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Q waves and coronary arteriography in cardiomyopathy[1]

G. T. Gau,[2] J. F. Goodwin, C. M. Oakley, E. G. J. Olsen, S. H. Rahimtoola,[3] M. J. Raphael, and R. E. Steiner

From the Department of Medicine (Clinical Cardiology), Royal Postgraduate Medical School and Hammersmith Hospital, London

This study shows that the Q wave pattern on the electrocardiogram provides insufficient evidence for the diagnosis of ischaemic heart disease. Apparent evidence of ischaemic heart disease on the electrocardiogram was misleading in 6 out of 10 patients with congestive cardiomyopathy and in 3 of 8 patients with hypertrophic cardiomyopathy. With the increasing application of surgical techniques to patients with atheromatous coronary artery disease, coronary arteriography will undoubtedly assume an increasing role in the more precise delineation of these cases.

Cardiomyopathy has been broadly defined as a subacute or chronic disorder of heart muscle of unknown or unusual cause often with associated endocardial and sometimes pericardial involvement (Goodwin, 1970). 'Primary' cardiomyopathy was used to denote that the disorder was confined to the heart but we now prefer to restrict the term cardiomyopathy to 'heart muscle disease of unknown cause or association' and to describe secondary heart muscle disease according to its cause (Oakley, 1971). Cardiomyopathies are divisible into two main groups: congestive cardiomyopathy in which there is systolic pump failure, and hypertrophic cardiomyopathy which is characterized by diastolic compliance failure with or without obstruction to left ventricular ejection. Hypertrophic cardiomyopathy has previously been known as obstructive cardiomyopathy (Goodwin *et al.*, 1960) or hypertrophic obstructive cardiomyopathy (Cohen *et al.*, 1964) in this country and as idiopathic hypertrophic subaortic stenosis (Braunwald *et al.*, 1960) or muscular subaortic stenosis (Wigle, Heimbecker, and Gunton, 1962) in Northern America.

Both congestive cardiomyopathy and hypertrophic cardiomyopathy can masquerade as coronary artery disease and cardiomyopathy can be diagnosed mistakenly in patients who really have coronary artery disease. This study underlines the importance of coronary angiography in order to make the distinction which has hitherto rested by default on the electrocardiogram backed up by circumstantial evidence based on the so-called coronary risk factors exhibited by the patient.

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[2] Present address: Division of Cardiology, Mayo Clinic, Rochester, Minnesota 55901, U.S.A.

[3] Present address: Department of Adult Cardiology, Cook County Hospital, 1825 West Harrison Street, Chicago, Illinois 60612, U.S.A.

Patients and methods

Group A Twenty-two patients, 8 women and 14 men, with ages ranging from 17 to 65 years and a mean age of 47 years, suspected clinically of having a primary cardiomyopathy, were fully studied including selective coronary angiography as part of their diagnostic investigation (Tables 1 and 2). Selective coronary angiography was carried out by either the Sones or the Judkins technique (Sones and Shirey, 1962; Judkins, 1967). The cineangiograms were independently reviewed by two of the authors (G.T.G. and M.J.R.). All other relevant clinical factors such as age of the patient, history of angina, family history of atheromatous coronary artery disease, presence of diabetes, abnormal blood lipids, raised blood pressure, and smoking history had been assembled and weighed as circumstantial evidence before the haemodynamic and angiographic study was undertaken.

Group B A further group of 22 patients in whom a diagnosis of primary congestive cardiomyopathy had been made during life and in whom we had adequate necropsy data was evaluated (Table 3). There were 7 female and 15 male patients with ages ranging from 15 to 69 years and a mean age of 43 years. The clinical records and

necropsy data of these cases were evaluated independently. Pathological specimens were examined both grossly and microscopically for evidence of occlusive coronary atheroma, small vessel coronary disease, focal myocardial scars, or diffuse myocardial disease.

Results

Group A Of the 22 patients with a tentative clinical diagnosis of cardiomyopathy, 14 were believed to have congestive and 8 were thought to have hypertrophic obstruction cardiomyopathy.

Five of the 14 patients considered to have congestive cardiomyopathy had infarct patterns on their electrocardiograms, and the remainder had nonspecific changes or left bundle-branch block. Selective coronary cineangiograms showed advanced diffuse coronary atheromatous disease in 3 patients while the remaining 11 had normal coronary arteries. All 3 patients with coronary artery disease had an infarct pattern on the electrocardiogram, but only one of these patients had a history of ischaemic chest pain.

Five of the 11 patients with normal coronary arteries had a history of angina and 2 of them had an infarct pattern on the electrocardiogram (Fig. 1 and 2). In this group of 14 patients there was no apparent correlation between any of the parameters listed in Table 2 and the presence of either atheromatous coronary artery disease or cardiomyopathy.

Three of the 8 patients with a clinical diagnosis of hypertrophic cardiomyopathy had infarct patterns on their electrocardiograms. The coronary cineangiograms showed normal coronary arteries in all 8 cases. Four of these 8 patients had a history of chest pain suggestive of ischaemic heart disease and 2 of the 4 patients complaining of chest pain showed an infarct pattern on their electrocardiograms.

Of 34 patients with congestive cardiomyopathy in whom we had adequate data either from necropsy (Tables 2 and 3) or from coronary angiography, there were 10 patients with an infarct pattern on the electrocardiogram. In addition to the 3 patients found to have advanced atheromatous coronary artery disease on coronary angiography, one further patient (Case 42, Table 3) was found to have diffuse coronary artery disease at necropsy. The remaining 6 patients with infarct patterns on their electrocardiograms were shown to have normal coronary arteries either at necropsy or on coronary angiography.

Ten of the 34 patients had a history of chest pain suggestive of ischaemic heart disease. Only 3 of these had both angina and infarct patterns on their electrocardiograms, of whom 2 were shown to have normal coronary arteries and one turned out to have atheromatous coronary artery disease. The only patient in the necropsy series who was shown to have atheromatous coronary disease had not given a history of chest pain. In 2 patients in this series both coronary angiography and necropsy data were available. The electrocardiogram of one of these patients is shown in Fig. 3 and the coronary angiograms are displayed in Fig. 4 and 5. The localized segmental lesion in the right coronary artery was judged to be a 50 per cent stenosis to which the generalized left ventricular hypokinesia and dilatation shown on angiography could not possibly be attributed. At necropsy this man was found to have diffuse disease throughout his entire myocardium as demonstrated by Fig. 6 which shows the histological appearance. There was no fibrosis, not even in relation to the right coronary artery distribution. The atheromatous plaque in the right coronary artery noted on the angiogram was readily identified at the time of necropsy but represented only about a 30 per cent narrowing of the lumen which was felt not to be significant. This was the only patient in the

TABLE I *Patients with clinical cardiomyopathy studied by selective coronary arteriography*

Case No.	*Age*	*Sex*	*Electrocardiogram*	*Alive or dead*	*Selective coronary cineangiogram*	*Necropsy*
Congestive cardiomyography						
1	50	M	Infarct	Dead	N*	Yes
2	48	M	Nonspecific	Alive	N	—
3	62	M	Nonspecific	Alive	N	—
4	56	M	Nonspecific	Alive	N	—
5	48	M	Nonspecific	Alive	N	—
6	48	M	Nonspecific	Dead	N	Yes
7	46	M	Nonspecific	Alive	N	—
8	45	M	Infarct	Alive	C	—
9	37	F	LBBB	Alive	N	—
10	52	M	LBBB	Alive	N	—
11	59	F	Infarct	Alive	C	—
12	37	F	Infarct	Alive	N	—
13	56	M	LBBB	Alive	N	—
14	55	M	Infarct	Alive	C	—
Hypertrophic cardiomyopathy						
15	53	F	Nonspecific	Alive	N	—
16	17	F	Infarct	Alive	N	—
17	65	M	Nonspecific	Alive	N	—
18	45	M	Nonspecific	Alive	N	—
19	35	M	Nonspecific	Alive	N	—
20	53	F	Infarct	Alive	N	—
21	35	F	Nonspecific	Alive	N	—
22	38	F	Infarct	Alive	N	—

LBBB = Left bundle-branch block.
N = Normal, or N* single non-occlusive plaque only.
C = Occlusive coronary atheroma.

TABLE 2 *Clinical information on patients studied by coronary arteriography*

Case No.	*Chest pain*	*Family history of coronary artery disease*	*Tobacco smoker*	*Congestive heart failure*	*Blood pressure*	*Heart x-ray*	*Fasting blood sugar*	*Glucose tolerance test**	*Fasting cholesterol*	*Fasting triglycerides*
Congestive cardiomyopathy										
1	o	+	+	+	95/60	Large	62	+	230	167
2	o	o	−	+	140/95	Large	155	+	180	236
3	+	+	+	o	130/90	Large	123	+	260	248
4	+	+	+	o	150/100	Large	101	+	220	102
5	+	o	o	+	120/60	Large	110	+	105	o
6	o	o	+	+	125/95	Large	111	−N	145	54
7	o	+	+	+	150/120	Large	96	+	215	302
8	o	o	+	+	120/95	Large	66	−N	150	68
9	+	o	o	o	110/80	Mod	78	−N	210	o
10	o	o	+	o	110/60	Large	88	+	240	o
11	+	o	+	o	130/70	Large	94	+	400	325
12	+	o	o	o	120/80	Large	69	−N	230	o
13	o	o	+	o	130/70	Large	141	+	210	o
14	o	+	+	o	120/70	Large	165	+	175	o
Hypertrophic cardiomyopathy										
15	+	o	o	o	130/70	Min (LV)	90	o	190	o
16	+	o	+	o	100/60	Min (LV)	98	o	o	o
17	o	+	o	o	110/70	Min	92	o	o	o
18	+	+	+	o	110/75	Min	89	o	340	o
19	o	o	o	o	120/70	Mod	o	o	170	o
20	+	+	o	o	170/90	Min	68	o	290	o
21	o	+	o	o	150/75	Min	o	o	200	o
22	o	o	o	o	120/80	Mod	58	o	145	o

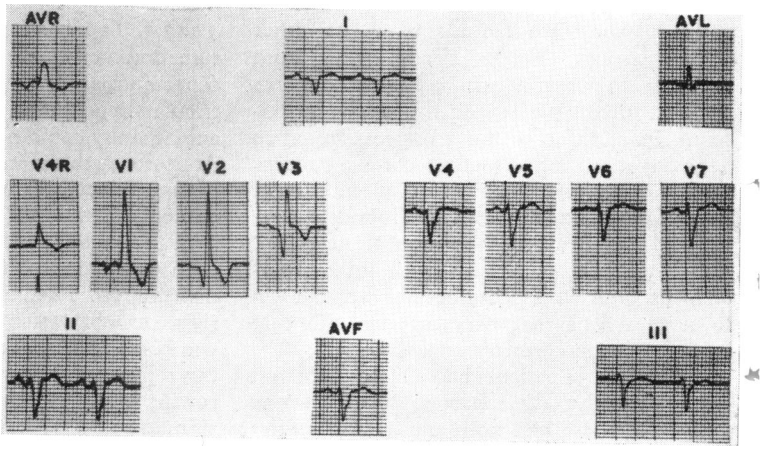
* + = positive; o = absent; − = not known.
Mod = moderately enlarged; Min = minimally enlarged; Min (LV) = minimal enlargement of LV only.

TABLE 3 *Patients diagnosed clinically as congestive cardiomyopathy on whom adequate necropsy data were available*

Case No.	*Age*	*Sex*	*Electrocardiogram*	*Selective coronary cineangiograph*	*Necropsy*
1	50	M	Infarct	Yes—N*	N*
6	48	M	Nonspecific	Yes—N	N
23	55	F	Nonspecific	—	N
24	43	F	LBBB	—	N
25	25	F	Infarct	—	N
26	25	M	Nonspecific	—	N
27	69	M	Nonspecific	—	N
28	15	M	Nonspecific	—	N
29	35	M	LBBB	—	N
30	40	M	LBBB	—	N
31	52	M	LBBB	—	N
32	27	M	Nonspecific	—	N
33	43	M	Nonspecific	—	N
34	48	M	Nonspecific	—	N
35	34	M	Nonspecific	—	N
36	51	F	Nonspecific	—	N
37	64	F	Nonspecific	—	N
38	28	M	Nonspecific	—	N
39	69	F	Infarct	—	N
40	22	F	Nonspecific	—	N
41	38	M	Infarct	—	N
42	59	M	Infarct	—	C

N* = single non-occlusive plaque only.

FIG. 1 *The electrocardiogram of a 37-year-old woman with congestive cardiomyopathy (Case 12, Table 2) and a bizarrely abnormal electrocardiogram suggestive of massive anterior infarction (described in Table 5).*



series in whom there was any question of a localized segmental coronary lesion. Without the necropsy data some might have doubted the diagnosis of cardiomyopathy, though unwisely because diffuse left ventricular power failure without aneurysm formation does not follow non-occlusive stenosis of a single coronary artery. Patients with cardiomyopathy are not immune from coronary atheroma, but we have observed that their coronary angiograms have usually been singularly free from disease. It is tempting to speculate whether the left ventricular dilatation and hypokinesia in congestive obstructive cardiomyopathy do not actually protect the coronary arteries from intimal cracks consequent upon the acute flexing stresses which occur during normal contraction. In hypertrophic obstructive cardiomyopathy coronary atheroma might be slower to produce obstruction in the unusually wide bore coronary arteries which some of these patients have.

Necropsy in the cases with congestive obstructive cardiomyopathy revealed overweight hearts with dilated ventricles. The muscle was pale and flabby in character and the coronary arteries were widely patent and free from atherosclerosis. No areas of localized fibrosis suggestive of focal myocardial infarction were present. Light micropsy revealed scattered fibrosis with evidence of muscle fibre necrosis and occasional round cell infiltration. These findings were all nonspecific but suggestive of chronic myocardial damage of unknown cause.

The positive criteria which are usually present consist of cardiomegaly due to hypertrophy and dilatation; endocardial thickening, which may be patchy or uniform (upon which thrombus is often found); as well as some fibrous replacement, particularly of the inner third of the myocardium. The coronary arteries are classically free from atherosclerosis. The histological appearances are similarly nonspecific and usually show the picture which was described above. Electronmicroscopical examination shows partial or complete loss of cristae in the mitochondria, dilatation of sarcoplasmic reticulum, and increased glycogen. That these changes are nonspecific in any group of cardiomyopathies has been confirmed by Van Noorden, Olsen, and Pearse (1971).

Table 5 illustrates the electrocardiographic findings in the 13 cases found to have infarct patterns on their electrocardiogram in this series. When a diagnosis of congestive cardiomyopathy was considered, as in all but the last 3 cases, the presence of left atrial enlargement favours a diagnosis of primary cardiomyopathy particularly when associated with a left

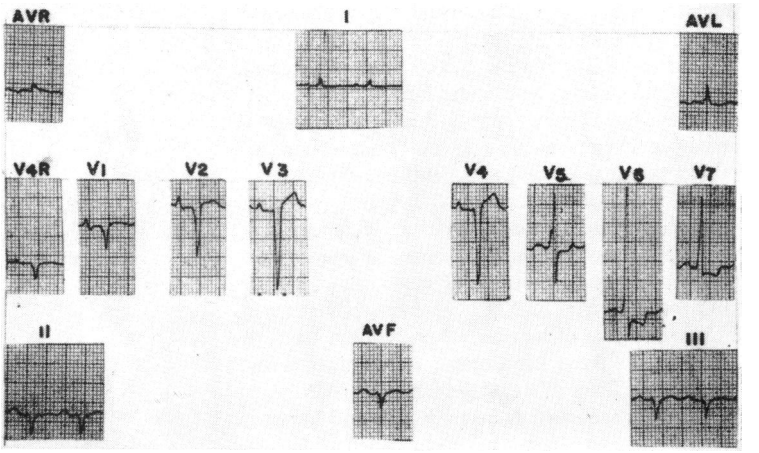


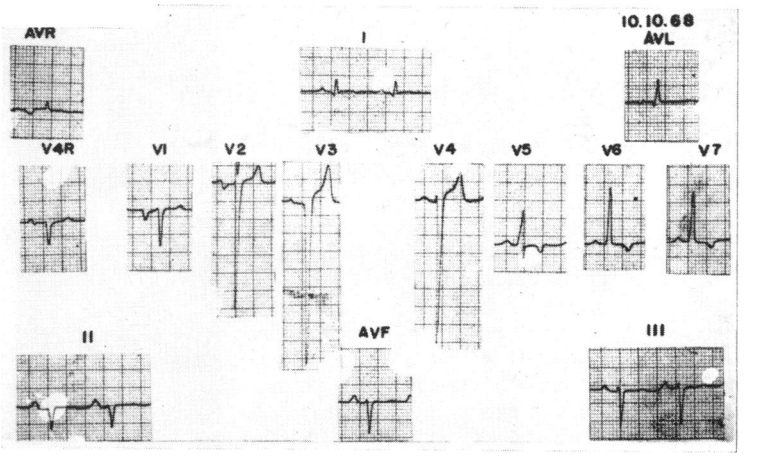
FIG. 2 *Electrocardiogram from a 38-year-old man with congestive cardiomyopathy (Case 41, Table 3) showing changes which would ordinarily be ascribed to old anterior infarction (described in Table 5).*

anterior hemiblock (Table 5). In general there appears to be no good electrocardiographic parameter which would enable congestive cardiomyopathy or hypertrophic cardiomyopathy presenting with an infarct pattern to be recognized from coronary artery disease.

Discussion

The presence of abnormal Q waves in the electrocardiogram leading to a mistaken diagnosis of myocardial infarction in patients with

FIG. 3 *Electrocardiogram from a 50-year-old man with congestive cardiomyopathy and angina showing changes characteristic of old anterior infarction and left anterior hemiblock.*



hypertrophic obstructive cardiomyopathy has been mentioned by several authors (Prescott, Quinn, and Littmann, 1963; Braudo, Wigle, and Keith, 1964; Frank and Braunwald, 1968; Wigle and Baron, 1966). An electrocardiographic infarct pattern is not uncommon in these patients. Frank and Braunwald reported that 69 out of 123 cases (56%) in their series had abnormal Q waves in the electrocardiogram. Three of our 8 patients in whom we found normal coronary artery anatomy had infarct patterns on the electrocardiograms. The presence of associated chest pain in 2 of these 3 patients highlights the clinical difficulties in excluding atheromatous coronary artery disease in such cases. Frank and Braunwald suggested that the absence of a history of myocardial infarction supports the contention that the Q waves are not a manifestation of myocardial necrosis. The finding of normal coronary arteries in our present study supports this view.

Possible explanations as to the genesis of these abnormal Q waves have been proposed. Wigle and coworkers attributed the abnormal Q waves in hypertrophic cardiomyopathy to massive hypertrophy of the interventricular septum, and suggested that since ventricular depolarization starts from the left septal surface, massive septal hypertrophy leads to a dominance of early septal forces producing the prominent Q waves. These workers, as well as Braunwald and his colleagues, have described the disappearance of these Q waves with the passage of time suggesting in support of this theory that the development of increasing hypertrophy of the left ventricular free wall counterbalances the electrical forces in the septum and diminishes the Q wave. Coyne (1968) suggested that increasing interstitial fibrosis in the septal area could also contribute to gradual electrocardiographic dominance by the left ventricular free wall through lessening the electrical forces of septal depolarization.

Burchell (1964) has pointed out that in some cases of multiple muscular ventricular septal defects (so-called Swiss cheese ventricular septum) similar Q waves can be seen on the electrocardiogram. This suggests that abnormal muscle structure itself can play a role in the genesis of the Q wave. Extrapolating this to hypertrophic cardiomyopathy, perhaps it is the specifically bizarre myocardial cells in these cases (Van Noorden *et al.*, 1971) which are responsible for the genesis of the Q waves.

One further possible explanation is premature activation of a portion of the ventricular septum by an anomalous conducting pathway.

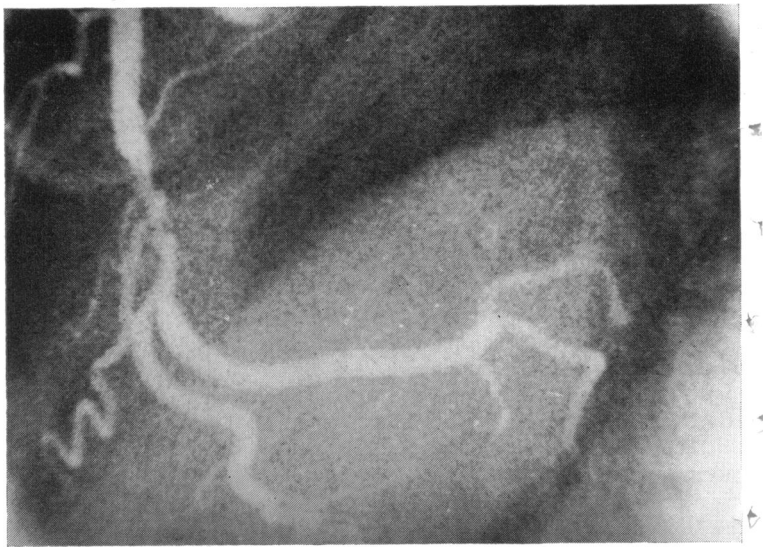
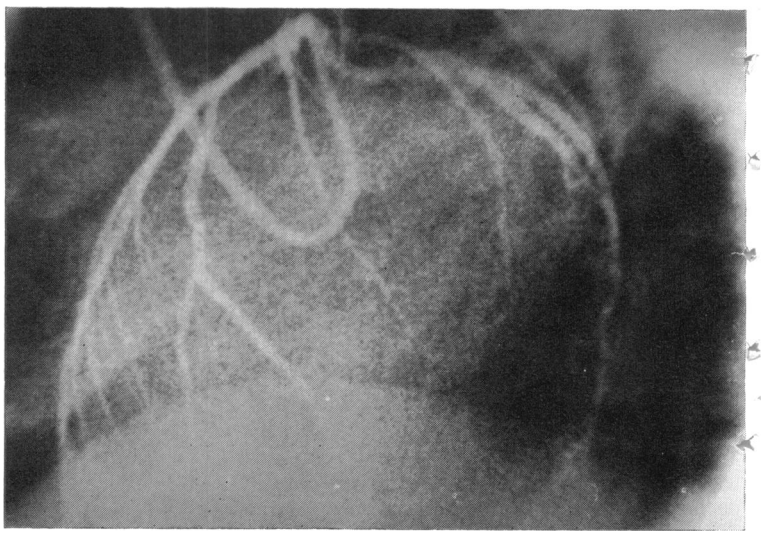
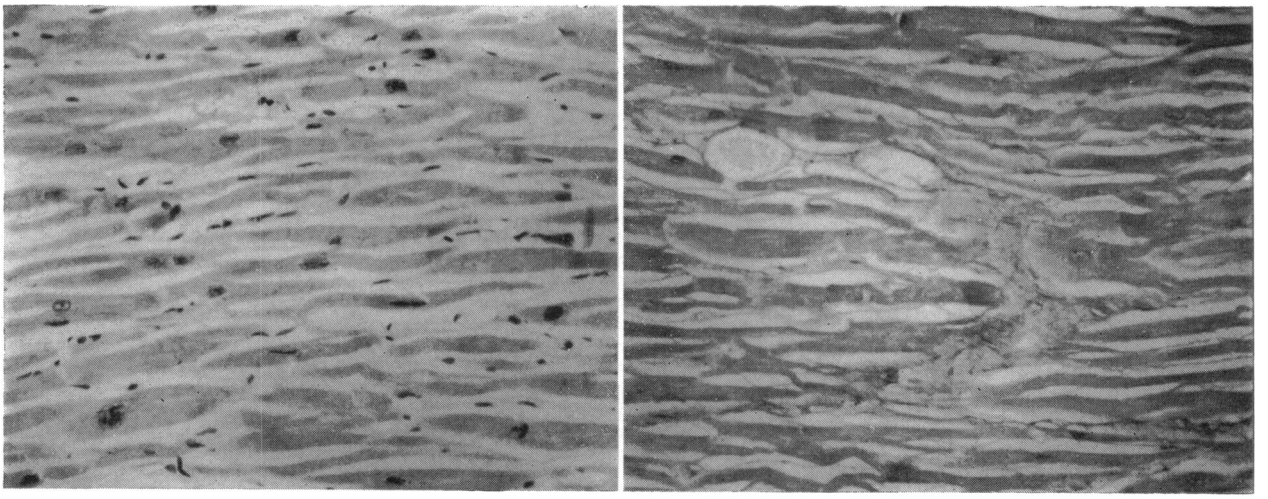


FIG. 4 *Right coronary angiogram left lateral view of patient whose electrocardiogram is shown as Fig. 3. A localized narrowing due to an atheromatous plaque is seen.* (See text.)

This association of hypertrophic obstructive cardiomyopathy and an electrocardiographic pattern suggesting pre-excitation has been noted but is not confirmed so far by intracardiac electrograph studies at the Royal Postgraduate Medical School, London, which have revealed no evidence of pre-excitation pathway (to be published).

FIG. 5 *Normal left coronary angiogram from the same patient as Fig. 3 and Fig. 4. Left lateral view.* (See text.)





a b

FIG. 6 (a) *Photomicrograph of myocardium showing attenuation of fibres and nuclear changes of hypertrophy. (Haematoxylin and eosin. × 200.)*

FIG. 6 (b) *Photomicrograph showing an area of degeneration which stains faintly for collagen. An occasional myocardial fibre can be traced through this area. (Weigert's elastic van Gieson. × 200.)*

The exact genesis of the Q waves in hypertrophic obstructive cardiomyopathy remains only an hypothesis, but realization of the frequency of this finding as well as the frequency of angina in these patients may prevent the erroneous diagnosis of coronary artery disease.

The clinical separation of patients with left ventricular dysfunction due to occlusive coronary artery disease from those with congestive obstructive cardiomyopathy can be extremely difficult. The clinical parameters usually employed are of little value in individual cases. In the group studied by coronary angiography in our series, 5 of the 11 cases with primary cardiomyopathy had complained of anginal pain, while only one of the 3 cases with atheromatous coronary artery disease had given a history of prior chest pain. The lack of specificity of anginal pain is more distinctly seen when we note that only one of our 10 patients with angina considered to have congestive cardiomyopathy turned out to have coronary artery disease.

Ten out of 34 patients showed infarct patterns on the electrocardiogram in this series. Four of these 10 cases with infarct patterns were found at coronary angiography or necropsy to have atheromatous coronary artery disease while the other 6 patients had normal coronary artery anatomy. All of the patients with coronary artery disease who were studied had infarct patterns on their electrocardiograms. Raftery, Banks, and Oram (1969) have reported cases with nonspecific electrocardiographic patterns simulating primary congestive cardiomyopathy who have been found to have coronary artery disease by coronary angiography. This was not our experience in the present series. In our series the finding that 6 out of 10 of the patients with left ventricular dysfunction and an infarct pattern on their electrocardiogram were found not to have coronary artery disease suggests that coronary arteriography is necessary for the correct diagnosis of patients with chronic left ventricular dysfunction. The overall incidence of an infarct pattern on the electrocardiograms of patients with primary congestive cardiomyopathy cannot yet be determined as we have shown that the diagnosis depends upon the exclusion of coronary artery disease by coronary arteriography.

Case 1 exemplifies one of the most challenging problems in this differential diagnosis. The association of angina, an infarct pattern on the electrocardiogram, and diffuse chronic left ventricular dysfunction makes it difficult not to attach significance to the evident coronary artery disease present. In this case, necropsy proved the presence of primary heart muscle disease and the absence of focal necrosis or fibrosis. Since the diagnostic

TABLE 4 *Clinical information on patients with congestive cardiomyopathy who came to necropsy*

Case No.	*Chest pain*	*Family history of coronary artery disease*	*Tobacco smoker*	*Congestive heart failure*	*Blood pressure*	*Heart x-ray*	*Fasting blood sugar*	*Glucose tolerance test**	*Fasting chol-esterol*	*Fasting trigly-cerides*
1	o	+	+	+	95/60	Large	62	+	230	167
6	o	o	+	+	125/95	Large	111	−	145	54
23	o	o	o	+	120/80	Large	110	o	225	o
24	o	o	o	+	85/50	Large	160	o	230	o
25	o	+	o	+	—	Large	o	o	o	o
26	o	o	+	+	90/75	Large	100	o	205	o
27	o	o	o	o	125/90	Large	o	o	o	o
28	+	o	o	+	100/65	Large	o	o	o	o
29	+	o	+	o	110/70	Large	o	o	210	o
30	o	o	o	+	90/80	Large	o	o	o	o
31	o	o	o	o	120/70	Large	o	o	200	o
32	o	o	+	o	110/75	Large	74	o	240	o
33	o	o	o	o	160/85	Large	o	o	300	o
34	o	o	o	+	110/70	Large	70	o	110	o
35	o	o	o	o	150/100	Large	o	o	o	o
36	o	o	o	+	130/70	Large	186	o	145	o
37	+	o	o	o	165/115	Large	o	o	o	o
38	o	o	o	o	105/85	Large	130	o	240	o
39	o	+	+	+	110/60	Large	185	+	240	o
40	o	o	o	+	70/50	Large	o	o	o	o
41	+	o	+	+	95/75	Large	o	o	o	o
42	o	o	+	+	130/80	Large	167	+	220	103

* + = positive; o = absent; − = not done.

TABLE 5 *Electrocardiographic features of patients with infarct patterns on their electrocardiograms*

Case No.	*Age*	*Diagnosis*	*P*		*PR (sec)*	*Frontal QRS axis*	*mm voltage*		*ST depression*	*QRS dur. (sec)*	*Site inf.*
			LA	*RA*			*V1 V5*	*V2 V6*			
8	45	CAD	−	−	0·18	−115	5	8	8	RBBB 0·16	ANT
14	55	CAD	−	−	0·20	0°	21	22	5	0·09	ANT
11	59	CAD	−	−	0·20	+50	30	38	7	0·09	ANT
42	59	CAD	+	+	0·18	+50	27	37	6	0·09	ANT
12	37	COCM	−	−	0·16	−118	2	8	8	RBBB 0·14	ANT
1	50	COCM	+	−	0·18	−60	20	50	10	LAHB 0·09	ANT
41	38	COCM	+	−	0·18	−50	21	43	5	0·08	ANT
25	25	COCM	+	−	0·16	−50	15	26	8	LAHB 0·16	ANT
39	69	COCM	+	−	0·16	−40	8	29	4	0·10	ANT
43	37	COCM	+	−	0·16	−55	3	3	6	RBBB LAHB 0·16	ANT
16	17	HOCM	+	++	0·14	+80	7	17	7	0·09	ANT LAT
20	53	HOCM	+	−	0·20	+25	37	41	12	0·10	ANT INF
22	38	HOCM	+	−	0·20	−15	20	23	10	0·11	INF

CAD = coronary artery disease; COCM = congestive cardiomyopathy; HOCM = hypertrophic cardiomyopathy; RBBB = right bundle-branch block; LAHB = left anterior hemiblock.

separation that we are discussing centres around patients in the 40- to 70-year age range, the association of various degrees of atheromatous coronary disease with primary cardiomyopathy is bound to be seen with increasing application of the techniques of coronary arteriography. It is well to remember that in the absence of focal aneurysm, septal rupture, or papillary muscle dysfunction, left ventricular pump failure is only seen when there is extensive occlusive coronary atheroma, usually with involvement of several major coronary vessels. Moreover, congestive obstructive cardiomyopathy is associated with generalized poverty of left ventricular movement on angiography, whereas in coronary artery disease with left ventricular failure some normal areas with increased shortening can usually be identified near to the base of the heart.

None of the 4 cases with congestive obstructive cardiomyopathy and an infarct pattern on their electrocardiogram who came to necropsy showed any focal area of scarring or myocardial wall thinning to explain the electrocardiographic Q wave pattern. As in hypertrophic obstructive cardiomyopathy, the true genesis of the Q wave pattern in congestive cardiomyopathy remains unknown. It is possible that despite generally poor ventricular function the most severely affected areas might act like a window relative to healthier areas of ventricular myocardium. Goodwin (1970) suggested that diffuse scattered fibrosis could alter interventricular conduction with loss of positive vector forces.

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Requests for reprints to Dr. Celia M. Oakley, Department of Medicine (Clinical Cardiology), Royal Postgraduate Medical School, Hammersmith Hospital, London W.12.