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THE EEG IN TWENTYONE CASES  
OF HEAT STROKE

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THE EEG IN TWENTY ONE CASES OF

HEAT STROKE

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RESEARCH.

July, 1967

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8. /...

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SUMMARY

1. The aims of this study were :
  - a) to establish whether heat stroke is accompanied by EEG changes;
  - b) to assess the significance of such changes in terms of brain function;
  - c) to assess the prognostic value of the EEG in heat stroke;
  - d) to determine whether heat stroke leads to permanent disturbance of brain function in cases that survive.
2. EEGs were obtained at different times after the onset of heat stroke from 21 patients, nine of whom had serial EEGs.
3. EEGs were also obtained from another four cases in whom the final diagnosis was epilepsy in one, hypoglycaemia in one and heat exhaustion in two.
4. Although the sample was small the following tentative conclusions were drawn :
  - a. There was a very high incidence of EEG abnormality in cases of heat stroke, especially soon after onset. In cases that survive, the incidence of abnormality is lower but may rise again at one month or more.
  - b. Early EEG abnormalities were mostly suggestive of generalized cerebral dysfunction and of subcortical disturbance. Electrical signs of the latter were most prominent two to five days after the onset of symptoms. Focal abnormalities were seen in only two patients.
  - c. Variations in the incidence and type of EEG abnormality, particularly in the early stages, may be related to level of consciousness and biochemical changes.
  - d. There was no consistent relation between the EEG pattern and clinical outcome. One of the reasons for this may be that an apparently normal EEG can be recorded in cases of localized cerebral destruction.
  - e. The subcortical signs in some EEGs might reflect cerebellar and/or basal ganglion damage.

(ii)

5. No clear evidence on the persistence of EEG abnormalities was obtained.
6. It is proposed that the EEG be included in the methods of assessment in heat stroke because of its potential value in differentiating between heat stroke and such disorders as epilepsy and to facilitate short-term monitoring and follow-up of patients.
7. It is also recommended that the G. A. B. be employed to assess mental deficit in heat stroke cases by comparison with achievement on recruitment.
8. It is further recommended that animal studies of brain function be carried out in an attempt to obtain information on the relative significance of cortical and subcortical changes following heat stroke.
9. Further EEG studies in cases of heat stroke, if carried out in a systematic manner might provide additional information on brain function in this disorder.

OPSOMMING /...



OPSOMMING

1. Die doelstellings van hierdie projek was :
  - a) om vas te stel of hittesteek deur EEG-veranderinge vergesel word;
  - b) om die betekenis van sodanige veranderinge in terme van breinfunksie te bepaal;
  - c) om die voorspellingswaarde van die EEG in hittesteek te bepaal;
  - d) om vas te stel of hittesteek tot permanente steuring van breinfunksie lei by gevalle wat hittesteek oorleef.
2. EEGs is na verskillende periodes na die aanvang van hittesteek van 21 pasiënte verkry, van wie nege opvolg-EEGs gehad het.
3. EEGs is ook van nog vier gevalle geregistreer, met 'n finale diagnose van epilepsie in een geval, hipoglisemie in een en hitte-uitputting in twee.
4. Alhoewel die monster klein was is die volgende tentatiewe gevolgtrekkings gemaak :
  - a. Daar was 'n baie hoë voorkoms van EEG-afwyking by gevalle van hittesteek, veral kort na aanvang. By gevalle wie oorleef, is die voorkoms laer, maar mag na 'n maand of langer weer styg.
  - b. Vroë EEG-afwykings het meestal op algemene steuring van serebrale funksie en op subkortikale steuring gedui. Elektriese aanduidings van laasgenoemde was veral na twee tot vyf dae prominent. Fokale afwykings is slegs by twee gevalle gevind.
  - c. Wisseling van die voorkoms en tipe van EEG-afwyking, veral in die vroë stadia, mag verband hou met bewussynspeil en biochemiese veranderinge.
  - d. Daar was geen konsekwente verband tussen die EEG-patroon en kliniese uitslag nie. Een van die redes hiervoor mag wees dat 'n blykbaar normale EEG in gevalle van gelokaliseerde serebrale beskadiging geregistreer kan word.

e. /...

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INTRODUCTION /...

- e. Die subkortikale afwykings in sommige EEGs mag op beskadiging van die kleinharsings en/or basale senuwee-knope dui.
- 5. Daar was geen duidelike bewys ten opsigte van die voortbestaan van EEG-afwykings nie.
- 6. Daar word aanbeveel dat die EEG by ondersoek-metodes vir hittesteek ingesluit word, weens sy potensiële waarde in die onderskeiding tussen hittesteek en afwykings soos epilepsie, en vir kort-termyn kontroleopnames en opvolgstudies van pasiënte te vergemaklik.
- 7. Daar word ook aanbeveel dat die Algemene Aanpassingstoetsbattery gebruik word om geestelike steuring as gevolg van hittesteek te bepaal, deur middel van vergelyking met prestasie by indiens-neming.
- 8. Daar word verder aanbeveel dat dierstudies van breinfunksie uitgevoer word ten einde inligting ten opsigte van die relatiewe betekenis van kortikale en subkortikale veranderinge by hittesteek te probeer verkry.
- 9. Aanvullende studies van hittesteek-gevalle, mits hulle op 'n sistematiese wyse uitgevoer sou word, mag verdere inligting ten opsigte van breinfunksie by hierdie sindroom verskaf.

## 1. INTRODUCTION

Heat stroke has been described as a syndrome "characterized by hyperpyrexia and severe disturbance of the central nervous system and caused by hot environmental conditions" (Barry and King, 1962). These authors emphasise disturbances of consciousness of a kind resembling cerebral decortication. "The patient is conscious", they say, "but the content of consciousness is severely restricted" (Barry and King, op. cit.).

The syndrome is thus of basic interest from the point of view of brain function. Barry and King contend that the features are compatible with microscopic lesions found at autopsy. Specifically they refer to petechiae and chromatolysis of the neurones of the cerebellum and cerebral cortex, but note that there is no such effect on the hypothalamus and postaqueeductal areas, although it has been suggested that the hypothalamic cells are damaged at a "biochemical and biophysical level". They stress that the reticular activating system continues to function, concluding that this explains why the disturbances of consciousness in heat stroke resemble experimental decortication.

Other types of behavioural disturbance described by Barry and King are confusion, delirium, stupor, semicoma and coma. The coarse tremors that often occur were ascribed tentatively to basal ganglia involvement. They suggest that these coarse tremors have been mistaken for epileptic tremors.

In the review of the literature, Peter (1963) notes that the earliest findings in the brain were of oedema, congestion, petechial haemorrhage and the absence of localized haemorrhage. The grey matter seemed to be more affected than the white matter, while there was less degeneration in other regions such as the thalamus. The hypothalamus, midbrain, medulla and cord showed no damage although there was slight impairment in various other regions, including the reticular formation. This latter finding is at first sight incompatible with Barry and King's assertion that the reticular system continues to function, but it is possible that the functional effects of slight impairment might well be minimal.

It would appear that the most striking changes take place in the cerebellum, where there is extensive neural degeneration, particularly involving the Purkinje cells.

These observations were considered sufficient to justify the application of the electroencephalograph (EEG) for the purpose of eliciting more information about such changes in the brain as accompany heat stroke .

Following approval of the project by the Research Advisory Committee of the Chamber of Mines of South Africa, the more specific aims set out in 2 were formulated.

2. AIMS /...

2. A I M S

The general purpose of this study was to use the electroencephalogram (EEG) to investigate possible changes in cerebral function accompanying and following heat stroke sustained in the South African Gold Mines.

The specific aims were :

- 2.1 to establish whether heat stroke is accompanied by EEG changes;
- 2.2 to assess the significance of such changes in terms of brain function;
- 2.3 to assess the prognostic value of the EEG in heat stroke;
- 2.4 to determine whether heat stroke is followed, in cases who survive, by a permanent disturbance of EEG pattern.

3. MATERIAL /...

3. MATERIAL.

The patients studied were 26 Bantu male adults. In the majority of cases age could not be reliably ascertained and this variable is accordingly omitted from the analysis.

The final diagnosis was heat stroke in 22 cases, heat exhaustion in two, hypoglycaemia in one and epilepsy in one.

Six of the heat stroke patients died, survival times being a few hours (in two cases), one day (one case), two days (one case) one month (one case) and two years seven months (one case). All the other patients are, as far as the writer is aware, still living.

4. METHOD /...

#### 4. METHOD

In general eight-channel EEGs were obtained by means of a portable Offner Type T electroencephalograph, using silver - silver chloride saline pad electrodes in selected positions according to the International ten - twenty system. In most cases the patients were supine during the recording.

The majority of the EEGs were recorded in mine hospitals, the apparatus being transported by car and once by aircraft. A number of follow-up EEGs were obtained at the NIPR.

In a number of examinations it was impossible to include the standard activating procedures of photic stimulation (blue - white flash, frequency being varied from three to 100 flashes per second) and voluntary hyperventilation (deep breathing for three minutes), - see Section D of Results.

It was originally planned to obtain the first EEG as soon as possible after the onset of the symptoms of heat stroke, but the first record was obtained within a few hours from only 15 patients. The first EEG examination of three of the remainder was recorded at intervals ranging from one to five days and of one 21 days after the onset of the symptoms. Two patients were seen for the first time after three and four and a half months respectively.

In most cases the reason for delay in obtaining the first EEG was tardiness in reporting the case to the NIPR.

#### 5. RESULTS /...

5. RESULTS

5.1 DATA OBTAINED

A total of 46 EEGs was obtained from the 25 patients, 41 of these being from the 21 cases with a final diagnosis of heat stroke. The distribution of records per patient is shown in Table I.

TABLE I

NUMBER OF EEGs OBTAINED FROM INDIVIDUAL PATIENTS

Final diagnosis	EEGs per patient					Total patients
	1	2	3	4	5	
Heat stroke	13	5	2	1	1	22
Heat exhaustion		2				2
Epilepsy	1					1
Hypoglycaemia		1				1
						26

5.2 EEG FINDINGS IN HEAT STROKE

5.2.1 Within 24 hours after onset of symptoms

The EEG was abnormal in 13 (87%) of the 15 patients tested at this time. The findings are summarized in Table II.

Only two patients had normal EEGs. One other had a doubtful record suggestive of a left-sided cortical dysfunction. All three survived but one of the patients with a normal EEG had marked sequelae.

Moderately /...



Moderately abnormal records with different types of electrical disturbance were found in five cases. Four of these patients recovered and one died a month later.

Six cases had severely abnormal records, all suggestive of diffuse cerebral dysfunction. Three of these patients died within a few days, two recovered and one survived with partial paralysis and ataxia.

The remaining patient (case 12) apparently expired as the EEG recording started and his record showed no electrical activity at all. However, case 10 also had a "flat" EEG after tracheostomy but he recovered (see 5.2.2).

There was a significant relationship between degree of EEG abnormality and the lapse of time between the onset of the symptoms of heat stroke and the EEG examination, in that the earlier records tended to be more abnormal ( $r = -.56$ ,  $p < .05$ , see Fig. 1).

#### 5.2.2 One to five days after onset of symptoms

Six patients were examined at this time. In three cases this was the second EEG recording. The findings are summarized in Table III.

Normal EEGs were found in three patients (50%), one of whom recovered, one (case 16) survived with partial paralysis and ataxia.

One patient had a mild EEG abnormality suggestive of subcortical dysfunction - he recovered. Another had a moderately abnormal record also suggestive of subcortical dysfunction; he survived but with marked mental defect (assessed clinically).

A series of EEGs obtained from Case 22 on the day after he sustained heat stroke showed a slight improvement when compared with the record of the previous day, although remaining severely abnormal due to fluctuating dominant rhythms in the theta range. He died on the following day before another EEG could be recorded.

FIGURE 1. /...

FIGURE 1

RELATION BETWEEN TIME AFTER ONSET OF  
HEAT STROKE AND DEGREE OF EEG  
ABNORMALITY

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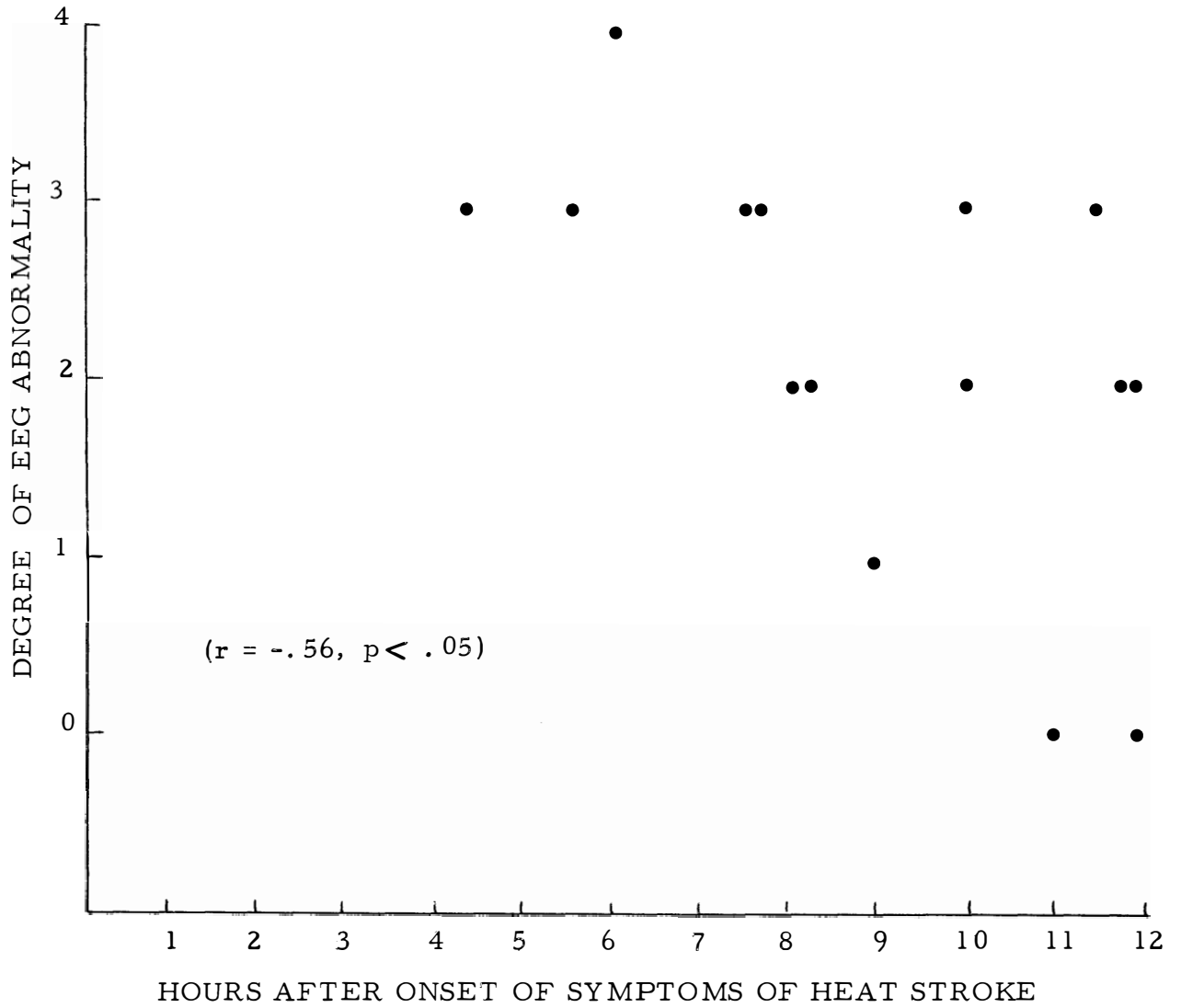


TABLE II /.

TABLE II

## EEG FINDINGS WITHIN 24 HOURS AFTER ONSET OF HEAT STROKE (15 CASES)

CASE NO.	TIME AFTER ONSET (HRS).	STATE	DEGREE OF ABNORMALITY	ABNORMAL SIGNS	INTERPRETATION	FOLLOW-UP
4	Approx. 12.00	Confused at first	Moderate	Excessive alphoid & asynchronous occipital delta	Sub-cortical dysfunction	Recovered*
5	9.00	? asleep	Doubtful	Alpha & beta confined to L hemisphere	? Left-sided cortical abnormality	Discharged with marked sequelae**
6	Approx. 12.00	Awake	Normal	- - -	Normal	Recovered
10	7.5	Comatose	Severe	Excessive very slow delta, then "flat" EEG after tracheostomy	Severe cerebral dysfunction, ?cerebral anoxia	Recovered - transferred to surface work**
12	Approx. 6.00	Dying	Extreme	No electrical activity	Death	Died
13	Approx. 10.00	Comatose	Severe	Dominated by delta activity	Severe diffuse cerebral dysfunction	Died on same day
16	Approx. 8.00	Semi-comatose	Moderate	Diffuse high-voltage delta	?Subcortical dysfunction	Survived with partial paralysis and ataxia*
17	11.75	Awake, raised muscle tone	Severe	Delta focus right fronto-temporal & occasional generalized paroxysmal theta	Right fronto-temporal epileptogenic disturbance	Recovered
18	4.25	Comatose	Severe	Continuous theta	Diffuse cerebral dysfunction	Recovered**
19	12.00	Confused	Moderate	Isolated delta discharges and diffuse theta	Non-specific	Recovered

Continued /...

TABLE II (Continued)

CASE NO.	TIME AFTER ONSET (HRS)	STATE	DEGREE OF ABNORMALITY	ABNORMAL SIGNS	INTERPRETATION	FOLLOW-UP
20	4.75	Semicomato- tose hypo- tensive	Severe	Fluctuating low dominant frequency	Compatible with fluctuating hypo- tension	Died on following day
21	8.00	Violent, irritable	Moderate	Left-sided beta	?Left sided cortical disturbance	Died one month later*
22	7.50	Comatose	Severe	Excessive frontal delta right parieto-temporal sharp waves diffuse very slow discharges	Severe cerebral dys- function with particu- lar involvement of right parieto- temporal area	
25	Approx. 10.00	Awake	Moderate	Left parieto-temporo- occipital delta distur- bances; excessive theta, especially on left	Left parieto-temporal dysfunction with sub- cortical involvement	Recovered
26	Approx. 11.00	Asleep	Normal	- - -	Normal	Recovered*

Footnotes : \* For further EEGs see Table IV  
 \*\* For further EEGs see Table V

TABLE III

EEG FINDINGS 1 to 5 DAYS AFTER ONSET OF HEAT STROKE

TIME AFTER ONSET (DAYS)	CASE NO.	STATE	DEGREE OF ABNORMALITY	ABNORMAL SIGNS	INTERPRETATION	FOLLOW-UP
1	7	Drowsy	Mild	Asynchronous and assymmetrical alpha activity; some focal theta right parieto-occipital area	? sub-cortical dysfunction	Recovered
1	11	Drowsy	Normal but unusual	(Persistent alpha, considerable alphoid)	Normal	Recovered
1	15	Semicomatose at first, then awake	Moderate	While semicomatose: considerable inter-hemispheric asynchrony; while awake: dominant theta and delta rhythms	?Sub-cortical dysfunction	Survived with severe mental defect **
5	16 (2nd EEG)	Awake, nervous	Normal	--	Normal	Survived with paralysis and ataxia
3	21	Unresponsive	Normal	--	Normal	Died 3 weeks later *
2	22 (2nd, 3rd & 4th EEGs)	Unresponsive	Severe, but improved	Dominant frequency still in theta range - no focal disturbance	Hypotension	Died on following day

FOOTNOTES :      \* For further EEGs see Table IV  
                         \*\* For further EEGs see Table V

5.2.3 Eleven to 24 days after onset of heat stroke

Five cases were studied within this interval. Case 9 had his first EEG at three weeks; cases 4 and 10 had their second EEGs at 11 and 21 days respectively, while case 21 had his third EEG at 14 days. The results are shown in Table IV.

Four cases had normal EEGs at this stage. The records of cases 4 and 10 had become normal while case 21 had had a normal EEG on the previous occasion. However, he died 25 days later.

The only EEG abnormality was seen in case 26 whose record contained excessive diffuse low-voltage theta activity. His previous EEG, a few hours after sustaining heat stroke, was normal (Table II).

TABLE IV /...

TABLE IV

## EEG FINDINGS 11 to 24 DAYS AFTER ONSET OF HEAT STROKE (5 CASES)

CASE	TIME AFTER ONSET (DAYS)	STATE	DEGREE OF ABNORMALITY	ABNORMAL SIGNS	INTERPRETATION	FOLLOW-UP
4 (2nd EEG)	11	Tense, then calmer	Normal	--	Normal	Recovered
9	21	Tense at first then sleepy	Normal	--	Normal	Recovered
10 (2nd EEG)	21	Drowsy	Normal	--	Normal	Recovered *
21 (3rd EEG)	14	Awake	Normal	--	Normal	Died 25 days later
26	24	Awake	Mild	Excessive diffuse theta activity	Non-specific	Recovered

Footnote : \* See Table V for subsequent EEGs.

5.2.4 More than one month after onset of heat stroke

Initial EEGs were obtained from two patients (cases 1 and 14) at 12 and 13 weeks respectively after they sustained heat stroke. Second EEGs were obtained from Cases 5 and 15 at eight and 12 weeks respectively. Case 10 underwent his third, fourth and fifth EEG examinations at six, seven and ten weeks after the onset of his symptoms.

Of the two cases studied for the first time at this stage, case 14 had a normal EEG and case 1 had a doubtful record suggestive of subcortical dysfunction. Case 1 survived for over two years, and case 14 recovered.

The second EEG of case 5 was normal, having thus improved. The second record of case 15 remained abnormal. Both survived with marked sequelae.

Case 10, whose EEG had virtually disappeared after tracheostomy on the day he sustained heat stroke, had a normal record on the second occasion, but his EEG showed deterioration at six, seven and 10 weeks. He recovered.

These findings are summarized in Table V.

TABLE V /...



TABLE V

EEG FINDINGS MORE THAN ONE MONTH AFTER ONSET OF HEAT STROKE (SIX CASES)

CASE	TIME AFTER ONSET (WKS)	STATE	DEGREE OF ABNORMALITY	ABNORMAL SIGNS	INTERPRETATION	FOLLOW-UP
1	12	Awake	Doubtful	Low-voltage theta and delta, appearing in L & R post temporal areas	?subcortical dysfunction	Died 2 yrs & 4 mths. later.
5 (2nd EEG)	8	Awake	Normal	- - -	Normal	Survived with marked sequelae.
10 (3rd EEG)	6	Awake	Mild	Excessive low-voltage delta & theta	?sub-cortical dysfunction	Recovered
10 (4th EEG)	7	Drowsy	Moderate	Irregular temporo-occipital delta & theta disturbances, independently on L & R.	? sub-cortical dysfunction	Recovered
10 (5th EEG)	10	Drowsy	Moderate	Similar to above	? sub-cortical dysfunction	Recovered
14	13	Awake	Normal	- - -	Normal	Recovered
15 (2nd EEG)	12	Awake	Moderate	Excessive low-voltage diffuse theta	?cortical depression	Survived with mental defect & ataxia
18 (2nd EEG)	6	Dull	Severe	Decelerated dominant frequency	?cerebral damage	Recovered

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### 5.3 RELATION OF EEG FINDINGS IN HEAT STROKE TO CLINICAL OUTCOME

#### 5.3.1 Fatal cases

Six of the 20 cases studied died, a mortality of 30%. Survival times and EEG findings are shown in Table VI.

Five of these patients had EEG examinations on the day of onset of heat stroke, four having severely abnormal records and one a moderate abnormality. One patient had an isoelectric or "flat" EEG and was probably already dead. Two of the other four had EEGs with diffuse slow activity and two had focal disturbances.

Serial EEGs were obtained from cases 21 and 22. In the former, two subsequent records were normal, although the patient died two weeks after the last EEG. Case 22, with a focal EEG abnormality on the first day had a diffuse abnormality two days later, dying soon after the second EEG.

The remaining patient (case 1) was first tested 12 weeks after he had sustained heat stroke followed by prolonged unconsciousness. He had a marked basal ganglion and cerebellar syndrome and his EEG was of doubtful normality, being suggestive of subcortical dysfunction. He died more than two years afterwards.

In this series of nine EEGs from the six patients who subsequently died there were only two normal records, both follow-up EEGs from one patient. This leads to the attractive hypothesis that a severely abnormal EEG might be an aid to prognosis in heat stroke. Considering the 15 records obtained within the first 24 hours, it will be seen that the 10 non-fatal cases had seven moderately or severely abnormal EEGs and three normal or mildly abnormal records. The five fatal cases all had moderately or severely abnormal EEGs. However this does not represent a significant difference in respect of EEG abnormality between the fatal and non-fatal cases ( $p = .26$ , Fisher's Exact Test). An additional reason for rejecting the hypothesis at this stage is provided by the improvement in the EEGs of Case 21 who died, and the severe abnormality of the later EEGs of Cases 10, 15 and 18, all of whom survived.

TABLE VI /...

TABLE VI

EEG FINDINGS IN SIX FATAL CASES OF HEAT STROKE

CASE NO.	SURVIVAL TIME	TIME OF EEG EXAMINATION	DEGREE OF EEG ABNORMALITY	TYPE OF EEG ABNORMALITY	INTERPRETATION OF EEG
1	2 yrs. 4mths.	12 weeks	Doubtful	Asynchronous bi-lateral post-temporal theta	?subcortical dysfunction
12	< 24 hours	Day of onset	Extreme	Isoelectric	Death
13	< 24 hours	Day of onset	Severe	Diffuse delta	Diffuse cerebral dysfunction
20	1 day	Day of onset	Severe	Diffuse slow activity	Compatible with hypotension
21	1 month	1) Day of onset	Moderate	Focal beta activity left hemisphere	Left-sided cortical dysfunction
		2) 3 days	Normal	- -	- -
		3) 14 days	Normal	- -	- -
22	2 days	1) Day of onset	Severe	Focal sharp waves	Right-parieto-temporal abnormality
		3) 2 days	Severe	Diffuse theta activity	Compatible with hypotension

5. 3. 2 Cases with neurological and/or psychological sequelae

Three patients had marked neurological sequelae : Case 1 had a basal ganglion and cerebellar syndrome and died after more than two years. Case 5 had marked but unspecified neurological abnormalities and case 16 was left with partial paralysis and ataxia. Case 15 was clinically assessed after physical recovery as a "simpleton".

The EEG findings are summarized in Table VII.

TABLE VII /...

TABLE VII

EEG FINDINGS IN FOUR PATIENTS WITH SEQUELAE AFTER HEAT STROKE

CASE NO.	SEQUELAE	TIME OF EEG EXAMINATION	DEGREE OF EEG ABNORMALITY	TYPE OF EEG ABNORMALITY	INTERPRETATION OF EEG
1	Basal ganglion and cerebellar syndrome	12 weeks	Doubtful	Asynchronous bilateral post temporal theta	? Subcortical dysfunction (Died)
5	Unspecified neurological sequelae	1) < 24 hrs	Doubtful	Alpha and beta confined to left hemisphere	? Left-sided cortical abnormality
		2) 8 weeks	Normal	- - -	- - -
15	"Simpleton"	1) 1 day	Moderate	Dominant theta and delta activity	? Subcortical dysfunction
		2) 12 weeks	Moderate	Excessive theta activity	? Subcortical depression
16	Partial paralysis and ataxia	1) 24 hours	Moderate	Diffuse high-voltage delta activity	? Subcortical dysfunction
		2) 5 days	Normal	- - -	- - -

EEGs were obtained soon after heat stroke in only three of these patients, two of whom had moderate diffuse abnormalities and one a doubtful record. A follow-up EEG after five days in case 16, whose first record was moderately abnormal, was normal. Follow-up records in the other three cases were obtained at 8 to 12 weeks after the onset of the symptoms. There was one normal, one doubtful and one moderately abnormal record.

Two trends emerge from these results : the EEG interpretation of subcortical dysfunction in three of the four patients : the apparent left-sided cortical disturbance in the fourth might also have reflected a subcortical disturbance. Such a cerebral abnormality would be compatible with the sequelae. The second trend is an apparent improvement in the EEGs of two of these patients with time, while one of the others had only a doubtful record at a late stage. Although both records from the remaining patient were moderately abnormal the dominant frequency increase after 12 weeks suggests that there was nevertheless some improvement.

#### 5.4 PERSISTENCE OF EEG CHANGES IN HEAT STROKE

Examination of the EEG findings at one month or more after heat stroke reveals only two patients with normal records. The remaining four comprised one with a doubtful record, two with moderate abnormalities and one with a severely abnormal EEG. Of the deviant records two were suggestive of sub-cortical dysfunction, one of cortical depression and one of diffuse brain damage.

All four abnormal EEGs contained excessive theta activity, diffuse in two and predominantly post-temporal in two, being dominant in one of the latter. Two records also showed excessive delta rhythms.

There was no consistent clinical relationship with these findings. Of the two patients whose EEGs contained excessive delta activity, one died more than two years after sustaining heat stroke and one apparently recovered. Of the other two with abnormal records due to theta activity alone, one recovered and the other survived with ataxia and mental defect.

Only two of these records (those with delta activity) directly raise the question of permanent brain damage. Of the remaining two the EEG with the more ominous sign was that of case 6, showing an apparently decelerated dominant rhythm below the alpha frequency range in a dull patient who apparently recovered.

Delta activity was seen at 12 and 10 weeks respectively in two cases, raising the possibility of permanent brain damage.

## 5.5 RESPONSE OF THE EEG IN HEATSTROKE TO ACTIVATION

### 5.5.1 Photic stimulation :

The normal response of the EEG to photic stimulation is in the form of medium to low voltage activity from the parieto-occipital areas at the stimulus frequency in the 3 to 15 f/sec. range. Responses at the fundamental frequency rarely occur above this range. Responses at harmonics of the stimulus frequency are common while subharmonic responses are more rare in normal adults.

Abnormal responses comprise a variety of features of the epileptic type such as spikes, waves-and-spikes and paroxysmal discharges at various frequencies. Other abnormal responses are excessive subharmonic activity and high-voltage recruiting responses, the former suggestive of cortical immaturity and the latter of cortical hyper-excitability.

No response to photic stimulation is not per se an abnormal sign.

#### 5.5.1.1 Responses to photic stimulation within 24 hours after onset of symptoms (Table VIII)

Photic stimulation was administered to ten of the 15 patients tested at this time. It was not applied to cases 5, 12, 16, 20 and 22.

Four patients (27%) - cases 10, 13, 18 and 19 showed no response at all. Five others (6, 17, 21, 25 and 26) had normal responses but case 17 had considerable subharmonic activity at 9 f/sec. Two of the four patients with no response were comatose and one was confused. Three patients with normal responses were conscious, but one (case 17) had a raised muscle tone and one was violent and irritable (case 21). Case 26 was asleep.

Case 4 had a doubtful response with excessive occipital theta and delta activity appearing independently in the hemispheres, suggestive of a disruption of subcortical regulating mechanisms. Similar disturbances appeared in his resting EEG.

#### 5.5.1.2 Responses on 2nd and 5th days (Table IX)

All but one of the six patients with EEGs at this stage were given photic stimulation. The response was normal in

three /...

three cases (7, 15 and 16) but there was no response in cases 11 and 21. Case 21 had previously responded to photic stimulation.

5. 5. 1. 3 Responses on the 11th to 24th days (Table X)

Four of the five patients tested at this stage had photic stimulation. Cases 4, 9 and 26 showed normal responses, case 4 having previously shown an abnormal electrocortical reaction.

Case 10 showed no response, as he had done on the day he sustained heatstroke, three weeks before.

5. 5. 1. 4 Responses at more than one month (Table XI)

Five of the six patients with EEGs at this stage had photic stimulation. The response was normal in Cases 1, 5 and 15 (previously also normal). Case 10 continued to show no response, at six and 10 weeks. The response of case 14 was doubtful due to a second subharmonic discharge in the right hemisphere at 12 f/sec.

TABLE VIII /...



TABLE VIII

RESPONSES TO PHOTIC STIMULATION WITHIN 24 HOURS AFTER ONSET OF HEAT STROKE  
(10 CASES)

CASE NO.	STATE	DEGREE OF RESPONSE	ABNORMALITY OF RESPONSE	INTERPRETATION
4	Confused at first	Marked	Doubtful, asynchronous	? Subcortical dysfunction
6	Awake	Slight	Normal	---
10	Comatose	None	Doubtful	Doubtful (EEG became isoelectric)
13	Comatose	None	Doubtful	Doubtful ('Resting' EEG severely abnormal)
17	Awake, raised muscle tone	Marked	Normal	---
18	Comatose	None	Doubtful	Doubtful ('Resting' EEG severely abnormal)
19	Confused	None	Doubtful	Doubtful ('Resting' EEG moderately abnormal)
21	Violent, irritable	Slight	Normal	---
25	Awake	Slight	Normal	---
26	Asleep	Good	Normal	---

TABLE IX

RESPONSES TO PHOTIC STIMULATION 1 TO 5 DAYS AFTER ONSET  
OF HEAT STROKE (5 CASES)

---

CASE NO.	STATE	DEGREE OF RESPONSE	ABNORMALITY OF RESPONSE	INTERPRETATION
7	Drowsy	Moderate	Normal	Normal
11	Drowsy	None	Normal	Normal ('Resting' EEG normal)
15	Semicomatose at first, then awake	Slight	Normal	Normal
16 (2nd EEG)	Awake, nervous	Good	Normal	Normal
21 (2nd EEG)	Unresponsive	None	Doubtful	Doubtful ('Resting' EEG severely abnormal)

TABLE X

RESPONSES TO PHOTIC STIMULATION 11 TO 24 DAYS AFTER  
ONSET OF HEAT STROKE (4 CASES)

CASE NO.	STATE	DEGREE OF RESPONSE	ABNORMALITY OF RESPONSE	INTERPRETATION
4	Tense, then calmer	Moderate	Normal	Normal
9	Tense at first then sleepy	Good	Normal	Normal
10 (2nd EEG)	Drowsy	None	Normal	Normal
26 (2nd EEG)	Awake	Moderate	Normal	Normal

TABLE XI

RESPONSES TO PHOTIC STIMULATION MORE THAN ONE MONTH AFTER ONSET OF  
HEAT STROKE (5 CASES)

CASE NO.	STATE	DEGREE OF RESPONSE	ABNORMALITY OF RESPONSE	INTERPRETATION
1	Awake	Moderate	Normal	Normal
5 (2nd EEG)	Awake	Moderate	Normal	Normal
10 (3rd EEG)	Awake	None	Normal	Normal
10 (5th EEG)	Drowsy	None	Normal	Normal
14	Awake	Slight	Doubtful (2nd Subharmonic discharge right hemisphere)	Doubtful
15 (2nd EEG)	Awake	Slight	Normal	Normal

In summary, of the 25 EEGs with photic stimulation (from 18 patients) not one was unequivocally abnormal. There were however seven doubtful responses in as many patients and there was a tendency for doubtful responses to be more common within the first few hours after sustaining heat stroke. Five of these seven doubtful responses were labelled as such because against the background of an abnormal EEG there was no electro-cortical response to photic stimulation. In the other two cases there was interhemispheric asynchrony (case 4) and a unilateral subharmonic discharge (case 14).

There was no response to photic stimulation in nine EEGs from six patients. As in the case of the doubtful responses, there was an apparently higher incidence of these unresponsive records in the first 24 hours after heat stroke was sustained.

In regard to a probable association between conscious and emotional state on the one hand and the type of response to photic stimulation on the other, while the seven doubtful responses were recorded, the patient was comatose in three instances, confused in two, unresponsive in one and awake (and behaviourally normal) in only one instance. In general photic stimulation does not make an EEG response in coma.

When there was no response to photic stimulation the patient was comatose in three instances, confused in one, unresponsive in one, drowsy in three and awake (and behaviourally normal) in only one instance.

By contrast the other 11 EEGs recorded with photic stimulation, while the patient was awake, all showed responses which were normal.

In regard to prognosis, only two patients (cases 13 and 21) who had no response to flicker (a phenomenon regarded as doubtful because of the abnormality of their "resting" EEGs) subsequently died.

#### 5.5.2 Hyperventilation

This activating technique requires the co-operation of the patient. For reasons of diminished consciousness, confusion and inability to grasp instructions hyperventilation was included in only 10 examinations of eight patients.

Cases 4 and 6 were seen on the first day, case 9 at one and at 21 days and cases 1, 5, 10, 14 and 15 at intervals of more than one month after sustaining heat stroke.

The only abnormal response occurred in case 4 : during hyperventilation there was excessive delta and theta activity which may well have been associated with some such factor as relative hypoglycaemia.

## 5.6 EEG FINDINGS IN HEAT EXHAUSTION, EPILEPSY AND HYPOGLYCAEMIA

### 5.6.1 Heat exhaustion

Records were obtained from two patients (cases 2 and 3) four days after onset of symptoms and again a week later.

Both initial EEGs were mildly abnormal due to excessive medium-low voltage theta activity often appearing independently on the left and right sides, thus suggesting a disturbance of subcortical function. In addition the EEG of case 2 contained one brief bilateral high-voltage paroxysmal theta burst, raising the additional question of a lowered convulsive threshold. This patient was in fact reported to have had an "epileptic fit" on admission.

On the second occasion the record of case 3 was normal, but in case 2 the EEG was mildly abnormal due to one occipital delta episode, starting in the right hemisphere and spreading to the left side. This is also suggestive of a subcortical dysfunction. Both cases recovered.

### 5.6.2 Epilepsy

Case 8 was reported with a provisional diagnosis of heat stroke. An EEG recorded three hours after admission to the mine hospital was severely abnormal due to numerous high-voltage frontal 1.5 - 3 per second wave-and-spike discharges, maximal on the right side. In addition the alpha rhythms were abnormally slow at 7 - 8.5 c/sec. The record was judged as strongly suggestive of an epileptic disorder. The subsequent clinical diagnosis agreed with this interpretation.

The patient returned to work.

### 5.6.3 Hypoglycaemia

Case 23 was reported as heat stroke, but subsequently diagnosed biochemically and clinically as hypoglycaemia. Two EEGs were obtained, the first seven hours after admission to the mine hospital and the second 24 hours later. Both records were normal and the patient recovered.

## 6. DISCUSSION /...

## 6. DISCUSSION

Any inference drawn from the observations made during this study is subject to the severe limitations imposed by the restricted sample. However the inclusion of the EEG in the investigation of heat stroke provided a unique opportunity of assessing brain function in a relatively rare disorder. To the best of the writer's knowledge these findings, although not far-reaching and are unique.

The **results** will be discussed in the light of the four main aims of the project.

### 6.1 EEG CHANGES IN HEAT STROKE

The incidence of EEG abnormality observed in the 15 patients studied on the day of the onset of the symptoms of heat stroke was 87 per cent. Although the sample is small, this figure is far in excess of the 17% incidence of abnormal records in the normal Bantu population (Nelson, 1967), the t value being 7.92,  $p < 10^{-7}$ .

At subsequent intervals after the onset of heat stroke the incidence of abnormality appears to decline (at 2 - 5 days and 11 - 21 days) but seems to rise again thereafter. However the numbers of records available at these stages are far too small to warrant statistical analysis.

### 6.2 SIGNIFICANCE OF THE EEG CHANGES IN HEAT STROKE

Within a few hours after the onset of heat stroke a common EEG abnormality was diffuse slowing suggestive of generalized cerebral dysfunction. This occurred in four of the 15 patients, excluding the one who died as the examination started. Equal in prominence come signs of localized cortical dysfunction, in another five cases, involving the left hemisphere (in two cases), the right parieto-temporal, the left parieto-temporal and the right fronto-temporal regions. This latter case was the only patient with a confirmed diagnosis of heat stroke who showed EEG disturbances of the epileptic type.

The remaining two patients with abnormal records showed signs of sub-cortical dysfunction and a non-specific deviation respectively.

From one to five days after heat stroke, EEG evidence of subcortical dysfunction was found in two out of six patients, the one other abnormal record showing diffuse electro-cerebral slowing.

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From 11 to 21 days normal EEGs were seen in four out of five patients. At the later stages of one month or more, EEG signs of diffuse and of sub-cortical dysfunction were equally represented (in two patients each out of a total of six).

Diffuse slowing as encountered in a number of these cases of heat stroke is not a specific sign of any particular pathology, but is encountered in association with a variety of factors such as severe head injury, hypoglycaemia, uraemia and protein malnutrition. It would be compatible with petechial haemorrhages of the cortex. It is perhaps worth noting that the EEG concomitants of kwashiorkor in which cortical petechiae have also been reported comprise mainly severe slowing of the brain rhythms, although these observations relate to patients of a few years of age (Nelson, 1959).

The EEG features suggestive of subcortical dysfunction comprise mainly fluctuating asynchronies and asymmetries of theta activity with no consistent lateral location. Such electrical features might well reflect hypothalamic damage in heat stroke as described by Barry and King (1962).

The focal disturbances, though rare, seemed to have a predilection for the parieto-temporal area. This further implicates subcortical dysfunction because of the anatomical proximity of the temporal lobes to these regions, (Strauss et al., 1952) but diffuse cerebral disturbance is most likely to be reflected first and often in greater degree in the temporal areas - these authors believe that the temporal lobe reacts electroencephalographically more severely to any cortical disturbance than do the more medial cortical regions. In an early paper Walter (1937) asserted that temporal lobe foci may represent the localization to this area of some such feature as cortical oedema, another factor known to be associated with generalized EEG slowing.

In evaluating the precise significance of the EEG abnormalities in general it is however essential to consider a number of possible sources of EEG disturbance in this context :

a) Level of consciousness

Many of the patients studied were comatose or confused. These variables are extremely difficult to measure accurately by methods other than the EEG itself (Nelson 1963) and in this situation it was impossible to disentangle completely the factors of lowered level of consciousness and of pathological changes in brain function. Moreover there were differences not only among the comatose patients with regard to depth

of /...



of coma, but also differences between these patients and those who were merely confused, in an apparently normal sleep or awake.

Although all four patients who were in a coma had severely abnormal EEGs, so did one who was semicomatose and one who was clearly awake. The three with moderate EEG abnormalities were semicomatose, confused and violent respectively. The patient with the doubtful record was apparently in a normal sleep, while the only patient who was awake had a normal record. Having regard to the variety of abnormalities seen, their degree would seem to be associated with the depth of unconsciousness in heat stroke.

#### b) Biochemistry

The profound and variable biochemical changes accompanying heat stroke are likely causes of generalized electrocerebral changes in the form of overall slowing of the EEG pattern. In particular alterations in blood chemistry consequent on impairment of liver function and hypoglycaemia may cause severe slowing of the EEG (Engel et al., 1944). In this connection it is noteworthy that the patient finally diagnosed as suffering not from heat stroke but from hypoglycaemia (case 23) had a normal EEG on the day of onset of the symptoms and on the following day.

A further source of variation in biochemistry is to be found in differences in method of treatment from one hospital to another and from one case to another. The 15 patients seen on the day of onset of the symptoms were in several different hospitals. Since the EEG is sensitive to such variables as acid-base and electrolyte balance, future studies should include precise comparisons of such assays with the EEG pattern at the time. A case in point is that described by Tarlau (1958) as having slow activity in the EEG in association with neurogenic acidosis, the EEG pattern, high blood CO<sub>2</sub> and somnolent state all being improved after the administration of sodium bicarbonate.

Returning to the general significance of the EEG findings in heat stroke, it would appear that the changes in the brain rhythms do not simply reflect raised cerebral temperature, because Hoagland (1936) who studied the effect of fever treatment on the EEG found that the brain activity (alpha rhythms) increased in frequency with increasing temperature between 38.5 and 40°C. (101.3 and 104°F). Ten Cate et al., (1949) raised the temperature of curarized rats, noting an increase in amplitude of brain rhythms at 40 - 41°C, (104 - 105.8°F), a rapid decrease thereafter and disappearance of electrical activity at 44 - 45°C (111.2 - 113°F). Teshan and Gellhorn (1949) directly heated the exposed cerebral cortex of cats and found that the potentials of the electro-corticogram (ECoG) increased in frequency and decreased in amplitude, suggesting excitation, above 45°C (113°F). Although heating to 50°C (122°F) did not abolish cortical electrical activity, the threshold of the motor

cortex /...

cortex to electrical stimulation was raised. The latter finding is apparently incompatible with the tendency to lowered cortical reactivity to photic stimulation encountered in some of the heat stroke patients of the present study.

This feature requires a consideration of the view of Barry and King (1962) that the reticular activating system continues to function in heat stroke. While this may well be the case, the absence of cortical desynchronization in response to photic stimulation in some patients of the present study suggests that the ascending activating system is not exerting its usual effect on cortical function.

With regard to the convulsions often reported to occur in heat stroke patients, particularly on admission to hospital, the contention of Barry and King (1962) that these are possibly coarse tremors rather than epileptic phenomena is supported by the absence of EEG abnormalities suggestive of epilepsy in all but one patient of the present sample. In this connection it is interesting that an attempt to elicit seizures in monkeys by hyperthermia (Schmidt et al., 1956) was not successful.

The EEG changes in 60 normal Bantu male volunteers experimentally exposed to very high temperature and humidity showed only minimal alterations (Nelson 1964). The incidence of EEG abnormality before exposure was 13% (8 cases) and in only one were the EEG disturbances more pronounced at high body temperature. No EEG abnormalities of any kind appear to have been evoked or caused by exposure to high heat and humidity. Basic EEG characteristics (mean alpha frequency, amplitude and index) showed no consistent changes. The EEG picture in these experiments was thus completely different to that found in the heat stroke patients, but rectal temperatures in the experimental study did not exceed 104°F (40°C).

In the heat stroke patients of the present study the EEG responses to activation appear to throw little further light on the problem of brain function in this disorder. Although there appeared to be an unusually high percentage of patients with no response to photic stimulation, this phenomenon was most probably related to diminished cerebral arousal level. On the whole the responses to both photic stimulation and hyperventilation suggest that there is no tendency in heat stroke for a rise in the cerebral excitability level, and certainly no significant reduction of convulsive threshold.

The EEG findings in one of these heat stroke patients (case 10) have implications which are far wider than those related to any specific disorder. This patient had a "flat" or isoelectric EEG for some hours,

subsequently /...

subsequently making a more or less complete clinical recovery with only minor residual EEG abnormalities. In general the total cessation of brain potentials on "electrical silence" is regarded as indicative of severe metabolic disturbance of the central nervous system (Arfel and Fischgold, 1961) often involving widespread cortical damage (Fischgold and Mathis, 1959). Similar conclusions were reached by Harvey and Rasmussen (1951) and Zeman and Youngue (1957), while Tentler et al, (1957) and Percy and Virtue (1959) emphasized that prolonged loss of electrocortical activity during anaesthesia is rarely followed by recovery.

However Fischgold and Mathis (op.cit.) felt that it was difficult to distinguish between electrical "silence" expressing death of the brain and that which comprises an electrical activity too weak to be registered by normal methods.

There is thus no complete agreement on the significance of a 'flat' EEG, although, other data being borne in mind, such a record is often an omen of imminent death (Schwab et al., 1963). A full discussion of this issue is not appropriate in the context of this report and the interested reader is referred to the literature cited.

### 6.3 PROGNOSTIC VALUE OF THE EEG IN HEATSTROKE

In the first few hours after heat stroke is sustained, the incidence and degree of EEG abnormality tend to be marked. In this sample most fatal cases were studied at this time and the majority of those with severe EEG disturbances died subsequently. However two other patients who died later had a moderately abnormal and a normal record respectively. The findings at longer intervals after the onset of the symptoms were also inconsistent.

On the basis of present evidence the only hypothesis that can be put forward is that a severely abnormal EEG soon after heat stroke occurs is suggestive of a poor prognosis. No prediction can yet be made on the basis of moderately abnormal or normal records.

A theoretical comparison with the EEG findings in severe head injury suggests other lines for future research, insofar as abnormalities may persist for many months after such an injury and the more rapidly the record returns to normal the better is the prognosis in general. However there is the occasional paradoxical situation in which the EEG abnormalities following a head injury increase although the clinical state is improving (Kiloh and Osselton, 1966). The opposite, sometimes called "Williams' paradox", is however also encountered when the EEG after a severe head injury returns to normal while neurological or behavioural

disturbances /...

disturbances persist. Such cases have a poor prognosis because of the implication of localized and irreversible destruction of brain tissue. Where this occurs the areas concerned are incapable of generating electrical activity and cannot therefore produce the slow waves or other disturbances that label the EEG as abnormal. Meanwhile the intact parts of the brain may continue to produce a normal pattern, giving the general impression of a normal record as a whole.

Kaada et al. (1961) for example, suggest that normal "waking" EEGs found in comatose patients after brain lesions are indicative of injury to the middle or rostral part of the pons.

If such selective destruction of cerebral tissue takes place in heat stroke, this would at least partially explain the two deaths that occurred in patients with normal records and the one where the EEG was only doubtful. The sequelae in two surviving patients with normal records and one with a doubtful record would also be easier to understand.

Finally the prominence in later EEGs of abnormalities suggestive of subcortical dysfunction may well be due to the known involvement of the cerebellum in heat stroke. Furthermore, abnormalities of this region may not be reflected at all in the EEG because a routine examination will in general only reveal changes in the cortex itself. An instance of this is that even in the case of tumours of the cerebellapontine angle only 50% of EEGs are abnormal when the patient is seen for the first time (Kiloh and Osselton, 1966). In fact EEG changes associated with infratentorial abnormalities are secondary effects of the involvement of the upper brain stem and the cranial thalamic nuclei, whose function is possibly disturbed in heat stroke.

#### 6.4 PERSISTENCE OF EEG ABNORMALITIES IN HEATSTROKE

Of the six patients studied at more than one month after they had sustained heat stroke, only two had normal EEGs. The other four showed records suggestive of subcortical dysfunction, cortical depression or cerebral damage. Although the latter patient (case 18) recovered, one of the others (case 1) died more than two years later, one (case 10) recovered and one survived with mental defect and ataxia (case 15). Cases 5 and 14 had normal records; while the latter recovered, case 5 was left with marked sequelae, possibly being an example of localized cerebral damage with a normal EEG as described in (3) above.

An analysis of the changes in the eight patients with serial records is not revealing: severely abnormal EEGs became normal over different periods in three cases (4, 16 and 21); one recovered, one survived with

sequelae /...

sequelae and one died. In another three cases the record remained severely abnormal (15, 18 and 22), although the period between records was only two days in case 22, who died; case 15 survived with a mental defect and case 18 recovered. Another patient (case 5) who had a doubtful EEG initially showed a normal record eight weeks later. He survived with sequelae. The last of these eight patients (case 10) had five EEGs, showing an improvement after three weeks with subsequent deterioration. He recovered.

The lack of any consistent relationship between the EEG findings and clinical outcome can be regarded as support for the hypothesis that firstly, permanent brain damage such as is accompanied by mental defect may be in the form of localized, and probably cortical, damage; and secondly, that the cerebellar damage responsible for such sequelae as ataxia will not necessarily be reflected in the EEG at all.

## 6.5 GENERAL VALUE OF THE EEG IN HEATSTROKE

6.5.1 Differential diagnosis : The inclusion of the EEG in the procedures applied on admission to hospital has the advantage of providing evidence, with minimal discomfort to the patient, for or against the alternative diagnosis of epilepsy. This was clearly illustrated in case 8 in whom the EEG confirmed the impression gained from the history. It is not known why the patient finally judged to be suffering from hypoglycaemia and not heat stroke should have had two normal EEGs.

6.5.2 Monitoring : Although it is hardly necessary to employ the EEG to determine whether a patient is in coma, the record will often indicate the depth of coma by the overall pattern of electrical activity and responsiveness to stimulation.

Relatively short-term EEG monitoring would provide valuable information on the efficacy of therapy, especially in cases where hypotension is a prominent feature, since the EEG is especially sensitive to deficiencies of cerebral oxygenation.

6.5.3 Follow-up studies : Systematic follow-up EEGs from a larger sample of patients would possibly reveal any association between EEG features and clinical progress.

6.5.4 Psychological assessment : There is little doubt that the study of both the short- and long-term EEG changes in heat stroke would benefit from parallel psychological assessments. Although the contribution of the clinically, especially neuropsychiatrically orientated, examination cannot be overestimated, its augmentation

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by objective psychometric techniques would be of equal value.

In situations where a psychological appraisal of the effects of trauma are necessary, as for example in medico-legal cases, there is very often an absence of precise data concerning the patient's cognitive, conative and affective characteristics prior to the trauma. However in the population of Bantu mineworkers in the South African gold mines there will in the very great majority of cases be at least some control information on some basic aspects of intelligence, in the form of scores on the General Adaptability Battery (G. A. B. ). This is administered to most, if not all, Bantu mineworker recruits for the purpose of preliminary classification.

This test battery, designed to assess certain basic aspects of intelligence in illiterate subjects might provide useful information if administered to cases of heat stroke. Such an approach would be essentially experimental at the outset but might prove valuable if data from an adequate sample of heat stroke cases and control subjects were collected.

## 7. CONCLUSIONS

Although the sample studied was small, the following tentative conclusions can be drawn :

1. There is a high incidence of EEG abnormality in heatstroke.
2. Severity of EEG abnormality tends to be maximal during the first few hours following the onset of the symptoms.
3. Soon after heatstroke the EEG abnormalities are mostly suggestive of diffuse cerebral dysfunction and/or subcortical abnormality, whilst the majority of abnormal records obtained at later stages are suggestive of subcortical abnormality.
4. Whilst there is no consistent relation between severity of EEG abnormality and prognosis, cases which terminate fatally tend to have severely abnormal records soon after sustaining heat stroke.
5. EEG abnormalities in heat stroke show no consistent trend with time; in some cases this may be due to the paradoxically normal appearance of the EEG in the presence of localized cerebral destruction.

6. /...

6. At this stage the main value of the EEG in heat stroke lies 1) in the provision of evidence in relation to the differential diagnosis of heat stroke and epilepsy; 2) in its value as a monitoring technique in, for example, hypotension, and 3) in follow-up studies for the assessment of brain function.
7. For the assessment of changes in specific aspects of mental ability the General Adaptability Battery might be of value in that it could be administered to surviving heat stroke patients, provided they were conscious, and the results be compared with their pre-heat stroke performance.

## 8. RECOMMENDATIONS

1. It is recommended that the EEG be included in the routine assessment of heat stroke patients.
2. It is further recommended that follow-up studies of such patients be carried out by means of the EEG, supplemented by tests of a cognitive kind, specifically the G. A. B. because pre-heat stroke data should, in theory, be available for comparison in the vast majority of cases.
3. Animal studies of brain function as measured by the EEG would probably supply valuable information on the relative significance of cortical and subcortical changes following heat stroke.

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APPENDIX

DETAILS OF CASES STUDIED

FINAL DIAGNOSIS	CASE. NO.	COMPANY NO.	MINE
HEAT STROKE	1	8013	Rand Leases
	4	3147	Pres. Brandt
	5	9867	Harties
	6	H127892	City Deep
	7	2332	F. S. Saaiplaas
	9	16395	Stilfontein
	10	1801	F. S. Saaiplaas
	11	48305	Durban Deep
	12	2908	Brakpan
	13	1010	Robinson Deep
	14	34436	Pres. Brand
	15	2641	Robinson Deep
	16	7915	Rand Leases
	17	62623	Vaal Reefs
	18	3284	Rand Leases
	19	14258	Grootvlei
	20	1450	New Kleinfontein
	21	9649	East Dagga
	22	3530	Robinson Deep
	25	--	Western Deep
	26	220787	ERPM

HEAT EXHAUSTION /...

DETAILS OF CASES STUDIED (Continued)

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FINAL DIAGNOSIS	CASE NO.	COMPANY NO.	MINE
HEAT EXHAUSTION	2	5927	Harties
	3	5861	Harties
HYPOGLYCAEMIA	23	321	Robinson Deep
EPILEPSY	8	5065	V' struis

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W N N R  
C S I R