of tenderness, and Steinbrocker *et al.* (1948) have described the painful stiff-shoulder syndrome in coronary disease. There is no doubt that fibrositic lesions of the chest wall and painful shoulder-joints are very common accompaniments of coronary disease, and it would be interesting to know how many of these patients are to be found attending orthopaedic clinics, their cardiac condition undiagnosed.

The summation of pain from the heart with pain arising from some local condition in the chest wall introduces a further difficulty in diagnosis, and Cohen (1947) has postulated this explanation of some cardiac pain being referred to particular areas of the same segmental supply. An old gunshot wound or some area of fibrosis may become the focal point for the expression of an underlying cardiac pathology; it is therefore important to realize the danger of being too readily satisfied with a local chest-wall explanation of a patient's symptoms in any case where he is a potential sufferer from heart disease.

The reverse mistake of diagnosing heart disease when the trouble lies entirely in the chest wall is, however, more frequent and disastrous. It might well be said that it is better that one patient should suffer a sudden cardiac death within a few hours of a reassuring prognosis than that one should go through life with a mistaken label of cardiac disease.

In elderly patients who have a doubtful chest pain and some arteriopathy no serious harm is done by reasonable limitation of activity on general grounds, but cases in the younger age group demand a more accurate diagnosis, and if after complete investigation there remains some doubt it is much better that the heart should be passed as normal.

Summary

The differential diagnosis of chest-wall pain is discussed and stress is laid on the need for due consideration to be paid to local structural causes as well as to the possibility of an underlying cardiac pathology. Some illustrative cases are briefly described.

REFERENCES

- Cohen, H. (1947). *Lancet*, **2**, 933.
 Copeman, W. S. C. (1949). *British Medical Journal*, **2**, 191.
 Davis, L., and Pollock, L. J. (1932). *Arch. Neurol. Psychiat.*, **27**, 282.
 Kellgren, J. H. (1947). *Lancet*, **2**, 440.
 Steinbrocker, O., *et al.* (1948). *Ann. intern. Med.*, **29**, 22.
 Warburg, E. (1938). *Subacute and Chronic Pericardial and Myocardial Lesions due to Non-penetrating Traumatic Injuries*. Oxford Univ. Press.

In the U.S.A. the Communicable Disease Centre is the division of the Public Health Service responsible for providing assistance to State health departments in meeting public health emergencies. According to Surgeon-General L. A. Scheele the most extensive investigation carried out by the Centre in 1949 was in Muskegon, Michigan, in August and September. An epidemiological team composed of 12 persons conducted there one of the most thorough studies ever made of a community in connexion with a poliomyelitis epidemic. Some phases of this research are to continue for several years. "An interesting feature of the poliomyelitis investigation in Arkansas in August," Dr. Scheele said, "was the use of a 'circuit rider,' not unlike the preacher on horseback who toured rural areas of the South around the turn of the century. The 1949 circuit rider in this case was a public health representative. He made rounds three times a week, collecting from field workers certain important specimens for laboratory study." A physician-veterinarian team was sent from the Communicable Disease Centre to investigate a baffling epidemic which afflicted human beings and horses at the same time last August and September in Colorado. They found that two virus diseases existed simultaneously, creating a complicated diagnostic problem. Poliomyelitis was prevalent among human beings at a time when encephalitis was occurring in both man and horses.

CANICOLA FEVER IN BRISTOL

CLINICAL, BACTERIOLOGICAL, AND EPIDEMIOLOGICAL NOTES ON SIX HUMAN CASES

BY

A. M. G. CAMPBELL, D.M., M.R.C.P.

Physician, Bristol Royal Hospital

J. MACRAE, M.D., F.R.F.P.S.

Resident Physician, Ham Green Hospital, Bristol

W. G. MANDERSON, M.B., F.R.F.P.S.

Deputy Resident Physician, Ham Green Hospital, Bristol

K. C. SUMNER, M.R.C.V.S.

Bristol

AND

J. C. BROOM, O.B.E., M.D.

Bacteriologist, Wellcome Research Institution, London

Canicola fever is an acute infective disease caused by *Leptospira canicola*. This organism belongs to the same genus as *L. icterohaemorrhagiae*, which produces Weil's disease (leptospirosis or infective jaundice), and is normally a parasite of dogs. It occasionally infects man, giving rise to an illness which may simulate influenza or meningitis, and which may be accompanied or followed by eye symptoms. The prevalence of the condition in the Low Countries has been demonstrated by a number of workers since the species was first isolated there by Klarenbeek and Schüffner (1933), but its existence in this country has been appreciated only within recent years. We here record six cases to add to the six previously reported in Britain by Baber and Stuart (1946), Laurent *et al.* (1948), and Weetch, Colquhoun, and Broom (1949).

In two of our cases canicola fever was not diagnosed during the febrile attacks, and was suspected only when ocular complications supervened. The patients, a husband and wife, were referred to the Bristol Eye Hospital on August 10, 1949, both complaining of dimness of vision in one eye following an influenzal illness. They were found to be suffering from iridocyclitis, and were sent on August 22 to the medical clinic of the hospital for complete examination by one of us (A.M.G.C.). When the history of the febrile attacks was reviewed it was noted that both patients had been nursing a sick dog before the onset of the illness. The husband, an engineer, constantly had abrasions on his hands, and the wife had a cut finger at that time.

The possibility that the pyrexial attacks had been canicola fever was considered, and specimens of serum were tested for the presence of antileptospirosis antibodies. The sera of both patients agglutinated *L. canicola* to high titre, so confirming the retrospective diagnosis of the cause of the febrile illnesses and the iridocyclitis.

These cases occurred in a so-called "squatters' camp." The term is likely to convey a completely wrong impression of the general hygiene and living conditions—which in fact are very good—so it will not be used. The community, inhabiting former American Army huts, was relatively circumscribed, and through the co-operation of its members we were able to investigate the agglutination reactions and to make ophthalmoscopic examinations of most of the adults. Serum from all the dogs on the site was also tested. The results obtained, and certain conclusions we think may be drawn from them, seem of sufficient interest to be given in some detail.

Four other cases which occurred recently in Bristol are described also. These patients had no known association with each other or with those already mentioned, although two of them live in the district in which the "camp" is situated. Their inclusion, however, gives a more complete picture of the disease in that city, and in addition one of them illustrates a different method of contracting the infection.

Disease in Man

The reports of the primary attacks of the first two cases are rather scanty because they were not treated in hospital and no written records are available. Both patients, however, gave every help in trying to make the information as accurate as possible.

Case 1

Mrs. A., aged 29, had a febrile illness which began abruptly on June 21, 1949, and was accompanied by very severe headache of meningeal type, pains in the neck and limbs, marked conjunctival injection, nausea, and vomiting. The fever ran a somewhat relapsing course over a period of 10-14 days, during which time the patient was severely ill; in all she was confined to bed for three weeks. On the eleventh day an erythematous rash appeared on the trunk and limbs, but this faded within twenty-four hours. On the thirteenth day she complained of dimness of vision affecting the nasal field of the left eye, which was red and inflamed. Spots and haziness in front of the eyes were also noted.

Eye Examination (F. Clifton).—On August 10 (51st day) the right eye was healthy. The left eye presented signs of low-grade iridocyclitis, and there were a few medium-sized grey keratic precipitates, occasional cells in the aqueous, and a slight flare, with a few flake-like opacities in the vitreous. The retina and choroid seemed normal. There were no visual field defects.

On August 22 (63rd day) the patient was free from all signs and symptoms except the above ocular disturbances. Blood examination showed: leucocytes, 7,800 per c.mm. (polymorphs 55%, eosinophils 4%, basophils 1%, lymphocytes 35%, monocytes 5%). Wassermann reaction and Kahn test were negative. The urine was normal on examination. Serum agglutinated *L. canicola* in dilutions of 1 in 3,000.

Case 2

Mr. A., a Post Office engineer aged 33, was taken suddenly ill on July 14, 1949. The symptoms and course of the complaint resembled those of Case 1, but pain and tenderness of the abdominal muscles were outstanding features. Vomiting was marked, and colicky pains accompanied by severe diarrhoea occurred on the seventh and eighth days of the disease. The total duration of his illness was about three weeks. On July 20 (7th day) photophobia and diminution of vision were noted in the left eye, which was red and painful.

Eye Examination (F. Clifton).—On August 10 (28th day) the right eye was healthy. The left showed iridocyclitis, numerous fine to medium-sized grey keratic precipitates, a faint injection of the iris, cells and a moderate flare in the aqueous. The vitreous showed a generalized haze and a number of filamentous floating opacities. The fundus appeared normal except for the presence of a small inactive paramacular pigmentary disturbance, which did not cause any detectable scotoma in the visual field.

On August 22 (40th day) this case presented exactly the same appearances as Case 1. Blood examination showed: leucocytes, 5,800 per c.mm. (polymorphs 40%, eosinophils 6%, basophils 1%, lymphocytes 48.5%, monocytes 4.5%). The urine was normal, and the blood Wassermann reaction negative. Agglutination tests with *L. canicola* were positive to a titre of 1 in 10,000.

Case 3

A schoolgirl aged 13 developed severe frontal headache on May 23, 1949, followed on May 25 by abdominal pain and vomiting. These symptoms persisted, and on May 27 (5th day) stiffness of the neck was noted; the patient was sent to hospital as a possible case of cerebrospinal meningitis.

On admission on May 27 the temperature was 100.4° F. (38° C.) with a pulse rate of 100 a minute. The patient appeared ill, and neck rigidity was pronounced, with a positive Kernig's sign and some enlargement of the axillary lymph glands. There was tenderness in the left iliac fossa. The eyes and conjunctivae appeared normal, and clinical examination revealed no further abnormalities.

Investigations.—Blood: Leucocytes, 8,000 per c.mm. (polymorphs 76%, lymphocytes 20%, monocytes 4%). Cerebrospinal fluid: Clear, colourless; 20 cells per c.mm. (lymphocytes 93%); total protein, 30 mg. per 100 ml., no excess of globulin; chlorides, 680 mg. per 100 ml.; sugar present; no bacteria seen in films; culture sterile. Urine normal, and sterile on culture.

Treatment was begun with sulphathiazole, 1 g. every four hours.

On May 29 (7th day) lateral nystagmus was noted in both eyes, but temperature and pulse were normal; abdominal pain, headache, and neck rigidity had disappeared, and Kernig's sign was negative. On May 30 the temperature was 101.2° F. (38.4° C.) and the pulse rate 90 a minute; nystagmus was still present. Headache and vomiting returned, and pharyngitis and conjunctival injection developed. Sulphathiazole was stopped, and penicillin substituted in a dosage of 200,000 units daily.

On May 31 (9th day) the conjunctival suffusion was less severe and the patient felt much better. A blood sample taken that day agglutinated *L. canicola* to a dilution of 1 in 30 (see Table). By June 3 (12th day) all symptoms had abated. Con-

Serum Agglutination Reactions of Cases 3 and 4

		Reciprocal of Titre with <i>L. canicola</i>		
Case 3	..	30 (9)	300 (16)	10,000 (30)
Case 4	..	30 (13)	300 (19)	1,000 (26)

The day of disease on which specimen was taken is shown in parentheses.

valescence thereafter was slow but uninterrupted, and the patient was discharged on July 4 (43rd day).

An unsuccessful effort was made to trace the source of the infection. Blood from a dog in the patient's house was negative for leptospirosis. A neighbour's dog had been sick and showing eye symptoms shortly before the onset of the child's illness, but it had been killed accidentally during the early part of the girl's stay in hospital.

Case 4

This patient, a schoolboy aged 11, who had often bathed in the River Frome, a very stagnant stream, became suddenly ill on July 14, 1949, with severe frontal headache and persistent vomiting. The boy was taken into hospital on July 21 (7th day) as a possible case of cerebrospinal meningitis.

On admission his temperature was 98.8° F. (37.1° C.) and the pulse rate 100 a minute. The patient was moderately ill and complained of headache. There was some neck rigidity, and Kernig's sign was positive, but no other signs of cerebral involvement were present.

Investigations.—Blood: Leucocytes, 8,000 per c.mm. (polymorphs 73%, eosinophils 2%, lymphocytes 21%, monocytes 4%). Cerebrospinal fluid: Clear; 180 cells per c.mm. (lymphocytes 66%); total protein, 65 mg. per 100 ml., with increased globulin; chlorides, 710 mg. per 100 ml.; sugar present; sterile on culture.

Treatment with 200,000 units of penicillin daily was begun, being continued until July 28 (14th day). On July 22 headache was still severe, and neck rigidity persisted. Blood taken on this day showed a low positive agglutination titre with *L. canicola* (see Table). On July 25 (11th day) the symptoms were beginning to abate. The lumbar cerebrospinal fluid contained 15 cells per c.mm. (lymphocytes 95%, polymorphs 4%, monocytes 1%); total protein, 10 mg. per 100 ml.; chlorides, 750 mg. per 100 ml.; sugar present; culture sterile. By July 27 (13th day) all symptoms had disappeared, and he made an uninterrupted recovery.

Case 5

A schoolgirl aged 15 had a febrile attack on October 2, which at first responded to treatment with salicylates. On

October 9, however, she developed a severe frontal headache, and her temperature rose rapidly. She was admitted to hospital as a suspected case of anterior poliomyelitis.

On admission her temperature was 101° F. (38.3° C.), pulse rate 86 a minute, and respirations 20 a minute. Neck and spinal rigidity was present, and Kernig's sign was positive. A cardiac systolic murmur was heard in the mitral area, the sequel to an attack of rheumatic fever 10 years previously. There were no disturbances of reflexes, no muscular weakness, and no abnormalities in the other systems.

Investigations.—Cerebrospinal fluid: Leucocytes, 780 per c.mm. (lymphocytes 90%, polymorphs 10%); total proteins, 65 mg. per 100 ml., with excess globulin; chlorides, 730 mg. per 100 ml.; sugar present; no organisms in stained films; culture negative. The serum agglutinated *L. canicola* to a titre of 1 in 300.

On October 10 (9th day) the temperature returned to normal; the headache and the neck and spinal rigidity improved, and had cleared completely by October 12. On October 14 (13th day) the temperature rose again to 103° F. (39.4° C.) and fever continued for three days. During this period nothing abnormal was found on examination of the central nervous system, but on October 17 (16th day) the cerebrospinal fluid contained 275 cells per c.mm. (lymphocytes 89%, polymorphs 10%, monocytes 1%). Thereafter the patient made a slow but uninterrupted recovery, and was discharged on November 15 (45th day). A specimen of serum agglutinated *L. canicola* at 1 in 1,000.

A dog belonging to the patient's household had been sick, with suffused eyes, about two weeks before the onset of the child's illness. The dog had had to be destroyed, so it was not possible to determine whether it was the source of infection.

Case 6

A railway employee aged 31 had a shivering attack on December 10, 1949. Next day he complained of severe frontal headache, and pains in the back, knees, calf muscles, and ankles. He had a dry persistent cough. During the next two days these symptoms continued, and profuse night sweats occurred. He was admitted to hospital on December 14.

On admission his temperature was 103° F. (39.4° C.), pulse rate 98 a minute, and respirations 28. The patient was acutely ill, pale, and in obvious pain. There was marked suffusion of both eyes. The axillary lymph glands were palpable on both sides. Movement of the right side of the chest was diminished; numerous rhonchi were heard throughout the lung fields and "sticky" rales over the left base posteriorly. No other abnormalities were noted during the clinical examination.

Investigations.—Blood: Leucocytes, 14,000 per c.mm. (polymorphs 76%, lymphocytes 24%). The urine contained albumin and granular and hyaline casts. A specimen of serum showed no leptospiral agglutinins.

Treatment with 200,000 units of penicillin daily was begun on admission and was continued for 14 days.

The temperature fell by lysis, and had reached normal by December 16 (7th day), but the headache persisted. Next day it became much worse, and was frontal and retro-orbital in position. The temperature rose to 100.6° F. (38.1° C.); the pulse rate was 96 a minute and respirations 28. Neck and spinal rigidity developed, and Kernig's sign was positive. Lateral nystagmus was present in both eyes. Cerebrospinal fluid: Clear; 225 cells per c.mm. (lymphocytes 79%, polymorphs 20%, monocytes 1%); total protein, 170 mg. per 100 ml.; sugar present; globulin increased; chlorides, 700 mg. per 100 ml.; no organisms seen in films; cultures sterile.

On December 18 (9th day) the temperature fell to normal, but the meningeal signs and symptoms cleared only slowly. The neck and spinal rigidity did not finally disappear until January 1, 1950 (23rd day). On this date the serum agglutinated *L. canicola* in dilutions up to 1 in 1,000.

On December 23 (14th day) the cerebrospinal fluid contained 250 cells per c.mm. (lymphocytes 81%, polymorphs 18%, monocytes 1%); total proteins, 130 mg. per 100 ml., globulin in excess: chlorides, 715 mg. per 100 ml.; sugar present.

On January 5, 1950 (27th day), the cerebrospinal fluid findings were: cells, 35 per c.mm. (lymphocytes 83%, polymorphs 16%, monocytes 1%); total protein, 50 mg. per 100 ml., with slight excess of globulin; chlorides, 740 mg. per 100 ml.; sugar present; no organisms were seen in films, and cultures remained sterile. At the time of writing (28th day) he still complains of frontal headache in the morning, but his general condition is good, and he is allowed out of bed.

The patient owns a dog which became ill with "distemper" some three weeks before the onset of the man's symptoms. The dog's conjunctivae were noticed to be yellow, and were covered with a film of mucus. The animal recovered but relapsed later, and on January 9 was again acutely ill. Serum taken from the animal at that time agglutinated *L. canicola* in dilutions up to 1 in 10,000, from which it can reasonably be assumed that the man's illness was contracted from his dog.

Disease in Dogs

A bitch belonging to the first two patients suffered from an attack of fever, with gastritis and vomiting, in December, 1948. It is impossible now to decide whether this was leptospirosis, but the animal's serum agglutinated *L. canicola* to a titre of 1 in 100 when tested in September, 1949. At that time no leptospirae could be demonstrated in the urine either by microscopical examination or by inoculation into hamsters. The same bitch produced a litter of six puppies on April 20, and both patients of course had often to mop up pools of urine. In September the sera of the two remaining puppies had positive titres of 1 in 1,000 for *L. canicola*, and leptospirae were seen on more than one occasion in the urine of one puppy. Urine was inoculated into hamsters, but only after treatment with penicillin had inadvertently been carried out, and no strain was isolated.

At the age of 5 weeks this puppy developed an abscess of the neck, and was under the care of one of us (K. C. S.) up to June 12. This has no aetiological significance, but it enables us to state quite definitely that neither bitch nor puppies showed any systemic disturbance. The importance of this point will be considered later, in relation to the carrier state.

Epidemiological Investigations

The living quarters of the families in the colony may be compared to the single-story prefabricated houses which are a common feature in bomb-damaged towns. The huts, substantially built of wood and raised two to three feet (60–90 cm.) above the ground, are provided with water and electricity. Sanitary arrangements are perfectly adequate. Separate rooms have been partitioned off, the interiors freshly decorated, and the rooms well furnished. The houses and attached gardens reflect the pride the inhabitants obviously take in their homes. The general conditions, indeed, appear much better than those found in many parts of industrial towns. This is important, because we think it makes our findings applicable in many ways to the community at large.

Survey of Canine Sera.—The sera of the other 10 dogs belonging to the colony were examined for the presence of leptospiral antibodies. Of these, seven (one being the father of the litter already mentioned) agglutinated *L. canicola* and three were negative. The positive titres were 1 in 100 for five dogs and 1 in 300 for two. To obtain some evidence of the general incidence of canine leptospirosis in Bristol, sera from a further six healthy dogs living at considerable distances from the camp were tested also. Positive titres of 1 in 100 were found in three and negative reactions in the rest.

Survey of Human Sera.—Blood samples from 43 adults living in the camp were examined in the same way. In every case the result was negative, showing that no other

human cases had occurred among grown-up persons in spite of the high rate of infection in the dogs. We had hoped to complete the survey by examining specimens from all the children. Unfortunate publicity, however, caused a change from the early co-operation which we received, and the project had to be abandoned. The close association between children and dogs would have made this information of great value, and it is very regrettable that we were unable to obtain it.

Discussion

The six cases outlined above illustrate the rather nebulous nature of the clinical picture in canicola fever. The features shown in common by these patients were the sudden onset of a febrile attack, which lasted for a week or more and was accompanied by conjunctival injection, very severe headache, some neck rigidity, muscular pains, and signs of alimentary disorder.

The relative severity of the different symptoms in individual cases may easily suggest a diagnosis of influenzal meningitis or poliomyelitis, or even some abdominal catastrophe, and cases of leptospirosis have been found in surgical wards. Jaundice, although rarer in canicola fever than in Weil's disease, may be present to complicate the picture still further.

A similar problem is often presented by the non-icteric meningeal form of Weil's disease, in which condition ocular or neurological manifestations may also occur. The value of these eye signs in indicating the correct diagnosis is seen in the first two of our cases.

The occurrence of the meningeal variety of Weil's disease was recognized by Costa and Troisier (1916), and the condition has been intensively studied by a number of French observers, who refer to it as "spirochétose méningée pure." This form has also been reported from many other countries, and the frequency of its recognition probably depends, as Walch-Sorgdrager (1939) suggests, on the degree of interest in leptospirosis shown by clinicians. Our last four cases demonstrate that this is equally true of canicola fever.

Scheid (1949) has recently reviewed the literature dealing with the neurological complications of leptospirosis, and he notes the following: subarachnoid haemorrhage, which is probably an indication of the haemorrhagic tendency of the disease; paralysis of facial, palatal, and oculomotor nerves; brachial and sciatic neuritis; paralysis of voluntary muscles of the legs.

Hegler (1936) describes two cases of Weil's disease: in one of these tetanic spasms occurred; in the other the original diagnosis was transverse myelitis. The latter patient suffered from paraplegia and paralysis of the bladder; recovery was very slow, and was still incomplete after 12 months.

The eye changes in Weil's disease were reviewed by Buzzard and Wylie (1947) and by Coutela (1948). Photophobia and injection of the conjunctivae are common, and a number of other symptoms, including mistiness of vision, diplopia, iritis, iridocyclitis, irregular pupils, papilloedema, vitreous opacities, nystagmus, optic neuritis, and retrobulbar neuritis have been described.

Optic neuritis, associated with iridocyclitis or choroiditis, occurs during convalescence. Though vision is usually markedly affected, it may be good even in the presence of a severe lesion. Bilateral papilloedema has been observed in the mild meningitic form of the infection due to *L. icterohaemorrhagiae*. The prognosis of the ocular symptoms is good with adequate mydriatic treatment. This was demonstrated in Cases 1 and 2 of our series.

The literature on canicola fever is much less extensive, but ocular and nervous symptoms have been reported. So far as can be judged they seem to occur with relatively greater frequency than in true Weil's disease.

The differential leucocyte counts deserve some comment, because they were carried out at different stages of the disease. It is well known that by the end of the first week the relative proportion of polymorphs is high, reverting to normal later. In many cases also the eosinophils disappear during the early stages and reappear as the severity of the illness diminishes, as occurred in our cases.

For the treatment of canicola fever no specific antiserum is available, and the sulphonamides are inactive. Penicillin, however, seems to exert a beneficial effect in some instances at least. For example, the cases recorded by Baber and Stuart (1946) and by Mackay-Dick and Watts (1949) were apparently improved by penicillin, and the course of the disease in our third case may have been modified by the drug. On the other hand, the fatal case described by Weetch *et al.* (1949) showed no response; though it should be noted that this patient died of uraemia, and that the leptospiral nephritis was superimposed on previously damaged kidneys, so that the effect of penicillin cannot be fully estimated. In Case 6 of our series, however, penicillin, begun on the fourth day and continued for 14 days, did not prevent a recrudescence of fever during treatment. Spontaneous recovery is the rule in human infections, and the number of cases treated is small; therefore much more evidence is needed before a definite decision about the value of penicillin can be reached.

The position is more clear-cut in the case of dogs, because canicola fever often produces a severe and rapidly fatal disease in this animal. As was first shown by Joshua and Freak (1947), early administration of penicillin is followed by dramatic clinical improvement. This is in marked contrast to the relative ineffectiveness of the drug in "yellows" (caused by *L. icterohaemorrhagiae*), in which liver damage is the outstanding feature. If administration of penicillin in canicola fever is not begun during the septicaemic stage—approximately the first five to seven days—and the leptospire have already established themselves in the kidney tubules, the infection cannot be eradicated. Brunner and Meyer (1949) found, however, that streptomycin, in doses of 40 mg. per kg. for three to four days, would eliminate the organisms completely from the kidney, even at that late stage.

In this connexion the case of Weil's disease described by Moreau *et al.* (1947) is of interest. On the sixth day of illness the patient was heavily jaundiced and showed signs of marked renal involvement, with albumin and casts in the urine, oliguria, and a blood-urea level of 210 mg. per 100 ml. Treatment with penicillin was begun on that day, and a distinct change for the better in the kidney condition was noticeable two days later, the output of urine rising to 3 litres. After a few days albumin had disappeared from the urine, and by the 12th day the blood urea had fallen to 25 mg. On the other hand, the jaundice and the liver enlargement were quite unaffected.

In addition to the gravest form, all lesser degrees of severity of *L. canicola* infection occur in dogs, and there is also a healthy carrier state. In these latter types of infection leptospire in large numbers may be shed in the urine for some months, the animals constituting a danger both to man and to other dogs. *L. canicola* has a very high infectivity for dogs, though the virulence of different strains seems to vary greatly. Stuart (1946) and Joshua and Broom (1949) found serological evidence of past infection in some

40% of unselected urban dogs in Glasgow and London respectively. The results we obtained in Bristol show that the incidence is at least as high in that city. Borg-Petersen (1949) found *L. canicola* much less common in country as compared with town dogs in Denmark, but Broom and MacIntyre (1948) noted only a suggestive, not a significant, difference between these two groups in England.

When one considers that we found nothing to suggest that any other cases had occurred among adults in the camp, two conclusions seem justified. First, that the infectivity of *L. canicola* is low for man, and, secondly, that something more than the usual association between dog and man is necessary to cause human infections. Our cases illustrate two of the factors which may be responsible. The first two patients had both frequently mopped up pools of urine made by puppies which were apparently healthy but were, in fact, carriers. As was noted above, the woman had a cut finger, and the man constantly suffered minor hand injuries at work. It has been proved experimentally that leptospirae can gain entrance to the body through cuts and abrasions, and such were doubtless the portals of entry in these two instances.

On the other hand, Case 4 had often bathed in a stagnant river, and it is well recognized that true Weil's disease may be contracted by bathing in stagnant fresh water fouled with rat urine containing *L. icterohaemorrhagiae*. Evidence is accumulating that canicola fever also can follow bathing—presumably because of contamination with infected dog urine. One cannot altogether rule out the possibility of infection through skin abrasions in bathing cases, but Romijn (1932) remarked that many of his patients used swimming strokes that kept eyes and nose much under water. Buzzard and Wylie noted that inflammation of the tonsils and fauces appeared commoner when the disease was contracted while bathing, and they considered that this observation supports a transmucosal route of entry. Commenting on the well-known preponderance of infection in boys, these observers suggest the marked proclivity of males for diving, ducking, and "larking" in the water as a possible explanation. It seems to us that a greater fastidiousness on the part of girls in the choice of bathing-places may be an equally important factor. For instance, we know of a boy who developed canicola fever after bathing in a disused quarry in company with the carcass of a dead dog.

An outbreak of Weil's disease in Leningrad was thought by Viskovsky (1944) to be caused by food contaminated by rats. Vegetables grown in gardens and allotments may often be fouled by dogs, and it seemed of interest to test the possibility of transmission by uncooked greenstuff. Approximately 0.5 ml. of a virulent culture of *L. canicola* (about 5,000,000 organisms) was sprayed over lettuce leaves, which were then fed to a hamster. When the animal was sacrificed 11 days later typical haemorrhages were present in the lungs, and large numbers of leptospirae were seen in microscopical preparations of the liver and kidneys. This was only one test, made on a single animal, and one cannot say that the result is applicable to man. It does indicate, however, the need to investigate the possibility more fully.

Problems of importance in public health arise with regard to the risk of transmission of canicola fever from dogs to man. In frank cases which are under the care of a veterinary surgeon advice may be given to the owners about the risk of infection and how best to counter it by disinfecting the animal's dejecta. But the large numbers of dogs which are infected without being acutely ill, and the healthy carriers, constitute potential sources of danger. We believe

that canicola fever is commoner in Great Britain than the numbers of recorded cases indicate, and that it will become more frequently recognized. This has been the experience in all countries where the disease has been found and carefully studied. In Denmark, Borg-Petersen uncovered six cases during the examination of 8,000 sera sent for routine Widal tests, and Broom (unpublished) has had the same experience when testing sera from patients believed to be suffering from meningitis of viral origin.

Summary and Conclusions

Six cases of canicola fever are described.

The febrile attacks in the first two patients, a husband and wife, were thought to be due to influenza, and the true cause was not suspected until iridocyclitis developed during the later stages.

The source of infection was a puppy which was found to be a healthy carrier of *L. canicola*.

Both patients suffered from skin abrasions of the hands, and both had mopped up pools of urine left by dogs.

The patients lived in a relatively circumscribed community, and 10 of the 13 dogs belonging to the inhabitants were serologically positive to *L. canicola*.

Samples of serum from 43 other adult members of the community were negative for leptospirosis.

The source of infection of the third case could not be traced.

The fourth case had apparently contracted the disease while bathing in a stagnant river, presumably fouled with dog urine containing *L. canicola*.

The fifth patient had possibly been infected from her own dog; the sixth almost certainly from his.

In discussing these findings, attention is drawn to the occurrence of ocular and neurological manifestations in leptospirosis, and to their value in differential diagnosis.

Some comments are made on the prevalence of the infection in dogs and the potential danger of transmission to man.

The conclusion is reached that *L. canicola* has a low infectivity for man, but that dog-owners should be advised to take precautions when dealing with the dejecta of sick animals, and even of apparently healthy puppies.

We wish to record our grateful thanks to Mr. F. Clifton and Dr. J. Pendergast, who supplied us with the reports of the examination of the eyes of the first two patients, and to Dr. Kelly, who carried out the eye examinations on the other members of the camp.

REFERENCES

- Baber, M. D., and Stuart, R. D. (1946). *Lancet*, 2, 594.
 Borg-Petersen, C. (1949). *Proc. R. Soc. Med.*, 42, 714.
 Broom, J. C., and MacIntyre, A. B. (1948). *Vet. Rec.*, 60, 487.
 Brunner, K. T., and Meyer, K. F. (1949). *Proc. Soc. exp. Biol. N.Y.*, 70, 450.
 Buzzard, E. M., and Wylie, J. A. H. (1947). *Lancet*, 2, 417.
 Costa, S., and Troisier, J. (1916). *C.R. Soc. Biol., Paris*, 79, 1038.
 Coutela, C. (1948). In *Modern Trends in Ophthalmology*, edited by Arnold Sorsby, 2, 525. Butterworth, London.
 Hegler, C. (1936). *Disch. Z. Chir.*, 248, 190.
 Joshua, J. O., and Broom, J. C. (1949). *Vet. Rec.*, 61, 711.
 — and Freak, M. J. (1947). *Ibid.*, 59, 595.
 Klarenbeek, A., and Schüffner, W. A. P. (1933). *Ned. Tijdschr. Geneesk.*, 77, 4271.
 Laurent, L. J. M., Norris, T. St. M., Starks, J. M., Broom, J. C., and Alston, J. M. (1948). *Lancet*, 2, 48.
 Mackay-Dick, J., and Watts, R. W. E. (1949). *Ibid.*, 1, 907.
 Moreau, R., Boudin, G., Cler, R., and Graveleau, M. (1947). *Bull. Soc. méd. Hôp. Paris*, 63, 588.
 Romijn, P. (1932). *Ned. Tijdschr. Geneesk.*, 76, 5832.
 Scheid, W. (1949). *Fortschr. Neurol. Psychiat.*, 17, 295.
 Stuart, R. D. (1946). *Vet. Rec.*, 58, 131.
 Viskovsky, S. V. (1944). Summary in *Bull. Hyg.*, 1945, 20, 196.
 Walch-Sorgdrager, B. (1939). *Bull. Hlth Org. L. o. N.*, 8, 143.
 Weetch, R. S., Colquhoun, J., and Broom, J. C. (1949). *Lancet*, 1, 906.

The Chelsea Hospitals Group publishes a periodical entitled the *Chelsea Hospitals Quarterly*. It is intended for people in Chelsea, and particularly for those connected with the hospital group as staff, patients, or friends. The periodical will tell them about the activities of their hospitals, and the January number includes a short history of St. Stephen's Hospital.