above our accepted upper limit of normal, and the range was wide. The use of the intravenous tolbutamide-tolerance test added little more of value. Here we found so many abnormal results, judged by the criteria originally laid down by Unger and Madison (1958), that it would appear as if these criteria are too strict for older people. The mean curves within the three groups, however, correspond very well indeed with the glucose-tolerance tests, being normal in the normal group and distinctly diabetic in the diabetic group (Fig. 2).

Effect of Diuretics on Carbohydrate Metabolism.—Shapiro *et al.* (1961) found no change in glucose tolerance after two weeks on *chlorothiazide* in those patients whose control glucose tolerance was normal, but a worsening in those who were originally mildly diabetic. We found almost the opposite—certainly no worsening in the abnormal and diabetic groups, and a higher mean two-hour level in the normal group, but individually only one normal curve became abnormal. Over a longer period of time, of course, more changes might have been seen. On *frusemide* there was an apparent improvement in mean glucose tolerance in the abnormal and diabetic groups, but we do not claim statistical significance for this. Two of the normal subjects became diabetic after three months of frusemide, yet repeat tests were normal in one and only slightly abnormal in the other. This indicates the possible degree of individual variability and danger of relying on a single glucose-tolerance test, though we must emphasize that in general the glucose tolerance is remarkably closely reproducible (Jackson, 1964). In summary we found no clear evidence in our patients that *chlorothiazide* in a three-week course, or *frusemide* over three months, caused deterioration in glucose tolerance or in response to intravenous tolbutamide. The conditions of our trial differed from those of other workers mentioned above, who reported upon individual cases or experiments in animals. We certainly do not deny that *chlorothiazide* can be diabetogenic in certain patients, but in pharmacological doses this effect would appear to be comparatively infrequent. We do not know why our results with *chlorothiazide* differed from those reported by Shapiro *et al.* (1961), in that the carbohydrate metabolism of our "subclinical diabetics" did not worsen on therapy.

Effect of Diuretics on Other Blood Levels.—Our patients showed no change in levels of circulating platelets, serum potassium, alkaline phosphatase, or S.G.O.T. In only one subject was there a rise in serum uric acid level.

Effect of Diuretics on Blood-pressure.—In general this was very slight, with a mean fall of only 4–5 mm. Hg in the dia-

stolic pressure, though rather more in the systolic—a mean of about 19 mm. Hg.

Summary

Of 19 selected moderately hypertensive subjects six were found to have diabetes by oral glucose-tolerance tests, previously unsuspected, and a further six gave "abnormal" results. Intravenous tolbutamide-tolerance tests were also frequently abnormal. There was good correspondence between the results of these two tests.

Chlorothiazide given for three weeks and *frusemide* given for three months produced no remarkable deterioration of carbohydrate tolerance in these subjects, nor was any substantial effect on serum potassium, S.G.O.T., uric acid, alkaline phosphatase, or platelet count observed. The serum uric acid rose in one patient only.

The drop in blood-pressure with both diuretics was very small. There was a mean fall of about 4–5 mm. Hg in the diastolic pressure and about 19 mm. Hg in the systolic.

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Canicola Fever in Man and Animals

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Leptospirosis is an acute infection caused by pathogenic members of the genus *Leptospira*. In this country *L. icterohaemorrhagiae* and *L. canicola* are the only serotypes which so far are known to cause disease in humans. Infection is transmitted to man by various animals, both wild and domestic. The Figure has been designed (Michna, 1962b) to illustrate the present state of knowledge regarding host susceptibility to *L. canicola*, the animals which may act as carriers or as vectors of the parasite, and the possible sources and modes of infection of man and animals.

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The carriage and transmission of *L. canicola* solely by dogs can no longer be regarded as one of the features distinguishing this organism from *L. icterohaemorrhagiae*. Cases of canicola fever have occurred where a canine origin could not be established. Baber and Stuart (1946) described the first instance of human disease in Britain in a boy who had no contact with dogs, and in whom the infection was thought to have been acquired by bathing—a possible source of infection later confirmed by Williams *et al.* (1953). Broom (1951) recognized that there was a proportion of cases in which neither the source of infection nor the method of transmission could be ascertained, and McIntyre and Seiler (1953) postulated the existence

of sources other than dog urine. Williams *et al.* (1956), in the United States, were the first to isolate *L. canicola* from the kidney of a pig, after an epidemic of human infection. In Israel Van der Hoeden (1956) described canicola infection in two herds of pigs, with associated disease in attendant workers. The following year Coghlan *et al.* (1957) recorded five cases among workers on farms with infected pigs; a serological survey of 47 piggery workers on 12 Edinburgh farms showed

that 19 had at some time been infected with *L. canicola*. Recently Pertzalan and Pruzanski (1963) published an account of 81 cases collected in Israel during the years 1952–60. Only eight of their patients were in contact with dogs, whereas 33 had contact with pigs.

The purpose of this paper is to put on record a further 10 cases of canicola infection among workers in piggeries in the Glasgow area during the years 1957–63, and to confirm what appears to be a major role now played by pigs in the transmission of the disease. Opportunity is also taken to describe the clinical picture in human beings and to add a brief summary of the infection in pigs and cattle.

All cases, with one exception, occurred among the owners or employees of pig farms in Glasgow and the surrounding district. The farms concerned were widely scattered within this area. During the period under review the infected piggeries were kept under surveillance by one of us (S.W.M.). Serological evidence of *L. canicola* in the pigs was obtained on all the farms, and in five the organism was isolated from the kidneys of pigs slaughtered for human consumption. All the patients were admitted to hospital, with the exception of one in whom a retrospective diagnosis was made and confirmed serologically. Three cases are described as illustrative of the clinical picture of canicola fever in man, after which all patients are considered in relation to the farms where they worked.

Case Reports

Case 1.—A youth of 16 was admitted to a city hospital on 8 December 1960 with a week's history of frontal headache, backache, and pains in the limbs. His temperature was 100° F. (37.8° C.). There was an exudate on the tonsils, generalized lymphadenopathy, and splenomegaly. The calf muscles were tender, and there were clinical signs of bronchitis with mild bronchospasm. The Schuffner test was inconclusive but the Widal reaction suggested typhoid fever and he was admitted to Ruchill Hospital on 13 December. On admission he was found to have widespread congenital ichthyosis. His fever had subsided, but the cervical, axillary, and inguinal glands were enlarged, the tip of the spleen was palpable, and the conjunctivae were injected. Further serological tests did not support a diagnosis of typhoid fever, whereas

agglutination tests with *L. canicola* were positive to a titre of 1:1,000. Though there were no signs of meningeal irritation, lumbar puncture on the 15th day of his illness produced cerebrospinal fluid containing 110 lymphocytes/c.mm. The patient made an uneventful recovery and was discharged from hospital on 31 December. He had been working in a piggery for 12 months.

Case 2.—A man of 37, who had been working with pigs for four years, was taken ill on 5 May 1962 complaining of severe headache, vomiting, and anorexia. He complained particularly of his eyes, which were "heavy and sore." He was not admitted to hospital but his employer, who already had experience of two of his workers and his wife (*vide infra*) with similar illnesses, asked the family doctor to call in the public health authorities. This was done and subsequent agglutination tests with *L. canicola* showed a titre of 1:3,000 (16 May) rising to 1:30,000 (22 May); a test in April 1963 still showed a titre of 1:3,000. The illness was complicated by spread to the right eye, and the patient was admitted to the Glasgow Eye Infirmary on 31 May, 26 days after the onset of canicola infection. He complained of pain and blurred vision of one day's duration. Examination revealed a considerable flare in the anterior chamber, adhesions between the iris and the lens, and a cyclitic membrane in the pupil. A diagnosis was made of iridocyclitis. Treatment consisted of atropine drops, betnesol ointment, and a subconjunctival injection of mydracaine. The pupil eventually dilated. The chest film was normal and the Wassermann reaction was negative. The patient was discharged well on 8 June.

Case 3.—A woman of 22, a former hospital nurse, lived on a farm with her father and had assisted him over a period of seven years. She was admitted to a city hospital on 28 October 1962 with a seven-day history of an influenza-like illness; she complained of severe headache, vomiting, photophobia, and neck stiffness. Her temperature was 102° F. (38.9° C.). Lumbar puncture produced cerebrospinal fluid with 500 lymphocytes/c.mm. and she was transferred to Ruchill Hospital on the following day. On admission the clinical findings were confirmed, and she was found to have herpes labialis and conjunctival injection. She was emotionally upset and wept repeatedly. She stated that the headaches were unbearable and she felt "as if the top of her head was coming off." She vomited all fluids. Bone tenderness rather than muscle tenderness was elicited in the lower limbs. After three days in hospital her symptoms disappeared and she made a rapid recovery. The Schuffner test demonstrated a rising titre with *L. canicola* from 1:500 (31 October) to 1:2,000 (6 November). The patient was discharged well on 8 November.

Infection in Animals

Pigs

The first evidence of *L. canicola* infection of pigs was presented by Williams *et al.* (1953), who isolated the organism from a sample of pig urine. At the time the authors were studying in Georgia the epidemiology of canicola fever among 26 human patients who had contracted the disease as a result of bathing in water contaminated by urine of farm animals. The voided urine of carrier animals constitutes a source of infection by *L. canicola*, since the organism has been shown to remain alive for about a week in the case of undiluted and uncontaminated urine (Michna, 1962b), and for a much longer period in urine diluted with rain water. Pertzalan and Pruzanski (1963) state that the alkalinity of porcine urine makes for a longer survival of leptospira, whereas acid canine urine has a leptospiricidal effect. In Russia Warfolomijewa (1957) described an outbreak of canicola fever among human beings contracted from water contaminated by pigs. The effluent from an infected piggery in the Glasgow area collected at a point where it joined a small stream flowing into a near-by pond was examined microscopically by one of us (S. W. M.). Many motile leptospirae were present.

The cultivation of *L. canicola* in 1957 from the kidneys of naturally infected pigs was the first recorded experience of this kind in the Glasgow area, and indeed in Britain (Michna, 1959a). Between March 1957 and October 1961 10 cultures of *L. canicola* were grown from the kidneys of clinically healthy pigs, the carcasses of which had been passed for human con-

sumption. The animals originated from eight different piggeries in the Glasgow area in which the presence of infection had already been established by serological means. In three instances the recovery of *L. canicola* from porcine kidney followed incidents of canicola fever among the employees of three piggeries (Farms A, B, and C).

Infected slaughtered pigs generally do not show any pathological changes and seldom are macroscopic lesions evident at post-mortem examination during routine meat inspection. Closer examination, however, may reveal whitish or yellowish foci varying in diameter from 1 to 3 mm. in the kidney cortex, and sometimes petechial haemorrhages (Michna, 1962a). Lesions consist mainly of interstitial foci of lymphocytes. There is some swelling of parenchymal cells. The histological picture is that of subacute interstitial nephritis. Clinical signs have been recorded by Bezdenzhnykh and Kashanova (1956) following an outbreak of *L. canicola* infection of piglets which occurred on collective farms in Russia. There was fever of three to five days' duration associated with dullness, loss of appetite, diarrhoea, and icterus, together with haemoglobinuria. There was a heavy mortality among piglets. In a series of experiments Michna (1962b) observed an undulant type of fever in some animals. Out of 23 piglets only three manifested transient dullness, anorexia, diarrhoea, and conjunctivitis. Cutaneous erythematous lesions were observed in all but one of the experimental animals. Recent evidence (Guida *et al.*, 1959; Kemenes *et al.*, 1962; Michna, 1962a) indicates that *L. canicola* may be responsible for abortion and breeding difficulties in the sow. In the United States swine leptospirosis is a cause of major economic loss (Edwards and Domm, 1960).

Cattle and Other Animals

Van der Hoeden (1955a, 1955b) encountered epidemics as well as sporadic outbreaks of *L. canicola* infection and described clinical signs such as fever, loss of appetite, reduced milk yield, weakness, jaundice, and haemoglobinuria. Heavy losses occurred among calves.

Other Animals.—Roth *et al.* (1961) isolated *L. canicola* from the skunk, and other workers demonstrated the presence of antibodies in the sera of sheep, horses, goats, and camels. One of us (S.W.M.) has recently isolated *L. canicola* from the kidneys of a brown rat caught on Farm B (Table II), and found it to be the same serotype as that infecting the pigs.

Epidemiology

All persons infected were working on the farms and had intimate contact with the animals. In all cases serological

TABLE I.—Leptospira Agglutination Tests

Case No.	Age (Years)	Dates of Tests	<i>L. ictero-haemorrhagiae</i>	<i>L. canicola</i>
1	16	19/12/60	1:300	1:750
		23/12/60	1:300	1:1,000
2	37	16/5/62	1:100	1:3,000
		22/5/62	1:100	1:30,000
3	22	31/10/62	1:30	1:500
		6/11/62	1:30	1:2,000
4	25	16/5/57	Neg.	1:10,000
		25/5/57	1:100	1:30,000
5	17	20/9/61	Neg.	Neg.
		26/9/61	1:100	1:30,000
6	20	25/4/62	1:30,000	1:30,000
		4/5/62	1:30,000	1:300,000
7	23	4/6/62	Neg.	Neg.
		15/6/62	"	1:30,000
8	23	23/6/62	"	Neg.
		4/7/62	"	1:1,000
9	26	1/8/63	"	1:30,000
		12/12/63	1:100	1:30,000
10	24	6/8/63	Neg.	1:30
		19/8/63	1:150	1:3,000

reactions were diagnostic. The importance of repeating agglutination tests when initial results are negative is illustrated in Cases 5, 7, 8, and 10 (Table I).

Table II shows the number of human cases in relation to the five pig farms A to E. It will be noted that of the 10 cases half occurred in Farm A between 1957 and 1963. We may conclude that *L. canicola* has persisted in this farm for a period of eight years; and this despite restocking of the herd on two occasions because of swine fever. Persistence of infection is probably of longer duration, since it is known that the farmer's wife suffered from an acute illness clinically resembling canicola fever in July 1954. Her symptoms consisted of fever, severe headache, and inflamed eyes. Meningitis was suspected but lumbar puncture was not performed. The fever lasted for eight days and the patient was treated at home. Eight years later, in September 1962, agglutination tests with *L. canicola* showed a titre of 1:1,000, but there was a negative reaction with *L. ictero-haemorrhagiae*. This was the only positive titre obtained in eight contacts in Farms A, C, and D.

Some months prior to his wife's illness in 1954 the farmer had acquired an Alsatian dog which was on the premises of Farm A for three months. At the end of this period the dog became ill and was diagnosed by a veterinary surgeon as suffering from advanced renal disease, and destroyed. No post-mortem examination was performed. It is possible that the dog was responsible for first introducing *L. canicola* to Farm A, resulting in the infection being transmitted from pig to pig and giving rise to six human cases in the space of 10 years. The sera of two dogs in Farm A were recently examined for agglutinins against *L. canicola* with negative results. In Farm B, where two cases occurred, one in 1961 and one in 1962, *L. canicola* was isolated from the kidneys of a brown rat killed on the premises (*vide supra*).

In addition to the five farms shown in Table II, three others are at present under surveillance where evidence has been obtained of natural infection in pigs, but not so far of humans. There are therefore in the Glasgow region eight known sources of canicola fever, all of them in pig farms. Edwards and Domm (1960) state that in a given area one animal species harbours only one serotype of leptospirae or, at most, a few serotypes. Our investigations suggest that in this area the pig has the greatest epidemiological importance for man.

TABLE II.—Canicola Infection in Relation to Farms

Pig Farm	Case No.	Age (Years)	No. of Years Working with Pigs	Date of Onset of Illness
A	2	37	4	5/5/62
	4	25	5	13/5/57
	7	23	2	31/5/62
	8	23	6	16/6/62
	10	24	5/52	29/7/63
B	3	22	7	21/10/62
	5	17	3	1/9/61
C	1	16	1	1/12/60
D	6	20	3	15/4/62
	9	26	3/12	22/7/63

Clinical Picture

The duration of the acute illness ranged from five to seven days, which corresponded approximately to the duration of fever. Table III shows the relative frequency of signs and symptoms. Headache and meningitis were the most common initial features. The headache was of striking severity and in some instances reduced the patients to a state of despair and alarm. The meningitis was characterized by minimal clinical signs. In two cases pathological changes in the cerebrospinal fluid occurred in the absence of meningeal irritation. There were no instances of jaundice, which is a rare complication of canicola fever. Splenomegaly was found in two patients. In none was hepatomegaly demonstrated. Persistent vomiting in five patients led to dehydration in the acute stage of the

illness. A transient rash, confined to the lower limbs, occurred in two patients (Cases 5 and 7). Edwards and Domm (1960) refer to the peculiar localization of the rash to the pretibial areas of the legs, with or without trunk involvement. Bradycardia, which could not be explained by jaundice, occurred in three patients.

TABLE III.—Signs and Symptoms in 10 Cases

Headache ..	10	Photophobia ..	5	Lymphadenopathy ..	2
Meningitis ..	8	Bronchitis ..	4	Splenomegaly ..	2
Injection of eyes ..	6	Bradycardia ..	3	Rash ..	2
Muscle tenderness ..	6	Albuminuria ..	3	Herpes labialis ..	1
Vomiting ..	6				

Conjunctivitis and photophobia were prominent. Marked injection of the sclera was noted in three cases and in one of these there was a striking ciliary injection. Case 2 was followed by iridocyclitis, an uncommon complication in this country. Eye changes in leptospirosis are usually confined to the conjunctivae and sclerae. Iridocyclitis in our patient occurred 26 days after the onset of the infection. This is in accord with reports in the literature where it is stated that iridocyclitis appears late in the disease, usually between the third and seventh weeks (Buzzard and Wylie, 1947). In a case described by Mackay-Dick and Watts (1949) iritis occurred on the 78th day of illness; in two cases in a series published by Campbell *et al.* (1950) iridocyclitis was diagnosed on the 51st and 28th day respectively. Other infrequent complications such as choroiditis and optic neuritis are associated with meningeal forms of leptospirosis. They are probably seen more often in canicola infection.

Leptospirosis is the cause of 3 to 6% of all cases of aseptic meningitis. Of the nine cases examined on admission to hospital seven presented with signs of meningeal irritation. Minkenhof (1948) noted that the cell count in the cerebrospinal fluid varied according to the day of illness on which the count was done. Edwards and Domm (1960) state that pathological changes are found in the fluid in 80% of cases if lumbar puncture is done during the second week of the disease. Four of our patients, examined by lumbar puncture within the first week of illness, had normal cerebrospinal fluid; of these, three showed increased cell counts on subsequent lumbar puncture carried out towards the end of the second week. Seven patients, in whom lumbar puncture was performed between the 9th and 21st days of illness, had cell counts ranging from 25 to 500 lymphocytes/c.mm. (Table IV). The sugar content was slightly raised, a finding which has been used to differentiate leptospiral and tuberculous meningitis when there is a doubt about diagnosis (Broom, 1951).

TABLE IV.—Findings in Cerebrospinal Fluid (8 Cases)

Case No.	Lymphocytes/c.mm.	Protein (mg./100 ml.)	Sugar (mg./100 ml.)
1	25	20	71
2	110	20	70
3	179	70	62
4	36	40	66
7	9	20	81
8	500	50	63
9	220	120	65
10	3	20	65

There was no marked anaemia. The white-cell count ranged between 4,200 and 13,000 cells/c.mm. with a normal distribution of neutrophils and lymphocytes. The erythrocyte sedimentation rate in five cases ranged from 13 to 50 mm. in one hour (Wintrobe). Blood urea was within normal limits, except in one case where there was a transient rise to 86 mg./100 ml. Liver-function tests were normal. These findings are in keeping with non-icteric leptospirosis.

Discussion

Table V, compiled from the annual report of the Medical Officer of Health of Glasgow, shows the incidence of infection

caused by *L. icterohaemorrhagiae* over the same interval of time as that in which the 10 cases of canicola fever occurred. The figures in parentheses show the incidence of leptospirosis during an 11-year period from 1945, when the first case of canicola fever in Britain was described by Baber and Stuart, to 1956, the year prior to the period under review. In recent years canicola infection has replaced icterohaemorrhagica infection as the common cause of leptospirosis in this part of the country. Between 1957 and 1963 there were 23 cases—10 traced to pigs and nine to dogs. Our results do not show a preponderance of pig-transmitted infection over dog-transmitted infection, but perusal of the literature suggests that the incidence of canicola fever in piggery workers is high in contrast with the low infectivity rate among dog owners (Coghlan *et al.*, 1957; Pertzalan and Pruzanski, 1963). A further guide to the incidence of leptospirosis in Glasgow and the west of Scotland is obtained from Table VI, which shows the number of cases diagnosed from complement-fixation tests carried out at the Regional Virus Laboratory on paired sera from cases of aseptic meningitis during the period 1959–64. Of 40 cases all but two were caused by *L. canicola*. Any assessment of the relative frequency of porcine infection here and in other countries must take into account factors such as hygiene and sanitation. Van der Hoeden (1955a) describes a piggery in Israel with a herd of more than 200 pigs attended by eight workers who, like the pigs, lived under poor hygienic conditions. The men worked barefooted, with the upper part of their bodies naked. Excretions and food remnants from the open concrete floor of the sties drained into a near-by lake in which the workers bathed and from which they took their drinking-water. It is perhaps not surprising that in Israel's population of 1.5 million reported cases of canicola fever amount to 8 per million, compared with a figure of 0.35 in Britain.

TABLE V.—Glasgow and District 1957–63

Source of Infection	<i>L. icterohaemorrhagiae</i>	<i>L. canicola</i>
Rat	5	—
Pig	1	10
Dog	—	9
Unknown	—	4
	6 (27)	23 (4)

The figures in parentheses are those for 1945–56.

TABLE VI.—Leptospirosis—West of Scotland—1959–64

Year	<i>L. canicola</i>	<i>L. icterohaemorrhagiae</i>	Total
1959	8	1	9
1960	8	0	8
1961	5	0	5
1962	4	0	4
1963	9	0	9
1964	4	1	5
	38	2	40

Six of the 10 cases occurred between the months of May and July, suggesting a higher morbidity in the summer months (Pertzalan and Pruzanski, 1963). There was no correlation between the onset of illness and duration of employment. It will be seen from Table II that the shortest period of contact was five weeks (Case 10), the longest seven years (Case 3). Healthy farm contacts who had experience of 30 years or more with pigs showed no agglutinins against *L. canicola* or *L. icterohaemorrhagiae*.

Sufficient evidence has now accumulated to suggest that canicola fever is an occupational disease among piggery workers. Coghlan and Norval (1960) have recorded infection by *L. canicola*, which persisted for as long as five years, in two piggeries on the outskirts of Edinburgh. Eleven out of 16 human cases were traceable to such sources. There is reason to believe that the infection of pigs has persisted for at least seven years in Farm A, where five employees developed canicola fever. Infection may spread by stream or effluent beyond the confines of infected piggeries to waters where bathers are at

risk. During the warm seasons of the year bodies of stagnant water, slow-moving streams, and wet soil may remain potential sources of infection for weeks after they are contaminated (Williams *et al.*, 1956; Edwards and Domm, 1960). It has been suggested that fish living in contaminated waters may play an important role in the epidemiology of leptospirosis. The organism has been recovered from the gills and the kidneys of artificially infected goldfish (Maestrone and Benjaminson, 1962).

Working conditions in piggeries no doubt predispose employees to infection. Many suffer minor skin abrasions, thus providing a portal of entry for leptospira. Case 1 would be at special risk on account of ichthyosis. The known dangers of immersion in, or taking drinking-water from, contaminated lakes or stagnant waterways point to transmucosal and conjunctival routes of entry (Buzzard and Wylie, 1947). Other possible origins of infection are the blood and internal organs of slaughtered carrier animals. The handling of these materials may be a hazard to man. Michna (1959b) has shown that *L. canicola* can be cultured post mortem from naturally infected porcine renal tissue kept at a temperature of 0–4° C. for at least 12 days, and for not less than six days at minus 8° C. From the artificially infected pig kidney kept at refrigerator temperature *L. canicola* was recovered up to 21 days later. Apparently healthy pigs are therefore not only a daily risk to their attendants but, after slaughter, could be a source of danger to abattoir workers, butchers, housewives, and domestic animals. Michna (1962b) infected a dog by feeding it with raw pig kidney containing *L. canicola*. Contamination of food by leptospirae is not a common source of infection but has been described by Borg Petersen (1944) in Denmark. A public health problem may arise in the future if, as we believe, more cases are brought to light because of a greater awareness of the disease and its origins. There is no doubt that the benign nature of canicola fever results in many cases being missed. This is particularly so when the patient does not present the clinical picture of the disease or is not employed in one of the occupations recognized as carrying a risk of infection (Broom and Alston, 1948).

Summary

Ten cases of canicola fever occurred in Glasgow and district during the seven-year period 1957–63. The source of infection was pigs, with which the patients had intimate contact during the course of their employment. Infection in pigs has been established serologically in eight pig farms. In three of the five farms where human infection occurred *L. canicola* was cultured from the kidneys of pigs slaughtered for human consumption. In one farm the organism was recovered from the kidney of a brown rat killed on the premises. In relation to another farm, motile leptospirae have been observed microscopically in the effluent from a near-by pond.

Of eight contacts investigated one, the wife of the owner of Farm A, showed serum agglutinins against *L. canicola*, sufficient to confirm a previous infection.

The clinical picture was typical, with headache, meningitis, and a characteristic suffusion of the conjunctivae forming a

diagnostic triad. In one patient the illness was complicated by iridocyclitis.

An approximate figure of the incidence of canicola fever in this area is given. It is suggested that canicola fever is an occupational disease among piggery workers. The recovery of live *L. canicola* from pig kidneys post mortem points to a hazard to persons other than those employed in occupations at risk.

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ADDENDUM.—Since this paper was written two further cases of canicola fever have occurred among employees in Farm A. This brings the total number of cases during the period 1957–65 to 12.

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