SECOND SESSION

The bacterial zoonoses

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Although, by derivation, the word zoonosis would seem to imply no more than 'a disease, or an abnormal condition of, animals', for many years now it has been invested —by both medical and veterinary epidemiologists—with an added meaning, viz. a disease, primarily of lower vertebrate animals, which may, at times and in certain circumstances, spread from them to man, giving rise to human infections.

Throughout history, plague and similar pestilences have always claimed anxious attention, but, in the post-Listerian era, so long as diseases such as diphtheria, typhoid fever and streptococcal infections continued to give cause for concern, interest in the zoonoses, at any rate in developed countries, tended to be desultory. Now that the 'common fevers' have been largely overcome, or at least brought under control, the zoonotic infections are being studied with renewed enthusiasm. Several factors have contributed to this:

1. The ever-increasing speed of transport (of men, animals and materials) across oceans and continents provides greater opportunities for the spread of these diseases.

2. Despite the progressive mechanization of our way of life we are still—perhaps now more than ever—a nation of stockbreeders, animal-lovers and pet-owners, and the pets seem to become ever more exotic.

3. Technological developments in agriculture are having a profound effect on the ecology of our native fauna, and no one can foretell how this may disturb established patterns of host-preferences among parasitic micro-organisms.

4. Modern diagnostic (especially immunological) techniques as applied to these infections make it possible for the medical and veterinary professions to work closer together—and to their mutual advantage—for the better understanding of the pathological processes involved.

5. Hopes raised by the advent of chemotherapy have given a new impetus to the study of these diseases, although it must be conceded that the hopes have often proved illusory; and, in this connexion, it is as well to remember that the uncontrolled administration of antibiotics to animals, no less than to our own patients, can have serious repercussions on the health and welfare of man (Anderson 1968).

For these and other reasons it is appropriate that those of us who are engaged, clinically, administratively, or in the laboratory, in the control of communicable diseases should address our minds to the zoonoses. Those of bacterial aetiology include:

- Anthrax
- Bovine (and, very rarely, Avian) tuberculosis
- Brucellosis
- Erysipeloid
- Leptospirosis
- Listeriosis
- Necrobacillosis
- Pasteurellosis and
- Salmonellosis

There are many others, but these are the ones that most merit our consideration in this country.

Brucellosis and salmonellosis, as befits their special importance, are the subjects
of separate contributions to this symposium and two of the others can be dismissed briefly.

**Bovine (and avian) tuberculosis**

In Britain, as in all countries where such measures have been adopted, increasing acceptance of the heat-treatment of milk and, in the last two decades, the spectacular success of the Attested Herds Scheme have all but eliminated the risk of human infection with *Mycobacterium tuberculosis* var. *bovis*, because the main mode of spread was, of course, by milk. Nevertheless some degree of occupational hazard remains, a point to bear in mind in the differential diagnosis of skin lesions in those whose work entails the handling of carcasses (and pigs as well as cattle are susceptible to infection with this organism), because cutaneous tuberculosis responds well to timely chemotherapy, thus obviating the distressing disfigurement which untreated lupus vulgaris can cause.

Tuberculosis is not an uncommon disease of poultry, but the causative organism, *M. avium*, is virtually non-virulent for man, and reports, which do appear from time to time, of human cases allegedly infected by the avian bacillus tend to arouse scepticism. However, a few—a very few—of them do seem to be well-authenticated.

**Necrobacillosis**

Necrobacillosis in animals presents in various forms, such as necrotic focal hepatitis in cattle, foot-rot in sheep and cattle, ‘calf diphtheria’ (in which there is a false membrane), equine gangrenous dermatitis etc., in all of which necrosis is the essential characteristic of the lesions caused by the strictly anaerobic Gram-negative fusiform bacillus, *Sphaerophorus* (or *Fusiformis*) *necrophorus*. The inclusion of the condition among the zoonoses is justified by the fact that the infection is sometimes acquired by those whose work brings them in contact with diseased animals and who consequently develop localized lesions of the skin and subcutaneous tissues. But a larger and more serious group of human infections are those where no definite history of contact with animals can be elicited. The upper respiratory passages (and sometimes the middle ear), the lungs and the female genital tract are among the sites affected. Thus lung abscess or empyema may result, and postpartum or postabortive infection may give rise to suppurative endometritis and so on. Another group of cases are those in which the infection supervenes on surgical conditions of the abdomen, such as appendicular abscess. In these patients the organism is presumably derived from the gut, although it can seldom be found in normal stools; very often the infection is mixed, with anaerobic streptococci or other organisms present in addition to the sphaerophorus, and this adds to the difficulty of direct culture, necessitating mouse-inoculation for diagnostic purposes. Although the organism is sensitive in *vitro* to penicillin, it may prove ineffective clinically (probably because of the other organisms present), in which case tetracycline is the drug of choice.

**Anthrax**

This disease of animals (chiefly herbivora), variously known as splenic fever, murrain and so forth, is essentially a septicaemia, but may occur in a less fulminating form with chronic fever and localizing pustular lesions. World-wide in distribution, it is specially prevalent among animals in India, Pakistan, other parts of Asia and in South America but its incidence among British and Western European livestock is, fortunately, low. The mode of infection in animals is mainly by ingestion from contaminated pasturage or artificial feeding-stuffs, which, once contaminated, can remain potentially infective for incredibly long periods of time. It is the readiness with which *Bacillus anthracis*, in the presence of oxygen, forms highly resistant spores when discharged from an infected animal that accounts for this persistence of infectivity.

Pathogenicity, however, is an attribute of the vegetative form of the organism;
its virulence depends on two main factors, the polypeptide, D-glutamic acid (a true hapten) contained in the well-known capsule, and an extracellular toxin made up of at least three components which, individually, are harmless enough but which, acting synergistically, give rise to severe oedema in animal tissues, thus facilitating the spread of the organisms, and can induce oligaemic shock with lethal effects.

In man anthrax presents in one of two main forms: Cutaneous anthrax and, less commonly, inhalation anthrax. (Intestinal anthrax can also occur, but this is rare.)

**Cutaneous anthrax.** The commonest sites for the local lesions are the head, face, neck and upper extremities, i.e., the exposed areas of the body. Within two or three days of contact with infected material—and probably some degree of trauma, however slight, is required to aid the implantation of the organisms—a reddish papule appears, which in another day or two becomes a blister, surrounded by an area of erythema. At this stage clinical diagnosis is not easy but, if the possibility is borne in mind, bacteriological examination of blister fluid will afford confirmation. Over the ensuing 24–48 hours the lesion develops into the characteristic *malignant pustule*. This is really a misnomer, because there is no true pus-formation, but a coagulation necrosis, leading to a central reddish-black discoloration, the so-called black *eschar*, surrounded by a ring of vesicles containing serous, often blood-stained, fluid and, beyond this, a wide zone of oedema which later becomes hard but not usually painful, except when touched. Occasionally, as in a recent case in Scotland (Lamb and Goudie 1967) there is a ‘spill-over’ of organisms, carried by lymphatic drainage of the oedema fluid, into the bloodstream and *B. anthracis* can be recovered on blood-culture; indeed some observers claim that, early in the disease, a transient bacteraemia occurs oftener than might be supposed. Otherwise diagnosis is made by bacteriological examination of material from the lesion, preferably fluid from intact vesicles.

**Inhalation anthrax.** This results from the inhalation of dust containing anthrax spores, which settle and germinate in the air passages. Initially, the symptoms are those of a deceptively mild upper respiratory tract infection, followed in about three days by acute respiratory distress and shock. By this time there is a fulminating haemorrhagic pneumonitis, more often than not accompanied by septicaemia (and, in some cases, a haemorrhagic meningitis) and, unless the condition is recognized and treated early—which seldom happens—death follows inexorably in a matter of hours.

The bacteriological diagnosis can be surmised when smears of material from the lesions are seen to contain large square-ended Gram-positive bacilli arranged end to end in chains; sometimes, especially in material from cases who have received antibiotic treatment, aberrant forms are found but, in any case, care must be taken to distinguish *B. anthracis* from saprophytic members of the genus *Bacillus* that might be present, hence the importance of isolating the organism on culture. It grows quite readily in air on simple media, producing the characteristic ‘medusa-head’ colonies made up of entwined chains of bacilli. Unfortunately, and this is particularly true of attempted culture from inanimate materials such as bone-meal, its growth may be outstripped by that of contaminants. This is why animal inoculation is usually required; even then it is necessary first to heat the intended inoculum to a temperature of 65–70°C for about 15 minutes, which kills at least some of the contaminants without harming the anthrax spores. The guinea-pig is the animal of choice and, again, if gross contamination is apprehended, it should be given prior protection with antitoxicidal serum. The test inoculum is injected into the deep subcutaneous tissues and if the result is positive the animal will die of anthrax in three to four days. The most striking post-mortem finding is extensive oedema of a characteristic gelatinous consistency, from which, and from the guinea-pig’s enlarged and congested spleen, *B. anthracis* can be cultured with ease. Also smears of the heart-blood stained with polychrome methylene blue give the well-
known McFadyean reaction. Finally, the cultures can be identified beyond doubt by their susceptibility to anthrax-specific phage.

Treatment with penicillin and tetracyclines, if started early enough, is effective in cutaneous anthrax and serum therapy is seldom required nowadays. Although most patients with inhalation anthrax die, the case fatality rate in cutaneous anthrax has been reduced from about eight per cent in pre-antibiotic days to one per cent or less at the present time.

In the past imported wools and hides were the chief sources of infection; thus inhalation anthrax, known as ‘wool-sorters’ disease’ was a formidable industrial hazard in the mills of Yorkshire. Infection, usually cutaneous, from contact with these materials still occurs but its frequency has diminished dramatically since the enforcement of the Anthrax Order whereby animal hairs and hides from countries in which the disease is prevalent must be imported through the Government Disinfection Centre in Liverpool. Other imported materials are now incriminated quite as often, if not oftener, as will be seen from table 1.

| TABLE I |

Examination of imported materials for B. anthracis at the City Laboratory, Glasgow, 1965–67 inclusive

<table>
<thead>
<tr>
<th>Material</th>
<th>No. of samples</th>
<th>No. positive</th>
<th>Per cent positive</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bone-meal, bone-grist, etc.</td>
<td>263</td>
<td>64</td>
<td>24.3</td>
</tr>
<tr>
<td>Goatskin</td>
<td>66</td>
<td>9</td>
<td>13.6</td>
</tr>
<tr>
<td>Wool</td>
<td>21</td>
<td>2</td>
<td>9.5</td>
</tr>
<tr>
<td>Sheepskin</td>
<td>13</td>
<td>0</td>
<td>—</td>
</tr>
<tr>
<td>Hides, various</td>
<td>12</td>
<td>0</td>
<td>—</td>
</tr>
<tr>
<td>Animal hair (various spp.)</td>
<td>11</td>
<td>1</td>
<td>9.1</td>
</tr>
<tr>
<td>Pigskin</td>
<td>3</td>
<td>0</td>
<td>—</td>
</tr>
<tr>
<td>Sundry samples</td>
<td>8</td>
<td>0</td>
<td>—</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>397</strong></td>
<td><strong>76</strong></td>
<td><strong>19.1</strong></td>
</tr>
</tbody>
</table>

Evidently imported bones and bone-meal are potentially very dangerous; indeed, in 1965, a London factory worker died of inhalation anthrax contracted after handling jute sacks which had previously been used for transporting imported raw bone (Enticknap et al. 1968). Workers known to be at risk can be artificially immunized with a ‘vaccine’ (in reality an antigenic extract of B. anthracis, not a true vaccine) but the real anxiety about bone-meal arises from its incorporation in artificial fertilizers which are handled unsuspectingly by so many people in trade, in agriculture and even in suburban gardens. Yet anthrax is a surprisingly rare disease as will be seen from table II.

| TABLE II* |

Notifications of anthrax, England and Wales 1 December 1960†–31 December 1966

<table>
<thead>
<tr>
<th>Suspected vehicle of infection</th>
<th>No. of cases</th>
<th>No. fatal</th>
<th>Notes on occupation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wool</td>
<td>19</td>
<td>2</td>
<td>17 in woollen mills</td>
</tr>
<tr>
<td>Hides and skins</td>
<td>14</td>
<td>1</td>
<td>8 in tanneries</td>
</tr>
<tr>
<td>Bones or bone meal</td>
<td>14</td>
<td>2</td>
<td>9 in animal by-products factories</td>
</tr>
<tr>
<td>Carcases†</td>
<td>11</td>
<td>—</td>
<td></td>
</tr>
<tr>
<td>Goat hair</td>
<td>2</td>
<td>—</td>
<td></td>
</tr>
<tr>
<td>Doubtful or unknown</td>
<td>10</td>
<td>—</td>
<td></td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>70</strong></td>
<td><strong>5</strong></td>
<td></td>
</tr>
</tbody>
</table>

Compiled from Reports (1965a, 1966a) †This is the date on which anthrax became notifiable.

In Scotland during the same period there were 22 cases, fortunately none of them
fatal, and of these ‘several had an association, usually occupational, with bone meal’ (Reports 1960–64, 1965b, 1966b).

Because of this relatively low incidence there is a belief that anthrax is of low infectivity for man but the fact that cases can, and do, occur sometimes after minimal exposure should dispel any complacency about the disease.

Erysipeloid

_Erysipelothrix rhusiopathiae_, a slender Gram positive rod-shaped, non-motile organism, closely related to the corynebacteria, is widely distributed in animals and birds and is found as a commensal in the slimy coating and scales of several species of fish (especially perch), although in all probability this is a result of contamination after the catch has been landed. It can cause disease in a variety of animals, but, among domestic species, it is the pig that most often acquires the infection.

_Swine erysipelas_ (which is wholly unrelated to human erysipelas), can occur in four main forms, two of them carrying a high mortality, _viz._ (1) the septicaemic form with a generalized, patchy, red rash and an 80 per cent case-fatality rate, and (2) the cardiac form, a true endocarditis with valvular vegetations, and two milder forms _viz._ (3) the urticarial form lasting a few days, with mild fever and deep red, rhomboidal patches (‘the diamonds’) on the skin, usually ending in spontaneous recovery, and (4) the arthritic form with joint swellings due to synovitis.

The natural disease in pigs is due mostly to intestinal infection and the organism is excreted in the faeces of sick, convalescent and (probably) carrier animals. But infection of man by direct transmission from pigs suffering from swine erysipelas seldom occurs. (Instances of veterinary surgeons and related personnel infected in this manner have been recorded, but such cases are rare.) Human infection is usually traced to contact with dead matter of animal, fish or vegetable origin, particularly in damp surroundings, and there is a frequent association with cuts, which may be quite small, _e.g._ scratches from fish scales or bone spicules.

The organism, which is very resistant to putrefaction—and supposedly to salting—can survive for long periods on dead matter. However, it is highly susceptible to skin cleansing agents, such as cetrimide (and possibly to local anaesthetic solutions) a point of some practical importance in regard to diagnosis by skin biopsy.

Clinically the lesions are mostly found on the hands, especially the fingers. After an incubation period of 1–4 days, but sometimes two weeks or more, the site of entry of the infection becomes purplish red, swollen and painful. The pain is burning or throbbing in character, but, on palpation, the part is never as acutely tender as a septic hand due to pyogenic organisms. The original trauma may have been so trivial that the patient has forgotten about it. There may be a lymphangitis and a regional adenitis, although this is more likely to be due to secondary infection. But the characteristic lesion is a sharply defined purplish-red area with a raised edge which advances peripherally as it desquamates centrally. It is often mistaken for a pyogenic infection and needlessly incised, but its chronicity should raise a suspicion. It tends to be self-limiting, spontaneous remission and resolution without suppuration occurring in 3–4 weeks, but it may last longer and relapses, or even second attacks, may occur.

This is the typical erysipeloid of Rosenbach. Among the general population it is not a common condition, although it is thought that a number of cases are ‘missed’, but in persons engaged in certain occupations there may be quite a high incidence. Thus Proctor, at the casualty department of Aberdeen Royal Infirmary, over a period of 13 months saw as many as 235 cases, most of them from among some 2,500 workers employed in the nearby fish-market and associated premises (Proctor and Richardson 1954); and ‘epidemics’ have occurred, one among fresh-water fish-handlers in Odessa in 1930, another affecting 247 workers engaged in sawing and polishing bones at an
American button factory in 1933. Sometimes, too, the organism proves more invasive, giving rise to more generalized cutaneous eruptions with arthritic and constitutional symptoms or more rarely, septicaemia with endocarditis; two such cases, both fatal, were reported in England three years ago (Morris et al. 1965).

Although the organism grows quite well, if a little slowly, in simple media, such as glucose broth, under aerobic conditions—and Morris and his colleagues had no difficulty in isolating it from blood cultures in their cases—bacteriological diagnosis is not easy. The problem is obtaining a suitable specimen from a cutaneous lesion. The usually recommended procedure is to inject sterile saline into the edge of the lesion and, without withdrawing the needle, re-aspirate the juice, which is then inoculated into glucose broth, but other workers (Sneath et al. 1951) found a pinch biopsy taken from the lesion’s edge a fairly successful method of obtaining material for culture. To go to such lengths to obtain a laboratory specimen may appear unnecessarily drastic when dealing with a condition which, if confidently diagnosed on clinical grounds, may be expected to respond to treatment with penicillin. But, certainly, if there is any question of septicaemia every effort should be made to isolate the organism from the blood-stream, so that its sensitivity to antibiotics can be tested. In these cases there is some evidence that a combination of penicillin and sulphonamide, which act synergistically on the organism, is the most effective form of therapy.

**Leptospirosis**

Members of the genus *Leptospira* of the order *Spirochaetales* are, as their name implies, thin spiral organisms. There is a central, relatively rigid, filament, or axistyle, around which the body of the organism is tightly coiled, and a hook at one or both ends is a characteristic feature. Another characteristic is motility, which mainly consists of rapid rotation around the long axis, but there is also a ‘gliding’ movement and, from time to time, the organism may be observed to bend into larger secondary coils. Some leptospiroa exist saprophytically, especially in water. These are known collectively as *L. biflexa* and they grow readily in ordinary media. The pathogenic ones, on the other hand, are strict animal parasites, which cannot long survive outside the host body and are relatively difficult to grow on artificial culture media; nor are they easy to stain for microscopic examination and thus dark-ground or phase-contrast microscopy is required for their study.

A great variety of wild and domestic animals, in almost every country in the world and under all manner of climatic conditions, act as reservoirs from which man can acquire the infection. The numerous synonyms for leptospirosis in different countries, such as Weil’s disease, mud fever, rice-field fever, swamp fever, Japanese seven-day fever, to name but a few, give an indication of its world-wide distribution. But, although there are at least 40 pathogenic leptospiroa serotypes (and, within these, several subtypes) many of which, in various parts of the world, infect man and some of these, e.g. *L. ballum* and *L. bratislava*, have been isolated from small rodents and hedgehogs even in Scotland (Broom and Coghlan 1958, 1960), only two serotypes have so far been implicated in human infections in Britain. These are *L. icterohaemorrhagiae* and *L. canicola*, the former being the more virulent.

Pathogenic effects in animals are variable, depending on host-parasite relationships between particular serotypes and different animal species; they include: haemolytic anaemia, hepatitis, meningitis, abortion and nephritis. Of these the most constant is a focal interstitial type of nephritis; it is also the most important from the point of view of dissemination of the disease to other animals and to man, because the

*The conventional nomenclature *L. icterohaemorrhagiae*, *L. canicola* (for “serotype *icterohaemorrhagiae*,” “serotype *canicola*”) etc., although no longer considered taxonomically valid, has, for the sake of simplicity been retained in this section.
organisms are excreted in large numbers in the urine of infected animals.

In man, infection due to *L. icterohaemorrhagiae* causes classical Weil's disease (haemorrhagic jaundice) in which the haemolytic, hepatotoxic, and nephritogenic potentialities of the organism predominate, and therefore, jaundice, liver damage associated with haemorrhages, and proteinuria with potential renal failure, are the respective clinical features. In *L. canicola* infections, on the other hand, lymphocytic meningitis is the more usual overt clinical syndrome and jaundice is seldom seen. Both diseases however may present in milder and, certainly as far as *L. canicola* infections are concerned, in subclinical or inapparent forms.

In this country rats are the main reservoirs of *L. icterohaemorrhagiae*, and dogs and pigs of *L. canicola*, and man is infected through the skin through which, especially if sodden or slightly damaged (e.g. minute abrasions), the leptospires enter.

Weil's disease can be diagnosed, with some difficulty, by blood-culture, by identifying the organism by dark-ground microscopy in the urine or by inoculating blood or urine into guinea-pigs, but these procedures are inapplicable to canicola fever. Indeed both conditions are more usually diagnosed by serological tests.

Penicillin is the treatment of choice in Weil's disease but, to be effective, it must be given early. As for canicola fever, despite the meningitis, it tends to be a self-limiting disease and fairly rapid recovery is the rule.

When one considers the opportunities for infection it is surprising that recorded cases of leptospirosis in Britain are so few. This is particularly true of canicola fever, because, quite apart from the occupational hazard amongst piggery workers, etc. (Lawson and Michna 1966), it has been shown that about one in every five dogs in this country show evidence of past infection with *L. canicola* (Alston and Broom 1958); clinically this presents as nephritis, jaundice being a variable sign, and the infected dogs continue to excrete the organisms in the urine for long periods after recovery. Figures are difficult to obtain, because the disease is not notifiable, but in Glasgow only five cases of Weil's disease and 24 of canicola fever have been known to occur in the last ten years (Ives 1968). Table III indicates that the picture for England and Wales is much the same.

There is a strong suspicion that a number of canicola infections are 'missed', but there is also good evidence that host susceptibility varies enormously among humans (McIntyre and Seiler 1953) no less than among animals.

**Listeriosis**

*Listeria monocytogenes*, like *Eryspelothrix rhusiopathiae*, is also now classed with the *Corynebacteriaceae* and is a Gram-positive, non-sporing, rod-shaped organism, but, unlike eryspeloethrix, it is motile, particularly at tempertaures rather lower than that of the human body.

It was from a disease in laboratory animals, characterized by monocytosis (especially in rabbits) that the organism was first isolated in Cambridge in 1926 (Murray et al. 1926). In the following year it was recognized as the causative organism of 'Tiger river disease' in rodents in South Africa and in 1931, from New Zealand, came the first report of disease in domestic animals caused by it; this was an encephalitis of sheep, called 'circling disease' (Gill 1931). It is not quite clear who was the first to isolate the organism from human patients, because many of the earlier reports (mainly from the U.S.A.) are confused by the fact that it was regarded as a diphertheroid of doubtful pathogenicity, but in Scotland, it was Gibson in 1935 who, while unsure of its exact taxonomy, was the first to report it as an undoubted cause of human meningitis (Gibson 1935).

During the intervening years infection with *L. monocytogenes* has been found to be widespread among different animals throughout the world. Its clinical features differ
according to the animal species affected. In ruminants infections of the central nervous system, especially meningo-encephalitis predominate, in fowls destructive lesions of the myocardium, in rodents septicaemia, with focal necrosis. Young animals are affected more than mature ones and, in all species, pregnancy predisposes to the infection which then causes metritis with premature expulsion of the foetus. The natural reservoir of infection is believed to be among rodents and fowls.

### TABLE III

**Cases of leptospirosis (all forms) recorded by the Epidemiological Research Laboratory (P.H.L.S.), Colindale**

<table>
<thead>
<tr>
<th>Year</th>
<th>No. of cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>1962</td>
<td>35</td>
</tr>
<tr>
<td>1963</td>
<td>45</td>
</tr>
<tr>
<td>1964</td>
<td>48</td>
</tr>
<tr>
<td>1965</td>
<td>43</td>
</tr>
</tbody>
</table>

**TABLE IV**

*Pasteurella multocida* isolations England and Wales 1957-59

(Figures from Epidemiological Research Laboratory, P.H.L.S., Colindale)

<table>
<thead>
<tr>
<th>Source</th>
<th>No. of cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wounds, animal inflicted</td>
<td>96</td>
</tr>
<tr>
<td>Wounds, not specified</td>
<td>5</td>
</tr>
<tr>
<td>Respiratory tract (bronchiectasis, etc.)</td>
<td>51</td>
</tr>
<tr>
<td>Meningitis (after head injury)</td>
<td>1</td>
</tr>
<tr>
<td>Brain abscess (secondary to otitis media)</td>
<td>1</td>
</tr>
<tr>
<td>Temporosphenoidal abscess</td>
<td>1</td>
</tr>
<tr>
<td>Ear swabs</td>
<td>3</td>
</tr>
<tr>
<td>Antrum washouts</td>
<td>2</td>
</tr>
<tr>
<td>Vaginal swab</td>
<td>1</td>
</tr>
<tr>
<td>Urine from patient with cystitis</td>
<td>1</td>
</tr>
<tr>
<td>Source not stated</td>
<td>3</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>165</strong></td>
</tr>
</tbody>
</table>

Human infection is most liable to occur in those in contact with animals, or who drink raw milk, but it is worth remembering that the organism is fairly resistant to heat and it has been claimed that it can withstand pasteurization. Like staphylococci, listeriae are unusually tolerant of salt and they can survive for long periods on hay and straw.

It has not been considered a common human infection and, up to about ten years ago, according to Seeliger, less than 300 cases had been reported altogether, but the impression is gained that it is now being reported rather more frequently (Lancet 1968). Thus, in Scotland alone, in the 18 months since the publication of the first “Communicable Diseases, Scotland” report no less than eight cases, two of them fatal, are known to have occurred (CDS, 1967, 1968). This probably reflects not so much a rise in its real incidence as an increased awareness of its possibility resulting in more certain diagnosis.

The commonest recognized form of infection is a purulent meningitis or meningo-encephalitis. It can sometimes present in other ways, eg. the anginose-septic form with conjunctivitis, sore throat and cervical adenitis; clinically this bears some resemblance to infectious mononucleosis and this at one time led to the belief, now known to be mistaken, that *L. monocytogenes* (which was isolated from the blood of some of these cases) was the causative organism of glandular fever.

A generalized infection may occur in pregnancy and this may be so mild as to pass unnoticed, in other cases there may be marked fever with rigors and, in these, there may be a metritis which can lead to the premature delivery of either a stillborn infant or one who dies soon after birth. But, although there is a high infant mortality, the mother nearly always recovers. The infant dies of a septicaemia associated with miliary granulomatous nodules in the lungs, spleen and other viscera, a condition known
as granulomatosis infantiseptica. In a recent Glasgow case, the baby died within two hours of birth and the organism was isolated from its pleural cavity and oesophagus and also from a swab taken six weeks after delivery from an erosion on the cervix of the mother (Scott and Henderson 1968).

In meningeal infections the diagnosis is made, presumptively, by microscopic examination of the cerebrospinal fluid and, conclusively, by isolating the organism on culture from the fluid. It grows aerobically at 37°C but colonies may take a few days to develop. Serological examination can also be helpful but this is complicated by cross-reactions with unrelated organisms with which L. monocytogenes shares partial antigens. It is sensitive to a wide range of antibiotics, including penicillin, but untreated listerial meningitis carries a heavy mortality.

**Pasteurellosis**

Oriental plague, in both the bubonic and the less common pneumonic form, the scourge of mankind throughout the ages, has in recent years been increasingly confined to a few small pockets of residual endemicity in various parts of the world, especially in Asia, but some disquiet is bound to arise from the knowledge of its occurrence in Vietnam during the war now in progress. However, there are sound reasons for believing that, even if it were to be imported to Britain, it would be unlikely to gain a foothold here under present day conditions. Nevertheless it is one of the hard lessons of history that it is never wise to be complacent about plague.

Infections due to members other than *P. pestis* of the *Pasteurella* group are of more immediate interest in this country. These, e.g. *P. multocida* (or *P. septica*) and *P. haemolytica*, are carried by many different animals, wild and domestic (including dogs and cats), and can cause disease in some e.g. haemorrhagic septicaemia in cattle (due to *P. multocida*) and pneumonia (due to *P. haemolytica*) in sheep and cattle.

These organisms, which are small non-motile Gram-negative bacilli, often oval in shape and exhibiting bipolar staining, are similar in morphological and cultural characteristics to *P. pestis* itself and all can be readily isolated on artificial culture. Infection in man may involve the lung (the organisms are not infrequently isolated from the sputum in acute exacerbations of bronchiectasis) or the abdomen (appendicular abscess etc.) but, as will be seen from table IV there are other manifestations.

Of particular interest are infections following animal bites and scratches. In one very unusual Glasgow case (McGeachie 1958) the biter was a lion, but infection following wounds caused by more homely animals, notably dogs and cats, are by no means uncommon. These can range from suppuration, with delayed healing of the wound, through cellulitis to more widespread infection with invasion of the blood stream. Penicillin is usually effective in treating these cases, but, especially in more generalized infections, other antibiotics may be required. A series of five cases, four following dog bites and one following a cat bite was recently reported from one Edinburgh laboratory in a period of two years (Maccabe and Conn 1968). For two of these patients the infection was their reward for helping their injured pets and the authors wisely caution those who would handle animals angered or frightened by pain, because for aught they know, the pets may be healthy carriers of pasteurellae.

**Conclusion**

Although some of the conditions described may not be common there can be no doubt that many of them occur far oftener than might be supposed from a perusal of the relatively few published reports. Not infrequently one finds that they occur as 'opportunistic infections' (Symmers 1965), i.e. complicating other pre-existing diseases or debilities, a point of growing importance these days when so many patients are
maintained on cytotoxic drugs, steroids and other forms of immuno-suppressive therapy. This is another reason why the zoonoses deserve our attention.

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Salmonellosis

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Saturday 26 June was a hot and balmy day. That afternoon a tennis tournament was held in one of the select areas of the city, and on Sunday the 27th, many of those who attended had diarrhoea and vomiting, quite a number being admitted to hospital. At the end of the week another batch of patients was admitted, many of whom were suffering from a severe pyrexial illness with or without gastrointestinal symptoms. *Salmonella virchow* was isolated from the faeces of all the patients and from the blood of nearly all those from whom a sample was taken.

At the tournament, chicken had been served both for tea and for late supper. This had been spit-roasted in a Liverpool cook shop earlier in the day and had been stored in a basket in the tennis pavilion from 2.00 p.m. onwards. As I have said, it was a very warm day and I was able clinically to distinguish those who had chicken for tea only