ECG abnormalities in patients with subarachnoid haemorrhage and intracranial tumours

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SUMMARY The incidence of and possible factors influencing ECG abnormalities were analysed in one patient group with subarachnoid haemorrhages ($n = 406$) and another with intracranial tumours ($n = 400$). The highest incidence of each ECG abnormality was always found in the patients with subarachnoid haemorrhages. In this group an ECG pattern, possibly attributable to the cerebral disease and comprising abnormalities of the T and U waves and prolongation of the Q-T_c interval, was frequently identified.

The existence of a relationship between intracranial disorders, particularly subarachnoid haemorrhage (SAH), and electrocardiographic (ECG) abnormalities is well established.¹⁻³ The incidence and type of ECG changes in previous reports vary widely⁴⁻¹⁴ probably owing to inadequate numbers of patients in the respective studies and to differences between the intracranial disorders, which are furthermore often not well defined. The large clinical series reported mostly consist of acute cerebrovascular accidents,¹⁵⁻¹⁷ often with SAH included as an insignificant subgroup. However, in the preoperative evaluation of patients with SAH from a ruptured aneurysm or other vascular malformation, an abnormal ECG may present diagnostic difficulties^{18,19} and thereby delay the impending surgical intervention and cause a significant risk of rebleeding. Accordingly, the present study was designed to elucidate in more detail the incidence and pattern of ECG abnormalities in a large group of patients with SAH and to analyse factors which may influence these abnormalities. The findings were compared with those occurring in a group of patients operated on for intracranial tumours and analysed in an identical manner. A further objective was to explore the possibility of a relationship between ECG abnormalities and a particular intracranial lesion.

Material and methods

The clinical series consisted of 806 consecutive patients oper-

ated on in the Neurosurgical Department of the Karolinska Hospital between 1975 and 1983. There were two groups, one with SAH ($n = 406$) and another with intracranial tumours (TUM) ($n = 400$). SAH was diagnosed by lumbar puncture and/or computed tomography and the source of bleeding was verified by angiography. Intracranial tumours were diagnosed by computed tomography (table 1).

A data-collection form was designed for recording the patient's diagnosis, history of previous diseases, drug therapy, laboratory investigations and ECG findings. All data were stored in a data bank for computerized processing. Clinical data from the two patient groups are presented in table 2. The mean age for the SAH group (49 ± 13 years) was significantly lower than for the TUM group (55 ± 12 years). The number of patients in the age category of 20-39 years was significantly larger in the SAH group, while the TUM group predominated in the age category of ≥ 60 years. The sex distribution was similar in the two groups. The incidence of cardiovascular disease was the same in the groups except for hypertension, which was more common in the SAH group. There was no significant difference with regard to drugs with cardiovascular effects. Identical numbers of patients ($n = 95$) in both groups had manifest or suspected papilloedema.

A standard 12-lead ECG was recorded at rest in all patients preoperatively. All ECGs were interpreted by one of us (SK) without identification of the diagnosis. The ECG was classified and coded according to the Minnesota Code.^{20,21} This code system was slightly modified by adding a few complementary codes to make possible a more detailed description of the T and U wave abnormalities (fig 1). The Q-T interval was measured routinely and corrected for heart rate (Q-T_c).²² A prolongation of the Q-T interval was defined as Q-T_c > 0.44 s.

In an attempt to eliminate, as far as possible, influences on the ECG other than the intracranial disorder, a subgroup within each of the two complete patient groups was generated (group SAHo ($n = 165$, 41%) and group TUMo ($n = 198$, 50%)) by excluding all patients with histories of hypertension, ischaemic, organic or congestive heart disease, abnormal serum values for potassium or total calcium, and

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Table 1 Location of intracranial lesion in the two studied patient groups with SAH (SAH group) and intracranial tumours (TUM group)

SAH group			TUM group		
Location of bleeding vascular pathology	No (n:406)	%	Location of intracranial tumours	No (n:400)	%
Aneurysms:			Tumours:		
internal carotid artery	109	27	frontal lobe	88	22
middle cerebral artery	102	25	temporal lobe	69	17
anterior cerebral artery	145	36	parietal occipital lobe	52	13
anterior communicating artery					
pericallosal artery					
vertebral artery, basilar artery	31	7.6	hypophysis	24	6
posterior inferior cerebellar artery			posterior fossa	62	16
Arteriovenous malformation of posterior fossa					
Other	19	4.7	Supratentorial meningioma	100	25
			Other	5	1.3

Table 2 Clinical data for the SAH group and the TUM group

Variable	SAH group		TUM group		Statistical differences between groups
Age: mean $\pm$ SD years (range)	49 $\pm$ 13 (14-74)		55 $\pm$ 12 (12-79)		p < 0.001
	No	%	No	%	
Patients	406	100	400	100	
Male	183	45	207	52	
Female	223	55	193	48	
Age distribution: (years)					
0-19	3	0.7	4	1	
20-39	102	25	47	12	p < 0.001
40-59	218	54	181	45	
$\geq$ 60	83	20	168	42	p < 0.001
History of previous diseases:					
Hypertension	78	19	48	12	p < 0.01
Angina pectoris	11	2.7	12	3	
Myocardial infarction	5	1.2	10	2.5	
Congestive heart disease	3	0.7	4	1	
Organic heart disease	2	0.5	0	0	
Other neurological disease	24	5.9	26	6.5	
Drugs:					
Digitalis	9	2.2	15	3.8	
Diuretics	31	7.6	41	10	
β -blockers	27	6.7	13	3.3	
Other antihypertensives	20	4.9	24	6	
Manifest or suspected papilloedema	95	23	95	24

those treated with digitalis, diuretics, beta-blockers or other antihypertensive drugs. The excluded patients with the above mentioned disorders and drugs are referred to as group SAHdis (n = 241, 59%) and group TUMdis (n = 202, 50%). The mean age of the patients in group TUMo (52 $\pm$ 13 years) was still higher than that of those in group SAHo (46 $\pm$ 13 years), and the sex distribution within these groups remained unchanged.

The incidences of identical variables in the different patient groups were analysed statistically using two-times-two tables and the chi-square test with Yates' correction.

Fig Schematic representation of T and U wave abnormalities classified essentially according to the Minnesota Code system. a: T amplitude negative $\geq$ 5 mm, b: T amplitude negative or diphasic with negative phase $\geq$ 1 mm but < 5 mm, c: T amplitude zero, negative or diphasic with negative phase < 1 mm, d: notched T wave, e: U amplitude > 1 mm.

Table 3 ECG data, essentially grouped according to the Minnesota Code for the SAH group, the TUM group, group SAHo and group TUMo

ECG items	SAH group		TUM group		Significant differences SAH-TUM	Group SAHo		Group TUMo		Significant differences SAHo-TUMo
	No	%	No	%		No	%	No	%	
Q and Qs patterns	13	3.2	18	4.5		5	3.0	5	2.6	
Left QRS axis deviation	9	2.2	17	4.3		2	1.2	7	3.5	
High-amplitude R waves	77	19	33	8.3	p < 0.001	25	15	13	6.6	
S-T segment depression	62	15	51	13		15*	9.1	18	9.1	
T wave abnormality	129	32	82	21	p < 0.001	45	27	22†	11	p < 0.001
Specific T wave abnormalities:										
T amplitude negative ≥ 5 mm	10	2.5	1	0.3		2	1.2	0	0	
T amplitude negative or diphasic with negative phase ≥ 1 mm but < 5 mm	39	9.6	24	6.0		14	8.5	4*	2.0	
T amplitude zero, negative or diphasic with negative phase < 1 mm	23	5.7	27	6.8		8	4.9	8	4.0	
T amplitude positive and T/R amplitude < 1/20	26	6.4	16	4.0		8	4.9	4	2.0	
Notched T amplitude	32	7.9	14	3.5		13	7.9	6	3.0	
U amplitude > 1 mm	190	47	40	10	p < 0.001	74	45	23	12	p < 0.001
P-Q interval ≥ 0.22 s	3	0.8	5	1.3		1	0.6	0	0	
P-Q interval < 0.12 s	5	1.2	13	3.3		3	1.8	8	4.0	
Frequent premature atrial, nodal or ventricular beats	12	3.0	5	1.3		1	0.6	4	2.0	
A-V nodal rhythm	7	1.7	0	0		1	0.6	0	0	
Sinus tachycardia (heart rate > 100 beats/min)	9	2.2	10	2.5		6	3.6	5	2.5	
Sinus bradycardia (heart rate < 50 beats/min)	20	4.9	7	1.8		11	6.7	4	2.0	
Infrequent premature atrial, nodal or ventricular beats.	53	13	24	6.0	p < 0.001	20	12	10	5.0	
Sinus arrhythmia										
Normal ECG	75	18	161	40	p < 0.001	36	22	88	44	p < 0.001
Q-T _c > 0.44 s	94	24	28	7.1	p < 0.001	27*	17	9	4.6	p < 0.001
Q-T _c interval (s) (range)	0.425 ± 0.0345 (0.35-0.56)		0.410 ± 0.0266 (0.34-0.55)		p < 0.001	0.417 ± 0.0324 (0.35-0.53)		0.405 ± 0.0237 (0.35-0.49)		p < 0.001
Heart rate (beats/min)	67 ± 14		75 ± 15		p < 0.001	67 ± 14		73 ± 14		p < 0.001

The statistical symbols (* = $p < 0.01$ and † = $p < 0.001$) in the columns for group SAHo and group TUMo are for comparisons of the incidence of the ECG item in group SAHo and the remaining patients in the SAH group (group SAHdis) on the one hand and in group TUMo and the remaining patients in the TUM group (group TUMdis) on the other.

Student's *t* test for unpaired data was performed to determine differences between mean values for age, heart rate and Q-T_c interval in the patient groups. $P < 0.01$ was considered significant. Values are given as mean ± SD.

Results

Table 3 shows ECG findings according to the modified Minnesota Code system for the SAH group, the TUM group, group SAHo and group TUMo. The figure illustrates coded abnormalities of the T and U waves.

The SAH and TUM groups

Seventy five (18%) patients in the SAH group and 161 (40%) in the TUM group had normal ECGs ($p < 0.001$). When all kinds of T-wave abnormalities were considered jointly, they were found to occur more frequently in the SAH group ($n = 129$; 32%) than in the TUM group ($n = 82$; 21%) ($p < 0.001$). The most pronounced difference was observed for the occurrence of a U wave of more than 1 mm. This abnormality was found in 190 (47%) patients in the SAH group

but in only 40 (10%) patients in the TUM group ($p < 0.001$). Other ECG abnormalities that appeared more frequently in the SAH group were high-amplitude R waves and a group of arrhythmias including infrequent premature atrial, nodal or ventricular beats and sinus arrhythmia. The Q-T_c interval was 0.43 ± 0.035 s for the SAH group and 0.41 ± 0.027 s for the TUM group ($p < 0.001$). More patients in the SAH group ($n = 94$; 24%) than in the TUM group ($n = 28$; 7.1%) had an abnormally prolonged Q-T_c interval ($p < 0.001$). The mean heart rate was lower in the SAH group than in the TUM group ($p < 0.001$). A higher incidence of patients with hypotassaemia and hypocalcaemia was observed in the SAH group (table 4).

Groups SAHo and TUMo

Virtually the same differences in ECG abnormality patterns were found between these groups as between the SAH group and the TUM group. There were no differences between the groups with regard to high-amplitude R waves, and the group of arrhythmias including infrequent premature atrial, nodal or

Table 4 Incidence of abnormal potassium and total calcium serum values and means $\pm$ SD of serum values for sodium, potassium and total calcium in the SAH group and the TUM group

Variable	SAH group (n:406)		TUM group (n:400)		Statistical differences between groups
	No	%	No	%	
K > 5.2 mmol/l	0	0	5	1.3	
K < 3.4 mmol/l	41	10	4	1	p < 0.001
Ca > 2.6 mmol/l	6	1.5	12	3	
Ca < 2.2 mmol/l	74	18	48	12	p < 0.01
	Mean $\pm$ SD		Mean $\pm$ SD		
Na mmol/l	139 $\pm$ 4		140 $\pm$ 4		
K mmol/l	3.8 $\pm$ 0.4		4.1 $\pm$ 0.4		
Ca mmol/l	2.29 $\pm$ 0.15		2.34 $\pm$ 0.15		

ventricular beats and sinus arrhythmia. With regard to T-wave abnormalities as a whole, there was the same difference as between the main groups. The difference in the Q-T_c interval also persisted (group SAHo = 0.417 ± 0.032 ; group TUMo = 0.405 ± 0.024 , $p < 0.001$).

In order to analyse the importance of the influence of the non-cerebral disorders on the ECG, group SAHo was compared with group SAHdis (table 3). On comparing these groups a lower incidence of S-T segment depression ($p < 0.01$) and Q-T interval prolongation ($p < 0.01$) was seen in group SAHo. In group TUMo the incidence was lower than in group TUMdis for all types of T-wave abnormalities ($p < 0.001$) and of the specific abnormality of a negative or diphasic T wave with negative phase of 1 mm or more but less than 5 mm ($p < 0.01$).

ECG: location of intracranial lesion

In table 1 the SAH group and the TUM group are broken down according to the location of the ruptured vascular malformation and of the tumour. The incidence of ECG findings grouped as all types of T-wave abnormalities, abnormal U wave, Q-T_c interval prolongation and normal ECG in groups with the same location of pathological change was compared with the corresponding figures for the remaining patients in the SAH group and the TUM group. However, except for a higher incidence of a prolonged Q-T_c interval for bleedings localised in the posterior fossa ($p < 0.01$), no major differences could be demonstrated. On the basis of the same ECG grouping it

was shown (table 5) that those patients in the TUM group with indirect signs of increased intracranial pressure, that is manifest or suspected papilloedema, had a slightly higher incidence of U-wave abnormalities ($p < 0.01$) as compared to the others in the TUM group without papilloedema. However, when those patients with papilloedema in the SAH group were compared with corresponding patients in the TUM group there were higher incidences of U-wave abnormalities ($p < 0.001$) in the SAH group and, consequently, a lower incidence of normal ECGs ($p < 0.01$).

ECG: time relation to onset of SAH

When the ECGs in the SAH group were divided according to elapsed days from bleeding to recording of the ECG, the number of patients in each group was found to be as shown in table 6. On the basis of this grouping, a higher incidence of Q-T_c interval prolongation ($p < 0.001$) was noted within 1 day. From 4 to 6 days after the bleeding a higher incidence of T-wave abnormalities ($p < 0.01$) was observed. After 13 days no Q-T_c prolongation and an increase in normal ECGs ($p < 0.01$) were observed.

Discussion

Incidence of ECG abnormalities

The finding of an approximately 80% incidence of ECG abnormalities in the SAH groups is in agreement with Stober and Kunze¹⁴ and Page *et al.*,²³ who examined SAH series of 89 and 100 patients,

Table 5 ECG abnormalities in patients with manifest or suspected papilloedema in the SAH group and the TUM group

Manifest or suspected papilloedema			T wave abnormality		U wave abnormality		Q-T _c interval prolongation		Normal ECG	
	No	%	No	%	No	%	No	%	No	%
SAH group	95	23	23	24	55	58	25	26	12	13
TUM group	95	24	20	21	17*	18	11	12	29	31
Significant differences between groups							p < 0.001		p < 0.01	

Significant differences of incidence of ECG abnormalities in these groups, compared with those of the remaining patients in the SAH group and the TUM group, are indicated as: * = $p < 0.01$.

Table 6 Grouping of patients in the SAH group according to days between bleeding and ECG recording

Days between bleeding and ECG recording	No	%	T wave abnormality		U wave abnormality		Q-T _c interval prolongation		Normal ECG	
			No	%	No	%	No	%	No	%
≤1	132	33	34	26	73	55	46†	35	18	14
2-3	143	35	48	34	65	45	33	23	26	18
4-6	66	16	31*	47	26	40	8	12	11	17
7-13	44	11	12	27	18	41	7	16	11	25
≥13	21	5.2	4	19	8	38	0	0	9*	43
Total SAH group	406		129	32	190	47	94	24	75	18

Each subgroup is compared with respect to the grouping of ECG items in the table with the remaining patients in the SAH group. Significant differences are indicated as: * = $p < 0.01$ and † = $p < 0.001$.

respectively. Other investigators with considerably smaller numbers of patients with SAH have reported incidences of 60–100%.^{9–17} In the present study, the observed higher incidence of ECG abnormalities in the SAH group than in the TUM group is in accordance with the findings of Hersch.⁸ Galloon *et al.*¹² on the other hand, found a 62% incidence for SAH patients ($n = 37$) and 68% for patients with space-occupying lesions ($n = 23$), but these figures were obtained after daily ECG tracings during hospitalisation and no other data about the series were reported.

When the SAH group and group SAHo were compared with corresponding tumour groups and a significant difference was found in the occurrence of a particular ECG abnormality, the higher incidence was always present in the SAH group. Moreover, the mean age of the tumour groups was higher, with a considerable number of patients over 60. It should be noted that when patients with other possible factors than the cerebral disorder influencing the ECG were excluded, the incidence of normal ECGs was found to be unchanged.

T and U wave, Q-T interval

In conformity with previous reports on SAH patients,^{6–11,17} most ECG abnormalities observed in the present study were disorders of the repolarisation process. The same total incidence of T-wave abnormalities was found both in the SAH group and group SAHo, whereas in the corresponding tumour groups the incidence was lower in group TUMo than in the TUM group, implying influences of other factors on the T wave than the intracranial disorder in this group. However, when patients with specific T-wave changes within the groups were compared only a slightly variable pattern of differences was observed. Obviously, this may be explained, to some extent, by the small number of patients in these subgroups. The most frequent ECG abnormality in the SAH group was a U wave with an amplitude of more than 1 mm (47%). In group SAHo 45% of the patients had an abnormal U wave. In the tumour groups only about

10% had an abnormal U wave.

The Q-T interval is, in the absence of conduction disturbances, an indirect measure of the duration of cardiac repolarisation. Prolongation of this interval is frequently reported in patients with SAH.^{6–11,14,24} The prolongation may be due to a disturbed regional myocardial sympathetic activity,^{25,26} which may be part of a general disorder of the sympathetic nervous activity following SAH.²⁷ In the present study the Q-T_c interval was longer in the SAH groups than in the tumour groups and the number of patients with an abnormally prolonged Q-T_c was also considerably larger in the SAH groups. The incidence of Q-T_c interval prolongation was lower, however, in subgroup SAHo than in the SAH group. This could be explained by the known influences on the Q-T interval of disturbances of serum potassium and calcium as well as cardiovascular disease among the patients in the complete SAH group.^{28,29}

S-T segment depression is another ECG change often reported in SAH patients.^{6–11,14} It was observed in 15% of the patients in the SAH group and in 13% in the TUM group. Significantly lower incidences were found in group SAHo, indicating that this ECG abnormality is probably due to extracranial factors.

Arrhythmias

A comparatively high incidence of arrhythmias including infrequent premature, atrial, nodal or ventricular beats and sinus arrhythmias were found in the SAH group, while other arrhythmias were rare in all groups. Goldstein¹⁷ has also reported a significantly higher incidence of sinus arrhythmia in SAH patients than in patients with stroke. A prolongation of the Q-T interval has been suggested to be associated with an increased susceptibility to cardiac arrhythmias.²⁸ Ventricular fibrillation has been reported in SAH patients with Q-T interval prolongation.^{30–32} However, when comparing incidences of arrhythmias between different reports, the ECG recording method is crucial. For a valid estimation a long and specified duration of ECG monitoring is indispensable.

Electrolytes

Early on electrolyte disturbances, particularly hypopotaemia, were postulated to be the cause of the ECG abnormalities seen in SAH patients⁸ but later reports did not confirm such a relationship.^{10 11} Cruickshank *et al*²⁷ found normal serum potassium in their patients with SAH, but when the total body potassium was examined, nearly all of the patients with abnormal ECGs had low levels, which returned to normal a few months later. In the present report high incidences of hypopotaemia and hypocalcaemia were observed in the SAH patients, significantly higher than in the tumour group. Hypopotaemia may cause a depression of the S-T segment, a decrease in the T-wave amplitude and an increase in the U-wave amplitude, as well as a tendency to precipitate arrhythmias. The Q-T interval may be difficult to measure because of U on T interaction.³³ Hypocalcaemia may increase the duration of the Q-T interval.³³ All these ECG abnormalities, with the exception of S-T segment depression, are similar to those appearing in association with cerebral disorders, particularly SAH. However, when patients with electrolyte imbalances as well as other factors with a possible impact on the ECG were excluded from the SAH and TUM groups no significant differences were observed in any of the groups with respect to the above-mentioned ECG abnormalities typical of cerebral disorders, except for the incidence of Q-T_c interval prolongation in the SAH group and group SAHo and for T-wave abnormalities in the TUM group and group TUMo. This implies that the majority of these ECG abnormalities were probably due to the cerebral disorder, particularly in the SAH patients, rather than to other factors. It must be pointed out, however, that the plasma total calcium concentration, which was measured in this study, does not correlate as closely as ionized calcium with the duration of the Q-T interval.³⁴

Intracranial pressure (ICP): ECG abnormalities

Patients with intracranial tumours are generally considered to have a higher incidence of sustained ICP elevation than patients with SAH. Papilloedema is considered to be a sign of elevated ICP, although it is sometimes absent despite a high ICP. On the basis of this sign, a higher incidence of ICP elevation in the TUM group could not be verified in the present study. An elevated ICP has been suggested as a cause of neurogenic ECG abnormalities.³⁵ However, the higher incidence of ECG abnormalities in the patients with SAH and the fact that the incidence of ECG abnormalities did not increase noticeably in the SAH and tumour patients with signs of elevated ICP, compared with those patients without this sign, did not support such a claim. However, it must be remembered that the development of the ICP elevation may

be of a fundamentally different nature in the two groups of patients, with a slow and progressive increase in tumour patients, and an acute increase immediately after the bleeding and thereafter possibly a fluctuant course in SAH patients.³⁶⁻³⁹

Location of intracranial lesion

Attempts to correlate the ECG abnormalities with the location of the cerebral disorder have been made by several authors but the reported results are divergent. No relationship of the ECG changes to the site of the bleeding aneurysm was found by Cropp and Manning,⁶ Shuster⁷ and Hunt *et al*.⁹ In studies focused on the intracranial vascular spasm after SAH and the appearance of ECG abnormalities, Wilkins *et al*⁴⁰ found no relationship, while Stober and Kunze¹⁴ were able to verify a correlation between spasms of cerebral arteries of the left hemisphere and negative T waves and Q-T interval prolongation. A correlation between arrhythmias and T-wave abnormalities and haemorrhages involving the anterior cerebral circulation has been reported in patients with cerebrovascular haemorrhages.^{41 42} Our major finding was an increased number of patients with Q-T_c interval prolongation and bleedings in the posterior fossa. Thus, from these various observations, it has not been possible to establish a clear correlation between the location of the intracranial disorder and a specific ECG abnormality.

Time factor: SAH

There have been few reports that take account of the number of days between bleeding and ECG recording. Generally, the ECG abnormalities are most marked immediately after the bleeding.^{7 8} The present study revealed a significantly higher incidence of normal ECGs after 13 days although the number of ECG tracings in this group was rather small. A large number of ECGs with Q-T interval prolongation and U-wave abnormalities were observed on the first day. This seems inconsistent with our previous study,²⁴ where Q-T interval prolongation was most pronounced on the 8th to 9th day after haemorrhage. However, in that study serial ECGs were followed for each patient and it was also shown that there was a relationship between Q-T interval prolongation and degree of impaired consciousness, which was not taken into consideration in the present study. These circumstances invalidate a direct comparison between the results of the two studies.

Conclusion

On comparing two large patient groups scheduled for neurosurgery, one with SAH and the other with intracranial tumours, the incidence of ECG abnormalities was significantly higher in the SAH group. ICP elevation, assessed as papilloedema, seems to be of

secondary importance for the appearance of the ECG abnormalities. A specific ECG abnormality correlated with a localised intracranial pathological change could not be established. Hypopotassemia and hypocalcaemia were much more common in the SAH group, but when patients with such disorders and other non-cerebral factors with possible influences on the ECG were excluded, the incidences of ECG abnormalities were nearly the same, particularly those of the U and T waves in patients with SAH. There was still a high incidence of Q-T_c interval prolongation in this patient group. These facts support a central nervous system origin of these ECG abnormalities. Further studies are required to clarify more precisely the causal connection between these abnormalities and the intracranial lesion.

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