J. Hyg., Camb. (1969), **67**, 147
Printed in Great Britain

An outbreak of 'winter vomiting disease' in a university hall of residence

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(*Received* 1 *August* 1968)

Where feeding is communal, epidemics of vomiting suggest food poisoning. It is usually possible to discover the food and the organism responsible, but in some cases it is not and then the episode tends to get the restrospective label of 'Winter Vomiting Disease'. This disease is thought by many to be due to an airborne virus which acts via the central nervous system; on clinical grounds it has been related to 'Epidemic Vertigo' and 'Epidemic Collapse', for which viral agents have also been postulated (Editorial, 1965). As at least some instances of 'Epidemic Collapse' are really hysterical, the possibility that an hysterical mechanism is involved in 'Winter Vomiting Disease' is worth investigation. An outbreak at Reading provided an opportunity to test this hypothesis.

The epidemic occurred at a Reading University Hall of Residence for Women Students. At the time there were 165 students present. The first two cases vomited at 4.15 p.m. and 5.30 p.m. on 31 January 1967 (day 1). By 8 p.m. 11 girls had been sick and the number rose steadily as the night progressed. At midday on day 2 the figure was 74 of whom two had required admission to the University Health Centre. During the early afternoon of day 2 there were no new cases but that evening and on the night of day 2/day 3 there were a further 12 cases. Five more occurred before the end of the epidemic on day 5.

The late afternoon start of the epidemic suggested that if food poisoning was the cause, lunch was the suspect meal. However, it was soon discovered that quite a number of the affected girls had not eaten lunch in Hall on day 1. A rather smaller number had not eaten dinner and three girls had not eaten either meal.

The outbreak was investigated by the Public Health and Welfare Department of Reading County Borough at the request of one of us (J.D.C.). No pathogens were isolated from specimens of food obtained from the Hall kitchen. No pathogens were seen on direct microscopy of stool samples taken from six of the students most severely affected and none were obtained by either aerobic or anaerobic culture.

From an operational point of view the negative results of the pathological investigations and the failure to obtain a correlation between Hall meals and vomiting put the epidemic in the category of 'Winter Vomiting Disease'. The hypothesis of an hysterical factor in the spread of the vomiting seemed particularly

worthy of investigation in this instance because the two girls admitted to the Health Centre had been in a state of tetany due to overbreathing. Also, there was a strong impression that the cases were geographically clustered within the Hall. Early reports indicated that along some corridors nearly all the girls had been affected whereas in other parts of the Hall and in some of the annexes there had been no cases at all. Such a response by social cells would, if substantiated, fit well with mass hysteria.

It was decided to test the hypothesis of epidemic hysteria by establishing the answers to the following questions:

(1) Did the affected girls have a higher score in neuroticism (N) on the Eysenck Personality Inventory than the unaffected?

(2) Did the affected have a higher frequency of attendance at the University Health Centre prior to the epidemic?

(3) Was there, in fact, a clear geographical clustering of cases?

METHOD

Within 2 weeks of the epidemic questionnaires were given to all the girls, both affected and unaffected, to establish if, when and to what extent they were affected and which Hall meals they had eaten on day 1. At the same time the girls were asked to complete an Eysenck Personality Inventory.

The records of attendance at the University Health Centre and a college room list were used to obtain the answers to questions 2 and 3.

All 165 residents who were given questionnaires completed them.

Table 1. *The distribution of cases by social cells*

Cell	Vomited	Nauseated	Unaffected	% vomited
1. Garden Annexe	21	8	5	60
2. 'Twelve'	5	3	3	45
3. South annexe	6	1	6	50
4. Redlands	7	2	4	55
5. Lydford	7	2	2	65
6. Woodville	9	2	6	55
7. Brierly	8	1	2	70
8. House	4	4	2	40
9*a*. Top corridor	11	0	5	70
9*b*. Middle corridor	8	4	6	45
9*c*. Bottom corridor	5	1	5	45
Total	91	28	46	Av. 55%

RESULTS

The N scores of the vomiters and non-vomiters are not significantly different. Ninety-one vomiters gave a mean N score of 12·2, and 74 non-vomiters a mean N score of 12·4.

The number of visits to the Health Centre was roughly the same for the two groups: 91 vomiters made 132 visits, or 1·45 visits per head, and 74 non-vomiters made 120 visits, or 1·67 visits per head. The records of 600 women randomly

selected from the whole female undergraduate population yielded a similar figure, the number of visits being 913, or 1·52 per head.

The geographical analysis (Table 1) also gave negative results.

These results effectively dispose of the idea that hysteria played a significant part in the spread of this epidemic of vomiting. Any over-emotional behaviour was a consequence of the epidemic and not a contributory cause.

There is some evidence that the small group of those who felt sick but did not vomit (and who are classed as non-vomiters in the analysis above) did satisfy the first two of the three criteria suggested for an hysterical reaction. Splitting the non-vomiting population into nauseated and unaffected groups we get:

(1)	Nauseated (28)	Mean N score 12·9
	Unaffected (46)	Mean N score 12·0
(2)	Nauseated (28)	66 visits to the Health Centre, i.e. 2·36 a head
	Unaffected (46)	54 visits to the Health Centre, i.e. 1·17 a head

This small element may therefore have contained a proportion of girls whose reaction was a neurotic one suggested by the illness around them. As this would merely be a fringe effect one would not expect it to meet the criterion of geographical concentration.

For the vomiters, who constituted the epidemic proper, we are forced back to an organic explanation and in particular to food poisoning. For this there is positive evidence because the analysis of the questionnaires established a definite relationship between vomiting and eating meals on day 1. Taking the actual times of vomiting given by the group of 71 who ate lunch and/or dinner on day 1 and vomited before midday on day 2, 8 women gave 8 p.m. on day 1 as the time they vomited, 7 women gave 2 a.m. on day 2, while the three next most frequently nominated times had only 5, 4 and 3 nominations. Lunch was served at 1 p.m., dinner at 7 p.m.; the intervals between the first meal and the first peak and between the second meal and the second peak are the same (7 hr.). Plotting out the vomiting times for the 71 cases shows that the distribution is readily analysable as two overlapping responses, the information as to meals eaten being compatible with the time of vomiting in the individual case (Fig. 1). The analysis into two curves is based on the following:

(1) That as no case occurred within 2 hr. of lunch, so no case due to dinner-time poisoning would manifest before 9 p.m. Therefore, all cases prior to 9 p.m. were due to poisoning at lunch.

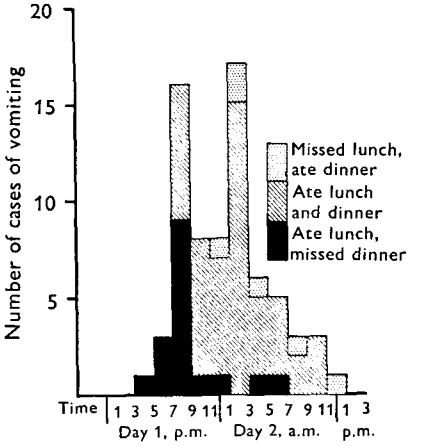
(2) That the causative agent was the same on both occasions and, as the peaks of the two responses were of the same height, the two response curves must have been identical.

(3) That decay from the peak incidence was exponential, the number of new cases halving in every 2 hr. period.

An additional point in favour of a connexion between eating in Hall and vomiting is the higher proportion of girls missing lunch on day 1, among those who

did not vomit (25 out of 91 = 28%) as compared to those who did (9 out of 74 = 12%).

There remain the three cases who did not eat either lunch or dinner on day 1, yet vomited before midday on day 2, and the 17 cases who vomited between late afternoon on day 2 and the evening of day 5. One explanation of these and of the epidemic as a whole would be that there was contamination of the food: (*a*) to a very mild degree at breakfast on day 1, causing 3 cases; (*b*) to a moderate degree at lunch on day 1, causing 35/36 cases; (*c*) to a moderate degree at dinner on day 1, causing 35/36 cases; (*d*) to a moderate degree at lunch on day 2, causing 12 cases; (*e*) to a mild degree on day 3, causing 3 cases; (*f*) to a very mild degree on day 4 and 5, causing 2 cases.



Number of cases	1	3	16	8	8	17	6	5	3	3	1	0	
Analysed as:													
Lunchtime cases	1	3	16	8	4	2	1	1	.	.	.	.	
Dinnertime cases	.	.	.	1	3	16	8	4	2	1	1	.	
Theoretical total	1	3	16	9	7	18	9	5	2	1	1	0	

Fig. 1. Time course of the epidemic. Number of women vomiting and meals eaten.

An alternative view would be to regard the mode of spread as 80% via food but 20% by contact. The later cases could then be put down to this second form of transmission which would presumably involve a smaller innoculum and a longer incubation period.

DISCUSSION

As all three of the predictions made from the hypothesis of hysterical spread turned out to be incorrect, this hypothesis can be regarded as disproved. Neurotic mechanisms may have played a part (together with subemetic poisoning) among the small group of those who felt sick but did not vomit, but 'Winter Vomiting Disease' is certainly an organic entity.

The hypothesis generally favoured is indeed an organic one—an airborne virus producing its effects via the central nervous system (Miller & Raven, 1936; Gray,

1939; Bradley, 1943; Haworth, Tyrrell & Whitehead, 1956). Food poisoning, it is admitted, cannot be excluded but no one seems to regard it with enthusiasm. The positive results of this study support food poisoning as the mechanism and suggest that it is unrealistic to demand that contamination be limited to a single meal before invoking spread via food. Our postulated agent is, of course, as mysterious as the alternative 'respiratory virus', but we would claim that the portal of entry suggested is inherently more likely in view of the rapid response of the majority of cases and the symptoms of vomiting and, to a less prominent extent, diarrhoea.

The above is written on the somewhat shaky presumption that 'Winter Vomiting' is a true disease entity. As the diagnosis is made on negative evidence it is more than possible that different agents are at work in different outbreaks. The episode described by Haworth *et al.* (1956), with a low attack rate and slow time course (18 cases in a month), certainly fits more readily with a viral theory than the 'explosive' type of epidemic described here. However, as the explosive category appears to include the majority of outbreaks reported in this country the observations in this paper are probably applicable to the syndrome of 'Winter Vomiting Disease' as currently recognized.

SUMMARY

1. An outbreak of 'Winter Vomiting Disease' is described involving women at a University Hall of Residence. Of the 165 women exposed, 74 vomited in the first 24 hr., and 17 more over the next 4 days.

2. Public health investigations were negative.

3. On the hypothesis that the vomiting was at least partly hysterical it was predicted that the affected would have higher N scores than the unaffected on the Eysenck Personality Inventory; that they would have a higher frequency of past attendance at the University Health Centre; and that they would be non-randomly distributed through the Hall and its annexes. All these predictions proved to be incorrect.

4. There is some slight but consistent evidence to suggest that there is a neurotic component in the small group of 28 who felt nauseated but did not vomit.

5. The histogram of time of vomiting has two peaks. It is shown that 71 out of the first 74 cases can be accounted for on the hypothesis of contamination of the food at both lunch and dinner on the first day of the outbreak, with the mean response coming 7 hr. after eating.

6. It is suggested that these results make a food-borne agent a more likely explanation of 'Winter Vomiting Disease' than the currently favoured airborne virus acting via the central nervous system.

The costs involved in the psychiatric side of this investigation were defrayed by a grant from the Clinical Research Committee of the Middlesex Hospital. We would like to thank the Chairman and members of the Committee for making these funds available and for their interest and confidence. We would also like to thank Dr A. W. Beard, Consultant Physician at the Middlesex Hospital for his advice and support.

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