MASSIVE T WAVE INVERSION

By B. L. Chia, M. B. Ghosh and H. H. Tay

SYNOPSIS

Contrary to popular belief, massive inversion of the T waves in the ECG is not always caused by ischaemic heart disease. It can also occur in a wide variety of other clinical states such as in ventricular hypertrophy, electrolyte imbalance, after electrical cardioversion, in bradycardia, acute pulmonary insufficiency, in cerebral diseases, pericarditis, severe anaemia and after lightning strike. This paper is written with a plea for a greater awareness of the numerous causes of massive inversion of the T wave, in order to minimise the number of errors made in the diagnosis of ischaemic heart disease.

The T wave in the ECG represents ventricular repolarization and occurs during the latter part of systole. Of all the various components of the PQRST complex, the T wave is the most labile, being affected by a host of diverse factors, such as after ingestion of a meal (Sears and Manning, 1958), smoking, drinking cold water, severe emotional upsets, drugs like digitalis, beri-beri, hypopotassaemia, hyperthyroidism (Friedberg, 1967) and ischaemic heart disease. In all these conditions, a non-specific change may be found in the form of a flattening or shallow inversion of the T wave, with or without ST segment depression.

In marked contrast to this, the intriguing but uncommon finding of massively inverted T waves in either the standard or praecordial leads has often been regarded as being distinctly ominous in nature, as they are thought to be usually due to coronary artery disease (Garcia Palmieri *et al*, 1956; Pruitt, Klakeg and Chapin, 1955 and Wood and Wolferth, 1934). However, according to Jacobson and Schrire (1966), deeply inverted T waves can be produced by several causes, of which coronary artery disease is but one example. We document in this paper the many instances of massive T wave inversion of varied aetiologies which we have encountered in the past few years.

CASE RECORDS

Case 1

The patient, a 54 year old Chinese man, was admitted to Medical Unit I in April 1970 for a classical history of acute myocardial infarction. He was found to be in shock with a blood pressure of 60/20. An ECG done on admission on 24.4.70 (Fig. 1), showed deeply inverted symmetrical T waves and poor progression of the R waves in the praecordial leads, but no Q waves. His serum SGOT was 220 King's units. He was diagnosed and treated as for an acute myocardial infarction. An ECG repeated 5 days later showed T waves which were much less deeply inverted. His condition improved and he was discharged from the ward 19 days later.

Comments

The symmetrical and deeply inverted T waves seen in the ECG of this patient were due to sub-endocardial infarction of the myocardium.

Case 2

The patient, a 37 year old Chinese woman, has been followed up in this unit for exertional dyspnoea since 1955. In 1967, a right heart catheterization was done and the results showed a left to right shunt at the atrial level. The pulmonary artery pressure was 96/42 and the pulmonary vascular resistance was 8 units/M^2. She was diagnosed as having an atrial septal defect with severe pulmonary hypertension. At no time did she present with any symptoms which might suggest that she could have ischaemic heart disease.

An ECG done on 7.8.65 (Fig. 1), showed gross right ventricular hypertrophy as evidenced by a qR pattern seen in V_1 and tall R waves seen in V_2 and V_3 and a rS pattern in V_5 and V_6. In addition, there was massive and symmetrical inversion of the T waves in V_5 and V_6.

Comments

The symmetrical and deeply inverted T waves in the ECG of this patient were due to the gross right ventricular hypertrophy which occurred as a result of severe pulmonary hypertension from an atrial septal defect.

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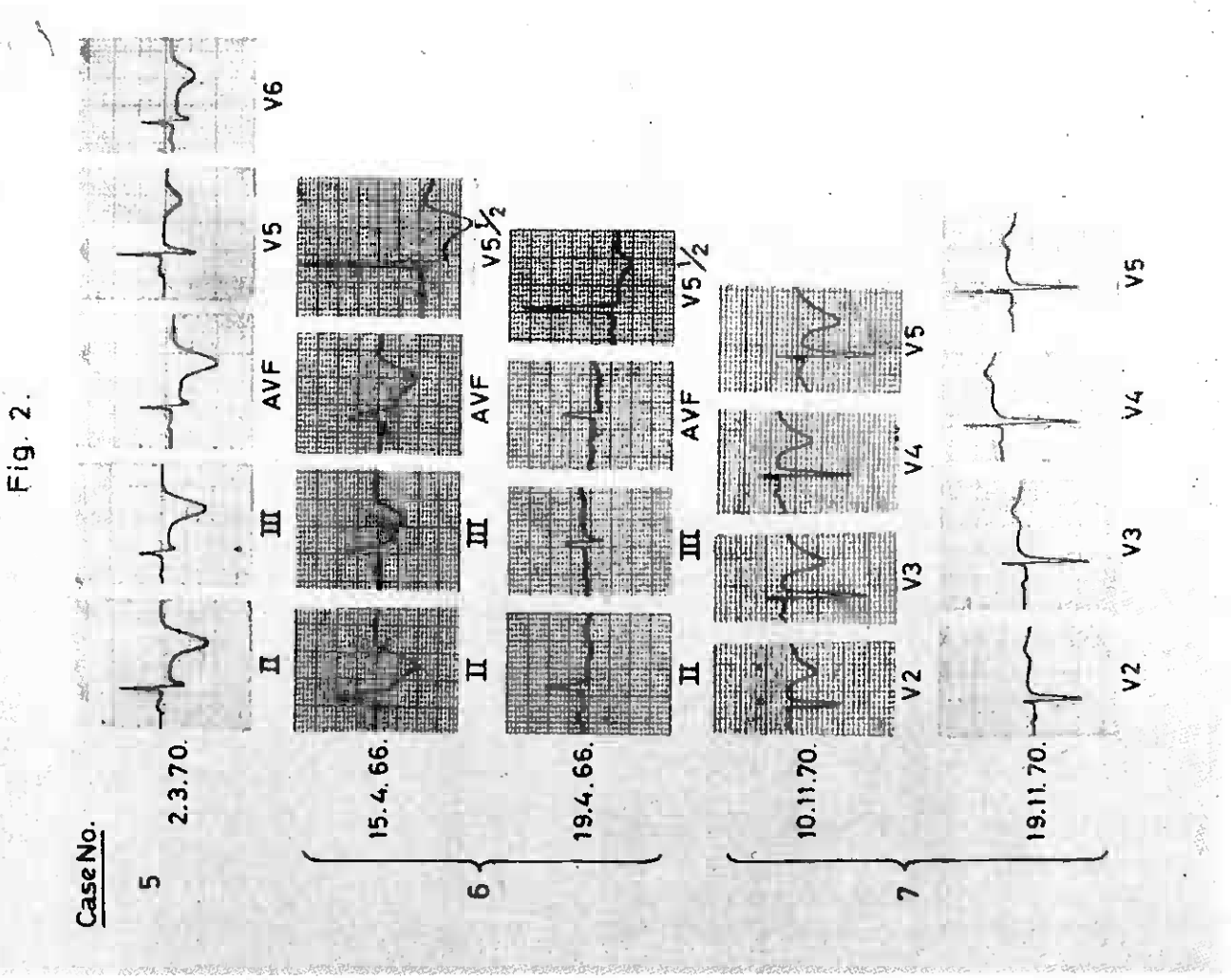

Case No.
5
2.3.70.
II
III
AVF
V5
V6
6
15.4.66.
19.4.66.
V5½
7
10.11.70.
19.11.70.
V2
V3
V4
V5

Fig. 2.

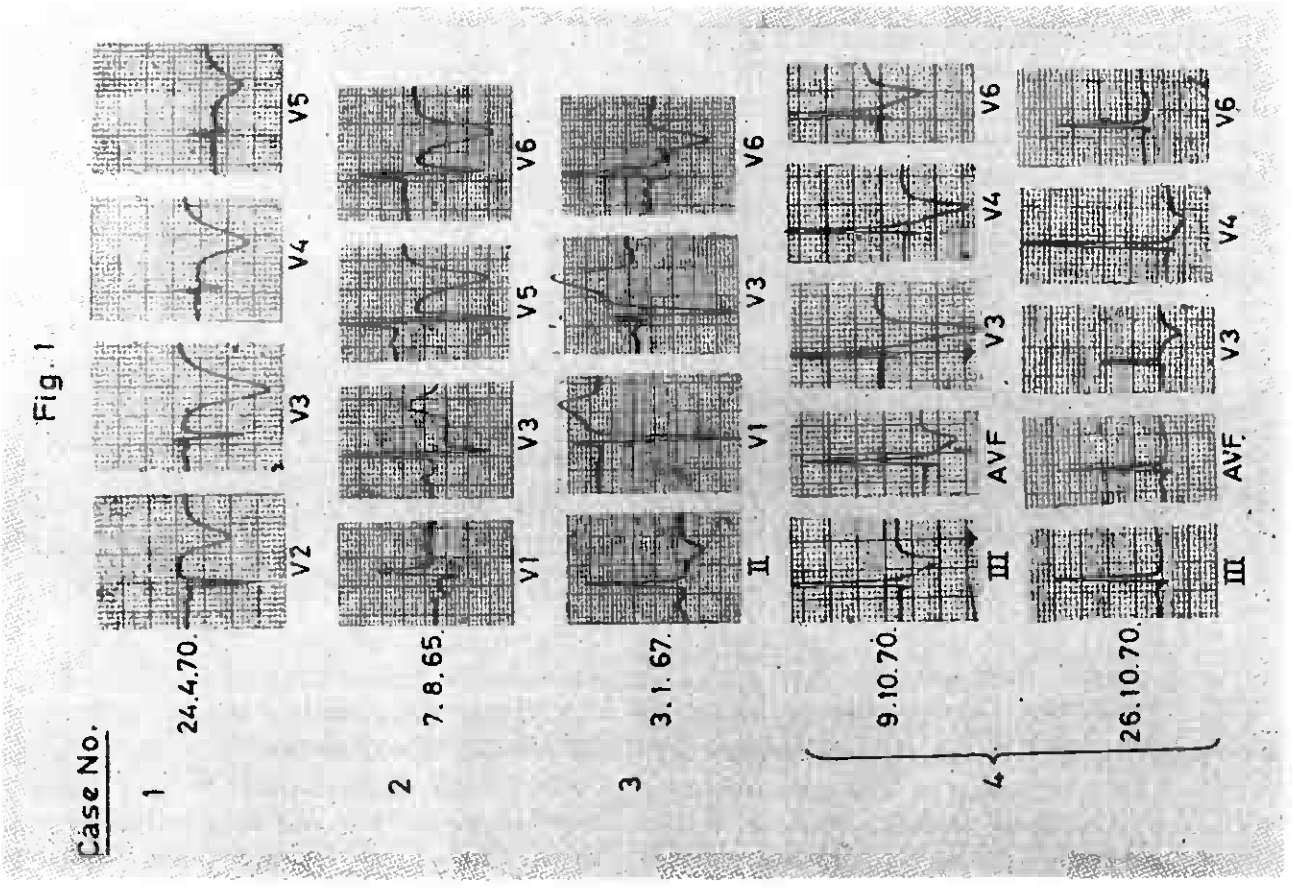

Case No.
1
24.4.70.
V2
V3
V4
V5
2
7.8.65.
V1
V3
V5
V6
3
3.1.67.
II
V1
V3
V6
4
9.10.70.
26.10.70.
III
AVF
V3
V4
V6

Fig. 1

Case 3

The patient, a 25 year old Chinese man, has been followed up closely in this unit since 1963 for mild aortic stenosis and severe aortic incompetence. At no time did he have any symptoms which could be attributed to ischaemic heart disease.

An ECG done on 3.1.67 (Fig. 1), showed changes of left ventricular hypertrophy. In addition there was gross inversion of the T waves over the left praecordial leads as seen in V_6. His ECG has remained the same since 1967.

Comments

The gross inversion of the T wave in the left praecordial leads in this patient was due to the severe left ventricular hypertrophy which occurred as a result of aortic valvular disease.

Case 4

A 48 year old Chinese woman was admitted to the unit for overt signs of thyrotoxicosis. She was also found to be hypertensive with a blood pressure reading of 190/90, uraemic with a blood urea of 239 mgm.%, and in congestive cardiac failure. The ECG on admission, showed shallow inversion of the T waves associated with slight ST segment depression. She was treated with digitalis and chlorothiazide.

On 9.10.70, the serum potassium was found to be 2·9 mequiv./L. and the serum calcium was 8 mgms./100 cc. The ECG (Fig. 1) then showed giant T wave inversion due most probably to the low serum potassium, as the SGOT was within the normal limits. With the correction of the hypokalaemia, a repeat ECG on the 26.10.70 showed only a residual shallow inversion of the T waves due either to the existing hypertension or to the digitalis therapy she was then receiving.

Comments

The massive inversion of the T waves which occurred on the 9.10.70 was due to hypokalaemia.

Case 5

The patient, a healthy 23 year old Chinese man, has been seen in this unit since 1964 for recurrent attacks of atrial tachycardia. In between the attacks of tachycardia, his ECG has always been perfectly normal, there being no Wolf Parkinson White Syndrome. Other causes of recurrent tachycardia have also been excluded.

On 1.3.70, he presented with an atrial tachycardia together with hypotension, the systolic blood pressure being 70 mm. Hg. and the diastolic pressure being unrecordable. He was successfully cardioverted with a synchronized D.C. shock and remained in sinus rhythm throughout his stay in hospital.

An ECG done on 2.3.70 (Fig. 2) one day after cardioversion, showed deeply inverted T waves in the standard and praecordial leads. When the ECG was repeated five days later, it was completely normal with upright T waves.

Comments

The deep inversion of the T waves seen was due to the electrical cardioversion done.

Case 6

The patient, a 65 year old Chinese man, was closely followed in this unit since 1962 for hypertension and chronic renal failure. His blood pressure was stabilized at around 190/120 with hypotensive therapy and his blood urea was maintained around 190 mgms.% with conservative treatment. His ECG showed changes of left ventricular hypertrophy, and there was slight depression of the ST segment in the left praecordial leads.

On 15.4.66, he developed complete heart block of unknown cause. His ECG then (Fig. 2) showed deeply inverted T waves in addition to the change in rhythm. The serum SGOT and electrolytes studied during the period when he was in complete heart block were all normal. He was treated with oral ephedrine and prednisolone and reverted to normal sinus rhythm on 19.4.66. A repeat ECG done at this time was identical to that before the onset of the heart block, showing only changes of left ventricular hypertrophy with slight depression of the ST segment in V_5 and V_6.

Comments

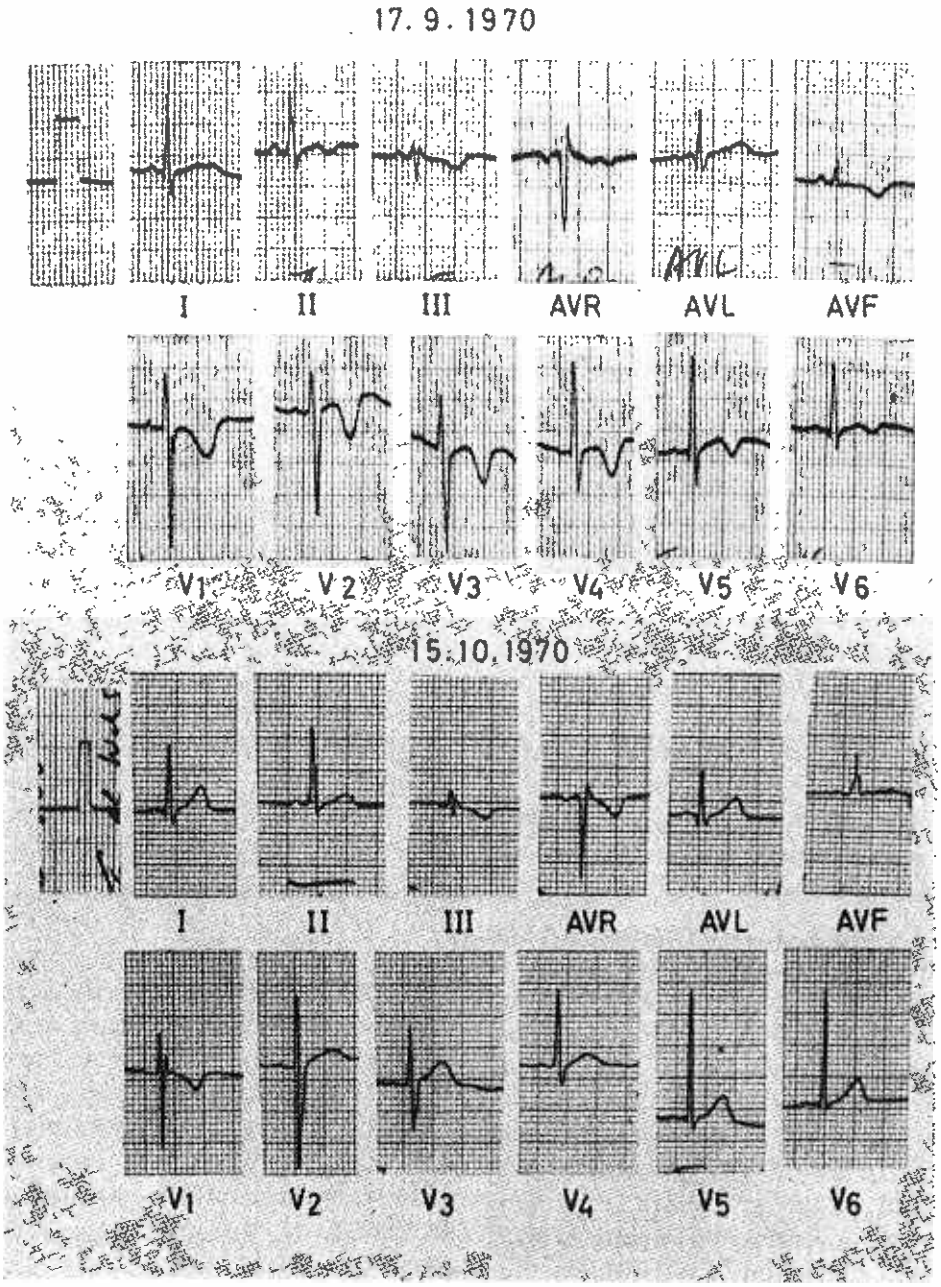
The deeply inverted T waves observed during the period of complete heart block were most likely due to the slow heart rate, rather than any myocardial disease.

Case 7

The patient, a 59 year old Chinese woman, presented on the 9.11.70 for an acute episode of respiratory insufficiency due to pulmonary infection. Previous to this, she had been suffering from exertional dyspnoea for 8 years. Clinical examination, radiography of the chest and lung function tests showed that she was suffering from an obstructive lung disease.

An ECG done on 10.11.70 (Fig. 2) showed deeply inverted T waves seen in the praecordial leads. The serum electrolytes and SGOT done

Fig. 3.



during this period was normal. Following vigorous therapy for the pulmonary infection, a subsequent ECG done 9 days later (19.11.70) showed normal upright T waves.

Comments

The deeply inverted T waves in this patient with chronic obstructive lung disease occurred during a period of acute pulmonary insufficiency.

Case 8

A 21 year old Chinese woman was admitted to the unit with a 5 day history of severe headache and vomiting. Clinical examination revealed signs of raised intra-cranial pressure as evidenced by bilateral papilloedema in the fundi and drowsiness. The heart was clinically normal and the blood pressure was 120/80. After an air encephalogram on 17.9.70 which showed a supra-sellar tumour, she lapsed into coma.

An ECG done on the same day showed deeply inverted T waves in most of the leads (Fig. 3). Serum SGOT and electrolytes estimated during this period were both normal. A shunt operation to decompress the brain was done on 19.9.70. The patient improved considerably after the operation. A repeat ECG on 15.10.70 showed a disappearance of the deeply inverted T waves and was essentially normal with a rSr pattern in V_1.

Comments

The deeply inverted T waves seen in this case were due to raised intra-cranial pressure associated with a brain tumour.

DISCUSSION

Massive inversion of the T waves has often been regarded as being usually due to ischaemic heart disease (Garcia Palmieri *et al*, 1956; Pruitt, Klakeg and Chapin, 1955 and Wood and Wolferth, 1934). However, the above eight cases illustrate the wide variety of conditions where this intriguing ECG finding may be seen.

According to Wood and Wolferth (1934), deeply inverted T waves which are strikingly symmetrical are typically seen in ischaemic heart disease. In the course of transmural myocardial infarction, associated Q waves are present as well, but solitary massive T wave inversion when due to ischaemic heart disease, usually denotes severe coronary insufficiency (Pruitt, Klakeg and Chapin, 1955) or subendocardial infarction (Levine, 1952). In Case 1, the typical history of coronary insufficiency together with signs of shock, a high serum SGOT and ECG signs of deeply inverted T waves and poor progression of the R waves in the precordial leads, all point strongly to a diagnosis of acute subendocardial infarction of the myocardium.

Cabrera and Monroy (1952) characterised the differences in the ECG findings between a systolic and diastolic overload of both the right and the left ventricle. According to them, a systolic overload of the right ventricle would show tall R and negative T waves over the right precordial leads in the ECG (as seen in Case 2) whereas similar changes would occur in the left precordial leads in systolic overload of the left ventricle (as seen in Case 3). Furthermore, Jacobson and Schrire (1966) commented that giant inversion of the T waves may occur in ventricular hypertrophy, particularly that of the right ventricle. In both Cases 2 and 3, it is reasonable to assume that the gross T wave inversion was secondary to the ventricular hypertrophy rather than to ischaemic heart disease, since both patients were relatively young and neither had any symptoms which could be attributed to myocardial ischaemia.

In Case 4, the massive T wave inversion which occurred on 9.10.70 was most likely due to hypokalaemia, since the serum potassium was 2·9 mequiv./L during this time. Myocardial infarction could be reasonably excluded because of a normal serum SGOT and absence of chest pain. Furthermore, when the hypokalaemia was corrected, the ECG showed only residual shallow ST segment depression due either to the existing hypertension or the digitalis therapy the patient was then receiving. Bellet *et al* (1966) described a wide variety of ECG patterns seen in hypokalaemia depending on the cause. Thus prominent U waves, depressed ST segment and deeply inverted T waves may be seen, the last occurring particularly in diabetic acidosis.

Resnekov and McDonald (1968) commented that following cardioversion from a D.C. shock, gross inversion of the T waves could occur. The serum SGOT done post cardioversion might also be raised due either to trauma to the skeletal muscle or some injury to the myocardium. In Case 5, the ECG done 1 day after cardioversion showed gross inversion of the T waves. However, another ECG done 5 days later was perfectly normal.

Massive inversion of the T waves may be found in the ECGs of patients who develop heart block not due to coronary artery disease (Jacobson and Schrire, 1966). In an analysis of the ECG pattern as related to the necropsy findings in a group of 42 patients with chronic complete heart block, Chatterjee *et al* (1970) found that whether underlying myocardial disease was present or not could not be predicted with any degree of certainty from the ECG findings. Thus widespread T wave inversion was noted not only in patients with myocardial disease, but also in those where the myocardium was relatively normal. Jacobson and Schrire (1966) described a patient with a Wenckebach phenomenon, whose ECG showed T wave inversion which became deeper as the R-R interval increased, as immediately after a dropped beat. Thus the deep inversion of the T waves in complete heart block and other bradycardias may be due to a slow heart rate, rather than being a reflection of underlying myocardial disease (Scherf, 1944). In Case 6, the massive T wave inversion which occurred when the patient was in complete heart block was most likely due to the slow heart rate rather than ischaemic heart disease as both the serum SGOT and the electrolytes done during this time were normal. Furthermore, when the patient reverted to sinus rhythm and a normal heart rate, there was a sudden disappearance of the deeply inverted T waves.

In a study of chronic obstructive airways disease in Singapore, Da Costa (1970) found that massive T wave inversion over the praecordial leads, simulating ischaemic heart disease closely, could occur in an episode of acute respiratory failure. With adequate therapy, the subsequent ECG showed reversion of the inverted T waves. In two patients showing such changes, necropsy

studies showed no evidence of coronary heart disease. This is further confirmed by the ECG changes seen in Case 7 who was suffering from a chronic lung disease and was admitted for acute respiratory failure due to lung infection. After vigorous therapy, the deeply inverted T waves became upright.

In subarachnoid haemorrhage, a variety of ECG changes, including deeply inverted T waves, have been amply documented since the first description by Burch, Meyers and Abildskov (1964). These same changes may also occur in a wide variety of other cerebral diseases, as after a grand mal-fit, following head injury, in encephalitis, in meningitis and in space occupying lesions (DePasquale and Burch, 1969). The present authors have encountered massive T wave inversion in subarachnoid haemorrhage, meningitis and cerebral tumours in young patients who show no evidence of ischaemic heart disease (Chia, Ghosh and Tay, 1971). The ECG in Case 8, 17.9.70, (Fig. 3) shows deeply inverted T waves and was taken from a 21 year old Chinese woman when she was in a coma due to raised intracranial pressure. With decompression of the brain following a shunt operation, the subsequent ECG recorded on 15.10.70 was normal. Since the serum SGOT and electrolytes estimated during the period the first abnormal ECG was recorded were both within the normal limits, the deeply inverted T waves seen could be assumed to be due to the cerebral disease.

The aetiological cause of the changes seen in the ECGs in patients with cerebral diseases has remained a source of controversy. Whilst Koskelo *et al* (1964) believe that these changes are indeed due to subendocardial injury, many others think that this is not so (Shuster, 1960; Burch *et al*, 1954 and Hersch, 1961). Furthermore, in most of the cases which showed these ECG changes and which went to necropsy, the heart had been normal. Hypokalaemia as a cause has also been excluded in many series (Hunt *et al*, 1969). De Pasquale and Burch (1969) have postulated that the ECG changes are due to intense sympathetic activity ("Sympathetic storm").

In addition to all the above causes, massive T wave inversion has been reported to occur in the ECGs of patients after termination of artificial cardiac pacing (Chatterjee *et al*, 1970) in pericarditis and severe anaemia (Goldman, 1967) and after lightning strike (Burda, 1966).

CONCLUSION

From all the above examples, it is clear that although the genesis of massive inversion of T waves is generally regarded as ill understood, it is vitally important to realise that it is not always due to ischaemic heart disease as is widely believed, but that it may also be encountered in numerous other conditions of diverse aetiologies, many of which are reversible, where there is a functional alteration of repolarization at the cellular membrane level. This fact should be constantly kept in mind in order to minimise the number of errors made in the electrocardiographic diagnosis of ischaemic heart disease.

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