Serial 3 orthogonal lead electrocardiographic abnormalities after pulmonary embolism

Computer assisted study

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The 3 orthogonal lead electrocardiogram has been evaluated with computer assisted interpretation in 20 patients with acute pulmonary embolism confirmed by pulmonary angiography. Initial 3-lead electrocardiographic abnormalities were found to be at least as helpful as the 12-lead electrocardiogram in supporting the clinical diagnosis. In addition, however, sequential changes in both the maximum QRS and T vector orientations in frontal and transverse planes were more often apparent than any sequential changes in the 12-lead electrocardiogram. While no specific 3 lead electrocardiographic criterion for pulmonary embolism could be determined, this technique was more helpful than conventional methods in following serial electrocardiographic changes in patients with this condition.

The recent availability of pulmonary angiography has facilitated a more critical appraisal of the 12 lead electrocardiographic appearances in acute pulmonary embolism than has hitherto been possible (Stein *et al.*, 1975). As far as is known, there have, however, been no studies to evaluate serial 3 orthogonal lead electrocardiographic abnormalities in patients with acute pulmonary embolism proven angiographically. With the recent availability of a routinely used computer programme for storing orthogonal lead data to enable comment on day-to-day electrocardiographic changes to be made (Macfarlane, Cawood, and Lawrie, 1975), it was decided to undertake a study of 3 orthogonal lead (and corresponding 12 lead) electrocardiograms in order to determine whether or not diagnostic criteria for a positive electrocardiographic change could be incorporated into the routinely used programme.

Subjects and methods

Twenty patients (12 male and 8 female—mean age 52 ± 13) were included in the study. In all cases a diagnosis of acute pulmonary embolism was suspected on clinical grounds and confirmed by pulmonary angiography. Routine clinical investigations were performed in all patients including chest

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x-ray, serial enzyme studies, full blood count and ESR. Patients were divided into two groups for purposes of analysis. Group 1 consisted of 14 patients with no previous history of cardiac or respiratory disease. Group 2 consisted of 6 patients who had a significant cardiac or respiratory history which *per se* had led to the development of an abnormal electrocardiogram. The details of age, sex, duration of symptoms before presentation, together with the underlying diagnosis are shown for groups 1 and 2 in Tables 1 and 2, respectively.

Pulmonary angiography was performed in all cases. A 7×125 Gensini catheter was passed via a right medial cubital vein into right atrium, right ventricle, and then into the main pulmonary artery. Pressure measurements were taken in these three sites. The catheter was then left in the main pulmonary artery and 45 to 55 ml (dose depending on body weight) angiografin were then injected using a Girdlund H-P injector. The angiogram was recorded on 35×35 cm film using an Elema Schonander rapid film changer in conjunction with a high speed fine focus tube. Films were exposed at a rate of 4 per second for 4 seconds and thereafter 1 per second for 9 seconds, allowing dynamic features of the angiogram to be assessed. All patients met one or more of the following major criteria for pulmonary embolism.

TABLE 1 Patients with no history of cardiac or respiratory disease (group 1)

Case No.	Age	Sex	Duration of symptoms before presentation (hr)	Underlying diagnosis
1	48	F	<24	Duodenal ulcer: post vagotomy
2	61	M	<24	Appendicitis: post appendicectomy
3	77	F	<24	Acute pancreatitis
4	53	M	<48	Thrombophlebitis
5	39	M	<48	Varicose veins: post ligation
6	47	F	<48	Nil
7	62	M	<48	Deep venous thrombosis
8	63	M	<24	Duodenal ulcer: post vagotomy
9	60	M	<24	Varicose veins: post ligation
10	48	M	<24	Varicose veins: post ligation
11	38	F	<48	Fibroids: post hysterectomy
12	42	F	<24	Obesity
13	25	M	<24	Carcinoma of oesophagus
14	71	F	<24	Deep venous thrombosis

TABLE 2 Patients with positive history of previous cardiac or respiratory disease (group 2)

Case No.	Age	Sex	Duration of symptoms before presentation (hr)	Underlying diagnosis
15	42	M	<24	Atrial fibrillation
16	48	M	<24	Previous myocardial infarction
17	66	M	<24	Previous myocardial infarction
18	57	M	<48	Angina
19	34	F	<24	Rheumatic heart disease
20	62	F	<48	Chondroma right lung; previous resection right middle and lower lobe

- (1) Cut-off vessels of large or medium calibre.
 - (2) Filling defects appearing consistently throughout the arterial phase of the injection.
 - (3) Vessel loss not related to bullous emphysema.
- Patients were subsequently grouped into three categories as follows according to the pulmonary arterial pressure noted during angiography:

- (a) Normal: systolic pulmonary artery pressure < 30 mmHg (4 kPa).
- (b) Moderate pulmonary hypertension: systolic pulmonary artery pressure 30 to 45 mmHg (4 to 6 kPa).
- (c) Severe pulmonary hypertension: systolic pulmonary artery pressure > 45 mmHg (6 kPa).

The orthogonal lead electrocardiograms (antero-lateral lead X, inferior lead Y, and antero-septal lead Z of I, aVF, V2) were recorded on each patient using the modified axial lead system (Macfarlane, 1969). Tape recordings were made using a 4-channel FM magnetic tape recorder and the electrocardiograms were replayed to a PDP8E computer for analysis using a programme which has been described in detail previously (Macfarlane and Lawrie, 1974). Included in the computer print-out were projections of the maximum QRS and T

vectors onto the frontal and transverse planes denoted QRS_F, QRS_T, T_F, and T_T, respectively, together with corresponding vector magnitudes.

The following relevant criteria were used for the 3 orthogonal lead electrocardiogram. These have been extracted from the detailed list of diagnostic criteria to be published elsewhere (Macfarlane and Lawrie, 1977) and are presented in simplified form.

- (1) RAD: QRS_F > 65°.
- (2) LAD: 270 < QRS_F < 360°.
- (3) Clockwise cardiac rotation: 180 < QRS_T < 360° and negative QRS_X area.
- (4) LVH: Max QRS vector amplitude > 2.5 mV or R_X > 2.1 mV or S_Z > 1.2 mV.
- (5) RVH: (R/S)_Z > 3 or R_Z > 0.85 mV or (S_X > 0.55 mV and S_X > R_X).
- (6) Myocardial infarction:
 - (a) Inferior: (Q/R)_Y > 1/3 and Q_Y > 0.03 s.
 - (b) Antero-septal: Q_Z > 0.02 s and Q_Z > 0.1 mV or (R_Z < 0.10 mV and (R/S)_Z < 1/10 and 180° < 0.03 sec QRS_T < 330°).
- (7) T wave abnormalities: T_X < 0.05 mV or T_Y < 0.05 mV or T_Z < -0.05 mV.

- (8) ST abnormalities: Visible ST junctional depression and ST segment flat or downward sloping in any lead.

Sequential changes

- (9) T vector: A day-to-day change in T_T or T_F of more than 30° .
 (10) QRS vector: A day to day change in $QRS_F > 20^\circ$ or in $QRS_T > 25^\circ$.

The normal limits of day-to-day change have been discussed elsewhere (Cawood *et al.*, 1974). If T wave inversion were found to be present in the anteroseptal lead with or without T wave abnormalities in the inferior leads, and if the clinical diagnosis were pulmonary embolism, the computer programme would report T wave changes 'consistent with the clinical diagnosis of pulmonary embolism'.

Twelve lead electrocardiograms were also recorded on each patient using a Siemens direct writing single channel machine. The paper speed was 25 mm/s and the calibration was adjusted such that a 10 mm deflection was equal to 1 mV. The 12 lead electrocardiogram tracing was recorded at the same time as the 3 lead electrocardiogram. The following diagnostic criteria were used in con-

junction with interpretation of the 12 lead electrocardiogram by two of the authors. The QRS axis in the frontal plane was determined from Tables (Goldman, 1970).

- (1) RAD: Frontal QRS axis $> 110^\circ$.
- (2) LAD: Frontal QRS axis $< -30^\circ$.
- (3) Clockwise cardiac rotation: transition line at V5 with S_{V_6} present.
- (4) RVH: $(R/S)_{V_1} > 1$ and $R_{V_1} > 0.7$ mV.
- (5) $S_1 Q_3 T_3$: $S_I > 0.15$ mV and $Q_{III} > 0.15$ mV and $T_{III} < 0$ mV.
- (6) LVH: $S_{V_1} + R_{V_5} > 3.5$ mV or $R_{aVL} > 1.1$ mV or $R_{aVF} > 1.5$ mV.
- (7) Myocardial infarction:
 - (a) Inferior: $Q > 0.035$ s and $Q > 1/4 R$ in any two of II, III, aVF.
 - (b) Anteroseptal: $Q > 0.20$ mV in any two leads V2 to V4 or $R < 0.10$ mV in any two leads V2 to V4.
- (8) T wave abnormalities: flat or inverted T waves in relevant leads.
- (9) ST abnormalities: visible ST junctional depression and ST segment flat or downward sloping in relevant leads.

Sequential T wave changes were diagnosed from 12 lead electrocardiograms in a somewhat subjective

TABLE 3 Initial 12 and 3 lead electrocardiogram in group 1

Case No.	Lead	Electrocardiographic changes	Pulmonary pressure	
			mmHg	kPa
1	12 } 3 }	Normal	35/11	4.7/1.5
2	12 } 3 }	$S_1 Q_3 T_3$ Q wave in Y lead; LAD	76/28	10.1/3.7
3	12 } 3 }	ST-T changes of inferior ischaemia	50/17	6.7/2.3
4	12 } 3 }	ST-T changes of anteroseptal and lateral ischaemia	43/20	5.7/3.7
5	12 } 3 }	ST-T changes of inferior ischaemia ST-T changes of inferior ischaemia; LAD	39/11	5.2/1.5
6	12 } 3 }	Normal	28/18	3.7/2.4
7	12 } 3 }	Left ventricular hypertrophy	56/14	7.5/1.9
8	12 } 3 }	Normal	37/12	4.9/1.6
9	12 } 3 }	Normal LAD	26/13	3.5/1.7
10	12 } 3 }	T wave inversion V1-V3 and in Z lead	27/6	3.6/0.8
11	12 } 3 }	T wave inversion in lateral leads	27/9	3.6/1.2
12	12 } 3 }	Normal	25/15	3.3/2
13	12 } 3 }	$S_1 Q_3 T_3$ CCR T wave inversion V1-V3 T wave inversion Z lead, CCR, RAD	50/15	6.7/2
14	12 } 3 }	$S_1 Q_3 T_3$ CCR T wave inversion V1-V3 T wave inversion Z lead, RAD, CCR	52/14	6.9/1.9

LAD, left axis deviation; RAD, right axis deviation; CCR, clockwise cardiac rotation.

manner by the two reviewers on the basis of the appearance of fresh T wave inversion or increase in inverted T wave amplitudes. Sequential QRS axis changes could not be reported as no known criteria were available.

Three and 12 lead electrocardiograms were recorded from almost all patients on at least 3 occasions, including the time of presentation, which was always less than 48 hours from the onset of symptoms, and the day of pulmonary angiography.

Results

Group 1

The main features of the initial 3 and 12 lead electrocardiograms together with pulmonary artery pressure are shown for each patient in Table 3. Three and 12 lead findings were the same in 9 patients while all patients were in sinus rhythm. Table 4 summarizes the frequency of selected electrocardiographic measurements.

Table 5 presents the sequential electrocardiographic abnormalities detected in group 1 patients. Sequential changes in either the QRS or T vectors were present in 8 patients according to 3 lead diagnostic criteria while sequential T wave changes in the 12 lead electrocardiograms were noted in 6 patients. QRS vector angles in the frontal and transverse planes are shown where an abnormal shift has occurred or where the vector has moved from a normal to an abnormal orientation.

Pulmonary artery pressure was normal in 5 patients in this group, moderately raised in 4 patients, and severely raised in 5. All three patients with S₁ Q₃ T₃ had severe pulmonary hypertension as had the two patients with marked QRS_F vector shifts detected by the 3 lead electrocardiogram.

Group 2

Three and 12 lead electrocardiogram results in group 2 patients are summarized in Table 6. There was no difference between initial 3 and 12 lead reports, but five patients subsequently showed

TABLE 4 Summary of 3 and 12 lead appearances, group 1

	3-lead	12-lead
Sinus tachycardia	5	5
Atrial ectopics	2	2
S ₁ Q ₃ T ₃	0	3
T wave inverted		
anteroseptal leads	3	3
Left axis deviation	3	0
Right axis deviation	2	0
Clockwise cardiac rotation	2	2

TABLE 5 Sequential changes in group 1

Case No.	T wave	Frontal	QRS Transverse
2 12	+ve	-ve	-ve
3 3	+ve		
3 12	+ve	-ve	-ve
3 3	+ve		
9 12	-ve	-ve	-ve
3 3	+ve		
10 12	+ve	-ve	-ve
3 3	+ve		
11 12	-ve	-ve	-ve
3 3	+ve		
12 12	-ve	8 → 26 → 72	331 → 291 → 340
3 3	-ve		
13 12	+ve	268 → 190	262 → 209
3 3	+ve		
14 12	+ve	182 → 163 → 136	223 → 225 → 237
3 3	+ve		

significant QRS or T vector changes on the 3 lead electrocardiogram whereas in only one patient were sequential changes noted by the 12 lead electrocardiogram. Two patients in this group had no pulmonary hypertension, 2 had moderate pulmonary hypertension, and the remaining 2 had severe pulmonary hypertension.

Discussion

Twelve lead electrocardiographic appearances after acute pulmonary embolism have been well documented. Few of these are diagnostic of pulmonary embolism though the S₁ Q₃ T₃ pattern (McGinn and White, 1935) and T wave inversion in the right praecordial leads (Sutton, Honey, and Gibson, 1969) are helpful confirmatory findings. The former is more likely to be present when there is major pulmonary vascular obstruction with pulmonary hypertension but even then this abnormality may not occur (McIntyre, Sasahara, and Littmann, 1972), and the latter appears to be particularly associated with acute massive pulmonary embolism (Sutton *et al.*, 1969). In the 3 patients in this study with S₁ Q₃ T₃, the 3 lead electrocardiogram did not show a specific abnormality with which this pattern could be equated but nevertheless showed striking right axis deviation in two, with a Q_y S_x pattern in the third. In respect of 5 of 20 patients with T wave abnormalities in the right praecordial leads V1 to V3, the computer interpretation of the 3 lead electrocardiogram also noted anteroseptal T wave abnormalities and commented on their relevance to the clinical diagnosis of pulmonary embolism. Three of these patients had significant haemodynamic changes, one had

TABLE 6 Electrocardiographic findings in group 2

Case No.	Initial	Sequential changes			Transverse	Pulmonary artery pressure	
		T Wave	QRS Frcntal			mmHg	kPa
15	3	Atrial fibrillation	3 +ve	18→7→48	324→294→274	25/18	3.3/2.4
12		Inferior myocardial ischaemia	12 -ve	-ve	-ve		
16	3	Sinus rhythm; LAD	3 -ve	-ve	-ve	43/23	5.7/3.1
12		Anteroseptal myocardial infarction	12 -ve	-ve	-ve		
17	3	Sinus rhythm, LAD	3 +ve	-ve	-ve	24/10	3.2/1.3
12		Inferior myocardial infarction	12 -ve	-ve	-ve		
18	3	Sinus rhythm	3 +ve	-ve	-ve	45/17	6.0/2.3
12		Widespread myocardial ischaemia	12 -ve	-ve	-ve		
19	3	Sinus rhythm	3 +ve	47→70→58	344→304→337	66/26	8.8/3.5
12		T wave inversion in anteroseptal leads	12 +ve	-ve	-ve		
20	3	Atrial fibrillation; RAD	3 -ve	156→119→76	178→239→231	84/55	11.2/7.3
12		Clockwise cardiac rotation	12 -ve	-ve	-ve		

moderately raised pulmonary artery pressure and one had normal pressure. Sequential T wave changes in group 1 were detected in 7 patients by the 3 lead electrocardiogram and in 5 patients by the 12 lead electrocardiogram. In group 2, it was extremely difficult to infer any relation between T wave changes and acute pulmonary embolism, but 3 out of 6 patients showed sequential T vector changes in the 3 lead electrocardiogram whereas no sequential T wave changes were evident on the 12 lead electrocardiogram.

The diagnosis of QRS axis shifts can be accurately estimated in the 3 lead system and an abnormality of the frontal or transverse maximum QRS vector occurred at some stage in 11 of 20 (55%) patients. All patients with initial right axis deviation showed subsequent abnormal QRS vector shifts and a further 3 showed significant sequential QRS vector shifts. Left axis deviation was noted on at least one occasion in 5 of 20 (25%) patients, in keeping with the findings of Lynch, Stein, and Bruce (1972) who reported that left axis deviation was twice as common as right axis deviation in patients with acute pulmonary embolism.

Clockwise cardiac rotation is known to be associated with acute pulmonary embolism (Cutforth and Oram, 1958; Phillips and Levine, 1950). All of the 3 patients with clockwise cardiac rotation in our series retained this abnormality throughout the period of the study, though the QRS_T vector rotated clockwise while the QRS_F vector correspondingly rotated in an anticlockwise direction as the time from the initial incident increased.

The temporal relation of electrocardiographic changes and acute pulmonary embolism is of great importance, and Spodick (1972) has emphasized the value of establishing a relation between likely time of the acute embolism and the initial electrocardio-

gram. In this study 13 patients (65%) had tracings recorded within 24 hours of initial symptoms while the remaining 7 patients had their first electrocardiogram recorded within 48 hours.

There was no specific 3 orthogonal lead electrocardiographic pattern which could be said to be typical of pulmonary embolism. Classical signs such as T wave inversion in the anteroseptal lead appeared simultaneously in both lead systems but could not be regarded as pathognomonic of pulmonary embolism. Likewise while significant QRS vector shifts outside normal day-to-day variation occurred in 6 of 20 (30%) patients, there was no pattern to suggest that one direction of rotation was prevalent. This is in keeping with findings of right and left axis deviation in patients with pulmonary embolism. Thus, there was no diagnostic criterion which could be regarded as a definite indication of pulmonary embolism.

Although there are additional difficulties in the assessment of electrocardiographic changes in patients with previously abnormal tracings, the exact effect of previous cardiopulmonary disease is not certain. It has been suggested, both in animals (Just-Viera *et al.*, 1965) and in man (Szucs *et al.*, 1971), that the incidence of electrocardiographic abnormalities may not be altered. It seems that the important factor in this situation is the demonstration of a changing electrocardiogram in the presence of a known pre-existing abnormality. We have found that in 5 out of 6 patients with known cardiac or respiratory disease, sequential 3 lead electrocardiographic changes occurred. In only one patient were definite sequential changes noted on the 12 lead tracings. These findings suggest that serial 3 lead tracings in this type of patient with suspected pulmonary embolism are of more value in supporting the diagnosis than the 12 lead electrocardiogram.

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