylene tube through this needle, left intra-ventricular electrocardiograms from the same point of the needle. From this fact, our above-mentioned inference may be thought to be probably valid. Camerini and Davies¹³, and Blount et al¹⁴ recorded unipolar chest leads extensively over the right precordium of adult subjects of every decade and of normal infant groups, respectively. In almost all the subjects, late R waves were observed in at least one of many exploring points. They attributed these late R waves to the activation of the outflow tract of the right ventricle. As indicated by Barbato et al¹⁷, however, late R waves are not observed over the outflow tract of the right ventricle and according to our foregoing inference that "linear conductor" plays a rôle, the presence of these late R waves at different right precordial sites can be well explained when intracardiac late R waves are likely to be transmitted incidentally to the right precordial exploring points. This idea will be applied to the analysis of an RSR' pattern in the next chapter.

4. As proposed by Barker and Valencia²³, the criteria of incomplete right bundle branch block are as follows:

1. QRS interval in limb leads is 0.08 sec. to 0.115 sec. in duration.

2. In unipolar right chest leads, especially in both lead V1 and lead VE, primary and secondary R waves are present. And when in other unipolar right chest leads such as lead V3R and lead V4R secondary R wave is present, the diagnosis is more accurate. Moreover, they

mentioned the presence of a prominent late R wave in lead VR and a relatively broad S wave in lead 1 as findings of useful reference but did not weigh these findings. According to Wilson, Rosenbaum and Johnston³⁹, the criteria are the existence of a clear-cut secondary R wave in lead V1 and/or lead VE when QRS interval is 0.08 sec. to 0.11 sec. in duration, and those for complete right bundle branch block are that QRS interval is over 0.12 sec. in duration and there are initial and secondary R waves in lead V1, always in lead V2 and nearly always in lead VE. Goldberger's criteria¹ using unipolar precordial leads are such that in complete right bundle branch block lead V1 and lead V2 exhibit an rsR' pattern of which final R wave is broad, and lead V5 and lead V6 show a qRS pattern whose final S wave is broad when QRS interval is over 0.12 sec. His criteria for incomplete right bundle branch block are the existence of the same morphologies as in complete right bundle branch block and of the duration of 0.12 sec. or less. Grant³⁶ chose as criteria for right bundle branch block both the QRS interval of 0.10 sec. or more and the existence of a S wave in lead 1 and an R' wave in lead V1, and mentioned that the restriction of QRS interval of 0.12 sec. or more was too strict for right bundle branch block and both S1 and R'-V1 were necessary for differentiating right bundle branch block from peri-infarction block. In regard to the QRS interval as one of each criteria, Lepeshkin⁴⁰ together with Grant stated that the QRS interval was not an absolute criterion and its overestimation was dangerous for the differentiation of incomplete and complete right bundle branch block. Because Goldberger allowed the QRS interval of about 0.10 sec. for complete right bundle branch block, his criteria are very equivocal for the decision of incomplete and complete right bundle branch block.

In view of the above-mentioned representative opinions for right bundle branch block, the satisfaction of the following three conditions may be thought to be necessary and sufficient:

1. The existence of a late R wave in

lead V1 greater in both duration and amplitude than an early R wave in the same lead.

2. The existence of a clear and broad S wave in lead V5.

3. QRS interval over 0.10 sec.

The reasons for the selection of these restrictions are as follows: When an RSR' pattern in lead V1 is present, following two instances can be considered in view of the intracardiac leads:

1. The peak of a late R wave in lead V1 occurs simultaneously with those of intracardiac late R waves.

2. The peak of a late R wave in lead V1 appears later than those of intracardiac late R waves.

Furthermore, the former can be subdivided into two instances.

1-A. The late R wave in lead V1 is of almost the same amplitude as the early R wave in the same lead or embryonic in morphology. The duration of this late R wave is short and the tendency to the plateau-formation of the apex is not significant. There is neither notching nor slurring in the ascending limb of these late R waves. The broad S wave in lead V5 is not observed.

1-B. The amplitude and duration of the late R wave in lead V1 are greater than those of the early R wave in the same lead. The tendency to the plateau-formation of the apex of such a late R wave is always observed. There are both notching and slurring in the ascending limb of this late R wave. The broad S wave in lead V5 is also observed. The latter has all the morphologic characteristics of complete right bundle branch block of Wilson et al, but the duration over 0.12 sec. is not observed in any case. Partly for this reason, the restriction of 0.12 sec. seems to be too rigid.

In the 1-A pattern as shown in Fig. 5, an early R wave is observed in any site in the intracardiac cavities, at times in the right pulmonary capillary as in normal subjects, and coincides in onset with an early R wave in lead V1. All the nadirs of S waves in unipolar right chest leads occur simultaneously with the peaks of unipolar left precordial leads, especially that of lead V5¹²⁾. The time-re-

lationships of the appearance of these S waves in right precordial leads are constant. Following this S wave, an R' wave appears. This R' wave occurs simultaneously with the nadir of S wave in lead V5 and the peaks of intracardiac late R waves as in normal cases (Table VIII). Furthermore, this S wave is not broad. For this reason, the late R wave in 1-A pattern must be ascribed to activation of the posterior base of the left ventricle. This pattern is observed in eight cases of which seven are cases of mitral stenosis and the other one is a case of pure mitral insufficiency. One of the subjects with mitral stenosis was complicated by old antero-septal myocardial infarction, but peri-infarction block of Grant³⁶⁾ was not observed. The existence of S1 is not constant. Though S1 is present, this negative wave is shallow and narrow. A S wave in lead V5 is always present, but their width is small. As shown

Fig. 5. Intracavitary and precordial leads in cases of so-called incomplete right bundle branch block. In each recording from above downward as regards electrocardiogram intracavitary lead, lead VI (case K. T.) or lead V2 (case H. A.), and lead V5. Pressure tracings, if recorded, are shown in the third or last tracing. Note that the peaks of early R in the intracavitary leads occur simultaneously with one another and the peaks of late R in both the intracavitary and precordial sites are synchronous with the nadir of the s of lead V5. A, B, C, D, Patient H. A. with mitral stenosis. E, F, G, H, Patient K. T. with mitral stenosis. Time lines=0.05 sec.; paper speed=40 mm./sec.

in tables, the QRS interval varies from 0.075 sec. to 0.10 sec., but almost all cases have the time-lag of about 0.08 sec.

In summary, the 1-A pattern shows late R wave in lead VI, but does not satisfy the above-mentioned three restrictions. According to the criteria of Barker and Valencia which are extensively used clinically, this pattern should be classified into incomplete right bundle branch block and exhibits the retardation of activation in the right ventricle. But actually this pattern may be thought to reflect the physiological retardation of activation of the postero-basal portion of the left ventricle.

In the case of 1-B pattern, right precordial late R waves do not occur simultane-

ously with all the intracardiac late R waves, but those recorded from the right precordium, right atrial cavity and superior vena cava are all synchronous and later than the peaks of late R waves recorded from the right ventricle and pulmonary artery (Table VIII and Fig. 6). As cited later on, this can be readily accounted for from the explanation of Wasserburger et al.¹⁹⁾ that an rsR' pattern in lead V1 is projected to the higher lead positions and retains the same morphology. For this reason, in the 1-B pattern precordial late R waves are contributed to by activation of the free right ventricular wall.

In the 2 and 1-B patterns, the RSR' in lead V1 exhibits all the features of so-called complete right bundle branch block whether or not QRS interval surpasses 0.12 sec. Early

Fig. 6. Patient K. S. with Roger's disease and so-called complete right bundle branch block (QRS interval: 0.127 sec.). In the upper row each recording shows from above downward intracavitary lead, lead V1, lead V5 and pressure, the last one tracing of which is not shown in the tracing from the SVC. In the lower row standard limb leads, a VR and right-sided precordial leads are presented. Note that in the RPC tracing ventricular complexes of rsR' and rSr' at VI appear. The rsR' pattern (the former) must be regarded as so-called complete right bundle branch block from, both the pattern point of view and the QRS interval, while the rSr' pattern (the latter) may be concluded as so-called incomplete right bundle branch block because both patterns are taken in the same tracing. Time lines = 0.05 sec.; paper speed = 40 mm./sec.

Fig. 7. Patient T. S. with mitral stenosis and so-called complete right bundle branch block (QRS interval: 0.136 sec.). In the upper row from left to right, recordings are taken from RPC, MPA, INF and RA, respectively. In each recording from above downward intracavitary lead, lead V2, pressure and lead V5, the last two tracings being inverted in the order in some recordings. In the lower row each recording shows right-sided precordial leads V1, V4R and V3R with lead V2 (above) and aVR (below) as monitor leads. Note that the peaks of the late R in various intracavitary sites occur earlier than the peaks of the late R in right-sided precordial sites. Time lines = 0.05 sec.; paper speed = 40 mm./sec.

R and S waves show the same time-relationships as in 1-A pattern. The peaks of late R waves recorded from the right precordium in 2-pattern occur later than those in any intracardiac lead. Intracardiac tracings, on the contrary, exhibits the broad, much or less deep negative wave which corresponds to the right precordial late R waves. And when there is at any rate a much or less deep negative

wave in lead V1, this relationship is satisfied by the late R wave in lead V4R and/or lead V3R.

In summary, the morphologies of complete right bundle branch block are shared by activation of the free right ventricular wall (Fig. 7).

In the 2 and 1-B patterns, the QRS interval ranges from 0.10 sec. to 0.136 sec. Time-

TABLE IX VENTRICULAR THICKNESS & STIMULUS ARRIVAL TIME

Patient	Age	Sex	Disease	Heart weight Gm	Thickness in mm.		Intrinsic Def. sec in.	
					RV	LV	CR ₂	CR ₅
Z. K.	57	M	Hypertensive heart disease			13		.051
S. K.	22	F	Rheumatic pancarditis	870	5	13	.035	.057
A. S.	41	F	Combined valvular disease		5	14	.042	.053
S. K.	51	M	Mitral valvular disease			8		.052
M. F.	33	M	Tetanus	285		11		.050
K. O.	57	M	Pneumococcal meningitis		4	12	.028	.050
H.	60	F	Typhoid fever		3	9	.036	.063
T. K.	25	F	Lung cancer	235	2	12	.032	.046
T. M.		M	Subdural hematoma	225	3	12	.030	
A. I.	25	F	Dyspepsia nervosa	300	2	10	.024	.053
M. U.	22	M	Brain tumor	170		10		.043
I. O.	57	F	Spinal cord tumor	280	3	10	.026	.047
Y. S.	63	M	Grawitz' tumor	360	2	10	.028	.048
K. F.	70	F	Apoplexy	310	4	12	.025	.061
I. Y.	40	M	Amyotrophic lateral sclerosis			9		.041
Y. N.	32	M	Renal hypertension	450	3	12	.031	.053
K. M.	25	F	Chronic nephritis		5	14	.039	.058
S. O.	44	M	Renal hypertension	490		12		.044
S. T.	46	M	Malignant hypertension		4	17	.035	.081
T. K.	39	F	Peptic ulcer	200	2	8	.026	.046
S. M.		M	Peptic ulcer		4	11	.028	.045
T. M.	53	M	Cancer of stomach	280	3	10	.025	.050
M. M.	53	F	Obstructive jaundice	415	4	14	.047	.055
M. E.	55	M	Liver cirrhosis	335		11		.053
H. K.	39	F	Liver cirrhosis	280	3	10	.045	.057
G. T.	61	M	Liver cirrhosis	250		12		.047
T. T.	64	M	Hepatoma	350	3	10	.029	.060
I. H.	68	F	Purpura, Hypertension		5	14		.057
K. I.	50	F	Banti's syndrome	245		11		.051
Y. O.	69	M	Multiple myeloma		2	11	.042	.046
K. N.	17	M	Lymphocytic leukemia	280		10		.058
S. F.	56	M	Monocytic leukemia		3	10	.033	.049

Note: This table is made from the material owned by the institute of pathology, Kyoto University Medical School. Heart weight measurement is somewhat inexact because some hearts have clotted blood mass in the ventricle, and also the larger part of the right ventricle including pulmonary conus is sometimes lost. Accordingly in such cases heart weight and/or right ventricular thickness are not measured.

lag of about 0.12 sec. is usually observed. Accordingly when an RSR' pattern in the right precordial lead is caused by a delay in activation of the free right ventricular wall, the above-mentioned three restrictions must be satisfied. The 1-A pattern should not be termed right bundle branch block, but merely an RSR' pattern. And as regards the differentiation between the patterns 1-A, and 2 and 1-B, it seems to be valid that the QRS interval of 0.10 sec. or more together with the morphology of S wave in lead V5 should be employed as criteria. One of the reasons for selecting such criteria is the fact that the greatest QRS interval of these eight cases exhibiting 1-A pattern is 0.10 sec., and on the contrary, that of all the cases showing 2 and 1-B patterns is over 0.10 sec. However, as shown in the RPC recording in Fig. 6, the amplitude and duration of late R waves vary even in the same lead. One should be properly termed right bundle branch block and the other should belong to 1-A pattern. In this case both patterns appear in the same lead and so the latter should be regarded as an intermediate form between the normal and the former pattern, namely incomplete right bundle branch block. When electrocardiograms exhibit the same morphologies as in 1-A pattern, it is assumed that there are two instances causing this pattern, and these two patterns are almost indistinguishable. But all cases belonging to the foregoing 1-A pattern are those of mitral valvular disease and this fact is worthy of notice.

5. At present, there is no authorized theory concerning the genesis of broad and high R waves in unipolar right chest leads in subjects with right ventricular hypertrophy verified at autopsy. On the thirty-two autopsy materials (Table IX) containing cases with non-cardiac disease but not congenital heart disease and yet not appearing in this article, we measured, on the one hand, the thickness of the right ventricular wall at its outflow tract and stimulus arrival time in lead CR2 (time interval from the onset of QRS complex to the intersection between the base-line and the descending

limb of the same QRS complex in unipolar chest lead), and, on the other hand, the thickness of the left ventricular free wall corresponding to the precordial site of lead CR5 and stimulus arrival time in the same lead. Correlation coefficients between the ventricular thickness and stimulus arrival time in each ventricular side are 0.39 (right ventricular side: lead CR2) and 0.55 (left ventricular side: lead CR5), respectively. Only in the left ventricular side, there is a significant correlation ($p < 0.01$) (Fig. 8). Because there is no cor-

Fig. 8. Relationship between ventricular thickness and stimulus arrival time in man.

relation in the right ventricular side, it may be assumed that the right ventricular thickness has no effect upon the morphology of QRS complex recorded from the right chest. As indicated by Kossmann et al²⁰, Kert and Hobler²⁵, and Rosenmann¹³, the right ventricular free wall seems to have little influence upon the morphology of unipolar right chest lead.

6. The morphological features of electrocardiograms exhibiting right ventricular hypertrophy are as follows:

High-voltage R wave, retardation in onset of the intrinsicoid deflection, and shallow or absent S wave in lead V1. This tendency is significant in cases with pulmonary stenosis as indicated in Table VI. In these cases, an rS pattern is recorded from any location in the right ventricle, but two different patterns are observed in the right atrium:

1. Such patterns as qR, qRs, Rs and R: these patterns are almost identical with those in lead V1 (Fig. 9).

Fig. 9. Patient J. K. with tetralogy of Fallot and classic electrocardiographic pattern of right ventricular hypertrophy. In the upper row simultaneous recordings of lead III (above) and intracavitary lead (below). Withdrawal tracing from the INF. to the RA (left) and RA (high portion) (right). In the lower row simultaneous recordings of lead III (below) and precordial lead CR1, CR2 and CR5 (above). Note that the forms of the QRS complexes both in RA and at right precordium are very similar with each other. Time lines=0.05 sec.; paper speed=40 mm./sec.

2. Qr pattern (Fig. 10)

Even when a single R wave of high-voltage is observed in the right atrium, the late R wave in the pulmonary artery is not so great as compared to that of normal cases and yet in a few cases this wave is not obtained in any location. The peaks of these late R waves are all synchronous but not simultaneous with the nadir of S wave in lead V5 as in normal cases. These peaks occur simultaneously with the nadir of S wave in leads V1 and V2 or their neighboring portion. In the cases with pulmonary stenosis of Barbato et al¹⁸⁾ and Wasserburger et al¹⁹⁾, the morphology of the direct-epicardial leads over the outflow tract of the right ventricle is an rsR' pattern and this rsR' pattern was assumed by Wasserburger et al to be projected to such lead-axes as leads aVR, V3R and VE, namely upper, lateral and lower sides of the heart. When this rsR' pattern is almost exclusively projected upwards, namely to the lead-axis of lead aVR, it is probable that this morphology is projected to the large right atrium occupying the anterior surface of the heart, and thus an R wave of high-voltage is obtained in this chamber. When intracardiac potentials are projected downwards

Fig. 10. Patient N. Y. with pulmonary stenosis and classic electro-cardiographic pattern of right ventricular hypertrophy. In the upper and middle rows simultaneous recordings of lead II and intracavitary lead. In the lower row lead CR1 and lead CR2. Note that tracings in the RA have QR pattern not similar to the pattern at CR1. Time lines=0.05 sec.; paper speed=40 mm./sec.

and/or down-lateralwards, a Q wave is expected to be predominant in the right atrium. The above-mentioned type 2 corresponds to this. In this case, the late R wave is perhaps due to activation of the left ventricular posterior base in view of the same time-relationships and morphologies as in normal cases. On the contrary to this type, the late R waves in type I fuse with single large R waves and are not manifest. We calculated correlation coefficient between the right ventricular thickness and activation time in lead V1 on the autopsy materials of Walker et al⁴¹⁾ exhibiting right ventricular hypertrophy (showing high-voltage single R wave and/or similar morphology in lead V1). This value is 0.52 ($p < 0.01$) and almost identical with the correlation coefficient of 0.55 between the left ventricular thickness and activation time in lead CR5 of our autopsy materials. Both are significant. In short, it seems that the right ventricular free wall thickness is much responsible for the genesis of right ventricular hypertrophy pattern when there are single high-voltage R waves indicative of right ventricular hypertrophy in unipolar right chest leads.

CONCLUSION

1. Time-relationships of those late R waves were principally reviewed which were recorded with right chest lead as monitor-lead from the right cardiac cavities and their neighboring vessels of ninety subjects.

2. Unipolar right and intracardiac late R waves which were frequently present in clinical electrocardiograms except for in cases with a single high-voltage R wave and/or right bundle branch block type whose QRS interval was 0.10 sec. or more were demonstrated to be derived from activation of the left ventricular posterior base.

3. It was presented that the concept of "linear conductor" of blood vessels was useful for interpreting mechanisms of the formation of these late R waves.

4. Single high-voltage R waves in lead aVR and unipolar right chest leads indicative of classical right ventricular hypertrophy, and late R waves characteristic of right bundle branch block with QRS interval of 0.10 sec. or more in the same leads were both proven to be attributed to a delay in activation of the ventricular free wall.

The data on which the observations in this article are based were obtained with the aid of the cardiac catheterization team of third medical clinic of the Kyoto University Hospital.

I wish to offer sincere thanks to Prof. M. Maekawa for his guidance and criticism. Many thanks are also to be expressed to the cardiac catheterization team, Drs. Y. Ogata, M. Sakai, C. Kawai, Y. Morita, K. Masuda and T. Ota.

The outline of this study was reported at 21st and 24th annual meetings of Japanese Circulation Society in 1957 and 1960, respectively.

REFERENCES

- Goldberger, E., Unipolar Lead Electrocardiography, Lea & Febiger, Philadelphia, 1953.
- Kossmann, C. E., Berger, A. R., Brumlik, J., Briller, S. A., *Am. Heart J.* **35**: 309, 1948.
- Kossmann, C. E., Berger, A. R., Rader, B., Brumlik, J., Briller, S. A., Donnelly, J. H., *Circulation* **2**: 10, 1950.
- Walker, W. J., Mattingley, T. W., Pollock, B. E., Carmichael, D. B., Inmon, T. W., Forrester, R. H., *Am. Heart J.* **52**: 547, 1956.
- Kjellberg, S. R., Mannheimer, E., Rudhe, U., Jonsson, B., Diagnosis of Congenital Heart Disease, Year Book Publishers Inc., Chicago, 1955. cited by 4)
- Sodi-Pallares, D., Rodriguez, M. I., Chait, L. O., Zuckermann, R., *Am. Heart J.* **41**: 569, 1951.
- Rodriguez, M. I., Sodi-Pallares, D., *Am. Heart J.* **44**: 715, 1952.
- Dickens, J., Goldberg, H., *Am. Heart J.* **56**: 8, 1958.
- "", *"* **56**: 372, 1958.
- Emslie-Smith, D., *British Heart J.* **18**: 78, 1956.
- Camerini, F., Davies, L. G., *British Heart J.* **17**: 28, 1955.
- Morris, T. L., Whitaker, W., *Am. Heart J.* **52**: 738, 1956.
- Rosenmann, R. H., *Am. Heart J.* **40**: 522, 1950.
- Blount, S. G., Jr., Munyan, E. A., Jr., Hoffman, M., *Am. J. Med.* **22**: 784, 1957.
- Lewis, T., Rothschild, M. A., *Phil. Trans. Roy. Soc.* **206**: 181, 1915.
- Wilson, F. N., Johnston, F. D. Hill, I. G. W., *Am. Heart J.* **10**: 176, 1934.
- Barbato, E., Pileggi, F., Debes, A. C., Fuzioka, T., Magalhaes, M. S., Tranches, J., San Juan, E., Décourt, L. V., *Am. Heart J.* **55**: 867, 1958.
- Barbato, E., Fuzioka, T., Debes, A. C., Pileggi, F., Filho, C. B., Silra, P. P., Décourt, L. V., *Am. Heart J.* **56**: 340, 1958.
- Wasserburger, R. H., Siebecker, K., Freeman, J., King, J. T., Young, W. P., *Am. Heart J.* **57**: 578, 1959.
- McGregor, M., *British Heart J.* **12**: 351, 1950.
- Latour, H., Puech, P., *Electrocardiographie Endocavitare*, Masson et Cie, Paris, 1957.
- Sokolow, M., Lyon, T. P., *Am. Heart J.* **38**: 273, 1949.
- Barker, J. M., Valencia, F., *Am. Heart J.* **38**: 376, 1949.
- Schmitt, O. H., Levine, R. B., Simonson, E., *Am. Heart J.* **45**: 416, 1953.
- Levine, R. B., Schmitt, O. H., Simonson, E., *Am. Heart J.* **45**: 500, 1953.
- Simonson, E., Schmitt, O. H., Levine, R. B., Dahl, J., *Am. Heart J.* **45**: 655, 1953.
- Frank, E., *Circulation* **11**: 937, 1955.
- Frank, E., *Am. Heart J.* **49**: 670, 1955.
- Frank, E., *Circulation* **12**: 406, 1955.
- Piersol, G. A., Human Anatomy, ed. 4, Vol I. Lippincott & Co., Philadelphia, 1913, cited by 34)
- Macleod, A. G., Barker, P. S., Alexander, J., *Am. Heart J.* **5**: 720, 1930.
- Oblath, R., Karpman, H., *Am. Heart J.* **41**: 369, 1951.
- Scherlis, L., Wener, J., Master, A. M., Grishman, A., Sandberg, A. A., *Am. Heart J.* **41**: 246, 1951.
- Levine, H. D., Goodale, W. T., *Circulation* **2**: 48, 1950.
- Kert, M. J., Hoobler, S. W., *Am. Heart J.* **38**: 97, 1949.
- Grant, R. P., Clinical Electrocardiography, McGraw-Hill Book Company, Inc., New York 1957.
- Luisada, A. A., Liu, C. K., *Am. Heart J.* **54**: 531, 1957.
- Liu, C. K., Aravanis, C., Testelli, M. R., Luisada, A. A., *Am. J. Cardiology* **2**: 379, 1958.
- Wilson, F. N., Rosenbaum, F. F., Johnston, F. D.,

- Advances in Internal Medicine 2:1, Interscience Publishers, Inc., New York, 1947.
- 40) Lepeschkin, E., Modern Electrocardiography, The Williams & Wilkins Company Baltimore, 1951.
- 41) Walker, I. C., Scott, R. C., Helm, R. A., *Circulation* **11**: 233, 1955.