# ELECTROCARDIOGRAPHIC CHANGES IN CEREBRAL DISEASES

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## SYNOPSIS

**The ECG findings in 13 patients with cerebral diseases due to meningitis, intracranial space occupying lesions or subarachnoid haemorrhage is reported. The ECG changes varied from tall peaked P wave, prolonged QTc, ST segment depression, tall and wide upright T wave, flattened and deeply inverted T waves. The mechanism of ECG changes are discussed.**


The ECG changes associated with cerebrovascular diseases, particularly subarachnoid haemorrhage, have been well documented in the past (Levine, 1953; Burch, Meyers, Abildskov, 1954 and Hunt, McRae, Zapf, 1969). However, little has been written about ECG changes occurring in other cerebral diseases such as meningitis or intracranial space-occupying lesions (Hersch, 1964).

This paper describes the abnormal ECG findings in 13 patients with cerebral diseases due to either meningitis, intracranial space-occupying lesions or subarachnoid haemorrhage.

## PATIENTS AND PROCEDURES

Table I summarises the clinical details of the 13 patients in this series. 4 patients presented with meningitis which was either to tuberculosis, leptospirosis, or acute lymphoblastic leukaemia. 3 patients were admitted for intracranial space occupying lesions, showing signs of raised intracranial pressure clinically. Arteriographic examination showed that the tumour was situated in the supra-tentorial region in Case 5, in the posterior cranial fossa in Case 6 and in the supra sella region (a craniopharyngioma) in Case 7.

Cases 8 to 13 were all admitted for subarachnoid haemorrhage. In every instance, lumbar puncture confirmed a uniformly blood-stained cerebro-spinal fluid. Cases 9 and 11 died in the acute stage of the illness. Carotid arteriography in Case 10 revealed a berry aneurysm arising from the junction of the left cerebral and the left anterior communicating branch artery. Bilateral carotid arteriography in Cases 8 to 12 failed to demonstrate any cause of the subarachnoid bleed.


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It is important to note that in this series, there was no clinical evidence of cardiac disease, e.g. pericarditis, coronary ischaemia or hypertension, in any of the patients except for Cases 10 to 12. Case 12 had clinical evidence of mild aortic incompetence and Case 10 had an initial blood pressure reading of 200/110 on admission, falling to 130/40 with anti-hypertensive therapy.

## RESULTS

### ECG Findings

No attempt is made in this study to assess the actual incidence of ECG abnormality encountered in either meningitis, intra-cranial space occupying lesion or subarachnoid haemorrhage, since a few of the cases in this series have been analysed retrospectively and serial ECGs have not been done routinely in the past in every patient with these cerebral disorders. However, a prospective survey to ascertain the actual incidence of each ECG change is being carried out at the present moment, the results of which will form the basis of a future communication.

The various ECG abnormalities found in this series are tabulated in Table II. Most of the ECGs were recorded within the first few days in patients presenting with either meningitis or subarachnoid haemorrhage. In the 3 patients presenting with intracranial space occupying lesions, the ECGs were done soon after they were admitted to the ward. The level of consciousness in each patient when the ECGs were recorded have also been tabulated in Table II.

12 patients showed a prolonged QTc interval ranging from 0·45 secs. to 0·50 secs. (average 0·48 secs.). Only Case 1 showed a tall P wave in Lead II measuring 3 mm. (Fig. 1). ST segment depression of more than 1 mm. was seen in Cases 2, 6, 7 and 12 (Figs. 1 and 2). Symmetrical deeply inverted T waves occurring in the precordial leads were present in Cases 1, 5 and 8 (Figs. 1 and 2). Minor but definite flattening of the T wave was found in 2 patients. In 4 patients (Cases 1, 3, 4 and 11) the T waves

TABLE I

CLINICAL DATA OF 13 PATIENTS WITH MENINGITIS, INTRACRANIAL SPACE-OCCUPYING LESIONS AND SUBARACHNOID HAEMORRHAGE

| Case No. | Age | Sex | Race | Diagnosis | Date of 1st ECG | Day of Illness | Level of Consciousness |
|---|---|---|---|---|---|---|---|
| 1 | 10 | M | CH | Leptospiral Meningitis | 3.1.66 | 4th | A |
| 2 | 20 | F | M | Tuberculous Meningitis | 5.9.70 | 9th | A |
| 3 | 15 | M | CH | Leptospiral Meningitis | 8.10.70 | 8th | A |
| 4 | 17 | M | CH | Leukaemic Meningitis | 13.9.70 | 2nd | B |
| 5 | 19 | F | CH | Supratentorial Tumour | 17.9.70 | 17th | B |
| 6 | 30 | F | M | Posterior Cranial Fossa Tumour | 5.9.70 | 3/12 | B |
| 7 | 15 | M | CH | Craniopharyngioma | 17.10.70 | 7/12 | B |
| 8 | 48 | M | CH | Subarachnoid Haemorrhage | 30.11.67 | 11th | B |
| 9 | 12 | F | CH | Subarachnoid Haemorrhage | 9.10.70 | 1st | C |
| 10 | 44 | F | CH | Subarachnoid Haemorrhage | 17.12.68 | 3rd | A |
| 11 | 65 | M | M | Subarachnoid Haemorrhage | 25.12.68 | 1st | C |
| 12 | 42 | M | CH | Subarachnoid Haemorrhage | 24.7.61 | 1st | B |
| 13 | 67 | M | I | Subarachnoid Haemorrhage | 5.11.70 | 7th | B |

CH = Chinese
M = Malay
I = Indian

A = Fully conscious
B = Drowsy or confused
C = Comatosed

were upright but very broad, measuring at least 7 mm. wide and stretching from the QRS complex to the P wave of the next heart beat, this not being accounted for by tachycardia (Figs. 1 and 2). In only one patient (Case 13) did the ECG, done 7 days after he had sustained a subarachnoid bleed, show tall, upright T waves measuring 12 mm. high with hyperkalaemia being excluded. A previous ECG done on admission 7 days earlier showed T waves of normal height measuring only 3 mms. A U wave was found in many of the cases, but in none was it considered abnormal.

**Duration of ECG Abnormalities**

Since most of the cases did not have serial ECGs, the exact duration of each abnormality could not be determined. However, repeated ECGs were carried out in 8 patients after they have recovered from their initial illness at varying periods of time after the first abnormal ECG—29 days (Case 1), 11 days (Case 2) 3 years (Case 3) 11 days (Case 4), 28 days (Case 5), 5 months (Case 8), 1½ months (Case 10) and 1 year 4 months (Case 12). In Case 5, a shunt operation to relieve raised intracranial pressure was carried out 24 days earlier. In every instance, the repeat ECG showed disappearance of the initial abnormal pattern (Figs. 1 and 2).

**Effect of Atropine**

IV Atropine 1/100 grain was given to Cases 2, 5 and 6 immediately after the first abnormal ECG was recorded. A subsequent repeat ECG done soon afterwards showed no change from the initial pattern.

**Effect of Electrolyte Imbalance**

Serum electrolytes done when the abnormal ECGs were recorded in Cases 2, 5, 6, 7 and 13 were all within the normal limits. In Cases 1 and 10, the serum potassium was 2·9 mEq./L.

## DISCUSSION

The ECG changes in patients presenting with cerebro-vascular diseases, particularly subarachnoid haemorrhage, have been amply documented in the past (Levine, 1953; Burch, Meyers, Abildskov, 1954; Cropp and Manning, 1960; Schuster, 1960; Hunt; McRae and Zapf, 1969 and Swiet, 1969). Various abnormalities have been described such as ST segment displacement (depression or elevation) tall P waves, T wave changes (deeply inverted, flattened or dimpled, wide and upright or tall and upright) U wave changes and prolongation or shortening of the QTc interval.

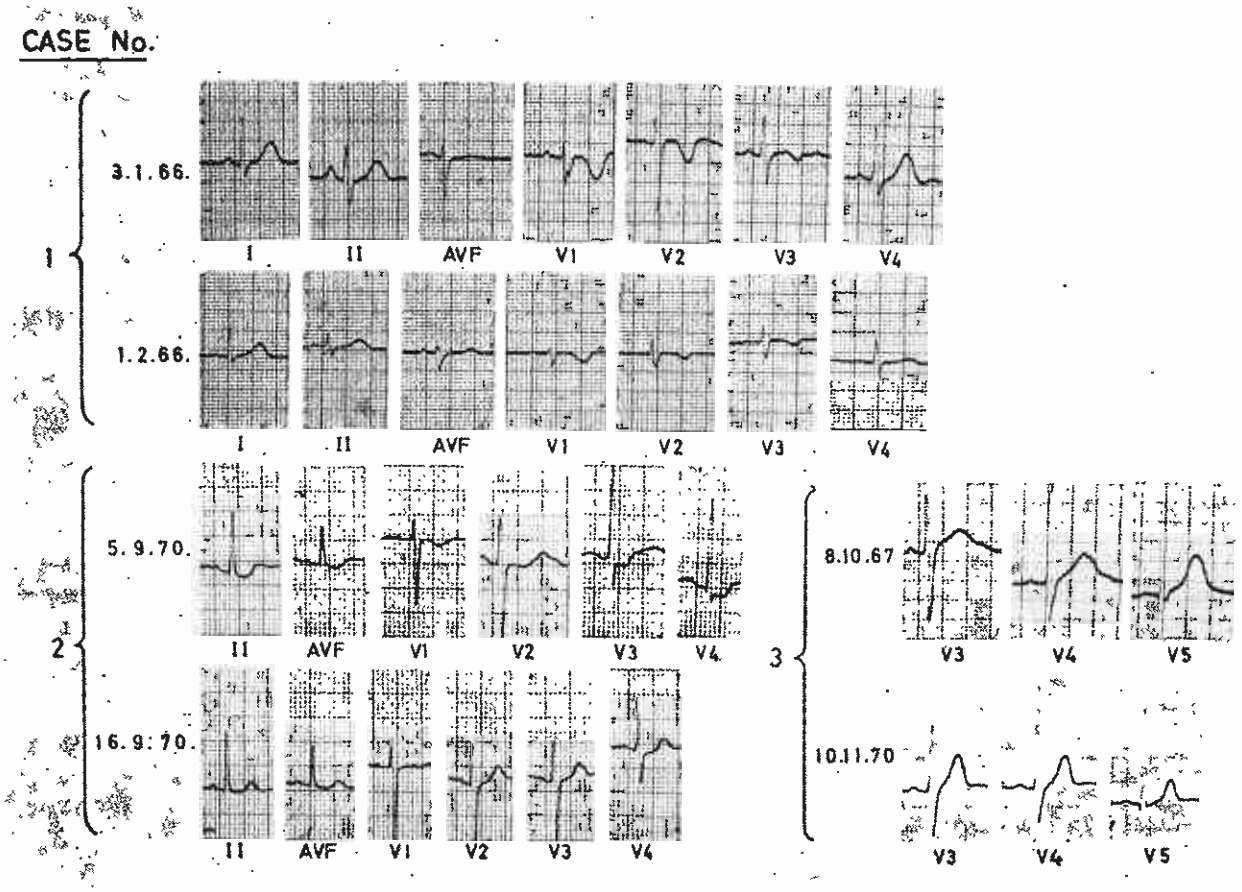


Fig. 1. E.C.G. changes in patients with cerebral diseases.

TABLE II

ECG ABNORMALITIES ENCOUNTERED IN 13 PATIENTS WITH MENINGITIS, INTRACRANIAL SPACE OCCUPYING LESIONS OR SUBARACHNOID HAEMORRHAGE

| ECG Abnormalities | Cases |
|---|---|
| T wave flattening | 7 (II), 9 (aVL, V4, V5) |
| Deeply inverted T wave | 1 (V1, V2), 5 (V1-V6), 8 (V1-V4) |
| Wide upright T wave | 1 (I, II, V4), 3 (V3-V6), 4 (V2-V4), 11 (I, II, III, V3-V5) |
| Tall upright T wave (more than 10 mm.) | 13 (V2, V3) |
| ST segment depression (>1 mm.) | 2 (II, aVF, V2-V4), 6 (II, III, aVF), 7 (III, aVF), 12 (II, III, aVF) |
| Prolonged QTc | 2 (0·49 secs.), 3 (0·48 secs.), 4 (0·47 secs.), 5 (0·46 secs.), 6 (0·49 secs.), 7 (0·49 secs.), 8 (0·45 secs.), 9 (0·46 secs.), 10 (0·49 secs.), 11 (0·52 secs.), 12 (0·49 secs.), 13 (0·50 secs.) |
| P II (>2·5 mm.) | 1 |

In contrast to the abundance of reports regarding ECG abnormalities in subarachnoid haemorrhage, documentation of ECG changes in other cerebral disorders such as meningitis or intracranial space-occupying lesions have been relatively scanty in the literature. Hersch (1964) described ECG changes in 60 patients, 20 with subarachnoid haemorrhage, 20 with meningitis and 20 with intracranial space occupying lesions of varied aetiologies. He found that ST segment depression of 1 mm. or more occurred in 25 percent of patients with subarachnoid haemorrhage, 5 percent with meningitis but none with intracranial space occupying lesions. In addition, inverted T waves were present in 20 percent of patients with subarachnoid haemorrhage and 15 percent of patients in both the meningitis and the intracranial space occupying lesion group. In their series, a high incidence of tall P and U waves and raised ST segment was seen in groups with meningitis and subarachnoid haemorrhage, whilst a prolonged QTc interval was seen only in subarachnoid haemorrhage, occurring in 45 percent of the patients.

In contrast to these findings, ST segment depression and deeply inverted T waves were found in all 3 groups of patients in this series. A prolonged QTc interval was also found in all 3 groups and was present in every patient in this series except for Case 1.

Large wide upright T waves have been documented in patients with subarachnoid haemorrhage (Burch, Meyers and Abildskov, 1954), but were found to be absent in the series reported by Hunt,

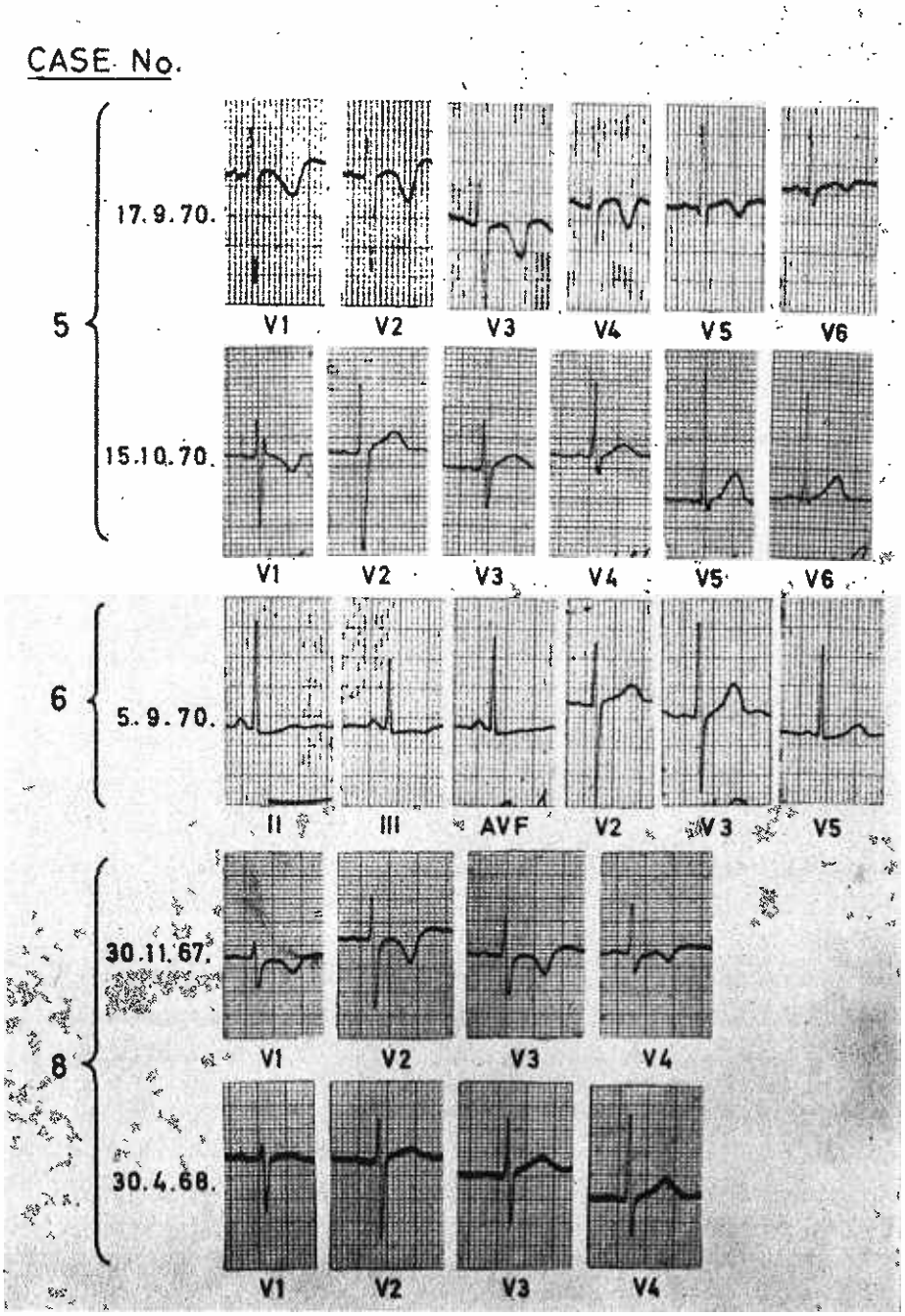


Fig. 2. E.C.G. changes in patients with cerebral diseases.

McRae and Zapf (1969) and Cropp and Manning (1960). Hersch (1964) did not find this change in his series of patients with subarachnoid haemorrhage, meningitis and intracranial space occupying lesions. It is interesting to note that in this series, very broad and upright T waves were present in Cases 1 and 3 (both leptospiral meningitis) and Case 11 (subarachnoid haemorrhage).

The exact incidence in this series of each of the ECG abnormalities in either subarachnoid haemorrhage, meningitis or intracranial space occupying lesions could not be determined for reasons already given (vide supra). Cropp and Manning (1960) found that T wave and ST segment changes suggestive of myocardial ischaemia were present in 40 percent of patients with subarachnoid haemorrhage. However Hunt, McRae and Zapf (1969) found T wave inversion in only 10 percent of patients, and postulated that many of the ECG changes attributed to subarachnoid haemorrhage were really due to concomitant hypertension or heart disease. However, it is reasonable to assume that in this series, all the ECG changes described have been due to the cerebral disease per series as none of the patients had any evidence of cardiovascular disease except for Cases 10 and 12. Case 10 showed only a prolonged QTc interval which could not be due to her hypertension. In Case 12, the ST segment depression seen during the actue subarachnoid bleed was most likely not due to the concomitant minimal aortic incompetence present, as a repeat ECG taken 1½ months later showed

that the ST segment has returned to the isoelectric line. Non-specific ECG changes have been described in leptospirosis without meningitis or overt clinical cardiac involvement (Parsons, 1965) but they do not in any way resemble changes described in Cases 1 and 3.

Since no serial ECGs were done in this series, the exact duration of each abnormality could not be determined. However, ECGs in 8 patients repeated at periods of time varying from 11 days to 3 years after the initial abnormal ECG showed a complete return to a normal pattern. In the series reported by Schuster (1960), serial studies done showed that the abnormal ECGs had returned to normal within 2 weeks in 5 of his 8 patients.

Several hypotheses have been suggested regarding the varied ECG changes seen in cerebral diseases. Gropp and Manning (1960) postulated that these changes are the result of lesions occuring around area 13 in the cerebral cortex, causing an increase of vagal stimulation. This was further supported by the fact that in a patient with ECG abnormality due to subarachnoid haemorrhage, atropine (given 1/100 grain IV) caused the depressed ST segment to return rapidly to the isoelectric line (Schuster, 1960). However, it is interesting to note that in this series, intravenous atropine 1/100 grain given to 3 patients elicited no change in the abnormal ECG whatsoever.

Another explanation given to the ECG changes has been that they are due to hypokalaemia, but this has been found to be absent in most series (Schuster, 1960 and Hunt, McRae and Zapf, 1969).

Since the ECG changes resemble ischaemic heart disease so closely, it has often been wondered whether they are indeed not the manifestations of myocardial ischaemia itself. Thus Kosskelo *et al* (1964) found subendocardial haemorrhages in the hearts of 3 patients who had subarachnoid haemorrhages and abnormal ECGs. However, in most of the cases reported, the heart and the coronary arteries have been found to be free of disease at necropsy (Cropp and Manning, 1960; Schuster, 1960; Harrison and Gibb, 1964 and Srivastava and Robson, 1964) although Connor (1968) claims that myocytolysis can be found in hearts of patients dying in a neuro-surgical unit. It is clear from this study that all the classical ECG changes that have been previously attributed to subarachnoid haemorrhage can also occur with meningitis and intracranial space occupying lesions. It is interesting to note that in this series 8 out of 13 patients were below the age of 30 years. The T inversion due to coronary artery disease in this young age group would be very unlikely.

In this era of cardiac transplantation where most of the donor material has been from the hearts of victims of subarachnoid and other cerebrovascular accidents, the keen interest shown regarding the exact significance of these ECG changes seen is certainly more than simply academic in nature.

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## REFERENCES

1. Burch, G.E., Meyers, R. and Abildskov, J.A.: "A new electrocardiographic pattern observed in cerebrovascular accidents." Circulation, 9, 719, 1954.
2. Chia, B.L., Ghosh, M.B. and Tay, H.H.: "Massive T wave inversion." Sing. Med. J., 12, 4, 210, 1971.
3. Connor, R.C.R.: "Heart damage associated with intracranial lesions." Brit. M.J., 3. 29, 1968.
4. Cropp, G.J. and Manning, G.W.: "Electrocardiographic changes simulating myocardial ischaemia and infarction associated with spontaneous intra-cranial haemorrhage." Circulation, 22, 25, 1960.
5. Harrison, M.T. and Gibb, B.H.: "ECG changes associated with a cerebrovascular accident." Lancet, 2, 429, 1964.
6. Hersch, C.: "ECG changes in subarachnoid haemorrhage, meningitis, intracranial space-occupying lesions." Brit. Heart J., 26, 785, 1964.
7. Hunt, D., McRae, C. and Zapf, P.: "Electrocardigraphic and serum enzyme changes in subarachnoid haemorrhage." Am. Heart J., 77, 479, 1969.
8. Levine, H.D.: "Non specificity of the electrocardiogram associated with coronary heart disease." Am. J. Med., 15, 344, 1953.
9. Parsons, M.: "ECG changes in Leptospirosis." Brit. Med. J., 2, 201, 1965.
10. Shuster, S.: "The electrocardiogram in subarachnoid haemorrhage." Bit. Heart J., 22, 316, 1960.
11. Srivastava, S.C. and Robson, A.O.: "Electrocardiographic abnormalities associated with subarachnoid haemorrhage." Lancet, 2, 431, 1964.
12. Swiet, J.D.: "Abnormal U waves in the electrocardiogram in subarachnoid haemorrhage." Brit. Heart J., 31, 526, 1969.