It is difficult to reconcile the occurrence of gastric ulcer after successful vagotomy with current views on the pathogenesis of the condition. There is after vagotomy a lowering of the gastric acid level and particularly of the volume and degree of acidity of the night acid secretion. This problem might be solved if an unequivocal test of complete vagal section were available; and the observations of Brooke (1949) with regard to the insulin test are supported by the findings of Walters and Brownson (1948) and Walters *et al.* (1949) that in their series the clinical results in those cases showing a negative post-operative insulin test were similar to those showing a positive response. Without therefore being able to say that vagotomy has any causal relationship to the gastric ulcers in the cases described above, it does seem that the lowering of acidity brought about by vagal section affords no protection against ulceration developing. Further confirmation is forthcoming from the report of Nuboer (1949), who has described the occurrence of gastric ulceration in some of his cases following resection of the oesophagus for malignant disease, when the vagi were necessarily completely divided.

These two cases are obvious failures following vagotomy in the treatment of duodenal ulcer. The Birmingham series referred to above contains cases which have had other undesirable sequelae of the operation: these will be recorded when the series is fully analysed.

Summary

In a series of 79 cases of duodenal ulcer, two cases developed gastric ulcer following successful vagal section. These two cases are described.

The view is expressed that vagotomy is unsuitable as a treatment of gastric ulceration and confers no protection against the development of a gastric ulcer.

In both cases the gastric ulcers developed within six months of the vagal section.

I am indebted to Mr. B. N. Brooke for permission to publish Case 2, and to Professor F. A. R. Stammers for his guidance and help in the preparation of this paper.

REFERENCES

Brooke, B. N. (1949). *Lancet*, **2**, 1167.
Cameron, D. A., Burt, C. W., and Tuttle, W. M. (1949). *J. Mich. med. Soc.*, **48**, 301.
Dragstedt, L. R., Camp, E. H., and Fritz, J. M. (1949). *Ann. Surg.*, **130**, 843.
Healy, M. J., jun., and Sauer, P. K. (1949). *Bull. N.Y. Acad. Med.*, **25**, 449.
Kugel, A. I., and Janzen, J. (1949). *Gastroenterology*, **12**, 993.
Moore, F. D. (1947). *Arch. Surg., Chicago*, **55**, 164.
—— (1948). *Gastroenterology*, **11**, 442.
Nuboer, J. F. (1949). *Acta gastro.-ent. belg.*, **12**, 380.
Orr, I. M., and Johnson, H. D. (1949). *British Medical Journal*, **2**, 1316.
Walters, W., and Brownson, B. C. (1948). *Bull. Chic. med. Soc.*, **51**, 135.
—— —— and Phillips, S. K. (1949). *Kentucky med. J.*, **47**, 254.

According to a press report the New York Academy of Medicine has arranged a series of "refresher" lectures for doctors in New York which will be given over the radio. Believed to be the first of its kind, the series is designed to bring the latest scientific developments in medicine to doctors who are unable to attend medical conventions, who have no hospital connexions, or who are too busy to read through the medical journals. The one-hour programmes, to be broadcast every Thursday evening, will actually be transcriptions of papers delivered at the Academy's regular Friday afternoon lectures. A test broadcast last April on A.C.T.H. and cortisone received such wide response that planning went ahead for the series. The New York radio authorities are now planning to start a similar series of "refresher" lectures for lawyers and are hoping to expand the idea to other professions.

EPIDEMIC OF BENIGN DRY PLEURISY

BY

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Since Sylvest's original monograph on Bornholm disease, or epidemic myalgia, was published in 1933, a similar condition has been described in countries as widely separated as Scandinavia, India, and the United States. A diagnosis of benign dry pleurisy has been made in cases with associated pleural friction.

In this country epidemics have been sporadic in appearance and often confined to localized communities such as schools (Attlee *et al.*, 1924; Carter, 1933), children's hospitals and convalescent homes (Lloyd, 1924; Williamson, 1924), or to single households (Howard, 1938), while Pickles (1933, 1939, 1948) has described several outbreaks in village communities in Yorkshire.

Scadding (1946) reviewed the literature on Bornholm disease and described a series of similar cases observed among soldiers in the Middle East. These were characterized by a high incidence of pleural friction (55% of 20 cases), and he consequently named them benign dry pleurisy.

This paper records an epidemic of benign dry pleurisy occurring in two households in the Bayswater district of London during September and October, 1949. Eleven cases in all were encountered, two of which were observed during the course of their illnesses.

Case 4

A healthy medical student aged 22 became ill on October 4: he complained of severe pain in both upper quadrants of the abdomen, which was lancinating and worse on coughing or deep breathing. It radiated first to the right, then to the left shoulder, and was accompanied by headache and sweating.

When first seen on October 5 his breathing was shallow and painful and his temperature 100.8° F. (38.2° C.), but, apart from upper abdominal tenderness, there were no abnormal physical signs. In view of the position and radiation of the pain, the absence of cough or abdominal rigidity, and his clean tongue, a tentative diagnosis of diaphragmatic pleurisy was made.

On October 6 his temperature had risen to 101° F. (38.3° C.) and his respirations were 40 a minute. The pain and tenderness were now localized in the region of the left costal margin. A chest radiograph showed no abnormality at the left base; there was, however, a slight but definite decrease in translucency at the right base, together with slight elevation of the right half of the diaphragm. The total leucocytes were 8,000—polymorphs 6,000 (75%), lymphocytes 1,600 (20%), monocytes 400 (5%).

On October 7 there was a pleural rub in the right axilla which persisted for three days. His E.S.R. (Westergren) was 18 mm. in one hour and the Mantoux reaction was positive to 1 in 1,000 tuberculin. The diaphragm moved normally on screening. Recovery was rapid; by October 11 he was free of symptoms and there was no relapse. A radiograph of the chest taken on November 16 showed no residual shadowing.

Case 10

On October 22, eighteen days after the above patient had fallen ill, a second medical student, aged 26, was seized with pleuritic pain in the right shoulder which wakened him in the night and which spread to the right upper abdomen when he got out of bed. The pain was worse on lying down and prevented him from lying on the affected side. His temperature was 99° F. (37.2° C.) and there were no abnormal physical signs. On October 23 the pain had spread to the left shoulder and

right lower chest, there was right upper abdominal tenderness, and a pleural rub which persisted for twenty-four hours was heard below the right nipple. By the 24th he was free of symptoms. The total leucocyte count was 7,000—polymorphs 4,340 (62%), eosinophils 770 (11%), basophils 70 (1%), lymphocytes 1,540 (22%), monocytes 280 (4%). The E.S.R. (Westergren) was 4 mm./hour. A chest radiograph, however, was normal.

The second patient stated that other residents at his lodgings had also suffered from similar symptoms. When they were interviewed it was evident from the typical history given in nine cases that there had been an epidemic of benign dry pleurisy similar to that described by Scadding (1946).

All the cases occurred in two lodging-houses under common ownership in adjacent streets in Bayswater. The patients were healthy adults between the ages of 19 and 28, in the proportion of nine males to two females. The onset of the epidemic was dated to September 28, when, in House A, the manageress, aged 24, wakened with pain under both costal margins. Four days later (October 2) her sister, living in House B, fell ill with similar symptoms. Seven further cases were identified in House A and two in House B. They are listed in Table I.

TABLE I

House A (16 Residents)				House B (20 Residents)			
Case No.	Age	Date of Onset	Occupation	Case No.	Age	Date of Onset	Occupation
1	24	Sept. 28	Manageress	2	19	Oct. 2	Music student
3	22	Oct. 3	Medical student	4	22	,, 4	Medical student
5	24	,, 5	Pharmacist	6	26	,, 6	Art student
7	21	,, 9	Medical student				
8	28	,, 19	Student				
9	21	,, 21	Medical student				
10	26	,, 22	,, ,,				
11	20	,, 24	,, ,,				

Summary of the Eleven Cases

Prodromal Symptoms.—One case only had catarrh preceding the attack of pain; there were no cases with sore throat (as have been reported by Attlee *et al.*, 1924). The onset was sudden in all cases, with pain as the presenting symptom. The pain was always suggestive of pleurisy and was worse on lying down. Unlike the pain of pleurisy associated with pneumonia or tuberculosis, it was worse when lying on the affected side, possibly because immobility of the intercostals in this position caused increased movement of the diaphragm. The situation of the pain at onset was right-sided in 9 of the 11 cases, and is set down in Table II. The position of the pain and its reference to the shoulders suggests its origin in the diaphragm, and it is interesting that in Case 7, whose pain was in the right subcostal region, referred pain in the right shoulder was produced by pressure below the right ribs.

TABLE II.—*Situation of Pain at Onset*

	No. of Cases
Right shoulder	3
Upper abdomen	3
Under right costal margin	2
In right arm and under right costal margin	1
In right chest and right shoulder	3

The average length of illness was three to four days, the minimum being two and the maximum seven days, and no relapses occurred. Pleural friction was heard in the two cases seen during the illness. It was of short duration and unilateral in both cases.

Radiographic and Blood Examinations.—Leucocyte counts were normal in both cases treated in hospital; the E.S.R. was normal in Case 10 (4 mm./hour) and raised in Case 4 (18 mm./hour). In previous series, chest radiographs have been normal and the diaphragm has moved normally on screening. In one of the two cases radiographed in the present epidemic abnormal findings were present. On the second day of illness Case 4 had a slight but definite decrease in translucency at the right base, which was more marked on the fourth day and was associated with slight elevation of the right half of the diaphragm. These *x*-ray changes were minimal but definite, and were possibly caused by pleural oedema.

Differential Diagnosis.—Finn (1947), in Boston, writing of a large series of cases, reports confusion with pneumonia, glandular fever, influenza, "non-paralytic poliomyelitis," lymphocytic chorio-meningitis, hepatitis, and acute abdominal emergencies. Pickles (1933) has emphasized that faulty diagnosis from the acute abdomen may lead to needless laparotomy. The pleural friction heard in the two cases here described clearly indicated the source of the pain.

Complications.—Pericarditis and orchitis have been quoted by Finn *et al.* (1949) in Boston and by Jamieson and Prinsley (1947) in Aden, while McConnell (1945), in Pennsylvania, recorded a high incidence of neurological symptoms (68% of 16 cases). There were no complications in this series.

Epidemiological Aspects

Seasonal Incidence.—The disease seems to be most prevalent in the summer and early autumn, when these cases occurred, resembling poliomyelitis in this respect. It is of interest that three cases of paralytic poliomyelitis and one of polio-encephalitis occurred among the hospital staff during the same period as the cases of epidemic pleurisy.

The incubation period is quoted by Finn (1947) as 8–10 days. In this epidemic it would appear to lie between 2 and 19 days. The cases occurred in two groups at intervals of ten days, the first group including occupants of both houses.

Case 2 may have been infected from her sister (Case 1), or both may have derived their infection from a common unidentified source. If Case 4 was infected from one of the preceding cases this could only have been from Case 2, making the minimum incubation period two days. The longest possible incubation period in these cases is provided by Case 11, who returned from holiday on October 5 and developed symptoms on the 24th (19 days).

The second group, Cases 8 to 11, consists of students who returned to London from holiday on October 5. Two of them, Cases 10 and 11, returned to share adjacent rooms with Cases 3 and 7 respectively, Case 3 being in bed with the disease at the time. Their accommodation on and after October 5 is shown in Table III.

TABLE III.—*Occupants of Adjacent Rooms in House A on October 5*

	Case	Onset of Symptoms
Room 1	3	October 3
	10	,, 22
Room 2	7	,, 9
	11	,, 24

Cases 10 and 11 returned from holiday on October 5. Prior to October 5, Room 1 was occupied by Cases 3 and 7, Room 2 being let to temporary visitors.

Causative Agent

Attempts to isolate any infective agent have met with little success. Small (1924) suggested a plasmodium; other workers have failed to isolate any bacteriological agent; nor has any virus been isolated from stools or nasal

washings. Henle (quoted by Finn *et al.*, 1949), in complement-fixation tests for the viruses of influenza and choriomeningitis, had negative results.

The absence of any other infective agent and the nature of the outbreaks suggest a virus as the probable cause. In the present series complement-fixation tests for the Coxsackie 2 strain of virus were kindly performed by Dr. G. M. Findlay in March, 1950 (five months after the last recorded case). Sera from five cases were tested, and the results are listed in Table IV. Three cases were strongly positive and two faintly positive; two normal students taken as controls were negative.

TABLE IV.—*Result of Complement-fixation Tests*

Serum	Antigen		
	Coxsackie 1	Coxsackie 2	Normal Mouse
Case 4	—	+ 1 in 5	—
Case 7	—	+ 1 in 20	—
Case 9	—	+ 1 in 20	—
Case 10	—	+ 1 in 20	—
Case 11	—	+ 1 in 5	—
Control I	—	—	—
Control II	—	—	—

This virus, which is transmissible only through newborn mice, was originally isolated by Dalldorf and Sickles (1948) and later by Melnick *et al.* (1949) from the faeces of cases of "non-paralytic poliomyelitis" in the United States. In mice the infection involves skeletal muscles, producing severe myositis and paralysis, while the central nervous system is unaffected. Death commonly occurs within twenty-four hours of the onset of paralysis.

Transmission

The exact mode of transmission is unknown. Melnick *et al.* (1949) isolated the Coxsackie strains from sewage and from flies in areas where epidemics of "non-paralytic poliomyelitis" occurred.

It is interesting that the first case in the present series was the cook for the whole of House A, in which seven further cases occurred, and that Case 2, the first in House B, assisted her parents in preparing food for this household. While this strongly suggests an alimentary spread at the onset of the epidemic, later cases may have arisen from droplet infection owing to the close proximity of their living quarters. On inquiry, however, no cases could be traced outside the two households among their day-to-day contacts at work.

Coincident with the epidemic, one sporadic case was also seen during the illness. A transitory bilateral pleural rub was present and the complement-fixation reaction to the Coxsackie 2 strain was strongly positive (1 in 20). This case also had a minimal unilateral pleuritic reaction on *x*-ray examination which did not persist.

Summary

An explosive outbreak of benign dry pleurisy is reported in two near-by households under common ownership. Two cases are described in detail.

The epidemiological aspects of the outbreak are discussed.

Five months after the illness the sera from five cases were shown by Dr. G. M. Findlay to give positive complement-fixation reactions to the Coxsackie 2 strain of virus as compared with negative results in two normal controls.

It is suggested that, in view of the seasonal incidence and the possible relationship to "non-paralytic poliomyelitis," the mode of spread may be similar to that of poliomyelitis itself and is possibly alimentary.

I wish to thank Dr. G. M. Findlay for his advice and for performing the serological tests, and Professor R. Cruickshank and Dr. T. A. Kemp, physician in charge of the Student Health Service, St. Mary's Hospital, for their guidance and helpful criticism.

References

Attlee, W., Amsler, A. M., and Beaumont, D. C. (1924). *Lancet*, **2**, 492.
Carter, A. H. (1933). *British Medical Journal*, **2**, 1186.
Dalldorf, G., and Sickles, G. M. (1948). *Science*, **108**, 61.
Finn, J. J. (1947). *New Engl. J. Med.*, **237**, 621.
—— Weller, T. H., and Morgan, H. R. (1949). *Arch. intern. Med.*, **83**, 305.
Howard, C. R. G. (1938). *British Medical Journal*, **2**, 1203.
Jamieson, W. M., and Prinsley, D. M. (1947). Ibid., **2**, 47.
Lloyd, E. I. (1924). *Lancet*, **2**, 272.
McConnell, J. (1945). *Amer. J. med. Sci.*, **209**, 41.
Melnick, J. L., Shaw, E. W., and Curnen, E. C. (1949). *Proc. Soc. exp. Biol., N.Y.*, **71**, 344.
Pickles, W. N. (1933). *British Medical Journal*, **2**, 817.
—— (1939). *Epidemiology in Country Practice*. Wright, Bristol.
—— (1948). *Lancet*, **1**, 201.
Scadding, J. G. (1946). Ibid., **1**, 763.
Small, J. C. (1924). *Amer. J. med. Sci.*, **168**, 570.
Sylvest, E. (1933). *Den Bornholmske Syge: Myalgia Epidemica*. Levin and Munksgaard, Copenhagen. English translation, *Epidemic Myalgia*, 1934. London.
Williamson, B. (1924). *Lancet*, **2**, 64.

ADRENAL INSUFFICIENCY AFTER SURAMIN TREATMENT OF PEMPHIGUS

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[WITH SPECIAL PLATE]

Suramin ("germanin") has been used for the treatment of pemphigus since 1931. It is a carbamide of a complex salt of trisulphonic acid, is also known as "naphuride" and "Bayer 205," and was originally introduced for the treatment of trypanosomiasis. It is known to have toxic effects on the renal tubules and haemopoietic system. Wells *et al.* (1937) have shown that when given to guinea-pigs in amounts comparable with human doses it causes damage to the renal tubules and adrenal cortex, but the full picture of so-called adrenal atrophy was not achieved in their experiments. Two cases of acute adrenal insufficiency following suramin treatment have been reported (Wells *et al.*, 1937; Tomlinson and Cameron, 1938), but any conclusion that these adrenal changes were solely due to the drug is confused by the fact that many of the features of pemphigus, especially in acute cases, strongly resemble those of adrenal insufficiency and are sometimes improved by the administration of cortical extracts.

Furthermore, Goldzieher (1945) has shown that signs of severe organic damage may be found in the adrenal cortex in deaths from pemphigus. None of the six cases he studied had received suramin,* and he regarded their lesions as terminal secondary processes such as occur in other severe debilitating diseases.

The paradox is a familiar pharmaceutical one—that a drug which mimics the effects of a disease should be beneficial for it—and perhaps the problem is one of correct dosage for the individual.

Wells *et al.* state that none of the persons who have been followed up for some time after suramin therapy for trypanosomiasis have shown any delayed symptoms attributable to adrenal insufficiency. We are therefore in somewhat

*Personal communication.