Electrocardiographic changes and their prognostic significance in subarachnoid haemorrhage

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SYNOPSIS In a study on the prognostic significance of catecholamine-linked ECG changes in 40 cases of subarachnoid haemorrhage, there were 16 men of mean age 41 years (range 13-56) and 24 women of mean age 42 years (range 11-65). There were six deaths, and five of these patients had consistently abnormal ECGs. Analysis of the abnormal components of ECGs revealed that the presence of either a pathological Q wave or a raised S-T segment indicated a poor prognosis. The two patients with pathological Q waves both died, and the three patients with raised S-T segments all developed cerebral arterial spasm, one of whom died. A high incidence of peaked P waves, short P-R intervals, a long Q-Tc, and tall U waves occurred in the ECGs of six patients who died, the 15 patients with cerebral arterial spasm, and the seven patients who were later to develop cerebral arterial spasm. Three of the latter eventually died, in contrast with the single death in the 22 patients who did not develop cerebral arterial spasm and had a low incidence of P wave peaking, short P-R intervals, a long Q-Tc, and tall U waves. Various combinations of these four ECG changes, in addition to peaking of the T wave, indicated a bad prognosis irrespective of the type of treatment. The criteria for prognosis on the role of catecholamines in causing the ECG abnormalities are discussed.

The mechanism by which changes in the electrocardiogram (ECG) associated with subarachnoid haemorrhage are brought about has been a matter for speculation. Possible explanations have included hypokalaemia (Hersch, 1964), stimulation of area 13 under the frontal lobes of the brain (Cropp and Manning, 1960), hypertension (Kreus *et al.*, 1969), ischaemic heart disease (Sarner and Crawford, 1965), increased parasympathetic drive (Shuster, 1960), and increased sympathetic drive to the heart (Tomomatsu *et al.*, 1964). Strong support for the last explanation was provided by Russian authors (Iliynski and Astrakhantseva, 1967) and by Cruickshank and his colleagues (1974). In the latter study a strong correlation was shown between urinary catecholamine levels and certain ECG changes—namely, peaking of the P wave, a short P-R interval, and peaking of the T wave; a weaker relationship was also demonstrated between urinary catecholamine levels and T wave inversion, a long Q-Tc, tall U waves, and patho-

logical Q-waves. Hypokalaemia as a possible cause of the ECG changes was ruled out.

If catecholamines are a common factor between ECG changes and cerebral arterial spasm in subarachnoid haemorrhage, then the above catecholamine-linked ECG changes might serve as prognostic indicators. The present study was designed to investigate this possibility.

METHODS

Forty cases of subarachnoid haemorrhage, proved by lumbar puncture, comprised 16 men of mean age 41 years (range 13-56) and 24 women of mean age 42 years (range 11-65). There were no cases with a history of hypertension or ischaemic heart disease. Thirty-seven cases had carotid angiography, and most had additional vertebral angiograms. Angiograms were reported on without knowledge of results of other investigations. Cases were classed into three angiographic groups: (1) cases with no cerebral angiographic spasm, termed the non-spasm group, (2) cases with cerebral angiographic spasm,

TABLE 1

INCIDENCE OF CEREBRAL ARTERIAL SPASM AND DEATH IN THREE ECG GROUPS OF 37 CASES WHO HAD CEREBRAL ANGIOGRAPHY

ECG group	Patients (no.)	Patients who developed spasm (no.)	Deaths	
			2nd haemorrhage	Spasm
All normal	7	1	1	—
Changing	12	4	—	—
All abnormal	18	10	1	3
Total	37	15	2	3

termed the spasm group, and (3) a third group who showed no spasm in the first angiogram but did so in a subsequent angiogram, termed the pre-spasm group.

A 12-lead ECG was performed on days 1, 2, and 3 after admission and thereafter every two to three days over a two week period. The criteria for ECG abnormality have been set out elsewhere (Cruickshank *et al.*, 1974). ECGs were reported on by one person (J.M.C.) without knowledge of the clinical state or the angiographic findings. For analytical purposes the 40 cases were divided into three groups: cases with all normal ECGs, with all abnormal ECGs, and cases with changing ECGs where some were normal and others abnormal. In the eight cases with normal ECGs six were men of mean age 36 years (range 13–54) and two women of mean age 43 years (range 42–44). The 19 cases in the abnormal ECG group comprised five men of mean age 48 years (range 43–56) and 14 women of mean age 46 years (range 16–65). The last group of 13 with changing ECGs included five men of mean age 40 years (range 16–54) and eight women of mean age 35 years (range 11–59).

RESULTS

Table 1 shows the high incidence of cerebral arterial spasm and death in the abnormal ECG group as compared with the normal and changing ECG groups. Of the six deaths in the study only five are mentioned in this Table, as cerebral angiography was not performed on the sixth. At the time of discharge from hospital, all patients in the normal ECG group were without neurological deficit, whereas in the changing and abnormal ECG groups, five of the 12 and six of the 18 respectively had varying degrees of neurological deficit.

TABLE 2

INCIDENCE OF ECG ABNORMALITIES IN THREE ANGIOGRAPHIC (37 CASES) AND DEATH GROUPS (FROM WHOLE GROUP)

Abnormal ECG component	Group			
	Non-spasm (22) (no.) (%)	Pre-spasm (7) (no.) (%)	Spasm (15) (no.) (%)	Deaths (6) (no.) (%)
Tachycardia (9) n=9	4 18	0	5 33	3 50
Peaked P waves (12)	4 18	4 71	8 53	5 83
P-R interval 0.14 s or less (16)	9 41	6 86	7 47	5 83
Raised S-T (3)	0	2 29	3 20	1 17
Depressed S-T (8)	3 14	2 29	5 33	2 33
Peaked T (6)	2 9	2 29	4 27	3 50
Inverted T (14)	7 32	3 43	7 47	4 66
Q-Tc 0.43 s or more (9)	4 18	4 57	5 33	5 83
Tall U waves (10)	4 18	4 57	6 40	4 66
Pathological Q waves (2)	0	1 14	1 7	2 33

Number of cases in parentheses.

TABLE 3

ECG ABNORMALITIES IN 40 CASES OF SUBARACHNOID HAEMORRHAGE AND INCIDENCE OF ASSOCIATED DEATH AND CEREBRAL ARTERIAL SPASM

ECG abnormality	Deaths (%)	Cerebral arterial spasm (%)
Tachycardia (10)	30	50
Peaked P (12)	38	61
Short P-R (16)	31	44
Raised S-T (3)	33	100
Depressed S-T (8)	25	63
Peaked T (7)	43	57
Inverted T (15)	27	47
Long Q-Tc (10)	50	50
Tall U (10)	40	60
Pathological Q wave (2)	100	50

Number of cases in parentheses.

Table 2 illustrates the incidence of various ECG abnormalities present on at least two occasions, in the three angiographic groups and in the six cases who died. The pre-spasm column consists of data obtained before the second angiogram: data after the second angiogram which showed spasm are included in the spasm column. The remarkable features are the con-

centration of most ECG changes in the death group, and the high percentage of cases with peaked P waves, short P-R intervals, peaked T waves, long Q-Tc, and tall U waves in the pre-spasm as compared with the non-spasm groups.

Table 3 shows the incidence of death and cerebral arterial spasm associated with individual types of ECG abnormality. With only one type of abnormality, a pathological Q wave (two cases), was death inevitable. The presence of a raised S-T segment (three cases) indicated a complicated course where all three developed cerebral arterial spasm and one died. No other type of abnormality possessed these degrees of specificity. However, certain combinations of ECG abnormalities (though not necessarily all on the same ECG) appeared to carry a particularly poor prognosis. Firstly, taking cases who were treated conservatively (28 cases), the two patients who showed the combination of peaked P waves, peaked T waves, large U waves, and a long Q-Tc both died after prolonged spasm. Of the three cases with combinations of three of these four abnormalities, one died after prolonged spasm, one developed spasm but survived, and the other had no spasm and survived. Of the other two deaths who were treated conservatively, one had a short P-R interval, long Q-Tc, and inverted T waves, and the other had a normal ECG, but died after a second haemorrhage (no ECGs were taken after the second subarachnoid haemorrhage). Secondly, of cases treated surgically (12 cases), six patients had at least two of the following ECG changes preoperatively—peaked P waves, short P-R interval, peaked T waves, long Q-Tc, and large U waves: one of these six patients developed spasm and died, two developed dense hemiplegias, two had prolonged impairment of consciousness, and one patient showed no change from the preoperative state. In contrast, of the remaining six cases, four showed no change from the preoperative state, one had transient impairment of consciousness, and one developed a dense hemiplegia.

The combination of an alert clinical state and the absence of angiographic spasm is normally taken to augur a good prognosis. In the present study there were six such cases who were clinically alert with no angiographic spasm, and who also had normal ECGs (none of these patients had operations). All made good recoveries except

one patient who had a second haemorrhage and died. On the other hand, there were 12 patients who were clinically alert and had no angiographic spasm, but had abnormal ECGs (eight of the 12 were operated on). Of these 12 patients, two died after developing prolonged spasm (one postoperatively), and three developed severe neurological deficit (two postoperatively). Examination of the ECGs of these five complicated cases revealed that one had a combination of peaking of P wave, short P-R interval, long Q-Tc and large U wave, three had combinations of these abnormalities, and one had only peaking of the P wave. The remaining seven cases who did not display these combinations of ECG abnormality had unremarkable recoveries.

DISCUSSION

A relationship between abnormal ECGs and a deteriorating clinical state in subarachnoid haemorrhage has been shown by other workers (Hersch, 1961, 1964; Iliynsky and Astrakhanseva, 1967; Galloon *et al.*, 1972). The findings of the present study are in broad agreement with this, but are at variance with Galloon *et al.* (1972), who state that cases with a changing ECG pattern are at highest risk. The explanation for this disagreement is probably to be found in the low number of cases with all abnormal ECGs in the series of Galloon and his colleagues, four out of 37, as compared with 19 out of 40 in the present investigation. It is possible that this difference is accounted for by a more rigorous definition of an abnormal ECG in the present study. On the other hand, Shuster (1960) found that ECG changes were not related to brain damage as assessed clinically, and Ananthachari and Anto (1967) concluded that the occurrence or persistence of ECG changes did not affect the course of the illness adversely. However, these conclusions were based on relatively small numbers of ECGs.

No study has demonstrated convincingly that certain types of ECG abnormality are linked with a poor prognosis in subarachnoid haemorrhage, but Hersch (1964) noted that, in seven cases on whom serial ECGs were carried out, there was an increased incidence of depressed S-T segments in those who died. Only one study

(Wilkins *et al.*, 1968) has attempted to correlate ECG changes with the onset of cerebral arterial spasm, and concluded that no correlation existed. However, in that retrospective study on a large number of cases, ECGs were recorded in only 35, and more than one ECG was performed in only 12 of the 35. In contrast, the present prospective study revealed a low incidence of ECG changes in the group who did not develop cerebral arterial spasm (non-spasm group) compared with those cases who were later to develop cerebral arterial spasm (pre-spasm group), and those who already had spasm (spasm group). Individual ECG abnormalities which were associated with the worst prognosis were pathological Q waves and raised S-T segments: the two patients with pathological Q waves both died and the three patients with raised S-T segments all developed cerebral arterial spasm with one fatality. Certain combinations of ECG abnormalities also indicated a poor prognosis, irrespective of the type of treatment. The three patients who had four of the following five abnormalities, peaked P wave, short P-R interval, peaked T wave, large U wave, and a long Q-Tc, all died. Combinations of two or three of these abnormalities were also associated with a high incidence of complications such as prolonged impairment of consciousness and the onset of dense hemiplegias. However, false positives did occur: one case had a combination of three of these five ECG abnormalities but did not develop cerebral arterial spasm and was well throughout. Likewise, false negatives occurred: one patient had normal ECGs throughout, but developed cerebral arterial spasm, and another who had all normal ECGs died after a second subarachnoid haemorrhage (but no ECG was performed after the second haemorrhage).

These results suggest that the five ECG abnormalities listed above indicate a poor prognosis regardless of the state of consciousness or angiographically demonstrated arterial spasm.

All the above ECG changes have been described in association with administration of catecholamines (Lepeschkin *et al.*, 1960; Damato *et al.*, 1969; Sparks *et al.*, 1970; Haft *et al.*, 1972), where both transient and more permanent changes, associated with endocardial necrotic

lesions, may occur. Also, a correlation between endogenous catecholamine excretion in subarachnoid haemorrhage and peaked P waves, a short P-R interval, S-T and T wave changes, a long Q-Tc, a tall U wave and pathological Q waves, has been demonstrated (Cruickshank *et al.*, 1974). The poor prognosis associated with combinations of these ECG changes is in keeping with the suggestions that catecholamines are closely linked with the development and maintenance of cerebral arterial spasm.

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