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Electrocardiographic abnormalities associated with cerebrovascular accidents

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ELECTROCARDIOGRAPHIC ABNORMALITIES ASSOCIATED WITH CEREBROVASCULAR ACCIDENTS

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I. INTRODUCTION

A. Previous Studies

Since 1947 several investigators have reported that cerebrovascular accidents may be associated with abnormal electrocardiograms even in patients without heart disease.[1-13] Although this observation had been made prior to 1954 in isolated examples, Burch and co-workers[3] were apparently the first to report this phenomenon in detail in 1954. In 17 cases, they described a prolonged QT interval and large, occasionally negative, T waves in the standard and chest leads. They stated that some of the widest and largest T waves seen in electrocardiography are seen in this syndrome. The T waves were reported to revert to normal with improvement in the clinical condition or changed to the pattern of any underlying heart disease prior to the intracranial insult. These workers also described U waves in the precordial leads which were usually located within the T waves. They believed that the close relationship between these 2 waves may have contributed to the QT interval. It was emphasized that subarachnoid hemorrhages were particularly

liable to produce these abnormalities.

Concurring data were provided by Wasserman and his associates[4] in 1956 in a study of 12 patients with acute cerebrovascular accidents, 11 of whom had previous heart disease. This group also described prolonged QT intervals, deep wide T wave inversions, and prominent U waves. Another report by Beard et al[5] described a 37 year-old white female with a spontaneous subarachnoid hemorrhage in which elevated ST segments and T wave inversions and severe substernal pain lead them to treat the patient with anticoagulants for myocardial infarction. The patient did not have a headache or nuchal rigidity until 3 weeks had elasped. She went on to expire but autopsy showed entirely normal coronary vessels without evidence of infarction.

The next detailed investigation did not appear in American publications until 1960 when Crop and Manning[6] reported ECG abnormalities to be associated primarily with subarachnoid hemorrhage. The most prominent features of their study of 29 patients were ST-T segment changes and T wave abnormalities. The typical pattern was one of flat or negative T waves in Lead I, aVL and V4-V6 along with ischemic RS-T segment changes. In a number of

cases these changes were suggestive of myocardial infarction. Shuster[7] in 1960 confirmed the studies of Crop and Manning; he also obtained results with T wave inversions and displacement of ST segments suggestive of myocardial infarction. Bradycardia, supraventricular arryhthmias, a short QT interval, and U waves were commom. Contrary to earlier studies Shuster noted a shortened QT interval in his series and it was his opinion that previous workers were in fact measuring the QU interval and not the QT interval.

In a comparatively large series of 90 cases, 69 of whom had acute cerebral infarction with the remainder having intracranial hemorrhage, Fentz[8] in 1962 also reported inverted T waves and prolonged QT intervals. The latter finding exceeding 0.45 second was observed in 5 of 69 cases of infarction and 7 of 21 cases of hemorrhage. However, this study differed from earlier ones by showing frequent large depressed RS-T segments in Leads I, II, and mid-precordial leads. Large or inverted U waves were not seen. In a smaller series of 6 cases, Hugenholz[9] in 1962 also observed prolonged QT intervals and inversion of T waves, but he reported prominent U waves in his results. Koskello[10] in his

report of 3 cases in 1964 also differed from earlier communications by noting inverted fused TU waves. All 3 of his cases had subarachnoid hemorrhages at necropsy. Contrary to earlier explanations, it is Koskello's contention that ECG changes found "in connection with intracranial bleeding are caused by cardiac lesions and that they do not simulate myocardial ischemia but are in fact signs of them." Harrison and Gibb,[11] also in 1964, reported a case of a cerebrovascular accident in a 17 year-old post-partum girl who showed ECG changes simulating those of severe ischemic heart disease. On the second day after admission the ECG showed widespread ST depression and T wave inversion with a QT interval of 0.49 seconds. The ECG pattern gradually returned to normal. ST segment depression was no longer present at 10 days and by 14 days the T waves became upright. The ECG tracing was normal on the 16th day except for the persistance of a QT interval of 0.44 seconds.

Srivastava and Robson[12] presented 4 cases of subarachnoid hemorrhage in 1964 in which the electrocardiographic changes consisted of abnormalities in the ST segment and T waves. They stated that inversion of the T waves occurred in 3 cases and

tall upright T waves appeared in 1 case. Significant prolongation of the QT interval was also noted. Menon[13] also reported a case of subarachnoid hemorrhage in 1964 which gave an ECG picture that simulated an anterior myocardial infarction.

A summary of the clinical literature appears in Table I.

B. Incidence of Abnormal ECG's in CVA's

It is apparent from Table 1 that few studies have been undertaken to reflect the incidence of this phenomenon. In addition, many authors have not indicated the frequency of these ECG changes. However, Crop and Manning[6] noted such abnormalities in 15 of 29 cases of subarachnoid hemorrhage. Shuster reported ECG irregularity in about one-half of 19 cases of subarachnoid hemorrhage. Fentz,[8] in 2 series of 67 patients with acute cerebral infarction and 21 patients with intracranial hemorrhage, 11 and 15 respectively showed electrocardiographic changes that could not be ascribed to cardiac causes.

Thus, it is not entirely clear how frequent these abnormalities occur; however, it is generally concluded that these changes are most often

associated with subarachnoid hemorrhage.[6,7,8]

C. Importance of Diagnosis

It is conceivable that surgical treatment of subarachnoid hemorrhage could be delayed in patients who reveal a classical pattern for myocardial infarction in the electrocardiogram. Such was the experience of at least one group[6] who found an entirely normal myocardium after the patient had succumbed from intracranial hemorrhage.

D. Purpose of This Paper.

The present investigation was made to study the frequency and character of electrocardiographic abnormalities in patients who succumbed to cerebrovascular accidents and to compare the results with those previously reported.

II. METHOD

This writer examined the post mortem records of patients at the Douglas County Hospital over a 2 year period from 10-1-62 to 10-1-64. After eliminating those patients in which ECG's were not taken or were lost, and those without complete post mortem examinations, 20 cases remained. At least one electrocardiogram is available for each case and in one case, 4 are available. Most ECG's were

Table 1 - Summary of the Literature (First 4 Items from Cropp and Manning[6])

Authors	No. of Cases	Intracranial Lesions	No. of Cases Having Heart Disease	Autopsy Heart	Electrocardiogram
Byer, Ashman, and Toth (1947)	5	4 cerebrovascular accident; 1 hypertensive encephalopathy	4	?	Large upright T waves; long QT interval
Levine (1953)	1	Subarachnoid hemorrhage	0	Normal	Myocardial infarction
Burch, Meyers, and Abildskov (1954)	17	Cerebral hemorrhage; subarachnoid hemorrhage; unclassified cerebrovascular accident	?	?	Large, wide T waves; long QT interval; prominent U waves
Wasserman, Choquette, Cassinelli, and Bellet (1956)	12	Subarachnoid hemorrhage; intracerebral hemorrhage; subdural hematoma; cerebral edema; cerebral thrombosis	11	1 case myocardial infarct	Long QT interval; deep, wide T wave inversion, prominent U waves
Beard et al (1959)	1	Subarachnoid hemorrhage	0	Normal	Elevated ST segments; T wave inversions
Cropp and Manning (1960)	29	Subarachnoid hemorrhage	9	5	RS-T segment and T wave changes

Table 1 - Summary of the Literature (First 4 Items from Cropp and Manning[6])

Authors	No. of Cases	Intracranial Lesions	No. of Cases Having Heart Disease	Autopsy Heart	Electrocardiogram
Fentz (1962)	90	Acute cerebral infarction (69); intracranial hemorrhage (21)	?	13	Abnormal ECG's (31); depressed RS-T segments; inverted T waves prolonged QT intervals.
Hugenholtz (1962)	6	Subarachnoid hemorrhage; cerebral thrombosis; intracranial hemorrhage	Could not be ruled out in (5)	1	Prolonged QT interval; wide inverted T waves; prominent U waves
Koskello, Punsor & Sipila (1964)	3	Subarachnoid hemorrhage (2); cerebral hemorrhage (1)	Subendocardial hemorrhage (3)	3	Inverted fused TU waves
Harrison, Gibb (1964)	1	Cerebral thrombosis?	?	0	ST depression; T wave inversion; prolonged QT interval; prominent U waves
Srivastava, Robson (1964)	4	Subarachnoid hemorrhage (4)	Calcific reduction of lumen(2) no myocard. damage	4	Prolonged QT interval; inverted T waves; tall upright T waves in one case
Menon (1964)	1	Cerebral infarction	0	1	Myocardial infarction

taken within 24 hours after admission; however, in one case, 15 days elapsed before the ECG was taken because the cerebrovascular accident did not occur until that time. All cases were examined at autopsy and the results, including ECG', are presented in Tables 2 and 3.

III RESULTS

A. Case Selection, Age, Sex, Diagnosis.

The electrocardiographic findings are presented in 20 cases of various types of cerebrovascular accidents in which the diagnosis was based on post mortem examination (Table 2). Ages range from 34 to 87 years with a mean of 64.0 years. There were 13 males and 7 females. Autopsy findings revealed 8 cases of cerebral thrombosis, 4 cases of subdural hemotoma secondary to trauma, and 4 cases of cerebral hemorrhage, 3 cases of spontaneous subarachnoid hemorrhage, and one case with encepholomaliela without a specific cause of death but who had neurological deficit consistent with a cerebrovascular accident.

B. Two Groups of Patients.

To evaluate the principal ECG findings in this study, all cases were divided into 2 groups,

one with minimal or no heart disease and the other with moderate to severe heart disease. Those cases in the first group have a heart weight less than 350 gms, minimal or no sclerosis, and a left ventricle not exceeding 20 mm in thickness. Those cases placed in the moderate to severe heart disease group have one or more parameters exceeding those of the normal limits in the first group. Table 3 and 4 compare the general findings of these 2 groups while tables 5, 6, & 7 compare the 2 groups more specifically with respect to serum potassium levels.

C. QT Intervals

In 6 tracings abnormally long QT intervals were found after correction for age, sex, and heart rate.[23] Two other tracings had QT intervals at the maximum upper extent of normal while 4 others were indeterminate. According to Table 5, there does not appear to be a relationship with serum potassium levels.

Normal serum calcium levels were observed in 2 cases (42542 and 49933) in which the QT interval was prolonged but these values were not obtained in the remaining patients. Therefore, it can not definitely be excluded that low serum calcium levels were respon-

sible for QT prolongation in the remaining 4 cases. However, the clinical history of these patients does not suggest abnormal serum calcium levels.

It has been proposed that prolonged QT intervals are caused by prominent U waves being incorporated into the Twave, but hypokalemia as a cause of this phenomenon was not suggested in this sstudy (Table 5).

If prolonged QT intervals are related to cerebrovascular accidnets in some way, the mechanism for this abnormality is not clear although several general theories have been advanced (see Discussion).

Table 5 seems to suggest that prolonged QT intervals occurred with greater frequency in the group with minimal or no heart disease. This may indicate that prolongation of the QT interval was caused by factors other than heart disease or abnormal serum potassium levels.

D. ST Segments

Eleven of the electrocardiograms have normal ST segments while the remainder are depressed, inverted, or absent, typically in leads I, aVL, and V5 - 6. These changes were usually nonspecific and often suggested hypokalemia, ischemia and/or

digitalis effect. Indeed, some of these suggestions are supported by Table 4. However, it is to be noted that most ST segment abnormalities appear in the group with moderate to severe heart disease, at least in those cases where serum potassium levels were obtained. Thus, there is no particular reason to attribute these changes to cerebrovascular accidents.

E. T Wave Changes

Thirteen of the 20 cases showed T wave abnormalities. Ten were depressed with the remainder being inverted or absent, usually in leads I, aVL, and V5 - 6. These changes appeared in 4 out of 6 patients with normal hearts and also in 2 out of 2 patients with normal hearts and normal serum potassium levels. It may be difficult to attribute these changes to cerebrovascular accidents, but this possibility can not be excluded. Moreover, it may be recalled that all previous investigators have reported some degree of T wave abnormality associated with cerebrovascular accidents (Table 1).

F. Myocardial Infarction

Four cases of special interest (42854, 49021, 43162, 49933) have Q waves or progression of T wave

changes suggestive of possible myocardial infarction, but only in the first 2 cases were recent myocardial infarctions demonstrated at autopsy. No evidence of infarction was found in the latter 2 cases. In addition, 2 other cases (45743 and 50141) are not suggestive of myocardial infarction on the ECG, but each case demonstrated a recent or old myocardial infarction at necropsy.

G. Miscellaneous

And finally, it is to be noted that 2 tracings (45743 & 48588) showed prominent U waves, both cases occurring in the moderate to severe heart disease group. Case 45743 had a normal serum potassium level while the remaining patient had a serum potassium of 3.9 mEq/L. Also, 9 of the 20 patients had a heart rate over 90 and the mean for all patients was 97.2. Rhythm disturbances, including one case of ventricular tachycardia, occurred in 10 of the 20 patients. There was no apparent relationship with heart disease; 3 of 6 in the normal group and 7 of 14 in the abnormal group had rhythm disturbances without relationship to electrolytes.

Table 2 - ECG findings in 20 patients with CVA's

Case No.	42517	42542
Age & Sex	76 F	34 M
Rate	75	118
Rhythm	Sinus	Sinus
PR Interval	0.14	0.14
QRS Interval	0.08	0.07
QT Interval	0.37	0.34 ↑
QRS Axis	+50 post.	+95 post.
T Axis	+60 N	0 ant.
P Wave	N	N
QRS Wave	N	Absence of normal R progression V1-4
ST Segment	N	N
T Wave	N	Slight ↓
Electrolytes	N	N
Coronary Art. Sclerosis	Prominent	None
ECG Diagnosis	Within normal limits	Sinus tachycardia. E.R.A.D. Nonspecific T wave changes
Subsequent ECG Changes		

Table 2 - ECG findings in 20 patients with CVA's

Case No.	42854	43070
Age & Sex	72 F	57 M
Rate	98	112
Rhythm	Sinus	Wandering pacemaker with frq. PAC's; runs of vent. tachycardia
PR Interval	0.21-0.22	Indeterminate
QRS Interval	0.08	o.14
QT Interval	Indeterminate	Indeterminate
QRS Axis	+50	+15 post.
T Axis	Indeterminate	Indeterminate
P Wave	N	Wandering pacemaker
QRS Wave	Q in II, III and a VF	Slurred
ST Segment	Sags - limbs and V4-6	↓
T Wave	↓	↓
Electrolytes	N	N
Coronary Art. Scerosis	Prob. recent subendocard. hemorrhage	Minimal
ECG Diagnosis	Sinus tachycardia. Nonspec. ST changes Dig. effect. ? old post. infarct. ↑ PR interval	Wandering pacemaker & frequent PAC's and PVC's. Complete LBBB
Subsequent ECG Changes	↑ PR to 0.24 Frequent PVC's.	Ventricular tachycardia

Table 2 - ECG findings in 20 patients with CVA's

Case No.	43162	45150
Age & Sex	39 M	37 M
Rate	130	106
Rhythm	Nodal rhythm with PVC's in bigeminy	Sinus
PR Interval	0.10 ↓	0.13
QRS Interval	0.09 ↑	0.07
QT Interval	0.26	0.35 ↑
QRS Axis	Initial + 75 Terminal -50	-60 post
T Axis	+65	+60 n
P Wave	Abnormal biphasic	N
QRS Wave	RBBB; Q in a VL	N
ST Segment	? elevation in a VL	N
T Wave	N	N
Electrolytes		Hypokalemia 3.5
Coronary Art. Sclerosis	Minimal	Minimal
ECG Diagnosis	Nodal pacemaker & PVC's; begeminy Incomplete RBBB Possible high anterolateral infarct with prei-infarct block.	Sinus tachycardia E.L.A.D.
Subsequent ECG Changes		

Table 2 - ECG findings in 20 patients with CVA's

Case No.	45181	45654
Age & Sex	59 F	59 M
Rate	54	60
Rhythm	Sinus bracardia	Sinus
PR Interval	0.14	0.14
QRS Interval	0.10	0.10
QT Interval	0.40	0.42
QRS Axis	0 post.	-10 N
T Axis	+40 N	+ 110 ant.
P Wave	N	N
QRS Wave	Tall R in aVL; electrical alternans	↑ voltage aVL
ST Segment	N	↓ I, aVL, and V5 - 6
T Wave	Slight ↓	Inverted I, aVL, V4 - 6
Electrolytes	Hypokalemia 3.3	Hypokalemia 3.9
Coronary Art. Sclerosis	Minimal	Marked
ECG Diagnosis	Sinus bracardia. Minimal voltage evidence of LVH. Pulses alternans	Abnormal tracing because of evidence of LVH.
Subsequent ECG Changes		

Table 2 - ECG findings in 20 patients with CVA's

Case No.	45743	46217
Age & Sex	87 M	56 M
Rate	64	95-100
Rhythm	Sinus	Atrial fibrillation Occ. PVC's
PR Interval	0.24	None
QRS Interval	0.08	0.14
QT Interval	0.38	0.34
QRS Axis	-5 N	mean -50 post
T Axis	Indeterminate	Indeterminate
P Wave	N	None
QRS Wave	Slurred (BBB)	Slurred
ST Segment	↓ I and aVL	↓ I, aVL, V5-6
T Wave	↓ throughout	----
Electrolytes	N	N
Coronary Art. Sclerosis	Moderate	None
ECG Diagnosis	1st degree A-V block Nonspecific ST-T changes consistent with hypokalemia and/or dig. effect. Prominent U Waves.	Atrial fibrillation with PVC's Complete LBBB
Subsequent ECG Changes		Increased vent. rate. Disappearance of PVC's

Table 2 - ECG findings in 20 patients with CVA's

Case No.	46523	46532
Age & Sex	67 F	54 F
Rate	140-160	65-70
Rhythm	Atrial fibrillation Occ. PVC's	Sinus arrhythmia
PR Interval	None	0.14
QRS Interval	0.8	0.07
QT Interval	0.28-0.32 ↑	0.41 ↑
QRS Axis	+10 N	+50
T Axis	Indeterminate	Indeterminate
P Wave	N	N
QRS Wave	N	N
ST Segment	↓ V4 - 5	↓ V4 - 5
T Wave	Depressed V4 - 6; inverted I, aVL	Depressed V4 - 5
Electrolytes	----	----
Coronary Art. Sclerosis	Marked	None
ECG Diagnosis	Atrial fibrillation with rapid vent. rate. Nonspecific ST-T changes consistent with LVH and probable digitalis effect. Volatge evidence of LVH	Nonspecific ST-T changes consistent with hypokalemia
Subsequent ECG Changes	↓ rate ↑ ST-T changes	

Table 2 - ECG findings in 20 patients with CVA's

Case No.	48118	48309
Age & Sex	76 M	60 M
Rate	82	64
Rhythm	Sinus	Sinus arrhythmia
PR Interval	0.16	0.14
QRS Interval	0.06	0.08
QT Interval	0.34	0.36
QRS Axis	0 post.	+60 ant.
T Axis	+50 post	+60 ant.
P Wave	N	Peaked in II, III and aVF
QRS Wave	N	SV_1 & RV_5 = 40
ST Segment	N	N
T Wave	N	↓ V5 - 6
Electrolytes	----	N
Coronary Art. Sclerosis	Moderate	Moderate
ECG Diagnosis	Within normal limits	Minimal nonspecific T waves - evidence of LVH. P waves suggest R atrial enlargement
Subsequent ECG Changes		

Table 2 - ECG findings in 20 patients with CVA's

Case No.	48588	49021
Age & Sex	60 M	70 M
Rate	62	75
Rhythm	Sinus arrhythmia	Sinus
PR Interval	0.14	0.13
QRS Interval	0.06	0.09
QT Interval	0.44 ↑	0.36
QRS Axis	-25 post	+30
T Axis	+50	+90 ant.
P Wave	Notched	N
QRS Wave	N	N
ST Segment	N	N
T Wave	N	AVL, I, V5 - 6
Electrolytes	Hypokalemia 3.9	N
Coronary Art. Sclerosis	Prominent	Marked
ECG Diagnosis	Not definitely outside normal limits; however prominent U waves are certainly striking	Nonspecific T wave changes
Subsequent ECG Changes		↑ rate; progressive T wave changes consistent with myocardial ischemia and/or subendocardial infarction

Table 2 - ECG findings in 20 patients with CVA's

Case No.	49188	49933
Age & Sex	77 F	63 M
Rate	160	86
Rhythm	Supraventricular or sinus tachy. & occ. PVC's	Sinus with occ. PVC's
PR Interval	0.12	0.18
QRS Interval	0.06	0.08
QT Interval	0.24	0.39 ↑
QRS Axis	+10 post.	+65
T Axis	Indeterminate ant.	+90 ant.
P Wave	Inverted I, aVL	? notching
QRS Wave	QS-VI & 2	0.04 Q in aVL
ST Segment	N	↓V5 & 6
T Wave	↓limb leads V4 & 5	↓ I, aVL, V4 - 6
Electrolytes	N	Hypokalemia 2.8
Coronary Art. Sclerosis	Minimal	Moderate
ECG Diagnosis	Sinus tachycardia. T wave changes consistent with rate	Nonspecific ST-T changes. Possible old anterolateral infarct. suggested by broad Q wave in a VL.
Subsequent ECG Changes		

Table 2 - ECG findings in 20 patients with CVA's

Case No.	50141	51127
Age & Sex	79 F	78 M
Rate	75	108
Rhythm	Sinus with occ PVC's	Sinus tachycardia
PR Interval	0.14	0.14
QRS Interval	0.09	0.06
QT Interval	Indeterminate	0.30
QRS Axis	0 post.	+30 post.
T Axis	N	0°
P Wave	N	N
QRS Wave	SV1 & RV5 = 38mm	N
ST Segment	↓ I, aVL, V4 - 6	N
T Wave	↓ limb leads, inverted, in V4 - 6	N
Electrolytes	Hypokalemia 3.8	----
Coronary Art. Sclerosis	Marked	Mild
ECG Diagnosis	Nonspecific ST-T changes consistent with dig. effect, ischemia or LVH. Minimal voltage evidence of LVH.	Within normal limits except for the rate.
Subsequent ECG Changes	15° left shift of axis. ↓ rate. ↑ ST-T changes	

Table 3 - Principal ECG findings in 6 patients with minimal or no heart disease (wt. less than 350 grms; minimal or no sclerosis; no evidence of infarction; but left ventricle up to 20 mm in thickness) with respect to serum potassium levels.

ECG Results	Normal Serum * Potassium	Hypoka-lemia (under 4.0)	Electro-lytes not determined
Prolonged QT Interval	1	1	1
Depressed ST Segment	None	None	2
Depressed or invert-ed T wave	2	1	1
Total Cases	2	2	2

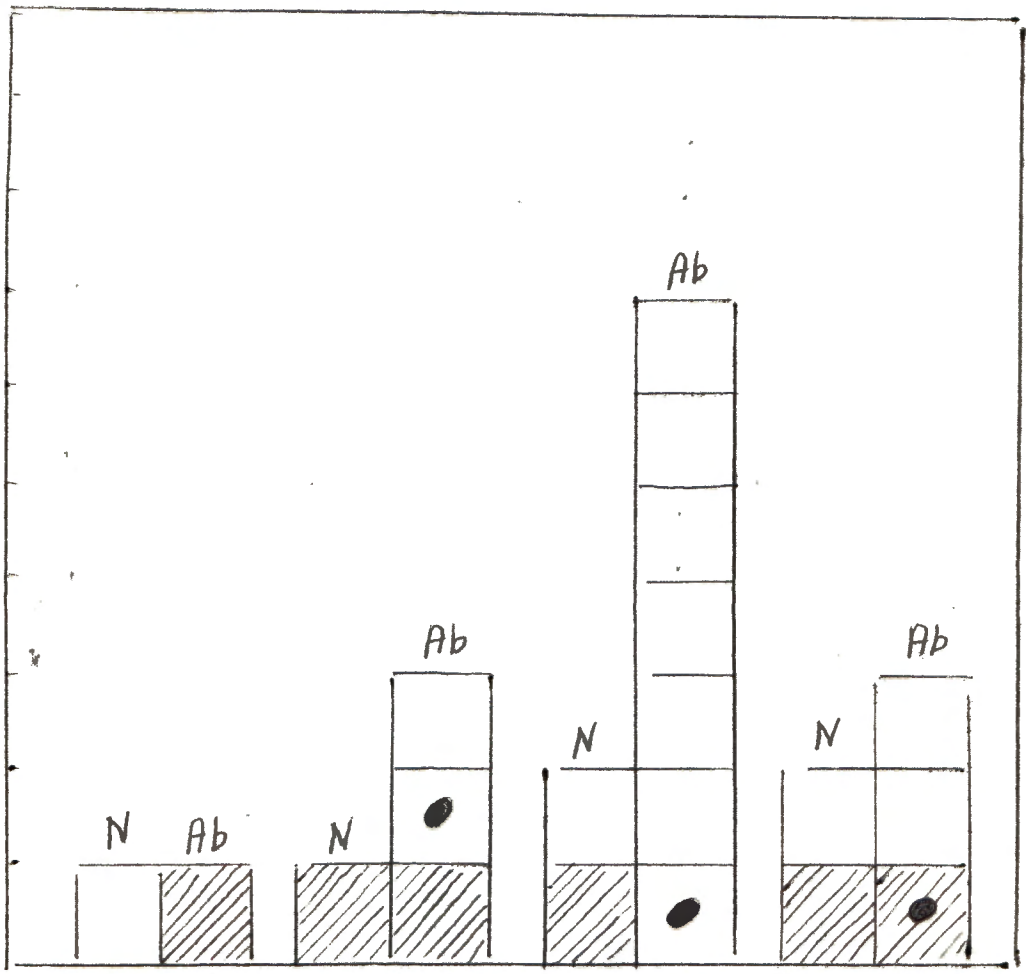
* Normal serum potassium level = 4.0 - 5.5 mEq/l

Table 4 - Principal ECG findings in 14 patients with moderate to severe heart disease (wt over 350 grms.; left ventricle over 20 mm in thickness; recent or old myocardial infarction present or a combination of the above) with respect to serum potassium levels.

ECG Results	Normal Serum * Potassium	Hypokalemia (under 4.0)	Electrolytes not determined
Prolonged QT Interval	None	2	1
Depressed ST Segment	4	3	1
Depressed or inverted T wave	5	3	1
Total Cases	7	4	3

* Normal serum potassium level = 4.0 - 5.5 mEq/l

Table 5 - Comparing QT intervals between a group with minimal or no heart disease and another group with moderate to severe heart disease with respect to serum potassium levels.



Prolonged QT interval

Receiving digitalis

N Group with minimal or no heart disease

Ab Group with moderate to severe heart disease

Table 6 - Comparing ST segment changes between a group with minimal or no heart disease and another group with moderate to severe heart disease with respect to serum potassium levels.

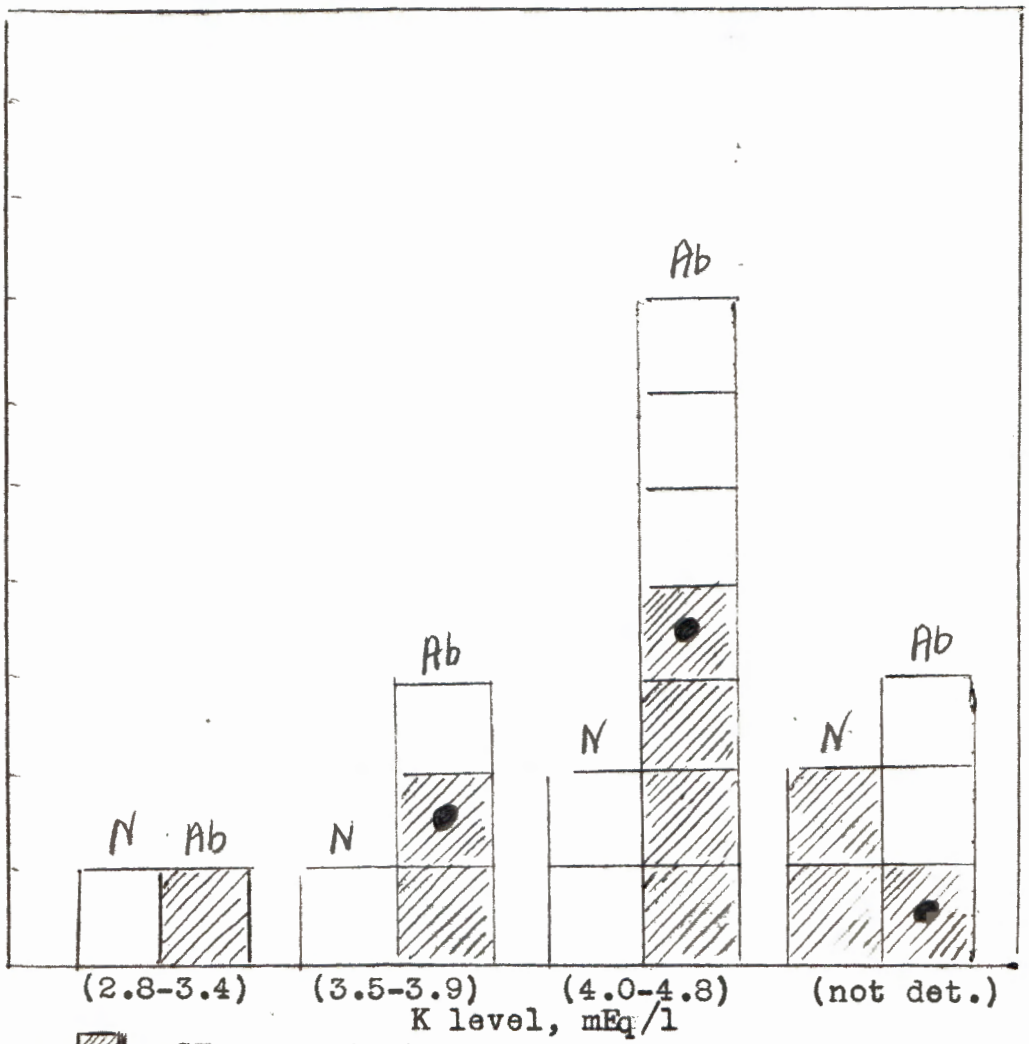
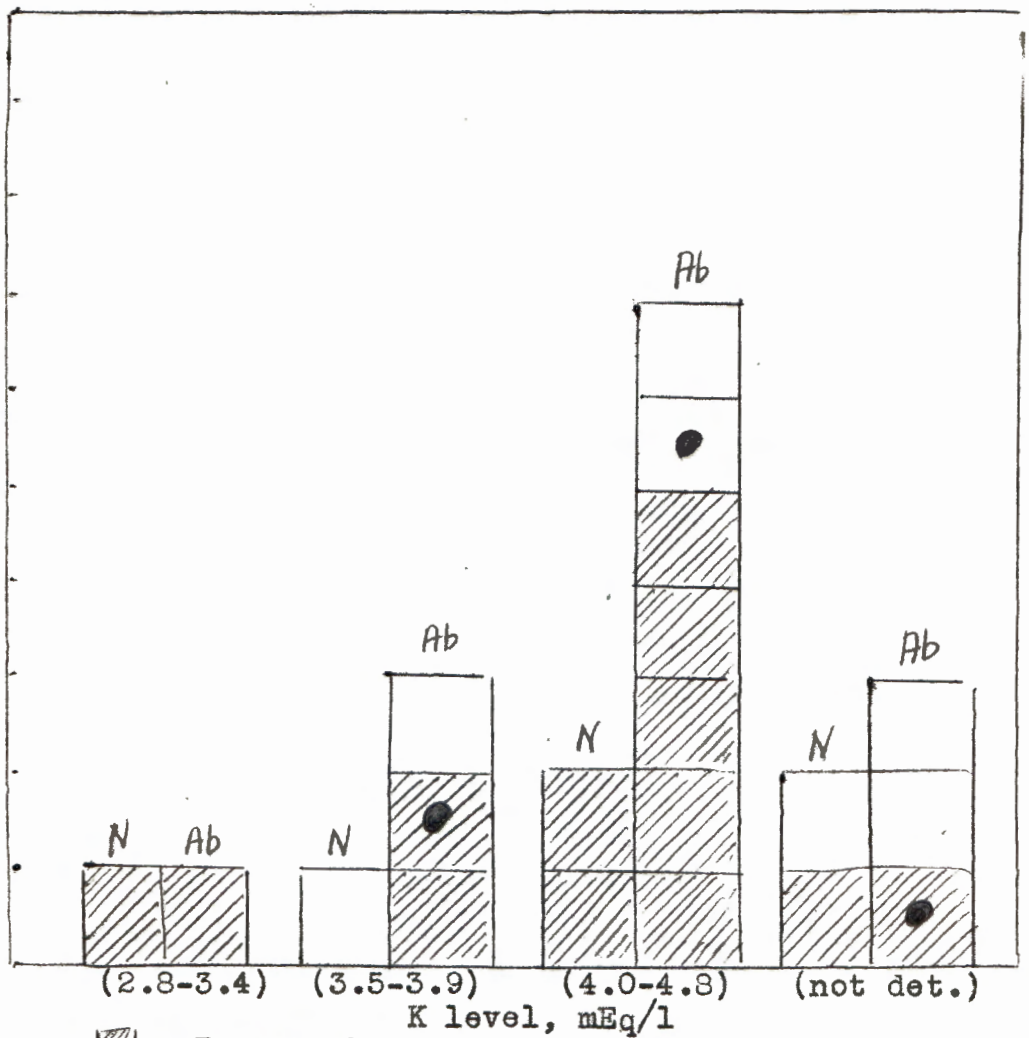


Table 7 - Comparing T wave changes between a group with minimal or no heart disease and another group with moderate to severe heart disease with respect to serum potassium levels.



XIV DISCUSSION

A. Comparison with Previous Studies

The principal ECG abnormalities encountered in this study are prolonged QT intervals (6 of 20 cases), depressed ST segments (9 of 20 cases), depressed or inverted T waves (14 of 20 cases),and rhythm disturbances (11 of 20 cases). On the surface these data appear to compare reasonably well with earlier reports (see Table 1). However, it has been emphasized in this study that ST abnormalities are not inexplicable on the basis of heart disease and normal serum electrolyte levels (Table 6). Inspection of Table 1 shows that of a considerable number of reported cases only a small percent have been examined at autopsy. Moreover, a significant percentage of patients not examined at autopsy were said to have heart disease. This suggests that perhaps many of the ST-T wave abnormalities reported are explicable on the basis of heart disease. In addition it is well known that T wave changes are not specific. Levine[2] has published an "incomplete list" of 47 different factors which influence the T wave.

The most persistent ECG parameter associated with cerebrovascular accidents appears to be pro-

longation of the QT interval. Seven of the 13 studies listed in Table 1 report this finding and this study seems to support this phenomenon. Shuster, however, reported shortened QT intervals in his study of 19 cases, and it was his contention that other workers were measuring the QU interval instead of the QT interval. ST segment changes were observed in 9 patients in this study and most of those abnormalities were related to the group with moderate to severe heart disease. Only 5 of the previous studies (Table 1) report this finding and 2 of those were listed as depressed while the remaining 3 were elevated and consistent with myocardial infarction.

Several investigators have reported the frequent occurrence of rhythm disturbances. In this study, these changes appeared uniformly in both groups and without apparent relationship to electrolyte changes. U waves also appeared casually in this study, but their small incidence does not permit a conclusion.

B. Etiology of ECG Changes

Several theories have been proposed to explain the cause of ECG abnormalities associated with cerebrovascular accidents. Burch[3] was the first to suggest abnormal electrolyte metabolism as a possible

cause. Hugenholz[9] suggested that intracellular electrolyte changes might be sufficient to alter the transmembrane potential but serum electrolyte determinations on their patients were in the normal range. Surawicz[18], in a well documented study showed that changing concentrations of potassium and calcium in extracellular fluid will be reflected in the ECG by changes in the ST interval and T wave.

One group[14] believes that area 13 may be responsible because cortical representation of the Vagus nerve is situated there. It was demonstrated that stimulation of the area leads to changes in heart rate and blood pressure. Another group[6] showed that manipulation of area 13 during an operation for subarachnoid hemorrhage caused multifocal ventricular ectopic beats to occur. Poole[15] demonstrated arrhythmias, ST segment and T wave changes during manipulation of the circle of Willis. He postulates that hemorrhage around the wall of an aneurysm or artery causes ECG abnormalities that are mediated through "vasovagal and vaso-cardiac reflexes." Burch[3] suggests "sympathetic storms" arising from cerebral injury. Menon[13] feels this view

is supported by finding "QRS changes consistent with islands of infarction or invasive block."

Shuster[7] showed that giving 1/150 grain of atropine to patients with subarachnoid hemorrhage restored depressed ST segments and T waves toward normal. Harrison and Gibb[11] feel this might explain the more common association of ECG abnormalities with subarachnoid hemorrhage. One investigator[10] claims that ECG changes in intracranial hemorrhage are due to cardiac lesions and "that ECG's do not simulate myocardial injury but are in fact signs of them."

A variety of other explanations are offered. Pollock[16] says "it is by no means uncommon to find abnormal ECG's showing decreased voltage, depressed T waves, sometimes inversion of T waves, and occasionally depression of the ST segment during the course of acute pancreatitis." Ippolia[22] found massive T wave inversion associated with syncopal attacks. Barger and his associates[21] were able to demonstrate ST segment depression and deeply inverted T waves by infusing epinephrine into coronary arteries of dogs. They concluded that sympathetic reflexes could produce persistent alterations in the

ECG without myocardial necrosis. In another interesting experiment[20], increased intracranial pressure was obtained by injecting an inert fluid into the CSF which produced prolonged ST segments and T wave inversion. Wiener and associates[17] report 3 entirely benign cases of the above phenomenon, all of which occurred in Negros. There was no evidence of myocardial or intracranial disturbance and they believe this phenomenon occurs in 1 percent of the adult population. And finally, Bellet and Finkelstein[19] present a list to provide explanation for prolonged QT intervals on the basis of abnormal serum electrolytes, heart disease, hypokalemia, and quinidine therapy.

V. SUMMARY

The literature concerning ECG changes associated with cerebrovascular accidents is briefly reviewed. The most commonly reported ECG changes are prolongation of the QT interval, ST segment elevation and depression, T wave abnormalities, and U waves. Less commonly reported findings are arrhythmias, shortened QT intervals, and bradycardia.

The electrocardiograms of 20 patients who expired from cerebrovascular accidents were studied.

(Table 2) All were examined at autopsy and the results are organized into 2 groups -- those with minimal or no heart disease and those with moderate to severe heart disease with respect to serum potassium levels. (Tables 3 - 7)

Results of this study suggest that a significant number of cases demonstrate QT interval prolongation that are caused by factors other than heart disease or abnormal serum electrolyte levels. Most of the ST segment depressions in this study are associated with moderate to severe heart disease. T wave changes appeared commonly in both groups, and although it is difficult to attribute these changes to cerebrovascular accidents, this possibility can not be excluded. Rhythm disturbances were frequently seen, but these changes appeared uniformly in patients irrespective of heart disease and serum electrolyte changes. U waves were infrequently present. Four cases had ECG tracings suggestive of myocardial infarction but only in 2 of these cases was an infarct actually demonstrated. In addition, 2 other patients had tracings which did not suggest infarction but in both cases an infarct was demonstrated at necropsy.

Many causes for these ECG changes have been suggested and they are briefly discussed. These include abnormal electrolyte metabolism, intracellular electrolyte changes altering the transmembrane potential, disturbances of cortical area 13, irritation of the circle of Willis, sympathetic storms, acute pancreatitis, and increased intracranial pressure.

VI. CONCLUSIONS

1. QT interval prolongation appeared in 6 of 20 cases, and this study suggests that these changes may be caused by factors other than heart disease or abnormal serum electrolytes.
2. ST segment depression occurred in 9 of 20 cases; however, these findings were generally associated with heart disease or abnormal serum potassium levels, and hence cerebrovascular accidents could not be implicated as a cause.
3. T wave changes were observed in 13 of 20 patients. Because these findings were observed both in groups with and without heart disease and in all ranges of serum potassium levels, the possibility that these changes are attributed to cerebrovascular accidents can not be

excluded.

4. Rhythm disturbances were noted in 11 of 20 patients, and these changes appeared in patients irrespective of heart disease and serum electrolyte changes. Therefore, cerebrovascular accidents could not be implicated as a cause.

5. Four cases had Q waves and progression of T wave changes suggestive of myocardial infarction, but only im 2 of these cases was an infarct actually demonstrated at autopsy. In addition, 2 other cases had ECG tracings not suggestive of myocardial infarction but in which an infarct was demonstrated at necropsy. These are interesting findings but can not be attributed to cerebrovascular accidents.

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