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Epidemic vomiting

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SUMMARY

Two outbreaks of epidemic vomiting are described. One affected 107 students and staff at a college of education out of a total of 398 persons. The other affected 172 pupils and staff out of 357 at a secondary school. Evidence is presented that in both cases infection was acquired in the dining hall of the institution concerned but no specific item of food was found as a likely cause. The literature is reviewed. Possible mechanisms of spread are suggested.

FIRST OUTBREAK

This occurred in May 1969, and 107 students and staff were affected. During the night of 10–11 May 74 cases occurred.

The college at that time had 320 students, all female, 36 teaching and administrative staff and 42 domestic staff. One hundred and eighty-four of the students and 5 of the staff were resident as follows.

Residence A	78 students	3 staff
Residence B	18 students	1 staff
Residence C	18 students	1 staff
Residence D	18 students	
Residence E	36 students	
Approved lodgings	16 students	

Residence A has the main dining room for the college and residents have breakfast, lunch and supper. Residents in B and C have lunch and supper at A. Residents of D and E and those in approved lodgings usually have lunch at A on weekdays as also do 15–20 of the teaching and administrative staff. The domestic staff are mainly part-time and employed in the residences. About two-thirds have lunch at Residence A.

Incidence

In Fig. 1 the onset is given at 6-hourly intervals according to whether or not meals were taken at Residence A.

Table 1 gives the incidence according to Residence and Table 2 gives the incidence according to meals taken at Residence A. Of the 107 persons affected

* Present address: 6 Long Meadows, Ponteland, Newcastle upon Tyne.

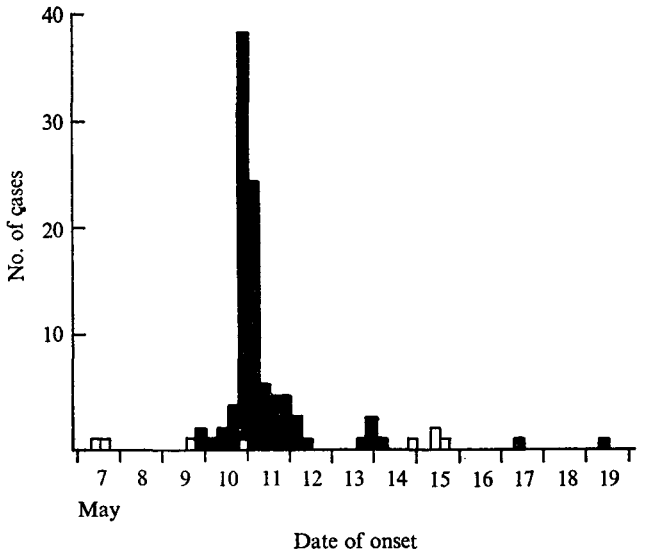

Fig. 1. Onset of cases in first outbreak, at 6-hourly intervals. □, No meal at Residence A; ■, meals at Residence A.

Table 1. *Incidence according to residence (first outbreak)*

	Total	No. affected	% affected
Students			
Residence A	78	46	60·3
Residences B and C	36	17	47·2
Residences D and E and approved lodgings	70	17	24·3
Non-resident	136	7	5·1
Staff			
Resident	5	2	40·0
Non-resident. Lunch on 9 May	15	13	86·6
Non-resident. No lunch on 9 May	16	1	6·3
Domestics			
Lunch on 9 May	28	4	14·3
No lunch on 9 May	14	—	—
	398	107	26·9

only 8 did not have meals at Residence A and of the 93 affected in the main outbreak only one did not have meals at Residence A in the previous 48 hr. Seventy-five of these had lunch on 9 May and this was the last meal which staff members and girls in Residences D, E and approved lodgings had before the outbreak started some 30 hr. later. In all 185 persons had this meal.

The heaviest incidence (87 %) was in the staff who had lunch on 9 May. On the other hand only 4 out of 28 domestics who had the same meal were affected and these 4 were all working in the kitchen. (The students and staff have two sittings and the domestics come in later.) The incidence in the students shows a progressive fall as their association with the dining room at Residence A diminishes.

Table 2. *Incidence according to meals taken at Residence A (first outbreak)*

	Meals at Residence A	No meals at Residence A	Total
Affected between 6 p.m. 9 May and midday 12 May	92	1	93
Affected other times	7	7	14
Not affected	133	158	291
Total	232	166	398

$\chi^2 = 81{\cdot}01$; $P < 0{\cdot}0005$.

Table 3. *Symptoms (first outbreak)*

	Staff		Students		Total	
	No.	%	No.	%	No.	%
Total numbers	16		65		81	
Nausea	8	50·0	59	90·8	67	82·7
Vomiting	7	43·8	57	87·7	64	79·0
Abdominal pain	5	31·2	32	49·2	37	45·7
Diarrhoea	11	68·7	26	40·0	37	45·7
Headache	2	12·5	11	18·5	13	16·0
Fainting	—		5	7·7	5	6·2
Malaise/fever/sweating	2		2		4	
Giddiness	—		2		2	
Weakness	2		—		2	

Three cases occurred before the main outbreak and there were 11 late cases. A similar type of illness was known to be prevalent in the district at the time.

The most interesting feature of this outbreak is that although persons visiting Residence A over the weekend had the impression of mass hysteria yet cases occurred simultaneously in 4 other residences and among staff and students at home or in lodgings.

Symptoms

Questionaries were not used. The number of cases, time of onset and main symptoms were ascertained by the college authorities. The 16 staff and 65 of the students were questioned personally. This was a total of 81 or 76% of those affected. The frequency of the symptoms is given in Table 3.

Among the students nausea and vomiting were most common while among the staff diarrhoea was the leading symptom. About half of those who vomited had preliminary nausea for 1–2 hr. but the remainder had precipitate vomiting, often occurring in the early hours of the morning. The vomiting was usually succeeded by colic and diarrhoea. Thereafter recovery was very rapid. Many described their symptoms as being very severe and yet were fit and well 24 hr. later.

Other symptoms were not prominent. Five students fainted at the outset. Eleven had headaches. In one case headache, stiff neck and vertigo were the leading symptoms.

Two cases who were felt to be part of the main outbreak complained of nausea on the evening of 9 May. They vomited 24 hr. later and thereafter the illness followed the usual pattern.

Incubation period

The cases after the main outbreak provide useful clues.

(1) Five students who were away for the weekend, leaving on the morning of Friday 9 May and returning on the Sunday evening, became ill as follows:

Nausea	Vomiting
Tuesday a.m.	Tuesday midnight
Tuesday 6 pm.	Wednesday 12.30 a.m.
Tuesday 6.30 p.m.	Wednesday 2 a.m.
Tuesday 11 p.m.	Wednesday 2 a.m.
Wednesday 11 a.m.	—

This suggests an incubation period of less than 48 hr. in at least four cases.

(2) The college was closed on Monday 12 May and Tuesday 13 May because of the outbreak. It reopened on 14 May at 9 a.m. Four day-students became ill at the following times: Wednesday late p.m., Thursday a.m., Thursday a.m., Thursday p.m.

This suggests an incubation period of about 24 hr.

(3) A girl at Residence D developed typical symptoms on 17 May. Her room-mate developed the same symptoms 48 hr. later.

(4) There were only two secondary cases in the families of staff who were ill at home. Both occurred about 48 hr. after the commencement of the index case.

(5) If one now goes back to the main outbreak and assumes that the 75 persons who had lunch on the Friday and subsequently became ill were infected at the time of this meal then the incubation period would be 12 to 66 hr. with a mode of 36 hr. The majority of those having lunch on 9 May also had lunch on the previous 4 days, but a party of students were in London for 4 days, returning on the morning of 9 May. The majority of these were ill.

Investigation

Two specimens of vomit and 12 of faeces were sent to the Public Health Laboratory. Neither specimen of vomit revealed any growth on culture.

Of the 12 specimens of faeces 1 was positive for *Salmonella*, the organism being *S. enteritidis* phage type 8. The other 11 were negative for *Salmonella* and all 12 were negative for *Shigella*, enteropathic *Escherichia coli*, *Staphylococcus* and *Clostridium welchii*. Subsequent culture for virus was undertaken in 9 of these specimens on HeLa and on monkey kidney tissue but all were negative.

The *Salmonella* isolation was almost certainly an incidental finding. The clinical and epidemiological pattern of the outbreak did not fit with infection with this organism. None of the cases had antibiotic treatment and if a *Salmonella* had been responsible further isolations would have been certain. The other types of food poisoning can also be excluded in spite of the small number of specimens taken.

Table 4. *Results of water sampling (first outbreak)*

	Date	Number of coliforms per 100 ml. Tap over dishwasher	Tap over food-preparation sink
Public Health laboratory	15 May	180+	50
	23 May	180+	0
	24 May	3	0
	29 May	5	5
Water Company laboratory	20 May	0	1
	2 June	0	0

The lunch on 9 May consisted of fried or baked haddock, peas and chips, blackberry crumble and custard, all freshly cooked and served hot. Jugs of water were placed on the tables. A few people had biscuits and cheese instead of fish or instead of sweet. No single item was common to more than 75% of those who subsequently became ill. Even among the 13 non-resident staff, for whom this meal was the only one eaten in common with the other affected persons, there was no common item. No food remained for examination. Cheese was again served at supper that night and a sample of the same block was sent to the laboratory. No pathogenic bacteria were grown.

The water supply to Residence A is by rising mains from the Newcastle and Gateshead Water Company. This supply is of constantly proven excellence as far as bacteriological sampling is concerned. Specimens taken in the kitchen after running and after flaming the taps were negative for coliforms.

Samples were also taken without running and without flaming the taps. The results are summarized in Table 4.

Any significance of these results is very much open to question but the matter will be referred to later.

Faecal specimens were taken from all members of the kitchen staff, 4 of whom were affected and 10 of whom were not affected. All were negative for Shigella and food-poisoning organisms.

SECOND OUTBREAK

This affected 161 pupils aged 12 to 16 and 11 staff including two domestics at a secondary school between 9 January and 3 February 1971. One hundred and ten of the cases occurred between midday on 26 January and 9 p.m. on 27 January. The school had 343 pupils, 18 teaching or administrative staff and 7 domestic staff, a total of 368 persons. Of these 11 were absent at the relevant time leaving 357 exposed to infection.

Incidence

During December and January there were reports of gastro-intestinal infections in the area, and between 9 and 26 January 16 pupils were affected (Fig. 2). The main outbreak began in the late afternoon of 26 January and reached its peak

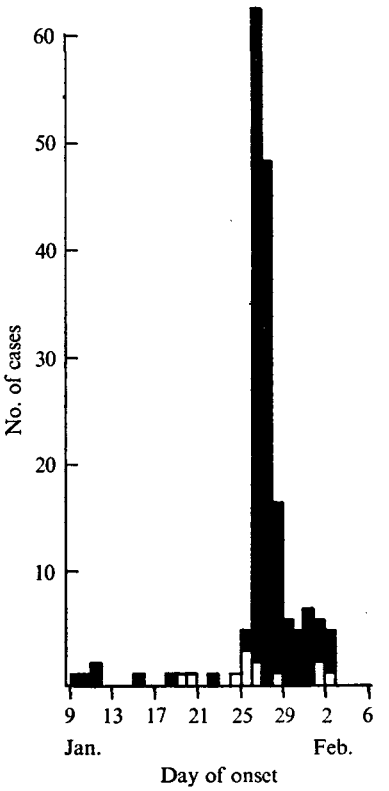

Fig. 2

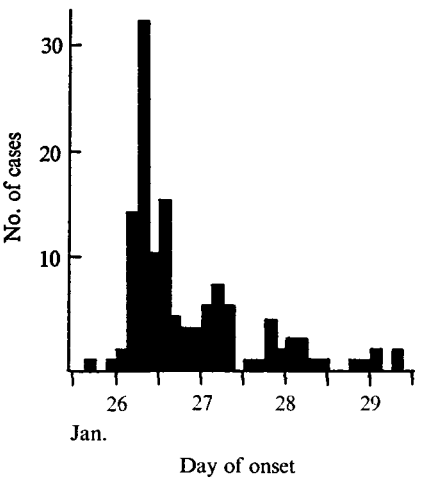

Fig. 3

Fig. 2. Onset of cases in second outbreak. □, Those not having school dinner; ■, those having school dinner.

Fig. 3. Onset of cases in second outbreak between 26 and 29 January, shown at 3-hourly intervals.

Table 5. *Incidence in relation to eating school dinner (second outbreak)*

	School dinner	No school dinner	Total
Affected between midday 26 January and midnight 27 January	109	1	110
Affected other times	51	11	62
Not affected	131	54	185
Total	291	66	357

$\chi^2 = 32{\cdot}97$; $P < 0{\cdot}0005$.

around 9 p.m. (Fig. 3) with a subsidiary peak at 5 a.m. on 27 January. Thereafter 46 cases occurred.

On 5 February a questionary was sent to all staff and parents. By interviewing those who had not completed the questionary information on all 357 persons was obtained.

The interesting feature of the outbreak is the preponderance of illness in those

Table 6. *Sex incidence in pupils (second outbreak)*

	M		F		Total	
Affected	94	52%	67	44%	161	48%
	58%		42%		100%	
Not affected	88	48%	84·	56%	172	52%
	51%		49%		100%	
	182	100%	151	100%	333	100%
	55%		45%		100%	

Table 7. *Symptoms (second outbreak)*

	Staff		Students		Total	
	No.	%	No.	%	No.	%
Total numbers	11		161		172	
Nausea	9	81·8	116	72·0	125	72·7
Vomiting	8	72·7	110	68·3	118	68·6
Abdominal pain	7	63·6	96	59·6	103	59·9
Headache	5	45·4	67	41·6	72	41·9
Diarrhoea	7	63·6	49	30·4	58	33·7
Giddiness	3	27·3	29	18·0	32	18·6
Fever	2	18·2	20	12·4	22	12·8
Cramp	—		11	6·2	11	6·4
Aching legs or back	1		5		6	
Sweating	2		—		2	
Shivering	1		—		1	

Table 8. *Duration of symptoms (second outbreak)*

	No.	%
Up to 1 day	92	53
1–2 days	38	22
2–3 days	24	14
3–5 days	9	5
5–7 days	5	3
Over 7 days	4	3
Total	172	100

taking school meals on 26 and 27 January (Table 5). Eighty-two per cent of children have school dinners and the proportion affected outside the main outbreak is the same. However on 26 and 27 January 99% of those affected took school dinner.

In most of the outbreaks described girls have been more commonly affected than boys, but in this outbreak there was a slight preponderance of boys affected (Table 6).

Symptoms

A typical illness was nausea followed within an hour by vomiting and then colicky abdominal pain and diarrhoea. This was accompanied by headache. In

many cases vomiting occurred precipitately. Most of those affected were recovering within 24 hr. The symptoms of the 172 affected are given in Table 7.

Diarrhoea was a more common symptom among the staff (63 %) than among the pupils (30 %) as in the other outbreak. Comparison of the symptoms between those affected before and those during the main outbreak does not reveal any real differences. With the later cases there was a tendency for the illness to become milder and abdominal pain was the leading symptom with only 31 % having vomiting.

The duration of the symptoms is given on Table 8. Though the symptoms were often described as being severe the family doctor was consulted in only 16 cases (9 %).

In three instances two episodes of similar illness were mentioned with an interval of 2–3 weeks.

Family spread

As has been mentioned gastro-enteritis was fairly widespread in the district at the time. Of the 185 persons not affected 11 (6 %) had cases of diarrhoea or vomiting in the household. Of the 16 affected before the main outbreak 6 (38 %) had associated cases in the family.

From 26 January to 3 February 27 (17 %) stated that one or more members of the family were affected within seven days, with a total of 38 persons affected. Of the 27, in 3 the family case was before the case in the school, and in 5 at the same time. This leaves 19 (12 %) where a case occurred in the family which is most likely to be secondary to the main outbreak. (There were a number of households where more than one member attended the school and in these it has been assumed that during the main outbreak infection occurred at the school.)

Incubation period

The questionary did not ask for the hour at which the secondary case started but merely the day. Thus no accurate estimate can be made of the time interval in the 19 cases mentioned above. The interval between cases is as follows: 2 days, 11; 3 days, 1; 4 days, 3; 5 days, 4.

If it is assumed that the 109 children who developed symptoms on 26 and 27 January were affected at lunchtime on 25 January, then the mean incubation period is 37 hr. Subsequent cases at the school could either have had a longer incubation period or could be secondary to the main wave.

Investigations

Stool specimens were obtained from 7 affected persons and in none of these was there any growth of *Salmonella*, *Shigella*, *Staphylococcus*, *Clostridium welchii* or enteropathic *Escherichia coli*. No virus isolation was attempted. The number of specimens taken was small but our recent experience of an almost identical outbreak (*q.v.*) led us to diagnose epidemic vomiting at an early stage and not to seek too much help from the laboratory which at that time was experiencing difficulties.

About 280 school meals were served daily. The meal on Monday 25 January consisted of sausages, chips, carrots, chocolate sponge and custard. Water was available. All but 3 (97 %) of those affected on 26 and 27 January ate the sausage, all but 2 (98 %) had chips, 89 % had carrots, 85 % chocolate sponge or custard and 60 % had water.

Tuesday's meal was beef stew, potatoes, cabbage, date tart and custard. 4 persons affected on 26 January did not have this meal. Of those affected, 92 % had stew or potatoes, 75 % had cabbage and about 80 % sweet: 60 % had water.

It seems most unlikely that chips could transmit an infective agent so the sausages are the only item to come under suspicion as the vehicle of infection. Pupils and staff tended to blame the sausages. As well as complaining about the taste many said they were dark in colour. They had been cooked in the oven at gas mark 3 for $1\frac{1}{2}$ hr. Inquiry was made at the firm manufacturing the sausages. No person on the premises gave a history of gastro-enteritis. Sausages manufactured at the same time had been distributed to other schools and eaten without ill-effect. On balance it seemed unlikely that sausages were the vehicle of infection although the possibility could not be excluded.

The catering staff were all interviewed individually and asked about symptoms in themselves or their families before 26 January. One member had vomiting and abdominal pain on 13 January, an illness lasting for 3 hr., but there was no illness immediately before the main outbreak.

Water samples were taken from the kitchen taps without flaming or letting the water run but there were no coliforms per 100 ml.

DISCUSSION

The terms winter vomiting disease, non-bacterial gastro-enteritis and epidemic vomiting have all been used to describe similar outbreaks in which the illness itself was insignificant but large numbers of cases occurring simultaneously have caused concern. Major outbreaks have been described by Bradley (1943) and Webster (1953) with 200 and 300 cases respectively. Lesser outbreaks have been described by Miller & Raven (1936), Dummer (1953), Cumming & McEvedy (1969) and Adler & Zickl (1969) among others.

While the symptoms of these outbreaks have shown some variation the epidemiological pattern has been similar. Cases have been reported in the district with one or two in the institution about to be affected. There follows an explosive outbreak involving $\frac{1}{3}$ to $\frac{2}{3}$ of the institution's members. Finally there are a few secondary cases. The incubation period is from 1 to 3 days. The illness, often of sudden onset and occurring during the night, is usually afebrile and recovery is rapid. There are no sequelae. No causative organisms are found. Poisoning by a specific item of food can be excluded.

Outbreaks with similar clinical and epidemiological features have been traced to the ingestion of sewage-contaminated water (Lobel, Bisno, Goldfield & Prier, 1969; Public Health Laboratory Service, 1974); or to shellfish harvested from such water (Gunn & Rowlands, 1969; Public Health Laboratory Service, 1970; Ratzan,

Bryan & Krackow, 1969; Dismukes, Bisno, Katz & Johnson, 1969). The incidence in these outbreaks is usually particularly high, e.g. 38 out of 40 in the outbreak associated with oysters described by Gunn & Rowlands. Secondary person-to-person spread has occurred.

In other outbreaks such as those described by Zahorsky (1929), Gordon, Ingraham & Korns (1947) or Hopkins (1958) symptoms have been similar but the onset has been more gradual and person-to-person spread has probably been the principal mode of transmission.

In most outbreaks hysteria has been considered either as a primary cause or as a secondary feature. Most observers however have concluded that hysteria played an insignificant part. Typically the majority of cases occurred during the night and at home when the possibilities of mass suggestion are at a minimum. Outbreaks of fainting among schoolgirls (Pollock & Clayton, 1964; Moss & McEvedy, 1966) occurred typically during school hours. An outbreak of abdominal pain at a children's gala (Smith & Eastham, 1973) was thought on clinical and epidemiological grounds to be hysterical. These outbreaks have little in common with those described although Pollock & Clayton thought they were dealing with an infection.

Mode of spread

If the waterborne outbreaks are due to the same micro-organism as the other explosive outbreaks it would seem that one must look for a common source for these also. Poisoning by a single food can be excluded but is it possible for an infective agent to spread itself over several items of food? If someone in the kitchen of the institution concerned were transmitting the organism could they infect a working surface or a tap at the food preparation sink and spread subsequently take place to several items of food? Or could contaminated washing-up water lead to spread via crockery or cutlery?

These are purely theoretical possibilities but seem more likely than the respiratory spread postulated by some observers. Proof will need to await the identification of the causative organism. Recent experimental work (Paver, Caul, Ashley & Clarke, 1973; Kapikian *et al.* 1972) raises hope that this will soon occur.

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