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Symptomless myocarditis and myalgia in viral and *Mycoplasma pneumoniae* infections

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Of 18 patients admitted to hospital with proved viral (influenza A, echo-9 and -30) or Mycoplasma pneumoniae *infections, 6 young adults between 15 and 27 years showed electrocardiographic evidence of symptomless myocarditis, which was invariably associated with myalgia.*

Myalgia is an important symptom presaging myocarditis and is an indication for an early electrocardiogram in all patients with suspected viral infections, despite the absence of cardiac symptoms or signs.

M. pneumoniae *myocarditis, we believe, was reported by us for the first time (Lewes and Rainford, 1970).*

Cardiographic signs, commonly maximal over the right ventricular surface leads, persisting for 2 to 14 days and occasionally for many months, may closely mimic coronary artery disease, septal cardiac infarction, or pulmonary embolism.

Correct electrocardiographic interpretation is essential if unwarranted cardiac invalidism is to be avoided, particularly in young patients with an indefinite history of infection and in those with protracted abnormalities in the electrocardiogram.

In July and August 1969 an outbreak of echo-9 virus infection was confirmed in the Bedford area: 7 such patients were admitted to hospital, and from 6 Echo-9 virus was isolated. Of these 7 patients only 1 suffered from myalgia and she alone showed cardiographic evidence of myocarditis.

A total of 18 patients are reported with echovirus or other infections (echo-9: 7 cases; echo-30: 1 case; influenza A: 6 cases; and *Mycoplasma pneumoniae*: 5 cases of whom 4 only were admitted to hospital). These patients were admitted to hospital between February 1969 and March 1970.

Over the years at Bedford Hospital evanescent electrocardiographic changes without cardiac symptoms or signs have been noted in patients suffering from Coxsackie viral infections: and for this reason during the outbreak of Echo-9 infection in 1969 routine cardiograms were carried out in all patients with suspected viral infection. Thus, 6 of the 18 patients reported showed cardiographic evidence of symptomless myocarditis which was *invariably accompanied by severe myalgia at the onset of the illness* (Table 1). Among 11 patients with proved viral or *M. pneumoniae* infections with normal electrocardiograms only one patient with an influenza A infection gave an history of transient muscle pain three days before developing pneumonia.

The association of myalgia with cardiographic evidence of myocarditis in our study was first noted by one of us (D.J.R.). We believe that myalgia in viral infections offers strong clinical evidence of simultaneous myocarditis in an abnormal cardiogram, despite the absence of cardiac symptoms or signs (Table 1).

We have been unable to discover, after an extensive search of the literature, any report of an aetiological relation between symptomless myocarditis

TABLE 1 *Myalgia and myocarditis in confirmed cases of viral and* M. pneumoniae *infections in patients admitted to hospital*

Organism	*No. of cases*	*Myalgia*	*Electrocardiogram myocarditis*
Echo-9	6	1	1
Echo-30	1	1	1
Influenza A_2	7	3 (1 transient 3 days before electrocardiogram)	2
M. pneumoniae	5	2	2
Totals	18	7	6

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TABLE 2a *Pattern of respiratory virus and Herpes virus infections in the Bedford area identified by isolation (Public Health Laboratory, Bedford) February 1969–February 1970*

Date	*Virus*	*No.*	*Male*	*Female*	*Clinical*	*Necropsy*
Feb. 1969	Influenza A	10	4	6	10	Nil
	Respiratory syncytial	5	1	4	5	Nil
	Adenovirus	2	0	2	2	Nil
	Herpes simplex	1	1	0	0	Nil
March 1969	Influenza A	3	1	2	3	Nil
	Adenovirus	3	1	2	3	Nil
	Herpes simplex	1	1	0	1	Nil
	Rhinovirus M. strain	1	0	1	1	Nil
April–May 1969	Influenza A	1	1	0	1	Nil
	Herpes simplex	2	2	0	2	Nil
	Mumps	2	1	1	2	Nil
July–Dec. 1969	Influenza A	2	1	1	2	Nil
	Adenovirus	2	0	2	2	Nil
	Herpes simplex	5	1	4	5	Nil
	Respiratory syncytial	1	0	1	1	Nil
	Parainfluenza	2	0	2	2	Nil
	Mumps	2	1	1	2	Nil
	Cytomegalovirus	1	1	0	1	Nil
Jan.–Feb. 1970	Influenza A	7	5	2	1	6
	Adenovirus	3	2	1	3	Nil
	Herpes simplex	1	0	1	1	Nil
	Influenza B	4	1	3	4	Nil
Total no. isolates		61	25	36	55	6

TABLE 2b *Pattern of respiratory virus and myoplasma pneumoniae infection in the Bedford area identified by serology (Public Health Laboratory, Bedford) February 1969–February 1970*

Date	*Virus*	*No.*	*Male*	*Female*	*Clinical*	*Single convalescent serum**	*Paired sera†*
Feb. 1969	*Myco. pneumoniae*	2	1	1	2	2	
Mar. 1969	*Myco. pneumoniae*	1	1	0	1		1
Apr./May 1969	Influenza A	1	1	0	1	1	
	Mumps	1	0	1	1	1	
July–Dec. 1969	*Myco pneumoniae*]	1	1	0	1	1	1
	Respiratory syncytial	1	1	0	1	1	
	Influenza A	3	1	2	3	2	1
Jan.–Feb. 1970	*Myco. pneumoniae*	1	0	1	1	1	
	Influenzia A	13	8	5	13	13	
Total No.		24	14	10	24	22	2

* Single convalescent serum – titres taken as significant are those accepted for inclusion in the communicable disease reports of the P.H.L.S. for *Myco. pneumoniae* and influenza titres are 1/256 or greater.
† Paired sera – showing more than fourfold rise in titre.

and myalgia in virus infections. Nevertheless, Sutton *et al.* (1967) and Zollner and Lydtin (1967) in their case reports on patients with viral myocarditis mention myalgia as a prominent symptom in the course of the illness. In fact, of 6 cases of myocarditis reported by Zollner and Lydtin (1967) 4 had myalgia.

Patients and methods

Patients in this study were either routine acute hospital admissions, or else were investigated as outpatients as suspected contacts of known infected cases.

In addition to viral studies, routine blood counts, ESR routine biochemical investigations including trans-

TABLE 3 *Pattern of enterovirus infection in the Bedford area identified by isolation (Public Health Laboratory, Bedford) February 1969–February 1970**

Date	*Virus*	*No.*	*Male*	*Female*	*Clinical*		*Necropsy*
July–Aug. 1969	Echo 6	3	0	3	3		Nil
	Echo 9	12	7	5	12		Nil
Sept.–Nov. 1969	Echo 6	1	1	0	1		Nil
	Echo 9	1	0	1	1		Nil
	Echo 30	2	0	2	2		Nil
	Echo 31	1	0	1	1		Nil
	Coxsackie A9	5	4	1	5		Nil
Total		25†	12	13	25		Nil
Grand total	Enteroviruses, respiratory viruses and Herpes group	109	45	64	102	7	Isolation 83 Serology 26

* There were no enterovirus isolations in the periods Feb.–June 1969 and Dec. 1969 – Feb. 1970.
† Of these 25 cases 7 were from specimens of cerebrospinal fluid, 14 from throat swabs, and 4 from faeces. Twelve cases were from the children's ward – mostly meningitis, some with pyrexia of unknown origin. Of 7 adult cases all had meningitis. Four cases referred by general practitioners to the public health laboratory were in children with pyrexia of unknown origin.

aminases, chest *x*-ray, and a 12-lead electrocardiogram were carried out on all hospital patients.

Virological methods

Specimens of faeces and throat swabs were inoculated into secondary tissue cultures of rhesus monkey kidney and primary tissue cultures of human amnion. The maintenance medium was Earles B.S.S. with 0·5 per cent lactalbumin hydrolysate with antibiotics, 0·2 per cent $NaHCO_3$, and 2 per cent horse serum. The tubes were incubated stationary at 36°C and were examined for cytopathic effect over a period of 10 days before being discarded. In case of doubt the tissue culture fluid was passaged.

Cytopathic agents were identified by neutralizing tests using sera prepared by Standards Laboratory at Colindale.

Complement-fixation tests were carried out by the technique described by Bradstreet and Taylor (1962).

The pattern of viral and *M. pneumoniae* infections in the Bedford area identified by isolation or serology between February 1969 and March 1970 is shown in Tables 2a, 2b, and 3.

Case histories

Echo infections

Case 1 Mrs. G.D., age 20 years, had echo-9 infection. On 10 July 1969 when 20 weeks' pregnant she complained of generalized muscle pains, followed 48 hours later by frontal headache with nausea and a rubelliform rash of face, abdomen, and legs.

On 14 July she was admitted to hospital with severe headache, vomiting, photophobia, and neck stiffness.

Examination revealed a pyrexia of 37·8°C, with meningism and muscular tenderness with the rash described above. Pulse 96 a minute and regular. No heart murmur, added sounds, pericardial, or pleural friction were heard. Blood pressure was 110/70 mmHg.

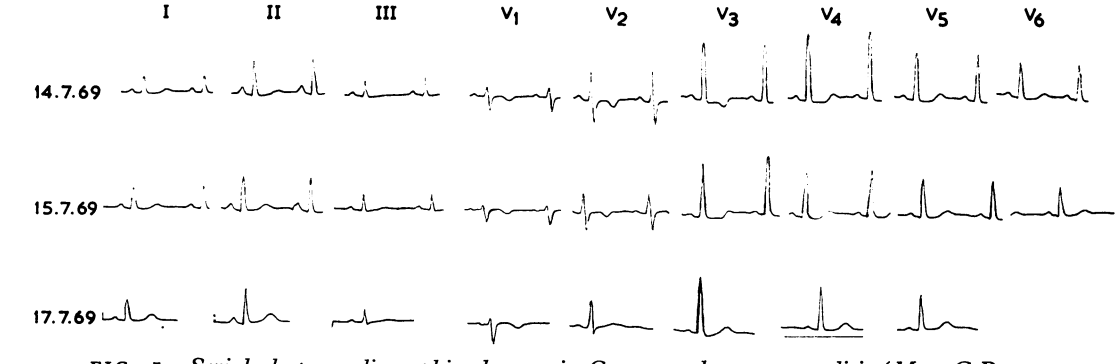
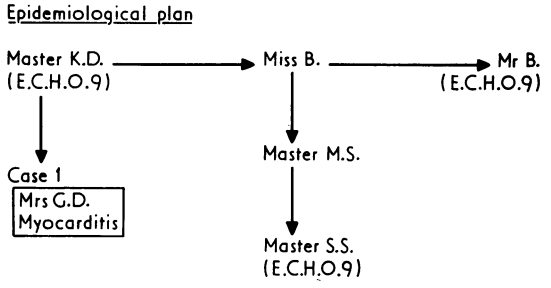


FIG. 1 *Serial electrocardiographic changes in Case 1: echo-9 myocarditis (Mrs. G.D. age 20 years).*

TABLE 4 *Symptoms of patients admitted with echo-9 viral infection*

Case	*Myalgia*	*Headache*	*Nausea/vomiting*	*Photophobia*	*Lassitude*	*Abdominal pain*	*Electro-cardiogram*
G.D.	⊞	+	+	○	+	○	⊞
M.S.	○	+	+	○	+	○	Neg.
S.S.	○	+	+	+	+	+	Neg.
S.B.	○	+	+	+	+	○	Neg.
M.G.	○	+	+	+	+	+	Neg.
M.C.	○	+	+	+	+	○	Neg.

TABLE 5 *Epidemiological plan relating to Case 1 (Mrs. G.D. and her family and contacts)*



Investigations Lumbar puncture revealed nothing abnormal; haemoglobin 68 per cent; ESR 63 mm in one hour (Westergren); WBC 5,500/mm^3; abnormal mononuclear cells 500/mm^3; Paul Bunnell: negative. Serial electrocardiograms (Fig. 1) showed ST changes maximal in V1 to V3, reverting to normal within 4 days.

Viral studies The epidemiological plan relating to Mrs. G.D. (Case 1), her family, and contacts is shown in Table 5.

Echo-9 virus was isolated from her son, K.D., the first to become ill, who had a similar rubelliform rash as did his mother, G.D. This child's infection was followed by a comparable illness in his girl playmate, B., whose father was admitted to hospital with an identical illness to G.D. (Case 1), apart from electrocardiographic evidence of myocarditis. From him echo-9 virus was isolated. Schoolgirl B's playmate, M.S., also became ill and infected his brother, S.S., age 12, from whom echo-9 virus was isolated from both throat swab and stools.

Case 2 Mrs. V.I., age 24 years, was admitted on 8 November 1969 with a 48-hour history of myalgia and of pain in the neck with nausea and vomiting.

Examination revealed pyrexia of 38°C, and muscle tenderness. The pulse rate was 100 a minute and regular.

There was no heart murmur or clinical evidence of pericarditis or of pleurisy, and no triple rhythm was present.

Investigations Lumbar puncture: 25 lymphocytes/mm^3; serum aspartate aminotransferase (SGOT) 10 units; Paul Bunnell: negative.

Electrocardiogram (Fig. 2) showed partial right bundle-branch block (perhaps left anterior hemiblock). Coving and depression of the ST segment from V1 to V6 and inverted T in V1 and V2; T was flat in V3. On 9 November isoelectric ST segments; TV2 flat; TCR1 inverted; and on 10 November TCR1 diphasic (Fig. 2).

Viral studies Echo-30 virus isolated from throat swab and stools.

Influenza infections

Case 3 Richard J., aged 14 years, was admitted on 3 March 1969 with severe influenza-like illness, characterized by sore throat, cough, mucoid sputum, and generalized myalgia.

On examination he was apyrexial. The pulse was 100 a minute and regular. There was no murmur or triple rhythm and no pericardial or pleural friction rub, but signs of consolidation were present at the left lung base.

Investigations On 5 March 1969 SGOT 45 units (normal 0 to 14 units); ESR 45 mm in one hour (Westergren); WBC 15,000/mm^3. On 15 March SGOT was 30 units; ESR 22 mm in one hour (Westergren).

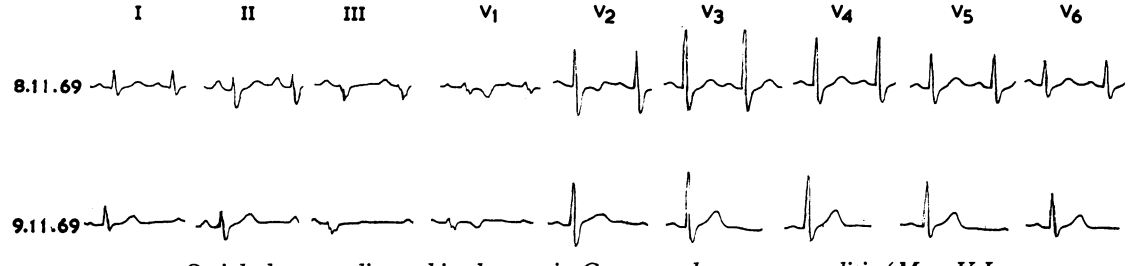


FIG. 2 *Serial electrocardiographic changes in Case 2: echo-30 myocarditis (Mrs. V.I. age 24 years).*

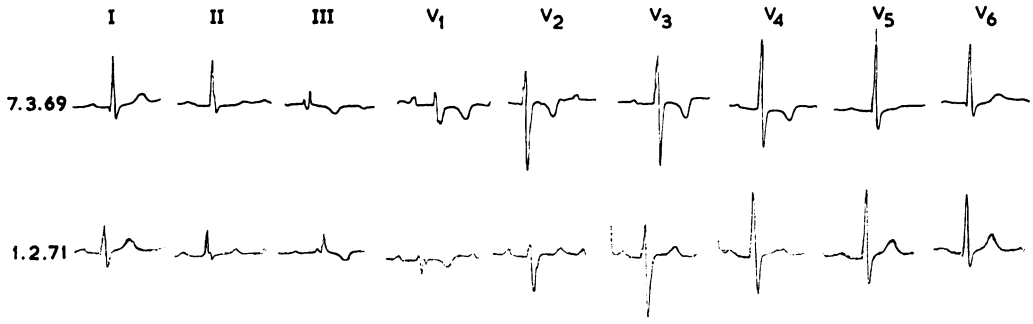


FIG. 3 *Serial electrocardiographic changes in Case 3: influenza-A_2 myocarditis (Richard J. age 14 years).*

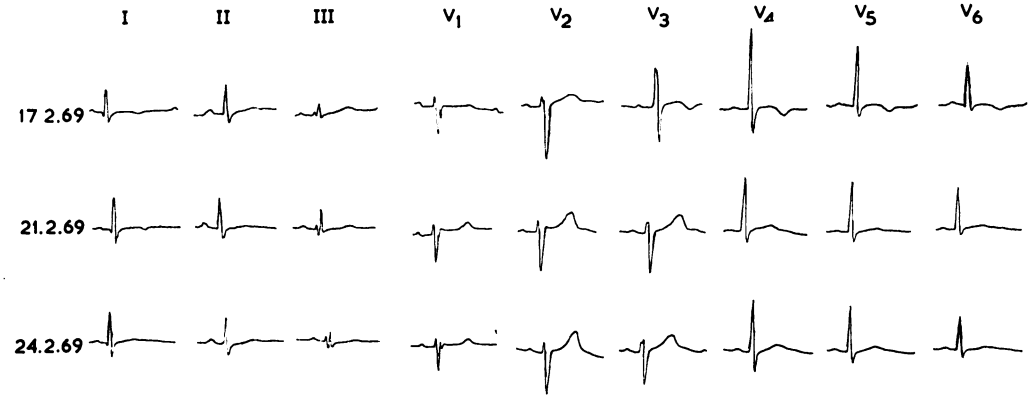


FIG. 4 *Serial electrocardiographic changes in Case 4: influenza-A_2 myocarditis (David S. age 25 years).*

Viral studies On 5 March influenza A CFT 1/640; on 15 March CFT 1/320.

Chest *x*-ray showed consolidation of lingula, which had cleared by 14 April. Electrocardiogram (Fig. 3) showed sinus rhythm at 100 a minute; partial right bundle-branch block, ST depression, and deep T wave inversion in V4R to V4, persisting for 8 days when the ST segment became isoelectric, but with T inversion from V1 to V4. On 11 April 1969 there was T inversion in V1 to V3; these changes slowly resolved over the next 6 months and by 1 February 1971 the tracing was normal in V leads, apart from the early partial right bundle-branch block but there was still inversion of TCR1.

This case is exceptional in retaining an abnormal electrocardiogram for nearly two years.

Case 4 David S., aged 25 years, was admitted to hospital on 17 February 1969 with a 3-week history of an influenza-like illness, with cough, generalized debility and severe muscle pains at the onset. His family had all suffered from a similar illness. On examination his general condition was good. There were signs of consolidation at the left lung base, which were confirmed radiologically. The pulse was 125 a minute and regular. No heart murmur or triple rhythm and no pericardial or pleural friction rub were heard.

Investigations WBC 12,000/mm^3; ESR 18 mm in one hour (Westergren); SGOT normal.

On 26 February, influenza A infection was diagnosed by CFT 1/320.

Electrocardiogram (17 February) (Fig. 4) showed sinus rhythm at 85 a minute; coving of ST and inversion of the T wave in I, aVL, and V3 to V6. There was steady regression of the ST changes and one week later the electrocardiogram was normal.

Mycoplasma pneumoniae

Of 5 cases of *M. pneumoniae* infection, two young women aged 19 and 27 years diagnosed on both clinical and serological evidence presented with a history of severe muscle pain and both showed abnormal electrocardiograms.

Case 5 J.O'B., aged 19, a known case of congenital heart disease with complete transposition of the great arteries; atrial septal defect; ventricular septal defect; valvular pulmonary stenosis; left superior vena cava

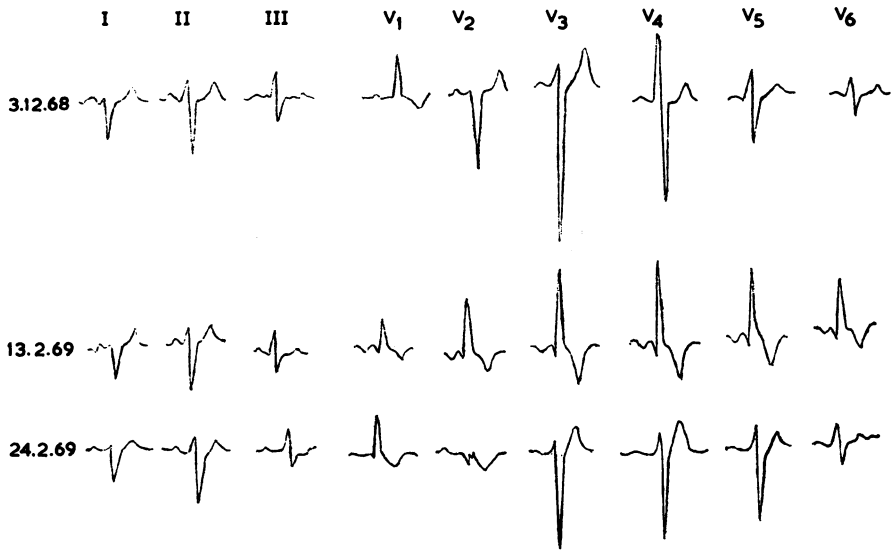


FIG. 5 *Serial electrocardiographic changes in Case 5 (Miss J.O'B. age 19 years).* Mycoplasma pneumoniae *myocarditis.*

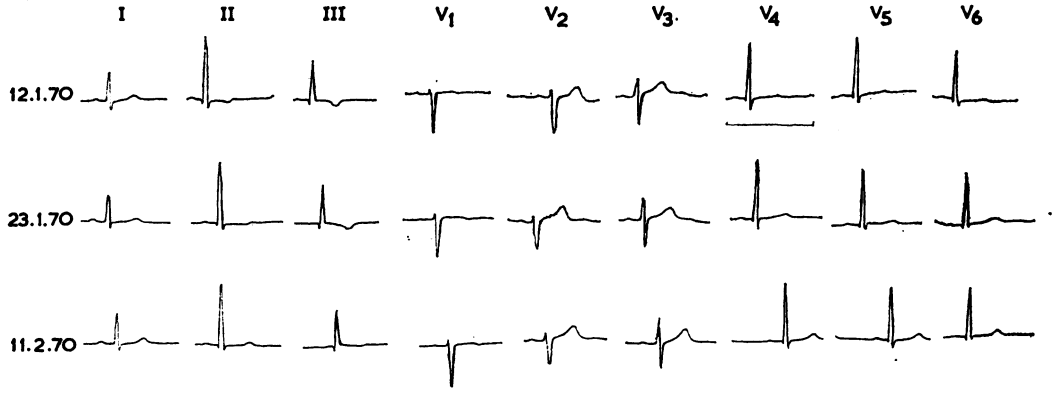


FIG. 6 *Serial electrocardiographic changes in Case 6:* M. pneumoniae *myocarditis (Patricia T. age 27 years).*

draining into the right atrium (investigated at Hammersmith Hospital by Professor J. F. Goodwin in June 1967).

This girl was seen at home by D.L. on 13 February 1969 after an 'influenza-like illness', with fever, severe myalgic pains, but no cough. A similar illness had earlier affected the family, who had all recovered when the girl was seen on a domiciliary visit. She was feverish with dusky cyanosis on examination and there was considerable cardiac enlargement, with a regular pulse, a right ventricular lift, and a widespread long, rough systolic murmur.

The electrocardiogram (Fig. 5) of 13 February shows striking changes compared with the earlier 'control' tracing, namely deep symmetrical T wave inversion from V4R to V6. She was admitted to hospital and within a few days of the home visit developed basal pneumonia. The last cardiogram taken on 24 February shows virtual recovery of the P and T wave inversion in the chest leads.

The constant ventricular rate in both limb and chest leads makes it unlikely that the bizarre complexes in the chest leads were due purely to the appearance of an abnormal atrial focus. There was no question of the leads being transposed between the recording of the limb and chest tracings.

Case 6 Mrs. P.T., aged 27. A staff nurse at the hospital, had for some time been subject to bouts of paroxysmal tachycardia and rhinitis. On 8 January

1970 she complained of headache and neck stiffness which was thought to be compatible with viral infection. There was a low-grade pyrexia during the illness and she complained of intermittent attacks of rapid heart beat. In addition, she had complained of nausea and giddiness, particularly on sitting up. These symptoms were transient and when examined on 26 January 1970 she gave a history of myalgic pains which were generalized, accompanied by headache and fever 5 days before admission: myalgia was chiefly in the lumbar region, shoulders, and neck.

Serial electrocardiograms (Fig. 6) on 12 January 1970 show abnormalities which are reflected chiefly in the limb leads. The tracing of 12 January showed coving and inversion of the T wave in leads II, III, IIIR, and aVF, with flattening of the T waves in V4 to V6. On 23 January the T waves were diphasic but upright and the coving of the ST segment had disappeared.

Discussion

Symptomless myocarditis in one-third of our patients suggests that the condition is common, but rarely diagnosed because electrocardiograms are seldom recorded in uncomplicated viral or *M. pneumoniae* infections. Thus, Bell and Grist (1970) mentioned cardiographic changes in only 2 of 833 proven cases of echovirus infection, one with rheumatic heart disease, the other with pericarditis.

Myocarditis is reported to be uncommon in adults or older children (Tricot *et al.*, 1966; Null and Castle, 1959; Connolly, 1961) and Coxsackie B infection is considered the commonest cause of viral myocarditis by Sutton *et al.* (1967), who cite extensive published material on its frequency.

Stevens and Underwood-Ground (1970) with unrivalled necropsy data on healthy subjects dying in aircraft and road accidents found evidence of asymptomatic focal myocarditis in 5 per cent of men between 18 and 50 years. Stevens' findings and the frequency of symptomless myocarditis in our patients suggests a possible aetiological link between his pathological and our clinical groups.

Myocarditis due to *M. pneumoniae* (Eaton agent), first reported by Lewes and Rainford (1970), is probably a common cause of symptomless myocarditis which was found in 2 of 4 patients with this infection in the present study. Recently Gerzén *et al.* (1972) have identified *M. pneumoniae* as the cause of frank myocarditis in several patients.

In southern England 19 per cent of the population possess complement-fixation antibodies to *M. pneumoniae* which can cause a wide variety of illnesses (Lambert, 1968, 1969). Infection may occur without overt illness. Thus, Chanock *et al.* (1961), reporting *M. pneumoniae* infections in 30 cases, found radiological evidence of pneumonia in only 1 subject. These observations and the present findings suggest *M. pneumoniae* infection to be a common cause, previously unrecognized, of symptomless myocarditis.

Myalgia was a presenting symptom in every patient with cardiographic evidence of myocarditis: of 12 patients with normal electrocardiograms and proved viral or *M. pneumoniae* infections only one complained of transitory muscle pains three days before developing pneumonia. The association of myalgia with an abnormal cardiogram was noted early in the study and each patient was closely questioned on this point.

We regard myalgia not only as a cardinal symptom presaging myocarditis in viral and *M. pneumoniae* infections, but also as an indication for an immediate electrocardiogram, despite the absence of cardiac symptoms or signs.

Myalgia is probably caused by short-lived viral involvement of voluntary muscle. In experiments with Coxsackie virus (type A9, strain 13) the virus soon loses its ability to replicate in striated muscle, but acquires cardiotropism in mice producing subclinical myocarditis (Lerner, Levin, and Finland, 1962). Myalgia features prominently in the case histories of patients with viral myocarditis reported by Zollner and Lydtin (1967), but the clinical significance of myalgia in relation to myocarditis appears to have escaped these authors and in no other case report has this association been recognized.

Cardiographic changes, sometimes so severe as to simulate septal cardiac infarction or pulmonary embolism, especially when accompanied by raised serum aspartate aminotransferase level, are usually present for 2 to 14 days, but occasionally persist for 6 months or more, as in our Case 3. In our cases and in those reported by Levander-Lindgren (1965) cardiographic changes dominate the right ventricular and septal cardiogram with coving and depression of the ST segment and deep inversion of the T wave. Similar but more evanescent changes may occur in the left ventricular surface leads; abnormalities, however, persist longer over the right ventricle, as in pulmonary embolism (Lewes, 1944). In none of our patients, however, was there clinical or radiological evidence of pulmonary embolism, acute cor pulmonale, or septal cardiac infarction. Moreover, the CR leads of Evans (1962) proved superior in our study to V leads in assessing final recovery of the right ventricular cardiogram. We did not consider that the cardiographic changes in our patients were likely to be due to acute pulmonary hypertension contingent upon the lung infection occurring in 3 of our patients in whom pneumonia was unilateral and strictly limited radiologically. Furthermore, none of our 6 patients showed triple rhythm which would be expected in

acute pulmonary hypertension and in myocardial infarction.

In symptomless viral myocarditis the standard and unipolar limb leads show little or no abnormality, though in one patient (Case 6) with *M. pneumoniae* infection a 12-lead electrocardiogram showed predominantly inferior myocardial involvement (Fig. 6).

Correct interpretation of the more florid and persistent cardiographic changes of symptomless myocarditis simulating myocardial infarction is of importance to the patient if unwarranted cardiac invalidism is to be avoided, especially in young adults. As our patients were 15 to 27 years old coronary artery disease was improbable. Nevertheless, the serial electrocardiograms sent to us by a cardiologist and an experienced general physician of 2 patients age 19 and 21 years, respectively, each with a diagnosis of ischaemic heart disease and probable cardiac infarction, were in our opinion characteristic of viral myocarditis. C. Oakley (1969, personal communication) recounts a similar patient in whom she replaced the original diagnosis of coronary artery disease by one of viral myocarditis, thus forestalling a life-long stigma and probable cardiac neurosis in the patient.

Bed rest for at least a week has been the rule of our patients. Unsuspected myocarditis may cause sudden or unexpected death (Stevens and Underwood-Ground, 1970; P. J. Stevens, 1970, personal communication; Suckling and Vogelpoel, 1958). Sutton *et al.* (1967) describe a 42-year-old man who complained of myalgia and dyspnoea attributed by him to obesity, and who went swimming to lose weight: rapid fatal heart failure supervened and at necropsy Coxsackie B4 virus was recovered from the myocardium. Monif, Lee, and Hsiung (1967) report a similar rapidly fatal case caused by echo-9 virus infection.

Exercise during incubation of certain virus diseases, e.g. poliomyelitis, increases the severity of the disease (Levinson, Milzer, and Lewin, 1945). Experimental animals are more liable to viral myocarditis if subject to hypoxia (Lyon, 1956), and mice made to swim after infection with Coxsackie type A9, strain 13 virus, show a higher incidence of virus isolation from the myocardium than do unexercised controls (Tilles *et al.*, 1964). Rabin and Melnick (1964), who showed that myocardial damage was caused by the cytopathic effect of virus replication within the myocytes, emphasize that extensive and fatal myocarditis in man may remain undetected unless specifically sought.

The question as to whether myocardial scarring from viral myocarditis may result in loss of cardiac reserve sufficient to contribute to heart disease at a later date remains uncertain. Somerville (1972), however, describes patients with known myocarditis in early life who on later follow-up developed frank cardiomyopathy with heart failure. Further research combining early electrocardiographic diagnosis of myocarditis and long-term follow-up with detailed pathological studies by workers such as Stevens and Underwood-Ground (1970), P. J. Stevens (1970, personal communication), Rabin and Melnick (1964), and Somerville (1972) may finally solve this important problem. It should be stressed, however, that the long-term prognosis in the majority of patients with viral myocarditis who survive the acute attack appears to be excellent (Gerzén *et al.*, 1972; *British Medical Journal*, 1972; Suckling and Vogelpoel, 1970).

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Addendum

In recent months, since the above report, we have seen two further proved cases of viral myocarditis (influenza A_2 infection and glandular fever) and a third case of *M. pneumoniae* myocarditis. Influenza A_2 infection occurred in a young airman in his 20's; *M. pneumoniae* in a woman of 23 years; and severe infectious mononucleosis in a man of 34 who developed anginose glandular fever with adenopathy, a rubelliform skin rash, hepatitis, and encephalitis.

In all 3 patients an electrocardiogram was taken on account of myalgic pains during the course of the illness. The initial cardiogram of each patient showed typical, but evanescent ST changes predominantly over the anteroseptal chest leads: and in none were there cardiac symptoms or signs or evidence of chest infection. All patients made a complete clinical and electrocardiographic recovery, with return of initially grossly abnormal cardiograms to normal within 1 to 3 days.

Follow-up of these 3 patients and those of the original series has revealed no evidence to date of cardiomyopathy or of permanent myocardial damage clinically or electrocardiographically.

Dr. David Verel and his co-workers, who have kindly allowed us to quote from their unpublished findings, read a paper at the Autumn meeting of the British Cardiac Society (1973) on 'Electrocardiographic changes in A2 England/42/72 influenza infection in patients treated at home'. *Of their 42 patients with cardiographic changes and proved viral infection, 40 gave a history of myalgia.*

We regard these findings of great significance in that they confirm independently our contention that myalgia accompanying viral infections is a reliable clinical indication of associated myocarditis as revealed by the cardiogram, even in the absence of cardiac symptoms or signs.

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