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'Eating epilepsy' — a reappraisal

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One hundred and fifty patients with eating seizures were detected over a 9 year period in two hospital clinics in Sri Lanka. The clinical and EEG features of 120 of them are compared to a control group of 120 patients with epilepsy. Patients with eating seizures showed a male predominance of 3:1. In more than 50% the onset of epilepsy was in the 2nd decade of life. A family history of epilepsy was obtained in 28.3% and 21 siblings themselves had eating seizures. The seizure type was simple or complex partial, secondarily generalised seizures were common. The EEG in 71.6% showed spikes, sharp/slow waves, focal in the temporal areas. The response to medication of eating seizures was similar to that of controls. Clobazam used in 17 patients as monotherapy or adjuvant therapy proved useful. The very high prevalence of eating epilepsy in the present series could pathogenically be related to genetic or ethnic factors and to the bulky meals rich in carbohydrates consumed by the patients.

INTRODUCTION

Charles Symonds[23] in his Hughlings Jackson Lecture, 'Excitation and inhibition in epilepsy' in 1959, suggested that precipitation of epileptic attacks by visceral stimuli was more common than generally appreciated. He cited the example of a man who suffered seizures during the day; these took the form of sudden confusion and a sense of familiarity which lasted 2 or 3 min while eating a heavy meal. A comparable case had been recorded by Allen[4] in 1945. In 1954, Boudouresques and Gastaut[5] reported on 4 patients with temporal lobe EEG foci who had postprandial seizures attributed to gastric distension. Subsequently, more case reports appeared from different parts of the world[1–3,7–10,12–21,24] and the condition came to be known as 'Eating epilepsy'. Of a total of 76 cases, 33 were from India.

The Epilepsy Clinic and the Neurology Clinic at Peradeniya register 150–200 new cases of epilepsy every year. During the past 9 years, we identified 150 patients from these 2 clinics whose seizures were related to a meal, either exclusively or in the majority of instances. In this paper we analyse the clinical and EEG features of these patients in comparison with a control group of epilepsy patients drawn from the same clinics. We also review the reported cases in the light of our experience.

PATIENTS AND METHODS

Patients who had more than 50% of the fits during or within 30 min of eating breakfast, lunch or

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dinner were considered to be suffering from eating epilepsy. The frequency of eating seizures was obtained from the history and, wherever possible, verified using fit-charts. The patients were interviewed personally using a standard questionnaire. A parent or an elderly relative and an eye-witness were also interviewed for information regarding birth, development, childhood illnesses and the seizures. Information was verified with clinic notes, especially with regard to medication and response to treatment. Previous EEGs were traced and reanalysed. New recordings were made where necessary. EEGs were recorded on a 10-channel recorder according to the 10–20 system. Routine recording included hyperventilation for 2–3 min and photic stimulation.

Of the 150 cases of eating epilepsy on record, complete and reliable data and EEG tracings could be obtained for 120 patients. These 120 cases are referred to as the 'test group' in subsequent analyses. From the same clinics, 120 cases of epilepsy matched with the test group for age and sex were randomly selected to serve as controls.

RESULTS

Forty of the 120 patients in the test group suffered only eating seizures. 23 of them had experienced more than 10 seizures each. The other 80 patients also had random seizures. 70 of them had experienced more than 10 seizures (eating or random) each and the remaining 10 patients a minimum of 6 seizures each. The age range of the patients was 13–43 years (mean 23.7 ± 6.1). The male:female ratio was 3:1. In the 40 patients who had exclusively eating seizures, the ratio was 6:1. 12 (10.0%) patients of the test group were married. The pattern of employment of the test and the control groups was similar. 117 (97.5%) patients of the test group were Sinhalese, 2 were Muslims and 1 was a Tamil.

The onset of epilepsy in 96 patients (80%) of the test group was in the 2nd decade of life. The age at onset of epilepsy for the control group was significantly different ($P = 0.0016$), the onset being in the 2nd decade in only 69 (57.5%). Factors of aetiological significance were similar in the two groups. However, there was a significantly higher incidence ($P = 0.0095$) of left-hand dominance in the test group (18 patients – 15%) compared to the control group (6 patients – 5%). The prevalence of epilepsy among male siblings was significantly higher ($P = 0.0009$) in the test group (20 patients – 16.7%) than in the control group (4 patients – 3.3%). The prevalence among other family members was similar in the two groups and the figures for the test group were: parents – 4 (3.3%), sisters – 9 (7.5%), others – 9. 13 of the male siblings and 8 of the female siblings were themselves patients with eating epilepsy belonging to 9 different families.

The type of epilepsy and the predominant EEG features are presented in Table I. In these two respects, the differences between the test group and the control group were highly significant. The common symptomatology of the partial complex

TABLE I

Type of epilepsy/seizures and predominant EEG features

	Test group (n = 120)	*Control group (n = 120)*
*(Ia) Type of epilepsy/seizures**		
Partial epilepsy	120 (100.0)	76 (63.3)***
Partial complex	5 (4.2)	10 (8.3)
Secondarily generalised		
Partial simple	20 (16.7)	18 (15.0)
Partial complex	91 (75.8)	38 (31.7)***
Undetermined symptomatology	4 (3.3)	10 (8.3)§
Generalised epilepsy	0	39 (32.5)
Unclassified	0	5 (4.2)
*(Ib) Predominant EEG features***		
Spikes, sharp/slow waves focal in		
Right temporal area	64 (53.3)	26 (21.7)***
Left temporal area	10 (8.3)	11 (9.2)
Both temporal areas	12 (10.0)	12 (10.0)
Generalised spikes, sharp/slow waves	3 (2.5)	21 (17.5)***
Focal and generalised abnormalities	8 (6.7)	0
Nonspecific abnormalities	19 (15.8)	16 (13.3)
Normal	4 (3.3)	34 (28.3)***

* Overall χ^2 test of association, $\chi^2 = 53.878$, $P < 0.0001$.
** Overall χ^2 test of association, $\chi^2 = 61.533$, $P < 0.0001$.
*** Difference significant at $P < 0.001$.
§ Difference significant at $P < 0.05$.
Figures in parentheses are percentages.

seizures experienced by the test group was: epigastric sensations – 12 (10.0%), gustatory/olfactory hallucinations – 10 (8.3%), fear – 39 (32.5%), dreamy states – 39, abnormal speech – 44 (36.7%), automatisms of face and mouth – 46 (38.3%) and automatisms of limbs – 20 (16.7%). The symptomatology was similar in the two groups except for epigastric sensations which showed a significantly higher incidence ($P < 0.01$) in the control group (35, 29.2%).

The relationship of eating seizures to the 3 main meals is shown in Table II. 106 (88.3%) patients had the seizures while eating, usually within 5 min of starting the meal. 47 (39.2%) had seizures immediately after the meal. 38 (31.7%) also, or only, had seizures during the postprandial period; these occurred within 30 min, but usually during the first 10 min. In 66 (55%) patients, the eating seizures occurred only in relation to rice meals, and in 3 (2.5%) only when eating meat or fish. The factors considered important by the patients in the causation of their seizures, together with the number of the patients who gave a positive response, were: eating late – 26 (21.7%), eating too much – 12 (19%), eating fast – 5 (4.2%), food with excess chillies – 6 (5%), oily food – 5 (4.2%), hot food – 4 (3.3%), cold food – 2 (1.7%). 16 (13.3%) patients had eating seizures only when eating alone, and 45 (37.5%) only when eating with others. 61 (50.8%) patients had eating seizures only when eating at home and 1 (0.8%) only when eating out. Only 3 (2.5%) patients avoided a particular food item (meat or fish) because of the fear of fits.

The number of antiepileptic drugs taken by the test group at the time of the survey was: 1 drug by 72 (60%) patients, 2 by 38 (31.7%), 3 by 3 (2.5%), no drugs by 7 (5.8%). The antiepileptic drugs used were: phenytoin in 75 (62.5%) patients, carbamazepine in 43 (35.8%), clobazam in 17 (14.2%), phenobarbitone in 16 (13.3%), primidone in 4 (3.3%), valproate in 1 (0.7%). The result of medication as expressed by the patients and verified with the clinic records was: free of fits – 44 (36.7%) patients, improved – 59 (49.2%), same – 9 (7.5%), worse – 3 (2.5%), unspecified – 5 (4.2%). The treatments received by the test group and the control group were significantly different but there was no significant difference in the response. Of the 17 patients treated with clobazam as monotherapy or adjuvant therapy, 2 were free of fits, 13 improved and 2 remained the same.

Of the 80 patients who suffered both random and eating seizures, the ratio between eating seizures and random seizures changed in 22 (27.5%) during the course of the illness. Some patients who initially had random seizures subsequently developed eating seizures and ended up with eating epilepsy. On the other hand, some who had mainly or sometimes exclusively eating seizures later began to develop mainly random seizures.

DISCUSSION

Definition

Eating seizures, for the purpose of this study, were defined as fits occurring during or within 0.5 h of eating a meal. Postprandial seizures were included because of the observation that a proportion of the patients who had seizures during eating also had postprandial seizures. Some patients who initially had seizures during eating subsequently began to have fits only in the postprandial state and vice versa. The time limit of 0.5 h for the postprandial period was arbitrary. In the majority, the seizures occurred soon after or within 10 min of a meal.

TABLE II

Relation of the eating seizures to the main meals

	Lunch only	*Dinner only*	*B'fast only*	*Most at lunch*	*Most at dinner*	*Most at b'fast*	*Not specific*
Number of patients (n = 120)	14	14	3	29	45	10	5
(%)	(11.7)	(11.7)	(2.5)	(24.2)	(37.5)	(8.3)	(4.2)

A patient takes about 15 min to eat an eastern meal. Even if one assumes that a meal lasts 30 min, together with the 30 min postprandial period, the total duration of meal-related behaviour in relation to the 3 meals is 3 h/day. The probability of a seizure occurring by chance during meal-related behaviour is therefore 3/24 = 0.125. The probability of a patient having 2 seizures, both related to meals, is then 0.125 × 0.125 = 0.0156, much less than the conventionally accepted criterion of 0.05 below which it is unreasonable to assume chance occurrence. 60 of our patients had mixed seizures, but each patient had a minimum of 6 seizures out of which at least 3 were eating-seizures. Applying the binominal test[22], given that the chance probability of each seizure being meal-related is 0.125, the probability that 3 or more of the seizures will be meal-related is 0.042, again below the conventional criterion. Thus, in the present series, the relationship between seizures and eating could not have occurred by chance alone.

Prevalence

Eating epilepsy, judging by the previous reports, is a rare disorder. Vizioli[24] from 20,000 examinations in the Laboratory of Electroencephalography of the Rome University, found only 9 cases whose seizures were related to eating. Nagaraja and Chand[15] found 13 cases amongst a total of 11,783 patients with epilepsy who attended the National Institute of Mental Health and Neurosciences in Bangalore – a prevalence of 1.1/1000 epilepsy patients. The prevalence at our centre is about 100 times the Bangalore figure. Even if one only considers the 40 patients who had exclusively eating seizures, the prevalence would still be very high, about 25/1000 epilepsy patients. Our awareness and interest are insufficient in themselves to explain this high prevalence. Could it be that there are host characteristics, ethnic and/or genetic, which predispose our patients to develop seizures in relation to eating? Or is there any peculiarity in their diet, physical or chemical, which is epileptogenic? Eating epilepsy with its high prevalence in the Kandy District of Sri Lanka is reminiscent of 'hot water epilepsy' which shows a geographical predilection to the Deccan plateau of the Karnataka State in South India[14].

Patient characteristics

The male preponderance and the frequent onset in the 2nd decade as in our series had also been noted previously[15]. An aetiologically related factor was only evident in a small percentage of cases, just as in the literature[19]. There was a high incidence of epilepsy among siblings of patients with eating epilepsy. Nagaraja and Chand[15] also found a high incidence (50%) of epilepsy among family members in their series. It is interesting that in our study 21 siblings themselves had eating epilepsy. This familial occurrence of eating epilepsy has not been reported before.

Among the previously reported cases, Aguglia and Tinuper[2] recognised 3 main types of seizure: (a) complex partial seizures: 1 of their cases, 4 cases described by Boudouresques and Gastaut[5], 7 by Vizioli[24], 1 by Forster[10] and 5 by Chemburkar and Desai[7]; (b) simple partial seizures: 1 case by Abenson[1], 1 by Robertson and Fariello[19] and 1 by Reder and Wright[17]; and (c) generalized myoclonic and/or atonic seizures: 2 of their cases and 1 by Cirignotta et al.[8]. Nagaraja and Chand[15] recorded temporal lobe epilepsy in 12 of their 13 cases. These reports confirm that partial epilepsy is the dominant type of epilepsy, and that partial complex seizures with or without secondary generalisation are the commonest seizure type. EEG in our series accordingly showed epileptiform abnormalities maximally in the temporal areas. The symptomatology of the partial complex seizures was not specific to eating epilepsy.

Relation to eating

In the majority of our patients, eating seizures accounted for more than 90% of all seizures. In the individual patient eating seizures may be replaced by random seizures and vice versa as described earlier[3,8]. In the majority (88.3%) of the patients, the seizures occurred while eating. Some patients who initially had postprandial seizures subsequently had seizures while eating, and vice versa. 21 (17.5%) patients had occasional seizures while getting ready for a meal even before touching food as described earlier[20].

Food and eating habits

In Sri Lanka the staple diet is rice, and the two

main meals, lunch and dinner, usually consist of rice served with several curries. Occasionally bread or a preparation made of rice flour or wheat flour may replace rice. The breakfast is usually a light meal consisting of bread or any other preparation made of flour served with a curry. Some people may also have rice for breakfast. Most of the patients who had seizures at breakfast were those who ate rice for breakfast. The predilection of the eating seizures for a particular meal had already been noted, e.g., breakfast[8] or lunch[2]. 66 (55%) of our patients thought that their eating seizures occurred only when they ate their staple diet, i.e., rice meals. Only 3 patients had noted that their seizures occurred only when they ate meat or fish. The relevance of the type of food is uncertain[2,8] considering an unusual case who developed fits only when eating an apple[1]. In the Bangalore series[15], 8 patients were strict vegetarians while the other 5 ate both vegetarian and non-vegetarian food. Factors such as eating late or eating too much were not considered important[23]. But Forster[10] in his experiments was able to eliminate the texture and temperature of food, time of eating, and the taste as specific or aggregate evoking factors. Nagaraja and Chand[15] thought that in 12 of their 13 cases, the seizures were brought on only by a specific pattern of eating a conventional Indian meal, which involved mixing and eating with the right hand a large bulk of rice with curries and vegetables.

Pathogenic mechanisms

Attempts to find a specific stimulus or a seizure-triggering mechanism have either been unsuccessful[10,15] or have produced variable results[2,8,20] including mastication and swallowing[20], passage of food along the oesophagus[12], gastric distension[5], richness of the meal[24] and a chemical substance[1], lifting food with the fork or cutting food[17]. The last case[17] subsequently lost the second, third and fourth digits of his right hand in an accident and ceased to have seizures. Our observations support the view that multiple stimuli may play a role in the pathogenesis of eating seizures. Several sites within the central nervous system have been suggested as the area responsible for initiating eating seizures. These include the diencephalon[19], hypothalamic areas[21] and the amygdala[3,9]. It is possible that during eating the amygdala becomes the site of additional stimulation, which may lower the seizure threshold and lead to rapid generalisation of seizures.

Treatment and prognosis

Eating epilepsy is considered difficult to treat[3,8,10,15]. Our experience was rather different; 85.9% of our patients were either free of fits or at least satisfactorily controlled. Many received clobazam[2,6,11] which shows promise in the treatment of eating-related seizures.

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