ance shows no evidence of infection (Fig. 2). Moreover, there was no evidence in the present two cases of maternal infection during pregnancy.

A prominent feature of this condition is extreme irritability, which may be due to pain in the bones; from observation of Case 1 it was thought that the child suffered from pain in the affected jaw. It is possible that, when the part becomes warm, pain is produced by vascular dilatation compressing the nerves in Haversian canals narrowed by new bone formation. This explanation is supported by Mossberger's (1950) finding of newly formed cancellous bone filling the Haversian spaces. The long course and marked irritability suggest an analogy with pink disease. It is notorious that cases of the latter are prone to secondary infection, and, to judge from reported cases, there seems to be a similar tendency in infantile cortical hyperostosis. One of our patients died in hospital of secondary infection, as did one of van Zeven's (1948). Mossberger's patient (1950) died suddenly at home, and necropsy indicated that an infection in the gut was the cause of death. Since three of the 41 recorded cases have ended fatally, this disease cannot be considered as benign as has been thought, and it would be sound practice to exclude these cases from hospital unless their admission was strongly indicated.

Summary

Two cases of infantile cortical hyperostosis are described; in one of these the patient died of secondary infection, to which these cases appear to be prone. The disease is therefore not invariably benign, and it would be prudent to avoid admission to hospital.

It is suggested that the bony changes are dependent on diminution in the blood supply to the bone due to intimal thickening, and that the disease is not primarily one of bone.

We wish to thank Dr. J. V. C. Braithwaite for the hospital notes of Case 1, which was admitted under his care, Dr. M. Bodian for the histological report, Dr. D. Forbes Lawson for the skiagrams, Mr. R. W. T. Gaze for the photomicrographs, and Drs. C. K. McCoan and T. M. Gibson for referring the patients to us.

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At the first annual general meeting of the Edinburgh Group of the Infantile Paralysis Fellowship the secretary gave a short outline of the history and work of the group (*Scotsman*, March 14). It is already acting as an information centre and has contacts with the Ministry of Labour and with the Edinburgh Council of Social Services. It has also offered to finance members who wish to take a degree by postal course. Priority at cinemas for members means that they do not have to wait in queues. Another project is a plan to supply television sets to patients in iron lungs. Also the group has managed to get increased supplies of steel needed for orthopaedic appliances.

OUTBREAK OF BORNHOLM DISEASE IN WEST FIFE PRACTICE

BY

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Quite a considerable amount of literature on the subject of Bornholm disease has accumulated within the past 20 years. The disease has now become more generally recognized as a definite entity.

The attention of one of us (W. W. W. McN.) was drawn to the symptomatology of the illness by Hopkins (1950), who described cases which had occurred in his practice. Similar cases had occurred from time to time in the practice here, but had gone unrecognized as Bornholm disease. In May, 1951, the appearance of the cases of two boys proved in retrospect to be the beginning of an outbreak, which continued during June and July. During that time 24 cases from a scattered community came under our care.

Bornholm disease, as we now know it, seems to be an infection of the diaphragm. Ronald (1942) made out a good case for this assumption and thought that the infection occurred primarily in the diaphragmatic muscle, but suggested it might also be primarily in the diaphragmatic pleura. Hopkins believed that when pleurisy did occur it was an extension of the disease from the muscle. The Coxsackie group of viruses is considered by Findlay and Howard (1950) to be the infecting agent.

The cases were investigated from the general practitioner's point of view. The first cases were seen at the end of May and the last on July 30. The outbreak was, therefore, almost exclusively confined to the months of June and July. Four patients were under 10 years of age, six were 10–19, seven 20–29, five 30–39, and two were 40 and over. There were seventeen males and seven females.

Symptoms and Signs

In almost every case the onset was sudden, pain in the upper abdomen or lower thorax being the outstanding feature. The site of pain and the frequency of occurrence are shown in Table I. The pain was definitely related

TABLE I.—Site and Frequency of Pain

Abdominal	20
Right upper subcostal	7
Bilateral subcostal	3
Vague upper	4
Central epigastric radiating to either side	3
Epigastric, spreading to lower left chest	3
Thoracic	4
Left lower anterior	2
Bilateral lower posterior	1
Right lower posterior	1

to respiration in most of the cases. In 20 it was much worse on breathing. Some complained that it was like a stitch in the side; others gasped on account of its severity and were afraid to breathe. Various adjectives were used to describe it: knife-like, terrible, sharp, shooting, unbearable. Some who initially had pain in the upper abdomen had pain in the lower thorax, on one or other side, within a few days. Others had transient pains in the lower thorax before the onset of the acute crippling pain in the abdomen. One patient had pain radiating into the right iliac fossa, which made him think he had appendicitis; another com-

plained of pain in the back of the neck as well as subcostal pain. Movement of the trunk and walking increased the pain in several cases. The pain was accompanied by sickness and vomiting in only two cases, and only one complained of constipation. Four patients had sore throat and nine had headache. All but three had a rise in temperature, the range being from 99 to 102° F. (37.2 to 38.9° C.).

Upper abdominal tenderness was present in nine, being in the right subcostal region in five. One patient had pain at the right shoulder-tip on deep palpation in the right hypochondrium. Tenderness over the lower thorax was noted in one case. Apart from these signs no other abnormality was noted on abdominal examination.

Examination of the chest at the onset of the illness revealed no definite abnormality except restricted breathing and occasionally inverse breathing. In five cases pleural friction was noted four to ten days after the onset (Table II). One patient

TABLE II.—Cases with Pleural Friction

Age in Years	Sex	Situation	Time from Onset of Symptoms	Duration
16	M	Left base anteriorly	8 days	Less than 7 days
26	F	Right " "	9 "	10 days
34	M	" " "	9 "	3-6 "
35	F	{ Left " "	4 "	" "
		{ Right base posteriorly	7 "	14 "
35	M	{ Left base anteriorly	" "	" "
		{ " " posteriorly	10 "	21 "

was entirely free from symptoms before the development of the pleural rub; another stated that she felt the rub. The symptoms usually subsided in a few days, most patients being fairly well within a week. Some took up to two weeks, and a few who developed pleural friction took longer.

Typical Cases

A girl aged 10 was brought to the surgery on the evening of June 21. She had complained of abdominal pain when home for lunch. After returning to school the pain got worse and she had to be sent home. Her main complaint was of severe pain across the upper abdomen, worse in the right costal region. The pain was excruciating whenever she took a deep breath. She also had a headache. Her temperature was 101° F. (38.3° C.) and there was marked tenderness in the right hypochondrium. She had no sickness or vomiting. There were no definite abnormal clinical signs in her chest. We thought she was in for pneumonia and sent her home to bed. Next day her temperature was normal; she was breathing quite freely and had no pain. The tenderness in the right hypochondrium had gone. A day later she was feeling so well that she was allowed up and in two days she was allowed out.

On June 22, when visiting this girl, we were asked to see her younger brother, aged 8. He complained of pain in the lower left chest anteriorly which was catching his breath. There was a slight rise of temperature to 99° F. (37.2° C.). No abnormal clinical signs could be detected in his chest. Next day he was feeling quite well again. The pain had gone and he was allowed up.

These two cases were direct contacts of two others which occurred in the same block of houses and probably of another one.

Observations on the Outbreak

Differential Diagnosis.—Pneumonia was excluded by the absence of abnormal clinical signs in the chest, and by the subsequent course of the illness. Pleurisy (tuberculous): all cases with pleural friction were x-rayed and declared negative. Abdominal conditions were ruled out by the absence of typical signs and symptoms, and by the fact that the pain in most cases was related to breathing.

When first seen more than half the cases were given "sulphatriad" until the possibility of pneumonia was ruled

out. On the non-appearance of definite signs the drug was usually discontinued after a very short course. Otherwise treatment was symptomatic. No complications were noted.

There were five families in which two members were affected. Contact could also be traced between another pair and probably between two others. No definite conclusions could be reached on the incubation period, but it was possibly between four and six days. When reviewed in October, five of the patients stated that pain lasting one day recurred two weeks after their first attack.

Pleural Friction.—The onset of pleural friction rub four to ten days after the onset of symptoms is noteworthy, as also is the age group—16-35 years. The incidence of pleural rub was almost one in five. Scadding (1946) reported cases which he called "acute benign dry pleurisy." Eleven out of his 20 cases had pleural friction. Finn, Weller, and Morgan (1949) reported 28 cases with pleural friction in their series of 114 cases.

Summary

The main features of an outbreak of 24 cases of Bornholm disease are described.

Observations are made on certain of the features, especially the occurrence of pleural friction rub.

We are grateful to Drs. P. Aitken and J. Rubin, radiologists at Dunfermline and West Fife Hospital, for their x-ray reports.

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Medical Memorandum

Pneumatic Rupture of the Colon

As pneumatic rupture of the colon has rarely been reported in British medical literature the case here described is of interest.

CASE REPORT

A lad aged 16½, employed as an apprentice fitter at an engineering works, was admitted to hospital on October 20, 1949. He complained of difficulty in breathing and of abdominal pain. His story was that while at work, about an hour before admission, two of his workmates had placed the nozzle of a compressed-air pipe between his legs and turned on the air. He said that he had felt suddenly blown up and had found that he could breathe only with difficulty. He had some pain in the lower abdomen but had not vomited.

Examination showed a pale, shocked boy. His lips were cyanosed and he was extremely dyspnoeic, with marked movement of his alae nasi. The respirations were shallow and grunting in type. The abdomen was very distended and tympanitic, with tenderness on deep pressure and absence of peristaltic sounds. An interesting feature of the abdomen was a curious reddish-blue mottling on the abdominal wall, particularly in the flanks. The blood pressure was 150/72. There was no evidence of any surgical emphysema. On rectal examination nothing abnormal was felt, but withdrawal of the finger was followed by a profuse discharge of blood-stained faecal-smelling fluid. A diagnosis of pneumatic rupture of the colon was made. A Ryle's tube was passed and the patient was prepared for operation.

Under general anaesthesia, the abdomen was opened through a left paramedian incision. This was easily done,