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Studies of Electrocardiographic Diagnosis of Right Ventricular Hypertrophy and of the Incidence of Arrhythmias and Left Ventricular Abnormalities in Patients with Chronic Obstructive Pulmonary Disease with Moderate to Severe Pulmonary Hypertension

(right ventricular hypertrophy/arrhythmia/pulmonary hypertension)

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In 28 hospitalized patients with chronic obstructive pulmonary disease (COPD) with a pulmonary artery mean pressure (PAm) over 18 mmHg, incidence of electrocardiographic pattern of right ventricular hypertrophy (RVH) and the prevalence of arrhythmias and left ventricular abnormalities were studied on a total of 146 electrocardiograms.

Our new single criteria for electrocardiographic diagnosis of RVH in COPD was mean QRS axis between 90° and 180° or between −180° and −80°. The majority diagnosed as RVH had a PAm of over 27 mmHg. In 7 patients, pulmonary artery wedge pressure (WP) was over the normal value. However, all patients except one with high WP showed no abnormality in left precordial leads. Twelve tracings of 146 (8%) showed arrhythmias. However, only one with transcient atrial fibrillation, the next day showed sinus rhythm with frequent supraventricular premature beats (SVPB). Other arrhythmias observed were all infrequent (less than 5 per minute) SVPB or VPB. Prevalence of arrhythmias on electrocardiograms of our patients with COPD was markedly less than the reported prevalences of hospitalized Americans with COPD. This difference may be attributed to the difference of the degree of clinical and subclinical ischemic degenerative changes of the heart. Autopsy findings and electrocardiograms of 4 cases with RVH were discussed.

The present work was undertaken with a view to clarifying the incidence of electrocardiographic pattern of right ventricular hypertrophy (RVH) and of the prevalences of arrhythmias and left ventricular abnormalities in patients of chronic obstructive pulmonary disease (COPD) with moderate to severe pulmonary hypertension.

MATERIAL AND METHODS

Twenty-eight patients hospitalized for COPD and with moderate to severe

pulmonary pulmonary hypertension were studied. All these patients were evaluated by us at Yamaguchi University Hospital. The patients ranged in age from 54 to 72 years of age and 4 were women (Table I).

TABLE I. *Clinical and Laboratory Data*

Case	Diagnosis	Age	Sex	History of RVF	PaO_2 (mmHg)	SaO_2 (%)	$PaCO_2$ (mmHg)	pH
1	PE+BA+TB	58	m	+	64	90	48	7.40
2	PE+TB+BCa	64	m	+	23	31	90	7.29
3	PE+bronch.	64	m	-	56	87	48	7.40
4	PE	61	m	+	52	83	52	7.39
5	PE	60	m	-	55	85	53	7.35
6	PE+CB	68	m	-	56	89	39	7.45
7	PE	67	m	-	52	89	39	7.44
8	PE	60	m	-	60	89	53	7.39
9	PE+bronch.	71	f	+	44	87	68	7.41
10	PE	79	m	-	85	95	37	7.49
11	PE+CB	64	m	+	47	92	59	7.46
12	PE+bronch.	59	m	-	50	82	60	7.41
13	PE	63	m	-	64	90	43	7.41
14	PE+CB	72	m	-	57	85	46	7.35
15	PE	54	m	-	60	91	53	7.43
16	PE	55	m	-	62	99	52	7.44
17	PE	70	m	-	70	92	48	7.37
18	PE(+EH)	67	m	-	63	90	56	7.39
19	PE	66	m	-	72	91	59	7.34
20	PE	55	f	+	64	92	39	7.47
21	PE+CB	68	f	-	58	91	41	7.46
22	PE	60	m	-	70	86	28	7.50
23	PE	64	m	-	60	93	34	7.48
24	PE+BA+TB	69	m	-	90	95	41	7.40
25	PE+CB	70	m	-	-	94	-	-
26	PE+bronch.	56	f	-	58	89	48	7.44
27	PE	64	m	-	60	93	35	7.53
28	PE	56	m	+	54	87	52	7.44

Abbreviations PE : chronic pulmonary emphysema, BA : bronchial asthma, TB : pulmonary tuberculosis, BCa : bronchial carcinoma, bronch. : bronchiectasis, CB : chronic bronchitis, EH : essential hypertension, RVF : right ventricular failure, PaO_2 : partial pressure of oxygen in arterial blood, SaO_2 : oxygen saturation of arterial blood, $PaCO_2$: partial pressure of carbon dioxide in arterial blood

Only one (case 18) had a history and showed clinical evidence of systemic hypertension, and in no patient was there coronary heart disease, valvular disease or other recognized causes of left ventricular abnormality.

Each of 28 patients had COPD and 4 also a bronchiectasis and 2 bronchial asthma. The antecedent respiratory disease was of 3 to 40 years' duration.

Each patient had moderate to severe obstructive or combined ventilatory disturbances. As shown in Table I, a low arterial partial pressure of oxygen (PaO_2) was found in 25 of 27 patients. Sixteen had hypercapnia ($PaCO_2$ 48 to 90 mmHg) and only one of these had an arterial pH below 7.35. Only case 5 had a hematocrit over 50 per cent. He had a hematocrit of 60 per cent. Seven patients had a history of right ventricular failure (RVF).

Pulmonary arterial pressures and flows were measured by cardiac catheterization in all cases. Because some of patients had previously had an acute

pulmonary infection, the clinical status was optimally stabilized before cardiac catheterization. With the mid-thoracic level used for zero reference, pressure were averaged over at least two respiratory cycles.

In the tables, the patients are arranged in descending order of pulmonary artery mean pressure (PAm).

All patients had at least one electrocardiogram, although the frequency of electrocardiograms varied considerably. Most of all electrocardiograms consisted of standard 12 leads and leads V3R and V4R. Conventional standarization of 1 cm = 1 mV. was employed.

Interpretation and quantitative evaluation of the electrocardiograms were made by an observer who had no knowledge of the case number of the corresponding hemodynamic data.

The criteria used for right ventricular hypertrophy (RVH) were a composite of those of Goodwin and Abdin (1) and Sokolow and Lyon (2) modified by Kilcoyne, Davis and Ferrer (3). Therefore, at least two of the following criteria were required for the ECG diagnosis of RVH : (a) mean QRS axis greater than $+90°$ or in serial records, the appearance of an axis shift of more than 30° to the right of the mean QRS axis previously shown by the patients ; (b) $R > S$ in leads V3R, V1 or aVR ; (c) ST segment depression in leads II, III and aVF ; (d) T-wave inversion or biphagic T-waves in the right precordial leads. (e) $R < S$ in lead V5 and (f) P-wave voltage greater than 2.5 mm in leads II or III.

In this study $S_1S_2S_3$ or S_1Q_3 pattern was also checked because it was a suggestive criteria of RVH in patients of COPD (4). Presence of slurred S in right precordial leads was also checked.

Moreover, Kilcoyn *et al.* (3) observed that when the arterial O_2 saturation fell below 85 per cent and PAm, which calculated by their prediction equation used only arterial blood gases for its solution, was 25 mmHg or greater, one or more of the following fluctuations were seen in patients with COPD : (a) a rightward shift of mean QRS axis of 30° or more ; (b) T-wave abnormalities in right precordial leads ; (c) ST depressions in leads II, III and aVF ; and (d) transitory right bundle branch block either complete (RBBB) or incomplete (IRBBB). They noted that these fluctuations subsided with recovery of more adequate arterial saturation and decrease in PAm calculated by their prediction equation.

In this study, we measured PAm directly and compared findings with incidences of T-wave abnormality in right precordial leads, ST depression in II, III and aVF and IRBBB. We also reviewed the serial electrocardiograms mounted in the clinical record, and checked these four fluctuations.

In 4 patienes with RVH as confirmed by autopsy, comparison of the final tracing with previous ones was feasible.

At the same time we checked the incidence of arrhythmia and left precordial abnormality in all 146 tracings of these 28 patients.

RESULTS

Pulmonary artery mean pressure (PAm) and ECG evidence of right ventricular hypertrophy (RVH) are summarized in Table II. In this study, we

TABLE II. *Pulmonary Hypertension and ECG Evidence of Right Ventricular Hypertrophy*

Case		PAm (mmHg)	mean* QRS axis (°)	R>S in* V3R or V1 or aVR	ST depr.* in II. III.aVF	T invert.* in right precord. leads	R<S* in V5	P>2.5* mm in II or III	$S_1S_2S_3$ pattern	S_1Q_3 pattern	IRBBB	Slurred S in V3R-V1
1	#	56	96	-	-	-	+	+	+	+	-	-
2	#	53	-164	+	-	+	-	-	-	+	-	-
3	#	36	-82	-	-	+	-	+	+	-	-	-
4		35	90	-	-	-	-	-	-	-	-	-
5	#	31	112	+	-	-	-	-	-	-	-	-
6	#	29	90	-	-	+	+	-	+	-	+	+
7	#	28	96	-	-	+	+	-	-	+	-	+
8	#	28	100	-	-	-	+	+	-	-	-	-
9		27	20	-	-	-	-	-	-	-	-	-
10		26	80	-	-	-	-	+	-	-	-	-
11	#	26	94	-	-	+	-	-	-	-	-	-
12		25	-62	-	-	+	-	-	-	-	-	-
13		25	85	-	-	-	-	-	-	-	-	-
14		25	70	-	+	-	-	-	-	-	-	-
15	#	24	109	-	-	-	+	-	-	-	-	-
16		23	72	-	-	-	-	-	-	-	-	-
17		23	-25	-	-	-	-	-	-	-	-	+
18		23	52	-	-	-	-	-	-	-	-	-
19		23	85	-	-	-	-	-	-	-	-	-
20		22	67	-	-	-	-	+	-	-	-	-
21	#	21	76	-	-	+	-	+	-	-	-	-
22		21	75	-	+	-	-	-	-	-	-	-
23		20	30	-	-	+	-	-	-	-	-	-
24	#	20	94	+	-	+	-	-	+	-	+	-
25		20	75	-	-	-	-	-	-	-	-	-
26		20	56	-	-	+	-	-	-	-	-	-
27		19	42	-	-	+	-	-	+	+	-	-
28		19	79	-	-	-	-	-	-	-	-	-

Abbreviations PAm : mean pulmonary arterial pressure, IRBBB : incomplete right bundle branch block

* : Criteria at least two of which are required for the ECG diagnosis of right ventricular hypertrophy (RVH)

\# : Cases diagnosed as RVH by sets of criteria marked*

number of mean QRS axis are underlined : A QRS axis is in conformity with a new single criteria for diagnosis of RVH

selected patients with moderate to severe pulmonary hypertension of PAm over 18 mmHg (normal value of PAm is under 17 mmHg) and in the tables, the patients are arranged in descending order of PAm, therefore case 1 had the highest PAm of 56 mmHg and case 28 the lowest of 19.

As described in methods, at least two of the first six criteria on the Table II (*marked) are required for the diagnosis of RVH. Only 11 cases (cases 1, 2, 3, 5, 6, 7, 8, 11, 15, 21 and 24) showed evidence of ECG diagnosis of RVH. In these 11 cases, 7 had PAm $\geq$ 28 mmHg and 4 had PAm $\leq$ 27 mmHg. When all patients are deliniated to two groups with the level of PAm over or under/or equal 27 mmHg, in the group with PAm $\geq$ 28 mmHg (group A) there are 8 cases (cases 1—8) and 7 of them (90%) diagnosed

electrocardiographically as RVH, contrarily in the group with PAm $\leq$ 27 mm Hg (group B) there are 20 cases (case 9—28) and 4 of them (20%) diagnosed as RVH. Therefore, it is clear that if at least two of the first six criteria are required for the ECG diagnosis of RVH, the majority of those diagnosed as RVH will have PAm $\geq$ 28 mmHg. Thus, these diagnostic ECG features of RVH become apparent only later. However in group A, by this set of criteria, one can detect RVH with a high frequency (90%).

The later 4 criteria in the Table II ($S_1S_2S_3$ pattern, S_1Q_3 pattern, IRBBB and slurred S in right precordial leads) were also positive in a higher frequency in group A than in group B. In eight patients, at least one was positive for these 4 criteria. Five of these 8 patients belonged to group A and electrocardiographically were diagnosed as RVH by the diagnostic set of criteria described above. Three of these 8 belonged to group B and one of these 3 had been diagnosed as RVH by diagnostic sets of criteria.

It was evident in Table II, that there was a new single criteria for diagnosis

TABLE III. *Pulmonary Hemodynamics and ECG Evidence of Arrhythmia and Left Precordial Abnormality*

Case	PAm (mmHg)	WP (mmHg)	Frequency of ECG	Left precordial abnormality		Arrhythmia	
				at the time of catheterization	at the time thereafter	at the time of catheterization	at the time thereafter
1	56	20	2	-		-	
2	53	16	4	-		-	→2 mo. after, atrial fibrillation with VPB 2/min and SVPB 1/min on next day return to sinus rhythm with SVPB 10/min
3	36	28	1	-		-	
4	35	9	1	-		-	
5	31	-	2	-		-	
6	29	14	1	-		-	
7	28	-	6	-		-	
8	28	6	2	-		-	
9	27	-	1	-		-	
10	26	10	7	-		-	
11	26	14	10	-		-	
12	25	15	32	inverted T in V4-V6	→changed its degree thereafter	-	→1 yr 4 mo. after, VPB 2/min, SVPB 1/min ↓1 yr 7 mo. SVPB 1/min
13	25	8	1	-		-	↓4 yr 4 mo. SVPB 5/min
14	25	10	4	-		-	↓4 yr 8 mo. SVPB 2/min
15	24	-	1	-		-	↓5 yr 10 mo. SVPB 3/min
16	23	5	5	-		-	↗4 mo. after, VPB 2/min
17	23	7	12	-		-	↓1 yr SVPB 2/min
18	23	6	1	-		-	↓1 yr 6 mo. SVPB 1/min
19	23	6	1	-		-	
20	22	-	5	-	→only digitalis effect	-	
21	21	5	1	-		-	
22	21	9	2	-		-	
23	20	12	1	-		-	
24	20	9	2	-		-	
25	20	10	2	-		-	
26	20	9	3	-		SVPB 2/min	
27	19	6	34	-	→1 mo. after, depressed T waves in V5, V6	-	→2 mo. after, VPB 1/min
28	19	-	1	-		-	
		total	146				

Abbreviations PAm : mean pulmonary arterial pressure, WP : mean pulmonary arterial wedge pressure, VPB : ventricular premature beat, SVPB : supraventricular premature beat

of RVH in COPD is the mean QRS axis between 90° and 180° or between −180° and −80°, in which range mean QRS axis of group A were scattered.

If one uses only this criteria for electrocardiographic diganosis of RVH, all (100%) of group A and 3 of 20 patients (15%) in group B could be diagnosed as RVH. By use this new RVH criteria of COPD, case 4 in group A is newly diagnosed as RVH, and contrarily case 21 in group B is omitted from those diagnosed as RVH by the set of criteria described above.

Table III is a summary of the pulmonary artery mean pressure (PAm) and pulmonary artery wedge pressure (WP) and ECG evidence of arrhythmias and left precordial abnormality. Frequency of electrocardiogram in the table shows the number of the serial electrocardiograms in the clinical records in each case.

WP could be measured in 6 of 8 patients in group A and 4 (cases 1, 2, 3, and 6) showed an elevated WP higher than normal 10 mmHg. In group B, WP also could be measured in 16 of 20 and 3 (cases 11, 12 and 23) showed elevated pressure. However, as seen in Table III, only one (case 12) showed inverted T-wave in leads V4-V6 at the time of catheterization and the degree changed thereafter. The other 6 patients with high WP showed no abnormality in left precordial leads at the time of catheterization and thereafter. The ECG of case 20 with a normal WP showed a left precordial ST depression due to the effect of digitalis after catheterization. One ECG in case 27 with a normal WP showed depressed T-wave in leads V5, V6 one month after catheterization but other following tracings were all normal in left precordial leads.

Arrhythmias were also looked for on the total number of 146 ECGs. Twelve ECGs of 146 (8%) showed supraventricular premature beats (SVPB)

TABLE IV. *Autopsy Findings and Electrocardiograms of Last Tracing*

Autopsy Findings									
Case	Right ventricular dilatation		Thickness of right ventricular wall		Thickness of left ventricular wall		Coronary artery disease	Survival period after catheterization	
2	+		0.6 cm		1.3 cm		-	4 mo.	
5	+		0.7 cm		1.4 cm		-	6 mo.	
12	+		0.8 cm		1.0 cm		-	6 yr	
27	+		0.7 cm		1.0 cm		-	4 yr 6 mo.	
Electrocardiograms of Last Tracing									
Case	mean QRS axis (°)	T inversion in right precord. leads	ST depre. in right precord. leads	R>S in $V3_R$ or V1 or aV_R	ST depre. in II. III.aV_F	IRBBB	Slurred S	Arrhythmia	Survival period after last Ecg tracing
2	96	$V4_R$-V1	$V4_R$-V1	+	+	-	-	-	10 days
5	112	-	-	+	-	-	-	-	5 mo.
12	-66	-	-	+	-	+	$V4_R$,$V3_R$	SVPB 3/min	3 mo.
27	80	$V4_R$-V1	-	-	-	-	$V4_R$-V1	-	3 mo.

Abbreviations IRBBB : incomplete right bundle branch block, SVPB : supraventricular premature beat

or ventricular premature beats (VPB) or atrial fibrillation. Case 2 had atrial fibrillation with VPB (2 per min) and SVPB (1 per min) 1 mo. after catheterization. This atrial fibrillation changed to sinus rhythm with frequent SVPB (10 per min). Other arrhythmias observed were all infrequent (less than 5 per min). Arrhythmias accompanied by pulmonary insufficiency were not observed.

Table IV shows autopsy findings and electrocardiograms of the last tracing of 4 cases (cases 2, 5, 12 and 27). As seen in this table, autopsy findings revealed right ventricular dilatation, abnormal thickness of right ventricular wall (thicker than normal of 4 mm or less), normal thickness of left ventricular wall (less than 15 mm) and no coronary disease. Survival periods after catheterization in cases 2, 5, 12 and 27 were 4 mo., 6 mo., 6 yr and 4 yr 6 mo., respectively. Cases 2 and 5 revealed a severe pulmonary hypertension (group A) and ECG pattern of RVH mentioned above at the time of catheterization and also on the last tracing, as seen in Table II and IV. However cases 12 and 27 revealed a relatively mild pulmonary hypertension (group B)and the ECG patterns of RVH seen in Table II were not apparent.

TABLE V. *In Case 12, ECG Fluctuations and Arterial Blood Gas and PH at or Near the Time of ECG Fluctuations*

ECG No.	Time after catheterization	mean QRS axis (°)	T inversion in right precord. leads	IRBBB	R>S in $V3_R$ or V1 or aV_R	ST depre. in II. III.aVF	$S_1S_2S_3$ pattern	Slurred S	Left precordial abnormality
1	0	-62	$V3_R$-$V4_R$	-	-	-	-	-	inverted T
		(PaO_2 50mmHg, SaO_2 82%, $PaCO_2$ 60mmHg, pHa 7.41)							
2	6 mo.	-38	-	-	-	-	-	-	-
3	11 mo.	-49	-	-	-	-	-	-	inverted T
4	12 mo.	-49	-	-	-	-	-	-	-
5	1 yr 2mo.	-34	-	-	-	-	-	-	-
6	1 yr 3 mo.	-48	-	+	-	-	-	-	-
12	2 yr 1 mo.	-60	-	+	-	-	-	-	-
13	2 yr 2 mo.	-17	-	+	-	+	-	-	ST depres.
14	2 yr 3 mo.	-17	-	+	-	-	-	-	ST depres.
15	2 yr 4 mo.	-10	-	+	-	-	-	-	-
16	2 yr 5 mo.	-10	-	+	-	-	-	-	ST depres.
22	3 yr 9 mo.	-39	-	+	-	-	-	-	-
		(PaO_2 42mmHg, SaO_2 77%, $PaCO_2$ 59mmHg, pHa 7.38)							
23	3 yr 11 mo.	-44	-	+	-	-	-	-	ST depres.
		(PaO_2 31mmHg, SaO_2 61%, $PaCO_2$ 71mmHg, pHa 7.40)							
24	4 yr 2 mo.	-77	-	Complete RBBB	-	-	-	-	-
		(PaO_2 30mmHg, SaO_2 61%, $PaCO_2$ 56mmHg, pHa 7.44)							
25	4 yr 3 mo.	-8	-	+	-	-	+	-	-
		(PaO_2 48mmHg, SaO_2 84%, $PaCO_2$ 56mmHg, pHa 7.42)							
26	4 yr 4 mo.	-28	-	+	-	-	-	-	ST depres.
	supraventricular premature beat 1/min								
	(PaO_2 31mmHg, SaO_2 64%, pHa 7.43)								
30	5 yr 1 mo.	-43	-	+	-	-	-	-	-
		(PaO_2 72mmHg, SaO_2 94%, $PaCO_2$ 73mmHg, pHa 7.41)							
31	5 yr 5 mo.	-58	-	+	+	-	-	$V4_R$	-
32	5 yr 10 mo.	-66	-	+	+	-	-	$V3_R$	-
		(PaO_2 50mmHg, SaO_2 86%, $PaCO_2$ 76mmHg, pHa 7.42)							

Abbreviations as in Tables I and II.

Therefore, we were interested in looking for the fluctuations of electrocardiograms in cases 12 and 27 and could measure the electrocardiograms on 32 tracings of case 12 and on 34 tracings of case 27. In case 12, electrocardiographic fluctuations and blood gas and pH at or near the time of electrocardiographic fluctuations are summarized in Table V, and in case 27 in Table VI. Criteria used but not presented in Tables IV, V, VI are those with which no tracing was in conformity.

As shown in Table V (of case 12) mean QRS fluctuated in the range of $-8°$ to $-77°$ (left axis deviation). T-wave inversion in leads V3R-V4R seen on ECG No. 1 was not seen in the following tracings, despite severe anoxemia (PaO_2 were between 38 mmHg and 41 mmHg) which was seen during the time between 3 yr 9 mo. and 4 yr 6 mo. after catheterization. In these hypoxemic periods, we could find infrequent SVPB (1 per minute) on only one tracing in 5.

IRBBB was first seen on ECG No. 6 taken 15 mo. after catheterization. This IRBBB was evident until the day last tracing taken. R > S in leads V3R, V1 and slurred S in leads V4R, V3R were seen at the first time on

TABLE VI. *In Case 27, ECG Fluctuations and Arterial Blood Gas and PH at or Near the Time of ECG Fluctuations*

ECG No.	Time after catheterization	mean QRS axis (°)	T inversion in right precord. leads	IRBBB	R<S in V5	ST depre. in II. III.aV$_F$	both $S_1S_2S_3$ and S_1Q_3 pattern	Slurred S	Left precordial abnormality
4	0	42	V4R-V3	-	-	-	-	-	-
	(PaO_2 60mmHg, SaO_2 93%, $PaCO_2$ 35mmHg, pHa 7.53)								
5	1 mo.	49	V4R-V3	-	-	+	+	-	T depression
6	2 mo.	56	V4R-V1	-	-	-	-	-	-
7	3 mo.	37	V4$_R$-V1	-	-	-	+	-	-
	ventricular premature beat 1/min								
18	1 yr 4 mo.	56	V4$_R$-V1	-	-	-	-	V4R-V2	-
19	1 yr 5 mo.	58	V4$_R$-V1	-	-	-	-	V4R-V2	-
	supraventricular premature beat 1/min								
20	1 yr 6 mo.	46	V4R-V2	-	-	-	+	V4R-V2	-
	supraventricular premature beat 1/min								
21	1 yr 7 mo.	72	V4$_R$-V1	-	-	-	+	V4R-V2	-
26	2 yr 7 mo.	59	V4$_R$-V1	-	-	-	+	V4R-V2	-
	(PaO_2 55mmHg, SaO_2 89%, $PaCO_2$ 40mmHg, pHa 7.44)								
27	2 yr 9 mo.	67	V4$_R$-V2	-	-	-	+	V4R-V2	-
	(PaO_2 46mmHg, SaO_2 82%, $PaCO_2$ 39mmHg, pHa 7.42)								
28	3 yr 1 mo.	65	V4$_R$-V1	-	+	-	+	V4R-V2	-
	(PaO_2 31mmHg, SaO_2 61%, $PaCO_2$ 68mmHg, pHa 7.39)								
29	3 yr 2 mo.	62	V4$_R$-V1	-	+	-	-	V4R-V2	-
	(PaO_2 31mmHg, SaO_2 61%, $PaCO_2$ 57mmHg, pHa 7.39)								
30	3 yr 3 mo.	54	V4$_R$-V1	-	-	-	-	-	-
	(PaO_2 53mmHg, SaO_2 87%, $PaCO_2$ 48mmHg, pHa 7.41)								
31	3 yr 7 mo.	61	V4$_R$-V2	+	-	-	-	-	-
	(PaO_2 54mmHg, SaO_2 85%, $PaCO_2$ 50mmHg, pHa 7.33)								
32	3 yr 8 mo.	70	V4$_R$-V2	+	-	-	+	-	-
	(PaO_2 48mmHg, SaO_2 83%, $PaCO_2$ 45mmHg, pHa 7.38)								
33	4 yr	90	V4$_R$-V1	-	-	-	+	V4$_R$-V2	-
	(PaO_2 32mmHg, SaO_2 59%, $PaCO_2$ 51mmHg, pHa 7.33)								
34	4 yr 3 mo.	80	V4$_R$-V2	-	-	-	+	V4$_R$-V1	-
	(PaO_2 32mmHg, SaO_2 63%, $PaCO_2$ 75mmHg, pHa 7.39)								

Abbreviations as in Tables I and II.

ECG No. 31 and also seen on last ECG No. 32. ST depression in leads II, III, aVF was seen on ECG No. 13 only once. $S_1S_2S_3$ pattern was seen on ECG No. 25 only once.

On ECGs No. 1 and 3, inverted T-waves in left precordial leads were observed. On ECGs No. 13, 14, 16, 23 and 26, ST depression in left precordial leads was seen. These left precordial abnormalities seemed to be nonspecific, and were not seen during the last one yr.

The electrocardiograms of the last tracing of this case are summarized in Table IV. This electrocardiogram was neither in conformity with set criteria of RVH nor with our new single criteria. On this ECG, not all of limb leads and lead V6 were less than 0.7 mm in QRS voltage.

As shown in Table VI (of case 27), mean QRS axis fluctuated in the range of 37° to 90°. On the ECG before last (No.33) mean QRS axis shift of over 30° to the right of the mean QRS axis shown 9 mo. before. T-wave inversions in leads V4R-V3 were seen on the ECG at the time of catheterization and the sphere of the inverted T-wave fluctuated thereafter until the day last tracing taken. IRBBB were first seen on ECG No. 31 and also on the next No. 32.

Severe hypoxemias (PaO_2 between 31 and 32 mmHg) occurred at or near time of ECG No. 28, 33 and 34. R < S in lead V5 was first seen on ECG No. 28 and seen on next tracing and never seen thereafter. ECGs taken during these severe hypoxic periods showed no evidence of arrhythmia.

ST depression in leads II, III and aVF was seen on ECG No. 5 only once. Both $S_1S_2S_3$ and S_1Q_3 patterns were first seen on ECG No. 5 taken 1 mo. after catheterization. These patterns were often seen thereafter. Slurred S in leads V4R-V2 (or V1) was seen on ECG No. 18 taken 16 mo. after catheterization. These slurred S were seen until the time of 37 mo. after catheterization. One yr later these slurred S reappeared and continued until the day of the last tracing. Only one ECG of No. 5 showed T-depression in left precordial leads.

Electrocardiograms of the last tracing of this case are summarized in Table IV. This electrocardiogram was neither in conformity with set criteria of diagnosis of RVH nor with our new single criteria. On this ECG, all of limb leads and lead V6 were less than 7 mm in QRS voltage. Therefore, these findings were in conformity with Selvester and Rubins' set criteria of RVH in cases of emphysema (5).

DISCUSSION

Electrocardiographic criteria for right ventricular hypertrophy (RVH) have been suggested by many workers (1—9). However, in patients with chronic obstructive pulmonary disease (COPD), the electrocardiographic diagnosis of RVH is more difficult than in patients of congenital heart disease or mitral stenosis.

In this report, we used Kilcoyne's diagnostic set of criteria of cor pulmonale

(3). Performing this study, we noticed a new single criteria, that is mean QRS axis between 90° and 180° or between −180° and −80°. This criteria is simillar to Millard's criteria which is a mean QRS axis between +90° and ±180° (6). We found that the majority of patients diagnosed as RVH, by our new criteria, and also by Kilcoynes' criteria, had a pulmonary artery mean pressure (PAm) over 27 mmHg. Thus ECG sings of RVH become evident only late during the course.

Two patients (cases 2 and 5) diagnosed as RVH by ECG criteria at the time of catheterization died 4 mo. and 6 mo. after catheterization, respectively. Autopsy findings of these 2 cases revealed RVH.

Two other cases (cases 12 and 27) not diagnosed as RVH by ECG criteria at the time of catheterization died 6 yr and 4 yr 6 mo. after catheterization, respectively. Their autopsy findings also revealed RVH. In these 2 cases, electrocardiographic fluctuations and blood gas changes were observed and described.

The last ECGs of both were neither in conformity with Kilcoyne's set criteria nor with our new criteria. However, the last ECG of case 12 showed IRBBB and $R > S$ in leads $V3_R$ and V1. Therefore, we think this set of criteria may be one criteria for RVH. Last ECG of case 27 showed low voltage (QRS in limb leads and lead V6 were less than 7 mm) and slurred S in right precordial leads. These findings were in conformity with Selvester and Rubins' set criteria of RVH in emphysema (5).

Regarding left ventricular abnormality, Rao *et al.* reported 8 patients with left ventricular failure secondary to chronic pulmonary disease (10). However, we did not experience such a case. Baum *et al.* reported abnormal left ventricular function in COPD (11). Contrarily, Williams *et al.* (12), and Frank *et al.* (13) reported normal left ventricular function in COPD.

In our study, we measured pulmonary artery wedge pressure (WP) and observed 7 patients with an elevated WP. However, 6 patients showed no abnormality in left precordial leads. Increased intrathoracic pressure may contribute to the increase of WP. Fishman reported that WPs were unreliable indexes of left atrial pressure in COPD since either vasoconstriction produced by hypoxia or anatomic changes in the intervening vessel might invalidate these relationships (14). In the autopsy of our 4 patients with RVH, neither left ventricular hypertrophy nor coronary heart disease were observed.

With regard to the prevalence of arrhythmias in patients of chronic cor pulmonale, Corazza and Pastor reported 31 per cent had one or more arrhythmias. In their study they omitted infrequent premature beats (less than 5 per min) from the number of arrhythmias (15).

Hudson *et al.* also reported that in their COPD patients, 70 per cent of those with ventricular arrhythmia (included ventricular bigeminy, AV dissociation, idioventricular rhythm, ventricular tachycardia and cardiac arrest) died during the hospital admission (16).

Holford and Mithoefer reported that by continuous tape recording of the electrocardiogram they observed cardiac arrhythmias sufficient in severity to

require therapy among the 57 per cent of hospitalized patients with COPD (17).

However, it was our impression that occurrence of arrhythmias was not so frequent in hospitalized patients with COPD during bouts of respiratory insufficiency. Therefore we studied retrospectively the prevalence of arrhythmias on 146 tracings. As Hudson *et al.* described, a review of electrocardiograms in the clinical record, the method used in this study, might underestimate the true prevalence of arrhythmias, but was identified in most of the sustained arrhythmias (16). The number of ECGs which revealed arrhythmias was 12 of 146 ECGs and of 5 of 28 cases. In case 2, transient atrial fibrillation and frequent supraventricular premature beats (SVPB) were seen. Arrhythmias of other cases were seen as infrequent premature beats. Therefore the prevalence of arrhythmia (except for the infrequent premature beats), was one in 28 cases (4%).

This observation seemed to confirm our clinical impression. Frequency of occurrence of arrhythmia of our patients of COPD was markedly less than the frequencies of arrhythmia of hospitalized Americans with COPD reported by Corazza and Pastor (15) or by Hudson *et al.* (16). Holford and Mithoefer described that 34 per cent of patients with COPD had coronary artery disease and that this incidence of coronary artery disease was consistent with findings of others (17). However, autopsy findings of our 4 patients with RVH revealed no evidence of coronary artery disease. Therefore, this difference in the prevalence of arrhythmias between in Japanese and Americans may be attributed to the difference of the degree of clinical and subclinical ischemic degenerative changes in the heart. To obtain the true prevalence of arrhythmia, continuous monitoring techniques are required. Such an approach is currently being used in our clinic.

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