THE ELECTROCARDIOGRAM IN ASPHYXIAL AND IN EMBOLIC ACUTE COR PULMONALE

BY

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Acute respiratory failure with alveolar under-ventilation is often due to obstruction to the airways occurring in acute exacerbations of chronic bronchitis and in severe asthma. The consequent hypoxæmia must lead to acute elevation of the pulmonary arterial pressure (Motley *et al.*, 1948; Fishman *et al.*, 1952), but the severity of the illness is such that this has only infrequently been demonstrated (Lenègre, 1950; Lovejoy *et al.*, 1954). This form of acute cor pulmonale may be termed "acute asphyxial cor pulmonale" to distinguish it from that due to pulmonary embolism.

The signs of acute respiratory failure and acute asphyxial cor pulmonale include cyanosis, tachycardia, sweating, hypotension, and gallop rhythm (Westlake *et al.*, 1955; Sieker and Hickam, 1956). The onset is frequently sudden, and often an electrocardiogram is taken to exclude pulmonary embolism or myocardial infarction. Little attention has been paid to the cardiogram in this form of acute cor pulmonale although Mack *et al.* (1950) and Turiaf (Turiaf and Thin, 1954; Turiaf and Blanchon, 1954; and Georges *et al.*, 1957) have emphasized its similarity to that of pulmonary embolism.

The purpose of the present investigation is to study the electrocardiogram in acute asphyxial cor pulmonale and to compare it with that in acute embolic cor pulmonale.

MATERIAL AND METHODS

A study was made of the case records of all patients who came to necropsy at Hammersmith Hospital between January 1, 1959 and March 31, 1961 with the diagnosis of chronic bronchitis, emphysema, asthma, or pulmonary embolism or infarction, irrespective of any other diagnosis recorded. The diagnosis of acute respiratory failure was made when a rapidly deteriorating clinical state characterized by dyspnœa, increased sputum, widespread pulmonary signs, cyanosis, tachycardia (heart rate of 100 a minute or more), altered consciousness (and in some cases, hypotension and muscular twitching) occurred in a patient with chronic bronchitis, emphysema, or asthma. The diagnosis was confirmed by the finding at necropsy of an adequate cause for acute respiratory failure, such as severe obstruction to the airways by widespread purulent bronchitis or mucus plugging of the bronchi, or extensive bronchopneumonia, and no other condition likely to have given rise to the clinical episode in question. In eleven patients an adequate electrocardiogram had been taken during an episode of acute respiratory failure. Eight of the eleven died within a week of the electrocardiogram studied.

In twelve patients with pulmonary embolism or infarction post mortem, a cardiogram had been recorded during an episode clinically characteristic of pulmonary embolism. Four of

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these twelve also had chronic bronchitis, and thus form a separate group, in which severe chronic pulmonary disease and pulmonary embolism are combined.

The electrocardiographic criteria and definitions used are given below. The mean frontal plane P wave axis ($\hat{A}P$) was measured in the same manner as the QRS axis from the relative amplitudes of the P wave in the standard leads, by the application of Einthoven's triangle. Calculation is made easier if 1 mm. of P wave amplitude is equated, in whichever diagram is used, with 5 or 10 mm. of QRS amplitude, according to convenience. In this study, the diagram of Carter *et al.* (1919) was employed.

<i>Electrocardiographic Sign</i>	<i>Definition</i>
<i>P waves</i>	
P wave axis ($\hat{A}P$)	Mean frontal plane P wave axis
Right atrial P wave	Pointed P wave of amplitude 2.5 mm. or more in lead II, III, or aVF
<i>QRS pattern</i>	
<i>Standard leads</i>	
Q wave	Amplitude 2 mm. or more
S wave	Amplitude equal to or greater than corresponding R wave
<i>Præcordial leads</i>	
Rs	R greater than S
rs	R equal to S
rS	S greater than R
<i>S-T segment</i>	Depression or elevation of 1 mm. or more
<i>T waves</i>	Classified as upright, flat, biphasic, or inverted

RESULTS

Acute Respiratory Failure. Table I shows the clinical and pathological data from the eleven patients with acute respiratory failure. Nine patients had chronic bronchitis, one asthma, and one a post-traumatic "frozen" hemithorax. Two had had a pulmonary lobectomy for bronchial carcinoma, and one had an untreated lung cancer.

The right ventricle was 5 mm. or more thick in the outflow tract in all but two, and the right atrium was abnormal in most. Three had left ventricular hypertrophy. The degree of coronary arterial disease was variable, but was usually slight, and the myocardium was normal in all but one. The degree of emphysema varied greatly.

Table II shows the electrocardiographic findings. All eleven patients were in sinus rhythm at the time of their episode of acute respiratory failure. The P wave axis was $+70^\circ$ or more in all but one, and $+90^\circ$ or more in five. A sharply pointed P wave indicating right atrial enlargement was present in four. One patient had complete right bundle-branch block, and his electrocardiogram will not be considered further. Significant Q waves were present in lead I in two patients, but were never seen in lead III. S waves were present in six, of whom two had S waves in lead I alone, three in leads I and III, one in leads II and III, and one in all three standard leads. A dominant R wave was seen in lead aVR in five. In the præcordial leads there was an rs or rS pattern in lead V5 in eight, in lead V6 in seven, and in lead V7 in six. T wave inversion in leads II and III was probably attributable to digitalis in Case 1 (Fig. 1); in no other patient was the T wave inverted in lead III, but one showed an inverted T wave in lead I (Case 4, Fig. 2). The S-T segments were elevated in the right præcordial leads in six. The T waves were inverted in leads V2 and V3 in only two.

Fig. 3 shows the reversion towards normal of the electrocardiogram after two episodes of extremely severe asthma in a middle-aged woman, whose case is not included in the main series. The P

TABLE I
CLINICAL AND PATHOLOGICAL DATA IN 11 CASES OF ACUTE RESPIRATORY FAILURE

Case No.	Sex	Age	Clinical diagnosis	Cardiac pathology						Pulmonary pathology
				R.A.	R.V.*	Chambers		Coronary arterial disease	Myocardium	
						L.A.	L.V.*			
1	F	69	Chronic bronchitis; left upper lobe tuberculosis	HD	10	N	15	Slight	Normal	Purulent bronchitis; old cavitation left upper lobe; much pleural thickening; no emphysema. (Secondary amyloidosis).
2	M	70	Chronic bronchitis; tracheostomy	HD	8	N	18	Moderate	Normal	Hæmorrhagic tracheobronchitis; confluent bronchopneumonia; abscesses both lower lobes; moderate emphysema.
3	M	63	Chronic bronchitis	D	7	N	17	Slight	Normal	Purulent bronchitis; bronchopneumonia; slight emphysema
4	F	49	Asthma; tracheostomy; IPPR	D	5	N	14	Slight	Normal	Gross mucus plugging of bronchi in all areas
5	F	60	Chronic bronchitis	HD	7	—	13	None	Normal	Much bronchial mucus; severe centrilobular emphysema
6	F	58	Chronic bronchitis	—	9	N	N	Slight	Normal	Purulent bronchitis; no emphysema
7	M	70	Chronic bronchitis	D	6	N	15	Slight	Normal	Purulent bronchitis; widespread emphysema
8	M	60	Carcinoma bronchus; left upper lobectomy and radiotherapy	D	5	N	15	None	Normal	Bronchopneumonia; centrilobular emphysema in R lung; left upper lobectomy; no recurrence of carcinoma
9	M	63	L. "frozen chest"; carcinoma bronchus	N	3	N	14	Slight	Normal	L. empyema with calcified pleura; fibrosis left upper lobe and right lower lobe; consolidation right upper and middle lobes; multiple secondaries
10	M	61	Chronic bronchitis; left lower lobectomy for carcinoma	N	3	N	15	Moderate	Normal	Left lower lobectomy; gross mucus plugging of all bronchi; no emphysema; no recurrence of carcinoma
11	M	61	Chronic bronchitis; morphine	D	7	—	17	Slight	Fibrosis posterior L.V.	Purulent bronchitis; severe bullous emphysema

IPPR=intermittent positive pressure respiration. —=not recorded. N=normal. R.A.=right atrium. R.V.=right ventricle. H=hypertrophied. D=dilated. L.A.=left atrium. L.V.=left ventricle.

* Thickness (mm.) in outflow tract.

† Greatest thickness (mm.).

FIG. 1.—Electrocardiogram of Case 1 (asphyxial cor pulmonale) showing $\hat{A}P + 70^\circ$, right atrial P wave, S in lead I, dominant R in aVR, rS pattern in leads V5 to V7, S-T segment depression, and T wave inversion in leads II and III (possibly attributable to digitalis), and upright T waves in leads V2 to V7.

TABLE II
ELECTROCARDIOGRAPHIC FINDINGS IN 11 CASES OF ACUTE RESPIRATORY FAILURE

Case No.	P waves		QRS pattern						S-T segments		T waves		Remarks
	ÂP	R atrial P wave	Standard leads	Lead aVR	Præcordial leads						Standard leads	Præcordial leads	
					V4R	V1	V5	V6	V7				
1	+70°	+	S ₁	R	R	qR	rS	rS	rS	Depr. II,III	Inv. II,III	Inv. V4R, V1; upr. V2-7	Digitalis, see Fig. 1
2	+70°	—	RBBB	RBBB	RBBB						RBBB		
3	+75°	+	S ₁ S ₂	rsR'	Qr	rsR'	rS	rs	Rs	Normal	Elev. V2-4	Inv. V4R, V1; upr. V2-7	Incomplete RBBB
4	+100°	+	Q ₁	Q	rS	rS	rS	rS	rS	Normal	Elev. V2-4	Upr. V4R-V7	See Fig. 2
5	+90°	—	QS ₁	QS	QS	QS	rS	rS	rS	Normal	Normal	Inv. V4R, V1; upr. V2-7	
6	+70°	+	S ₁ S ₂	qR	rS	rS	rS	rS	rS	Normal	Elev. V2-3	Inv. V4R, V1; upr. V2-5; flat V6-7	Digitalis
7	+90°	—	Normal	QS	QS	QS	Rs	Rs	Rs	Normal	Elev. V1-4	Inv. V4R-V3; upr. V4-7	Q in V4 See Fig. 5
8	+75°	—	S ₁ S ₂	qR	qr	qR	rS	rS	rS	Normal	Elev. V1, Depr. V7	Inv. V4R, V1; upr. V2-7	Incomplete RBBB
9	+60°	—	S ₂ S ₃	rsr'	rS	rS	rS	Rs	Rs	Normal	Normal	Inv. V4R, V1; upr. V2-7	
10	+90°	—	Normal	rS	rS	rS	Rs	Rs	Rs	Normal	Depr. V3-7	Upr. V4R to V4; flat V5-7	
11	+90°	—	S ₁ S ₂ S ₃	R	rS	rS	rS	rS	rS	Normal	Normal	Inv. V4R-V3; flat V4-7	

+ = present. Elev. = elevated. Upr. = upright. ÂP = P wave axis. ÂP = P wave axis. — = absent. Depr. = depressed. Inv. = inverted. RBBB = Right bundle-branch block.

TABLE III
CLINICAL AND PATHOLOGICAL DATA IN 12 CASES OF PULMONARY EMBOLISM

	Case No.	Sex	Age	Clinical diagnosis	Cardiac pathology						Pulmonary pathology
					Chambers		L.A.	L.V.	Coronary artery disease	Myocardium	
					R.A.	R.V.					
Cases without chronic lung disease	12	F	75	Carcinoma of rectum; lung metastases	N	3	N	12	Slight	Normal	Small metastases in both lungs; massive pulmonary embolism
	13	F	81	Paget's disease; amputation of L. leg	N	5	Thr	15*	Slight	Normal	Embolus infarct right upper lobe
	14	F	75	Temporal arteritis	N	2	N	15	Slight	Normal	Massive pulmonary embolism
	15	F	90	Fractured femur	N	4	N	20	Severe	Posterior fibrosis	Multiple old emboli in left lower lobe artery; recent emboli in right upper lobe
	16	F	38	Aortic and mitral valve disease, aortic and mitral valvotomy	D	5	H†	16†	None	Normal	Embolus infarction of both lower lobes
	17	F	67	Primary biliary carcinoma	N	3	N	13	None	Normal	Old emboli left lower lobe; massive pulmonary embolism
	18	F	80	Rheumatoid arthritis; myxoedema, chronic myeloid leukaemia	N	3	N	15	Slight	Normal	Multiple embolic infarcts in both lower lobes
	19	F	60	Carcinoma of ovary	—	3	—	20	Slight	Normal	Massive pulmonary embolism; infarction right middle and upper lobes
Cases with chronic lung disease	20	M	66	Carcinoma of lung; inferior vena caval thrombosis	N	3	N	14	Slight	Normal	Multiple small emboli with septic infarcts in right upper lobe and right lower lobe; moderate centrilobular emphysema
	21	M	65	Chronic bronchitis; acute renal tubular necrosis	N	8	N	17	None	Normal	Diffuse bronchiectasis; panacinar emphysema; purulent bronchitis; multiple embolic infarcts
	22	M	54	Chronic bronchitis	H	7	N	18	Slight	Normal	Severe emphysema; multiple emboli in small pulmonary arteries
	23	M	63	Chronic bronchitis	D	9	—	18	Moderate	Normal	Purulent bronchitis; moderate emphysema; embolic infarcts in both upper lobes

Symbols as in Table I. Thr=thrombus.

* Slight mitral stenosis; acute bacterial endocarditis of aortic valve.

† Mitral and aortic stenosis relieved.

waves diminish in amplitude in leads II and III, and the P wave axis shifts to the left. The rS pattern disappears from the left precordial leads. The T waves are upright.

The usual electrocardiographic pattern in acute respiratory failure consists of sinus tachycardia with a P wave axis of $+70^\circ$ or more, an rS pattern in leads V5 to V7, and upright T waves in leads V2 to V7 (Fig. 1-3). A P wave axis of $+90^\circ$ or more (Fig. 2), deep S waves in leads I and II, and dominant R waves in lead VR are often seen (Fig. 1). These changes may be transient (Fig. 3). Q waves were not seen in lead III, and inversion of T in lead III was infrequent.

Pulmonary Embolism. Table III shows the clinical and pathological findings in the twelve patients with pulmonary embolism, including the four (Cases 20 to 23) in whom this occurred in the presence of severe chronic pulmonary disease. The heart was anatomically abnormal in seven patients.

Of the eight patients without chronic pulmonary disease, three showed atrial fibrillation (Table IV). The P wave axis was $+70^\circ$ or more in three of the five with sinus rhythm. None showed a right atrial P wave. One patient had left bundle-branch block, and her electrocardiogram will not be considered further. Significant Q waves were present in lead III in two, and S waves in the standard leads in four. Two patients had an rs pattern in lead V5, and one in lead V6, but none in lead V7. S-T segment depression was seen in the standard leads in five, and in the left precordial leads in six. The T waves were inverted in one or more standard leads in five, and in lead V3 in three.

The pattern seen in the four patients with both chronic pulmonary disease and pulmonary embolism is of great interest. In Case 22 there is doubt about the relationship in time between the

FIG. 2.—Electrocardiogram of Case 4 (asphyxial cor pulmonale), showing $\hat{A}P + 100^\circ$, right atrial P wave, Q in lead I, rS in all præcordial leads, and T inverted in lead I but upright in all præcordial leads.

electrocardiogram studied and the many small pulmonary emboli found at necropsy; he had chronic hypoxæmia and hypercapnia, and repeated episodes of severe dyspnœa and tachycardia. All the three patients with sinus rhythm showed right atrial P waves. One had S waves in all three standard leads (Fig. 4), another S in lead I. Two had dominant R waves in lead aVR. All four had an rs or rS pattern in lead V5 (Fig. 4), and two an rS pattern in leads V6 and V7. The T wave was inverted in lead III in one, and in leads V3 and V4 in one.

DISCUSSION

Comparison between the electrocardiogram of acute asphyxial cor pulmonale and that of pulmonary embolism can be made if our data on the electrocardiogram in pulmonary embolism are amplified by reference to the substantial literature on the subject (McGinn and White, 1935; Phillips and Levine, 1950; Zuckerman *et al.*, 1950; Laham and Gerbaux, 1951; Soulié *et al.*, 1951; Eliaser and Giansiracusa, 1952; and Cutforth and Oram, 1958).

Cardiac Rhythm. None of the patients with acute asphyxial cor pulmonale showed an arrhythmia during the episode in question, although Case 11 had a variety of arrhythmias on other occasions. Thus, although supraventricular arrhythmias are not rare in chronic pulmonary disease, their presence should suggest a complication other than acute respiratory failure. Atrial fibrillation (Sagall *et al.*, 1954; Short, 1952) and other arrhythmias (Zuckerman *et al.*, 1950; Laham and Gerbaux, 1951) are of course not infrequently precipitated by pulmonary embolism.

P Wave Changes. A shift to the right in the P wave axis is characteristic of the electrocardiogram of patients with airways obstruction (Novelo, 1945; Spodick, 1959; Caird and Wilcken, 1961). Table III shows that the same is true of acute asphyxial cor pulmonale, the most striking example being Case 4, with a P wave axis of $+100^\circ$ (Fig. 2). Turiaf and Thin (1954) and George,

TABLE IV
ELECTROCARDIOGRAPHIC FINDINGS IN 12 CASES OF PULMONARY EMBOLISM

	P waves		QRS patterns							S-T segments		T waves		Remarks
Case No.	ÂP	R atrial P wave	Standard leads	Lead aVR	Præcordial leads				Standard leads	Præcordial leads	Standard leads	Præcordial leads		
					V4R	V1	V5	V6	V7					
Cases without chronic lung disease	12	+70°	—	rs	S	rs	Rs	Rs	Rs	Depr. 1, 2, 3	Depr. V3-7	Inv. 1, 2, 3	Upr. V4R-V7	Digitalis
	13	+90°	—	QS	QS	QS	R	R	R	Normal	Depr. V4-5	Normal	Inv. V4R-V3 upr. V4-7	
	14	A.F.	—	qr	—	rs	Rs	Rs	—	Depr. 1, 2	Depr. V5-6	Inv. 1, 2, 3	Biphasic V6	
	15	+60°	—	LBBB	LBBB	LBBB	LBBB	LBBB	LBBB	LBBB	LBBB	LBBB	LBBB	
	16	A.F.	—	S ₁	qR	qr	rs	rs	Rs	Depr. 2, 3	Depr. V4-7	Inv. 2	Inv. V4R-V5; upr. V6-7	
	17	+30°	—	Q ₃	Qr	QS	rs	RS	Rs	Rs	Normal	Elev. V1-3	Normal	
Cases with chronic lung disease	18	A.F.	—	QS	rs	rs	rs	rs	Rs	Depr. 1	Depr. V5-7	Inv. 1	Upr. V4R-V7	See Fig. 4
	19	+90°	—	QS	qr	rs	R	R	R	Depr. 3	Depr. V5-7	Inv. 3	Inv. V4R-V4; upr. V5-7	
	20	+90°	+	Normal	rSr'	rs	rs	rs	Rs	Normal	Normal	Normal	Upr. V4R-V7	
Cases with chronic lung disease	21	+90°	+	S ₁ S ₂ S ₃	Q	Rs	rs	Rs	Rs	Normal	Depr. V5-7	Normal	Inv. V4R, V1; biphasic V2; upr. V3-6	Digitalis
	22	+75°	+	S ₁	rs	rs	rs	RS	rs	Depr. 3	Elev. V2-4	Inv. 3	Inv. V4R, V1; upr. V2-4; biphasic V5-7	
	23	A.F.	—	Normal	qR	QS	rs	rs	rs	Normal	Elev. V4R-V3; depr. V6-7	Normal	Inv. V4R-V4; upr. V5; biphasic V6-7	

A.F. = atrial fibrillation. Symbols as in Table II. LBBB = Left bundle-branch block.

FIG. 3.—Electrocardiograms of a 58-year-old woman with asthma. Records of 23/9/58 and 23/11/60 taken during episodes of severe status asthmaticus show tall P waves and extreme clockwise rotation (rS in leads V5 to V7). Records of 29/9/58 and 5/12/60 taken during remissions show reversion towards normal.

et al. (1957) record similar findings in status asthmaticus. A shift in P wave axis is usually stated to be rare in pulmonary embolism (Laham and Gerbaux, 1951), but an axis of $+90^\circ$ can certainly occur in pulmonary embolism in the absence of chronic pulmonary disease (Delanoë, 1951; Eliaser and Giansiracusa, 1952, Case 3; Cases 13 and 19 in this series). Too much reliance cannot therefore be placed on this sign for the exclusion of pulmonary embolism, although a P wave axis of less than $+60^\circ$ should be regarded as evidence against a cardiographic diagnosis of acute asphyxial cor pulmonale.

A right atrial P wave is considered rare in pulmonary embolism, though there were six examples among Cutforth and Oram's (1958) fifty patients. It was found in four of our eleven patients with acute asphyxial cor pulmonale (Fig. 1) and in three of the four with pulmonary embolism complicating chronic pulmonary disease (Fig. 4). Thus a right atrial P curve should suggest chronic pulmonary disease rather than pulmonary embolism. It cannot assist in the differentiation of acute asphyxial from acute embolic cor pulmonale in patients with chronic pulmonary disease.

QRS Patterns. The characteristic alteration in QRS pattern in the standard leads in acute embolic cor pulmonale consists of the development of an S wave in lead I and sometimes lead II

FIG. 4.—Electrocardiogram of Case 21, showing $\hat{A}P + 90^\circ$, right atrial P wave, S waves in all standard leads, dominant R in lead aVR, rS in lead V5, and biphasic T in lead V2. (Necropsy examination showed multiple embolic pulmonary infarcts in addition to severe emphysema.)

and of a Q wave in lead III (McGinn and White, 1935). Mack *et al.* (1950) noted similar changes in respiratory failure. Deep S waves were seen in lead I in six of our eleven patients with acute asphyxial cor pulmonale, and in lead II in four. Q waves occurred in lead I in two, but not in lead III in any case. The presence of Q in lead III may be evidence against a diagnosis of acute asphyxial cor pulmonale.

A dominant R wave may develop in lead aVR in pulmonary embolism (Friedberg, 1956). This sign was present in five of the eleven patients with acute respiratory failure and two of the four with both pulmonary embolism and chronic pulmonary disease.

In pulmonary embolism there is frequently a shift to the left in the transition zone in the præcordial leads (Phillips and Levine, 1950; Laham and Gerbaux, 1951; Soulié *et al.*, 1951; Eliaser and Giansiracusa, 1952; Cutforth and Oram, 1958). The electrocardiograms illustrated by these authors suggest that the S wave rarely becomes dominant in leads V5 or V6. Indeed two of the cases showing clockwise rotation of this degree had in fact not pulmonary embolism but respiratory failure (Phillips and Levine, 1950, Case 7; Laham and Gerbaux, 1951, Case 14). Of our ten patients with acute asphyxial cor pulmonale and normal QRS complexes, there was an rs or rS pattern in lead V5 in eight, in lead V6 in seven, and in lead V7 in six. An rS pattern in lead V5 was seen in three of the four patients with pulmonary embolism and chronic pulmonary disease, and in leads V6 and V7 in two patients. Extreme "clockwise rotation" would thus seem to be one of the most frequent electrocardiographic signs of acute asphyxial cor pulmonale, though there may on occasion be no clockwise rotation at all, as Fig. 5 shows. It will not, however, serve to exclude pulmonary embolism in patients with chronic pulmonary disease. In both instances there is an exaggeration of the clockwise rotation with an rS pattern in lead V5, which is often the first and only sign of right ventricular hypertrophy in chronic pulmonary disease (Carouso *et al.*, 1951). This exaggeration may be due to dilatation of the right ventricle (Zuckerman *et al.*, 1950), or to positional changes associated with an increase in lung volumes consequent on obstruction to the airways, or to both. Studies of the electrocardiogram in acute respiratory failure in conditions not accompanied by obstruction to the airways might clarify this point.

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FIG. 3.—Electrocardiograms of Case 7. Record of 23/3/60 taken during acute respiratory failure shows Q in lead V4, S-T elevation in leads V1 to V4, and no clockwise rotation. Record of 6/4/60 shows disappearance of Q in V4, but further T wave changes in the præcordial leads. Necropsy examination showed no evidence of myocardial infarction.

Fig. 3 illustrates the regression of extreme clockwise rotation on recovery from acute respiratory failure. Thus the electrocardiographic signs tend to be transient.

Conduction Defects. Complete or incomplete right bundle-branch block is seen on occasion in pulmonary embolism (Soulié *et al.*, 1951; Cutforth and Oram, 1958). Complete right bundle-branch block was present in one and incomplete in two of our eleven patients with acute asphyxial cor pulmonale.

S-T Segment and T Wave Changes. In pulmonary embolism the standard leads may show depression of the S-T segment with upright T waves, giving rise to the "staircase pattern" (McGinn and White, 1935). In the præcordial leads there may be depression on the left (Soulié *et al.*, 1951; Laham and Gerbaux, 1951; Cutforth and Oram, 1958), and elevation on the right (Laham and Gerbaux, 1951). S-T segment depression was seen in lead I in only one case of acute asphyxial cor pulmonale, and then was probably attributable to digitalis (Fig. 1). Changes in the præcordial leads were more frequent, since elevation of the S-T segment occurred in the right præcordial leads in six cases (Fig. 1 and 2).

T wave inversion in lead III and over the right præcordium is characteristic of acute embolic cor pulmonale. Only one of the eleven patients with acute asphyxial cor pulmonale showed T wave inversion in lead III (and this was probably due to digitalis) (Fig. 1), and only two patients showed inversion in leads V2 or V3 (Fig. 5). Of the four patients with pulmonary embolism and chronic

pulmonary disease one had T wave inversion in lead V3. Thus T wave inversion in the right præcordial leads is rare in acute asphyxial cor pulmonale. Its presence may be used as evidence against that electrocardiographic diagnosis, but it is often absent also when pulmonary embolism complicates chronic pulmonary disease (Fig. 4).

SUMMARY

A comparison has been made of the electrocardiogram of 11 patients with acute respiratory failure, 8 with pulmonary embolism, and 4 with pulmonary embolism complicating chronic pulmonary disease.

In acute respiratory failure the electrocardiogram characteristically shows sinus tachycardia, a P wave axis of $+70^\circ$ or more, extreme "clockwise rotation" (rS pattern in leads V5 to V7), and upright T waves in leads III and V2 to V7. A right atrial P wave is frequent.

In acute respiratory failure, in contrast with pulmonary embolism, there is only rarely a Q wave in lead III, S-T segment depression in leads I and II, or T wave inversion in lead III or over the right præcordium.

A dominant R wave in lead aVR and deep S waves in leads I and II are seen in both conditions, and in both the electrocardiographic signs tend to be transient.

When pulmonary embolism complicates chronic pulmonary disease, the electrocardiogram often shows changes resembling those of acute respiratory failure, rather than those of pulmonary embolism.

The term "acute asphyxial cor pulmonale" is proposed for the cardiovascular manifestations of acute respiratory failure.

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