

Electroencephalogram during and after an Acute Attack of Asthma in Children

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EEGs were recorded in 12 children who were admitted to hospital because of an asthmatic attack. One EEG was recorded during the attack, one immediately after, and a third EEG several weeks later. The severity of the attack was classified as moderate in eight children, severe in three and very severe in one child. There was a corresponding change in the blood gases, but other laboratory values (electrolytes, glucose, etc.) were normal. Contrary to previous findings, no change was seen in the three successive EEG recordings which had been related to the attacks. It is therefore concluded that an asthmatic attack as such does not give rise to changes in the EEG, and a complicating factor should be suspected if such changes are seen.

Key words: anoxia; asthma; electroencephalography; water-electrolyte balance.

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The occurrence of an anoxic brain injury after an acute attack of asthma is very rare in children (3). At times, however, this possibility must be considered in the differential diagnosis after a very severe attack. An electroencephalogram is one of the first investigations likely to be performed in such circumstances.

It is, however, not known how much the EEG might be altered during and after an uncomplicated asthmatic attack. Several factors, such as hypoxia, disturbances in the fluid and electrolyte balance and perhaps the allergic mechanism itself (7, 9, 10) might give rise to abnormalities in the EEG. Consequently, it is difficult to estimate when the abnormality in the EEG is secondary to the asthmatic attack and when it should be regarded as an indication of some complicating factor.

We therefore report the results of EEG recordings during and after an acute attack of asthma in 12 children.

MATERIAL AND METHODS

Twelve children, six boys and six girls, age range 4–13 years, were investigated. Two of them had had no previous attacks of asthma, while four children had had 1–3, two 4–10 and four more than 10 previous attacks. None had neurological symptoms. Informed consent for the study was obtained from the parents in each case.

Five children had definite allergic asthma and in other five the allergic aetiology was probable. In two children there was no positive evidence of allergy. This conclusion was based

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on seasonal occurrence of symptoms, positive skin tests and elevated eosinophil count or IgE level.

In all 12 children the severity of the attack indicated admission to hospital. The duration of the attack prior to the first EEG was 0–1 days in six children, 2–6 days in four and more than 6 days in two children. For analysis, the severity was further classified:

- I) Clear bronchial obstruction, with prolonged expirium and some dyspnoea (8 cases).
- II) Severe obstruction, accessory muscles in use, possibly some cyanosis (3 cases).
- III) Assisted ventilation necessary (1 case).

The attacks were treated according to conventional principles. The EEGs were recorded with 16-channel Siemens-Elema or 8-channel Siemens-Elema type Junior apparatus using silver-silver chloride cup electrodes held in place with rubber mesh. Four of the 37 EEGs were recorded in the intensive care unit.

In each child three EEGs were recorded: the first as soon as possible after admission (within a few hours), the second 4–7 days later and the third more than 3 weeks later. In the most severe case (Fig. 2) four EEGs were recorded.

For this study the EEGs were interpreted visually without any knowledge of the sequence of the records or the clinical situation. At the time of the first and second EEG a blood sample was taken for analysis of the arterial blood gases, electrolytes, glucose, creatinine and transaminase values.

RESULTS

The results are summarised in Fig. 1 which represents two important parameters (the frequency of occipital rhythmic activity and background activity) in the three successive EEGs in the 12 children.

The frequency and amount of the occipital rhythmic activity (alpha rhythm) did not change appreciably in the three successive EEGs recorded in each child. There were no changes in this activity, which had been clearly related to the attack.

In eight cases the occipital rhythmic activity was well formed and appropriate for age in all three EEGs. In three cases it was slightly slow for age, and in one case (4-year-old girl) there was no definite rhythmic activity in the posterior regions. These differences did not show any clear relationship to the severity of the attacks or the allergic disposition of each particular child.

The amount of generalised slow or intermediate slow activity in the three EEGs was considered to be excessive in three cases, moderately increased in three cases and within the normal range in six cases. Neither the amount nor the variation of the slow activity in successive records showed any relationship to the attacks. There was, however, depression of the amplitude of the EEG during assisted ventilation in the most severe case (Fig. 2). This represents an extreme situation where the treatment included sedation. The three successive EEGs in Fig. 3 illustrate a more typical course.

There was no local abnormality in any of the EEGs.

In one case (5-year-old-girl who had never had epileptic seizures) there were definite paroxysmal features in the form of numerous generalised, irregular bursts of spikes and slow waves in all three EEGs. This abnormal activity was much more obvious during the attack than after it.

The relationship of the EEG features to the number of previous attacks of asthma and to the length of the present attack is presented in Table 1. There was no correlation between these parameters.

The mean ($\pm$ SD) PaO_2 during the first EEG was 77.8 ± 13.7 mmHg ($n = 10$) and during the second EEG 92.8 ± 14.7 mmHg ($n = 8$). In the six cases where the puncture was successful both times, the first PaO_2 was always lower. Other laboratory values remained well within normal limits. The boy in Fig. 2 had the lowest PaO_2 , 55 mmHg.

DISCUSSION

There are only a few reports of EEG recordings during an asthmatic attack in children.

Table 1

Number of children having various EEG features, and their relationship to the number of previous attacks of asthma and to the duration of the present attack before the first EEG

		Alpha-rhythm			Slow activity			
		Normal	Slightly slow	None	Normal	Moderately increased	Excessive	
Previous asthma attacks	0	2	—	—	2	—	—	2
	1-3	3	1	—	2	1	1	4
	4-10	1	—	1	1	—	1	2
	> 10	2	2	—	1	2	1	4
Duration of attack before first EEG (days)	0-1	4	1	1	3	1	2	6
	2-6	3	1	—	3	—	1	4
	> 6	1	1	—	—	2	—	2
		8	3	1	6	3	3	12

Severity grade	III	II	II	II	I	I	I	I	I	I	I	I
Age (years)	4	4	7	13	4	4	5	5	9	9	11	11
Male / Female	M	M	F	F	F	F	M	F	M	F	M	M
Etiology A/P/N	N	P	A	A	A	A	P	P	A	P	P	N
frequency of alpha rhythm c/sec					no							
frequency of background activity c/sec												

Fig. 1. The severity grade and probable aetiology (allergic, A; probably allergic, P; no evidence of allergy, N) of the asthma attacks, and the age and sex of the 12 children are plotted against two selected parameters of the three successive EEGs recorded in each case. The frequency range of alpha rhythm and background activity of the EEGs is indicated by vertical lines.

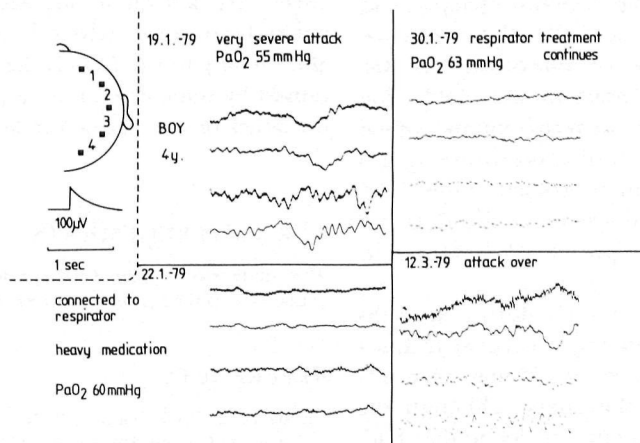


Fig. 2. Four successive EEGs during and after the most severe attack in this material. Assisted ventilation was necessary for over 1 week. The EEG is unaltered at the beginning of the attack, despite the low PaO_2 . During assisted ventilation there is clear amplitude depression and some increase in intermediate slow activity.

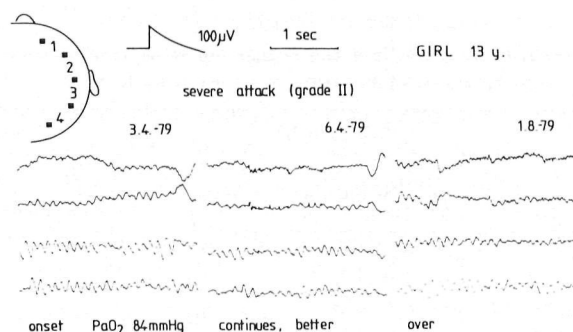


Fig. 3. Three successive EEGs during and after a severe asthmatic attack. The bronchial obstruction was not severe enough to lead to significant hypoxia. The EEG is not influenced by the attack.

Usually a generalised slowing has been recorded during the attack (6, 7). This abnormality resolved after the attack. The observation that asthmatic children often have abnormal EEGs also between the attacks (2), has not been confirmed in later recordings (8).

No increase of generalised slow activity was observed in the present study during the asthmatic attack, and in this respect the results do not agree with the earlier studies (6, 7). The most likely explanation for this difference seems to be the advent of intensive care treatment and better maintenance of electrolyte and fluid balance.

The paroxysmal activity in the EEG of one child remains unexplained. The literature concerning the relationship between epilepsy and asthma is extensive (4, 5, 8), but the results are controversial. The use of anticonvulsants was not considered appropriate in this child. The problem resembles that of recurrent abdominal pain (1) where the effect of anticonvulsants also has not been tested in controlled studies. An increase of paroxysmal activity in the EEG has been reported during periods of frequent attacks of asthma (4).

The recording of an EEG during an asthmatic attack was not an easy procedure technically. On the other hand, it did not adversely affect the course of the attack, although the recording was an additional burden to the child. For this reason, we had to restrict the material to a minimum. Similarly, the recording of an EEG during an asthmatic attack can be recom-

mended only in selected situations, not as a routine monitoring procedure, at least if the routine, rather cumbersome apparatus is used.

Despite the severity of the clinical situation in a few cases in this material, no complications were suspected clinically. In this respect the recording of the EEG only confirmed the clinical impression. It seems possible, however, by EEG, to differentiate whether or not an attack of asthma or its treatment has affected the brain function.

The results of the present study indicate that an attack of asthma as such does not give rise to EEG abnormalities. This finding is important, because the usual complications of an attack and its treatment, hypoxia and electrolyte disturbances, are known to increase the amount of generalised slow activity in the EEG (9). An abnormality in the EEG is therefore likely to be caused by some complicating factor, and not by the attack or the mechanism leading to it.

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