STUDY OF THE ELECTROCARDIOGRAPHIC CHANGES IN ACUTE BRAIN DISORDERS

THESIS

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By

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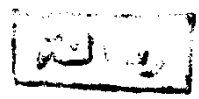

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ARABIC SUMMARY.

AIM OF THE WORK

Electrocardiographic abnormalities have been reported to follow brain trauma and cerebro-vascular accidents. There is a great controversy as regards the incidence, pattern and pathogenesis of such changes.

In the present work, we plan to study the E.C.G. abnormalities in relation to head injuries and different types of cerebro-vascular strokes, to identify the frequency and rate of reversion of such abnormalities under the different conditions and to explore whether extra cellular derangements of the electrolytes play any significant role.

It has been suggested that brain injury could lead to myocardial damage, this stimulated us to study the serum enzyme levels that are known to increase with myocardial necrosis in an endeavour to explore the validity of such hypothesis.

One of our aims also is to try to correlate between the E.C.G. abnormalities in acute brain disorders and the electroencephalographic abnormalities, if present, in such cases.

The type of E.C.G. abnormalities will be correlated also with the type and site of brain lesion in a trial to find whether specific brain areas are responsible for specific electrocardiographic changes.

I N T R O D U C T I O N

A N D

R E V I E W O F L I T E R A T U R E

E.C.G. CHANGES IN CEREBRO-VASCULAR ACCIDENTS, SUBARACHNOID HAEMORRHAGE AND HEAD INJURIES

The first account on this subject was made by Byer, Ashman and Toth[1] who in 1947 described large upright T. waves and prolonged QT interval in the E.C.G. of a patient with subarachnoid haemorrhage.

Levine[2] in 1953 in an extensive review of non-specific T. wave changes reported a case of subarachnoid haemorrhage whose E.C.G. showed initially widespread T.wave inversion and later ST segment elevation in V_5 & V_6 and the heart was normal at P.M.

Watt's and Fulton[3] in a complete review of the literature, summarized the effect of frontal lobe simulation. The area that appears most intimately linked with the cardio vascular system was the orbital surface of the frontal lobe (area 13) and the anterior cingulate gyrus (area 24). The result of stimulation of these areas was a rise or fall in the blood pressure and alterations in the heart rate. Respiration was also affected when area 13 was stimulated. Evidence was quoted that area 13 contains the chief cortical representation of the vagus nerve in man.

Burch and Meyers[4] in 1954 studied 17 patients with subarachnoid haemorrhage, intracerebral haemorrhage and other cerebro-vascular accidents. They drew attention to prolongation of QT interval and T wave abnormalities. In their series the increase in the QT was from 7 - 66 % above normal. The T waves were described as large with the configuration found in myocardial ischaemia. It was usually negative in the standard and chest leads although large positive T waves were encountered in the chest leads to the right of the transition zone. With improvement of the condition, T waves either returned to normal or changed to a pattern depending on the underlying heart disease prior to the cerebral insult. They also described prominent U waves. They believed that the large U wave contributed frequently to the height of the T waves.

Wasserman in 1956 studied 12 patients with various cerebro-vascular accidents. He demonstrated prolonged QT interval, inverted wide T waves and prominent U wave without co-existent electrolyte disturbances.

Kung[5] in China in 1958, examined 44 patients with cerebro-vascular accidents including 7 cases of subarachnoid haemorrhage, he described prominent U wave in 33 (75 %),

[illegible] in 2 (+.5 %), in all patients, the electrolytes were normal.

Shuster[6] classified 19 patients with subarachnoid haemorrhage in two types :

Those with primary subarachnoid haemorrhage and those with subarachnoid haemorrhage secondary to intra-cerebral haemorrhage.

In both groups, the findings were :-

1- Bradycardia, sinus arrythmia not related to the phases of respiration.

2- Transient auricular fibrillation in one patient, 3 had multiple atrial ectopics, ventricular ectopics in one patient.

3- Corrected QT interval was short.

4- U waves taller than 0.5 mm in 10 of 19 patients. It occurred in the standard and precordial leads of both groups.

5- T wave and ST changes :

a- In patients with secondary subarachnoid haemorrhage: In all patients, T wave was depressed or inverted in V_5 & 6 and in lead 1. There was slight ST segment depression in these leads.

All [illegible] high blood pressure with clinical or radiological evidence of hypertensive heart disease and the E.C.G. abnormalities could possibly be attributed to L.V. strain.

b- Primary subarachnoid haemorrhage :
In 6 (50 %) out of 12 patients, T wave was inverted with ST segment changes in lead AVF and precordial leads.

He found that the E.C.G. changes were not related to the site of the aneurysm, the degree of brain damage as assessed clinically or by the C.S.F. pressure.

Cropp and Manning[7] in 1960 reported that their interest in the E.C.G. changes simulating myocardial infarction or ischaemia in cases of subarachnoid haemorrhage and aroused since 1956 when in a case of subarachnoid haemorrhage proved by bloody C.S.F. and cerebral angiogram, the E.C.G. revealed a classical pattern of anterior myocardial infarction. Surgical treatment was delayed because of this finding and the patient suffered further intracerebral haemorrhage and succumbed. At P.M., the coronary arteries were patent and no evidence of myocardial infarction was found. Since then,

[illegible] studied [illegible] cases of R.A.A. comprising 18 females and 11 males. Their findings were as follows :

1- Heart rate : was variable being 59 or less in 4 patients, 60 - 100 in 23 and 101 or more in 2 cases.

2- Rhythm : Wandering pacemaker in 2, nodal rhythm in 1, nodal extrasystoles in 1 and ventricular ectopics in 3.

3- P wave : Normal in 25, spiked in 3 and in 1 (nodal rhythm) P was absent.

4- P-R interval : Varied from 0.1 - 0.18 sec. and was considered within normal.

5- Q R S complex :

Duration : Normal in 28 and in one patient it was 0.12. High voltage of QRS was present in 6 patients, 4 of these revealed other evidence of left ventricular hypertrophy, one patient revealed a pattern of right B.B.B. and one patient left axis deviation without other evidence of left ventricular hypertrophy.

Q wave : 4 patients revealed abnormal or questionable Q in II, III and A.V.F.

6- Axis : Left axis deviation in 10 patients and right in one patient.

7- Q-T interval, prolonged in 83 % of cases.

8- ST segment : Elevation or depression was found in 15 cases.

9- T wave : 16 of 29 patients showed T wave abnormalities mostly seen in lead I, aVL, mid and lateral precordial leads.

10- U wave : 24 of 29 patients showed U wave, 8 of which were considered abnormally large.

Hugenholtz[8] in 1962 described similar E.C.G. changes consisting of prolonged QT interval, changes of ST segment and T wave in cases of cerebro-vascular disorders.

Fentz and Gormsen[9] in 1962 studied 69 cases with acute cerebral infarction and 21 cases with intracerebral haemorrhage. The changes were considerably more common in patients with cerebral haemorrhage than in patients with primary infarction : 71 % as compared with 15 %. The E.C.G. changes often resembledthe findings in acute coronary occlusion i.e. alterations of the ST segment, usually depression, large inverted T waves. Prolongation of the QT interval above 0.45 sec. was observed in 5 (8 %) of 69 cases of

infarction and [illegible] cases of haemorrhage. They did not observe large or negative U waves. They stated that the changes of ST segment, T and U waves were completely or partially reversible but the changes in the QT usually persisted. They claimed that their results support the previous reports on the E.C.G. changes in cases of intracranial haemorrhage but they were able to find such E.C.G. changes also in cases of acute cerebral infarction.

Harrison and Gibb[10] in 1964 described a case of 17 years old female who was admitted in a state of stupor and upper motor neuron lesion of face and right hemiparesis. She delivered 4 months before. Her E.C.G. 2 days after admission revealed widespread ST depression, T wave inversion and prolonged QT interval, changes suggestive of ischaemic heart disease but returned to normal by the 16th day except for a prolonged QT interval. This patient was a case of cerebral venous thrombosis that developed during the puerperium.

Srivastava and Robson[11] in 1964 had presented a series of cases with similar E.C.G. changes, but they particularly referred to a case in their series who had pronounced, persistent and generalised vasoconstriction of the skin.