(1947) in which the local condition cleared up in a matter of days but the general malaise was considerably prolonged.

Acute tenderness caused by pressure over the zygoma was noticed in Case 2, and thereafter was elicited in Cases 3 and 4. It was not noticed in Case 1, perhaps because that area was not palpated, but in Case 2 the provisional diagnosis of osteitis of the zygoma was made, owing to the situation and degree of the tenderness. This point was about 1 in. (2.5 cm.) behind and below the external canthus—a point quite distinct from that found in tenderness due to enlarged preauricular gland—and presents an interesting feature for speculation. It may be that the sensory nerve supply for this area of skin is derived from the zygomatic branch of the maxillary nerve, and as a result of the communication between the lacrimal and zygomatic nerves tenderness in this region reached a high degree. No reference to this phenomenon could be found in the literature, yet it appears to be characteristic of acute dacryoadenitis at an early stage, when swelling of the lid is too tense to permit palpation of a lump.

Ptosis was present on each occasion, and owing to the situation of the swelling, which was maximal at the lateral aspect of the lid, a sinuous outline was given to the lid margin. This appearance has been noted by Richardson (1942) and by Rosen (1944).

Restriction of eye movement was found in all patients in the early stages of the gland infection; in two it amounted to almost complete immobility of the eye, and a notable feature was the comparative slowness in recovery of full eye movements.

Proptosis was a pronounced feature in one instance. This patient had had the most severe general reaction, and one might visualize an abscess in the gland which had extended backwards to cause an orbital cellulitis.

Chemosis was seen in all patients, chiefly affecting the temporal bulbar conjunctiva. In two it caused bulging of the conjunctiva between the lid margins.

In conclusion, it may be stated that acute dacryoadenitis is not uncommon in some districts and at certain times of the year. Schmidt (quoted by Schirmer, 1877), stated that the condition was not uncommon; Wright and Nayar (1937) described three cases which seemed to be caused by *Diplobacillus morax-axenfeld*. Epidemics have been reported from time to time (Galezowski, 1875; Groenouw, 1889 (quoted by Nicolau, 1933); Nicolau, 1933; Toulant, Benhamou, and Foissin, 1947). The seasonal incidence is a matter of some interest; Nicolau described 10 cases which occurred during the winter months, and, on investigating 81 instances previously reported, she found an increase in numbers during the months of October to March, those months in which respiratory infection is most common. Respiratory infection may be a frequent cause of unilateral acute dacryoadenitis.

It appears that there may be a latent interval of two to three weeks during which the patient feels quite well before orbital symptoms become manifest; only careful history-taking will reveal that there has been a period of headache, malaise, sore throat, and feverishness. Fralick (1935) makes the following comment: "Seldom is the practising ophthalmologist called upon to treat conditions affecting the lacrimal gland. Consequently, little is written in the standard textbooks on the subject of acute inflammation of this structure. It is therefore conceivable that a few cases may leave our offices

improperly diagnosed because of our unfamiliarity with this particular group."

Summary

Four patients with acute dacryoadenitis have been seen during a six-months period. The clinical features are described and discussed. Attention is drawn to the following points: (1) the rarity of acute dacryoadenitis, (2) its association with respiratory infection, (3) its seasonal incidence, and (4) an early diagnostic sign of acute dacryoadenitis consisting of tenderness over the malar bone.

I would like to thank Dr. Maxwell Telling for much valuable criticism and for access to Case 4, and Mr. John Foster and Mr. George Black for their help and for allowing me to describe their patients.

BIBLIOGRAPHY

- Benford, M. C., and Brunner, H. (1947). *Amer. J. Ophthalm.*, **30**, 297.
 Boley, J. P., and Taylor, R. E. (1940). *J. Canad. med. Ass.*, **43**, 66.
 Byers, W. G. M. (1912). *Trans. Amer. ophthalm. Soc.*, **13**, 135.
 Foster, M. L. (1925). *Ibid.*, **23**, 50.
 Fralick, F. B. (1935). *Amer. J. Ophthalm.*, **18**, 19.
 Galezowski, X. (1875). *Recu. Ophthalm.*, **2**, 270.
 — (1886). *Ibid.*, **8**, 415.
 — (1892). *Ibid.*, **14**, 594.
 Nicolau (1933). *Ann. Oculist., Paris.*, **170**, 772.
 Pooley, T. R. (1884). *Trans. Amer. ophthalm. Soc.*, **3**, 729.
 Richardson, J. M. (1942). *Arch. Ophthalm.*, **Chicago**, **28**, 93.
 Rosen, E. (1944). *Amer. J. Ophthalm.*, **27**, 276.
 Schirmer, R. (1877). In A. Graefe and E. T. Saemisch's *Handbuch der Gesamten Augenheilkunde*, **7**, 1. Engelmann, Leipzig.
 Thomson, StC., and Negus, V. E. (1948). *Diseases of the Nose and Throat*, 5th ed., p. 297. Cassell, London.
 Toulant, Benhamou, and Foissin (1947). *Bull. Mém. Soc. franç. Ophthalm.*, **60**, 23.
 Weeks, J. E. (1911). *Treatise on Diseases of the Eye*, p. 190. Kimpton, London.
 Wright, R. E., and Nayar, K. K. (1937). *Brit. J. Ophthalm.*, **21**, 367.

BORNHOLM DISEASE

BY

T. GEFFEN, M.D., M.R.C.P.

Late Medical Registrar, Hampstead General and North-west London Hospital

Outbreaks of Bornholm disease have been described in this country at infrequent intervals since the work of Pickles (1933) in Yorkshire. The subject was recently reviewed by Hopkins (1950), who reported a further series of cases; and the possible connexion between this disease and the Coxsackie group of viruses was discussed by Findlay and Howard (1950). Very shortly after the publication of the latter two papers a small group of patients attended this hospital with clinical histories corresponding closely to those reported in previous descriptions of Bornholm disease. Dr. G. M. Findlay very kindly attempted to isolate Coxsackie virus from the nasal washings of some of the patients and to carry out the serological antibody reactions on them from the earliest stages of the outbreak. The clinical histories of these patients are briefly described, together with a summary of Dr. Findlay's findings.

Case Reports

Case 1.—A girl aged 18 first noticed mid-thoracic pain and pain behind the right shoulder on May 28, 1950. It was worse on inspiration but was not severe, and she did not mention her symptoms until three days later, when two

other members of her family had been similarly affected. She was found to have a temperature of 101° F. (38.3° C.), but the fever settled within two days, and she was then free of all symptoms. This girl did not attend hospital at any time and so no investigations could be carried out on her. A fortnight later, however, she was reported to be completely well.

Case 2.—On May 29 a schoolboy aged 14, a brother of Case 1, complained of a stabbing pain on the left side of the chest on inspiration. He had previously had a heavy head cold for a few days but had had no other symptoms. The onset of his pain was quite abrupt while he was sitting in a chair. On admission his respiration was shallow and was obviously causing pain. His temperature was 104.4° F. (40.2° C.), pulse 110, and respirations 28. Movement and air entry on the left side of the chest were diminished and a friction rub could be heard over a small area in the front of the chest, just to the left of the sternum. No other abnormal physical signs were found. No chemotherapy was given, and within a day the pleural rub had gone, though the pain on inspiration persisted; on the second day he developed epigastric pain on respiration. The temperature was 101.4° F. (38.55° C.) on June 1 and then slowly settled to normal during the following week. A blood count showed: haemoglobin, 80%; leucocytes, 9,000 per c.mm. (69% polymorphs, 25% lymphocytes, 6% monocytes). The sedimentation rate was 15 mm. in the first hour. A chest radiograph showed no abnormality. The patient was discharged perfectly fit on June 19.

Case 3.—On May 30 another brother of Case 1, aged 12, complained of epigastric discomfort, headache, and general malaise, and was found to have a temperature of 101° F. (38.3° C.). Later in the day he developed a sharp pain in the left upper chest, which, though not severe, was increased by deep breathing. On the next day his chest pain was worse and his abdominal pain was also exacerbated by breathing, so he was admitted to hospital. On admission he was hot and flushed but not unduly distressed. His temperature was 100.6° F. (38.1° C.), pulse 110, and respirations 26. A few lymph nodes were palpable in both axillae, but no other abnormal physical signs were found in the chest or elsewhere. Within a day his pain had gone and the temperature settled after a further three days. A blood count showed: haemoglobin, 78%; leucocytes, 9,000 per c.mm. (neutrophils 45%, lymphocytes 38%, monocytes 14%, eosinophils 3%). The sedimentation rate, uncorrected, was 21 mm. in the first hour. A chest radiograph was clear. He was discharged fit on June 10.

Case 4.—On June 1 a married sister aged 21 complained of cough and of sharp pain in the right side of the chest and behind the lower end of the sternum. The pain was increased by deep breathing and by coughing. She felt feverish all that day, and was still feeling ill three days later. Despite this she attended hospital to visit her brothers and was examined there. She then had a temperature of 99.6° F. (37.55° C.), but no other abnormality was found on physical examination. As she was breast-feeding her 5-weeks-old infant it was impossible for her to come into hospital, but she was seen again on subsequent visits to her brothers. Although on her second visit on June 11 the fever and pain had settled, she still felt unwell, but further physical examination again revealed no abnormality and the chest radiograph was clear. This patient did not feel completely well for a further three weeks.

There were seven members of this family. The mother, aged 43, shared a room with a son aged 4, and Case 1. Case 2 shared a second room with a 12-year-old brother. Case 3 shared a room with a 7-year-old adopted brother. Case 4 and her baby lived in a near-by flat, her husband at the time of the outbreak being away from home. Case 4 was a daily visitor to her family. It is curious that only one occupant of each room was affected, whereas, in the outbreak re-

ported by Hopkins, every member of two affected households had symptoms of the disease.

Case 5.—On June 11, 10 days after the admission of Case 3, a boy aged 7½, who had been admitted on May 26 with a boil on the arm and who occupied a bed next to Case 3, developed a sudden spike of fever of 102° F. (38.9° C.) and complained of pain in the left side of his chest increased by inspiration. On examination the respiration rate was 24 and the pulse rate 120. There was diminution of movement and air entry on the left side of the chest, but there was no friction rub nor any other abnormal findings. His temperature was still 99.6° F. (37.55° C.) on June 13, but settled completely, and he was perfectly well when discharged on June 16. On June 12 a blood count showed: haemoglobin, 90%; leucocytes, 7,000 per c.mm. (neutrophils 55%, lymphocytes 20%, eosinophils 12%, basophils 2%, monocytes 10%, plasma cells 1%). The sedimentation rate, corrected, was 18 mm. in the first hour. A chest radiograph was clear. The patient attended hospital on August 22; he then felt perfectly well and a chest radiograph was again normal.

Case 6.—A boy aged 16, the last patient in this series, had no connexion with any of the earlier cases nor was there a history of contact with anyone else with a similar illness. Although the serological investigations were negative, the course of his illness was so like that of the others that he is included in the series. He was admitted on June 16 with a one day's history of pain across the upper part of the chest, worse on deep inspiration, and of epigastric pain. During that time he had also noted nausea and anorexia and frontal headache, but had not vomited. On examination his temperature was 102° F. (38.9° C.), pulse 95, and respirations 24. Palpable glands were present in the posterior triangle of the neck on both sides. There was tenderness to palpation right across the lower abdomen. No abnormalities were detected in the chest or elsewhere. Investigations: haemoglobin, 98%; white cells, 6,000 per c.mm. (neutrophils 65%, lymphocytes 28%, monocytes 6%, eosinophils 1%). A blood count on June 30 was substantially the same. The Mantoux intradermal test was positive at a dilution of 1:1,000, and a chest radiograph was normal. This patient ran a swinging pyrexia until June 25, after which he settled down completely and was well on discharge on July 5.

No meningeal or paralytic signs were observed in any case in this series.

Further Investigations

Attempts were made to isolate Coxsackie virus from the nasal washings of Cases 2, 3, and 4. The results were negative in the first two cases, but the virus was isolated in the nasal washings from Case 4 on the second day of her illness.

The complement-fixation reactions against Coxsackie virus 2 were as follows:

		Day of disease	Titre
Case 4	...	2	Negative
		5	1:10
		18	1:20
Case 3	...	3	1:5
		6	1:10
		19	1:20
Case 2	...	2	1:5
		5	1:5
		18	1:10
Case 5	...	3	1:5
		77	1:10

One of the contacts gave a titre of 1:10 during the first and third weeks after the outbreak, but the other two members of the family who were tested never showed a titre greater than 1:5.

Case 6 gave negative results on the second and nineteenth days of his illness.

Discussion

These six patients all had clinical histories and findings which were essentially similar, and which, furthermore, closely resembled previous descriptions of Bornholm disease. The condition was in fact suspected on the admission of Case 1 even before the occurrence of further cases in the family helped to establish the diagnosis. The first five patients, forming as they did a small group, could be fairly confidently diagnosed as suffering from Bornholm disease. In these cases the connexion previously described between Bornholm disease and Coxsackie-virus infection (Curnen, 1950) was confirmed by the isolation of virus in one case and by a rising agglutination titre in all four of the five patients who were available for testing; no such confirmation was obtained in Case 6, and the final diagnosis of this patient cannot be regarded as established.

Coxsackie virus was first isolated from two paralytic patients during an epidemic of poliomyelitis (Dalldorf and Sickles, 1948). During 1947 and 1948 there were reports from the United States of various outbreaks of febrile illness in which many cases resembled non-paralytic poliomyelitis. In some of these cases poliomyelitis virus was isolated, but in others faecal specimens and nasopharyngeal washings tested for poliomyelitis virus were non-infective for monkeys (Dalldorf, 1950). During these outbreaks Coxsackie virus was isolated from patients with various minor illnesses, with illness resembling epidemic myalgia (Bornholm disease), and with illness suggesting non-paralytic poliomyelitis. In some but not all of the last group poliomyelitis virus was also isolated (Curnen, 1950).

In some previously reported cases of Bornholm disease signs of meningeal irritation have been noted. DeRudder (1937) drew attention to the similarities between epidemic myalgia and poliomyelitis, although his views were to some extent disputed by Lindberg (1938). The similarities were also stressed more recently by Ronse (1949).

There is of course no reason why patients should not harbour both poliomyelitis and Coxsackie virus at the same time, and this association has in fact been recorded (Melnick, 1950).

The similarities and differences between these two groups of virus are summarized by Melnick (1950), who concludes that, though antigenically distinct, their biological behaviour is surprisingly similar. He points out, furthermore, that members of the Coxsackie group of viruses are now being repeatedly found where poliomyelitis is epidemic.

I am grateful to Drs. H. V. Morlock and E. W. Hart, under whose care these patients were treated, for their permission to publish this article, and to Dr. L. Cohen, who brought four of the patients to our notice. I am also very much indebted to Dr. G. M. Findlay, who carried out all the virological investigations, for his generous assistance.

REFERENCES

- Curnen, E. C. (1950). *Bull. N.Y. Acad. Med.*, **26**, 335.
 Dalldorf, G. (1950). *Ibid.*, **26**, 329.
 — and Sickles, G. M. (1948). *Science*, **108**, 61.
 DeRudder, B. (1937). *Klin. Wschr.*, **16**, 585.
 Findlay, G. M., and Howard, E. M. (1950). *British Medical Journal*, **1**, 1233.
 Hopkins, J. H. S. (1950). *Ibid.*, **1**, 1230.
 Lindberg, G. (1938). *Klin. Wschr.*, **17**, 532.
 Melnick, J. L. (1950). *Bull. N.Y. Acad. Med.*, **26**, 342.
 Pickles, W. N. (1933). *British Medical Journal*, **2**, 817.
 Ronse, L. (1949). *Brux.-méd.*, **29**, 3411.

TUBERCULOSIS OF LIVER TREATED WITH STREPTOMYCIN

BY

IAN D. MELVILLE, M.B., Ch.B.

House-physician, Western Infirmary, Glasgow

The following case is of interest on account of its rarity and its clinical course.

Case Report

The patient, a student aged 20, was admitted to hospital on March 14, 1950. He enjoyed good health until June, 1949, and was living in a college pleasantly situated in the country. He noticed, however, that during the first two weeks of his summer vacation in 1949 he was becoming easily tired, could find little energy for his usual holiday activities, and had diarrhoea. Some few days later his stools became very pale and offensive, and he noticed that his eyes "were becoming yellow." A few weeks later he was drenched in a rain-storm, after which severe pain developed in the right loin accompanied by anorexia and malaise. He was confined to bed for seven days, and was seen to be slightly jaundiced. His condition improved with rest, and he resumed his usual activities. Before his return to college in September, however, two of his teeth were extracted; haemorrhage was profuse and persisted over four or five days in spite of plugging. His general condition as a result became very poor.

Lest any absence due to illness should mean the sudden end of his scholastic career, he returned to college, although he felt very weak. He had been advised to take iron, and with this therapy his pallor became less severe. His jaundice persisted, and during the last months of 1949 gradually became more intense. He began to have frequent sweats, often profuse, and especially at night. At the end of December he suffered a minor cut on one of his hands, and again the bleeding was prolonged. A vitamin-K analogue—self-prescribed—made no difference to this tendency to bleed. Bleeding from the gums occurred, becoming more and more frequent until it was a daily event. His appetite at this time was fairly good, and he was passing two or three loose pale motions each day. The urine was deep yellow in colour. He had no cough, and, so far as he could judge, had not lost weight.

His past history was of appendicitis at the age of 7 years and section of peritoneal adhesions at 15, when there was suspicion of mesenteric tuberculous adenitis. No proof of this was ever obtained. The family history shed no light on his illness.

Examination on admission showed him to be a tall lean youth, pale and slightly jaundiced. He was febrile and was sweating freely; his fingers were clubbed; enlarged lymph nodes were not detected. The left elbow-joint was painful and tender, its movements being limited to about a right-angle. The abdomen was distended and the liver slightly enlarged. The spleen and kidneys were not palpated. Examination of the chest disclosed no abnormality, and the cardiovascular and central nervous systems appeared to be normal.

Blood examination revealed an iron-deficiency anaemia: Hb, 76% Sahli (10.64 g.); red cells, 4,580,000 per c.mm.; colour index, 0.8; white cells, 8,000 per c.mm. The differential count gave average readings. The bleeding-time was 4½ minutes by Duke's method, and the coagulation time 8 minutes by the method of Lee and White. The red-cell-fragility test showed commencing haemolysis at a concentration of 0.5% NaCl, complete at 0.35%. The urine contained bile and urobilin. No blood or casts were seen on microscopical examination.

Diagnosis was at this point obscure, and many investigations were carried out. Radiological examination of the