

V or who married into Classes IV and V from a higher class must have been highly selected, either because their environment or training was particularly unfavourable or because their abilities were poor. If the Aberdeen experience is relevant, these movements would tend to concentrate in Classes IV and V women with poor physique and health, who have relatively high prematurity and obstetric death rates. At the same time continuous recruitment of healthy women into higher classes would lower the prematurity and obstetric death rates of these classes. In this way the process of social selection may continuously obscure the effects of social levelling by keeping open the gap between classes.

The inevitable delay in translating social improvements into improved health and the greater rapidity with which the upper social classes take advantage of new welfare services and advances in medical knowledge also tend to keep open the gap between classes,\* so that it is impossible to assess quantitatively the influence of selective mobility on differences of mortality between the social classes. The Aberdeen figures and the amount of interclass movement occurring in the last 20 years suggest that the influence of selection may have been considerable. The Aberdeen data, however, relate only to an urban population in a single city over a short period of time, and the number of cases in the upper social classes is small. While there is no reason to suppose that either the occupational structure or the prematurity and obstetric death rates of Aberdeen are at all exceptional for an urban community, it is obviously desirable that the study should be repeated and extended to rural areas.

Nevertheless it is clear that a comparison of social class differences at two points in time—for example, between census years—is misleading, because of changes in the composition of classes and selective interchange between classes. The persistent gap between the mortality of the various social classes, defined in occupational terms, does not necessarily mean that the social levelling of recent years has been ineffective. The use of another social classification less affected by selective mobility or by changes in occupational structure might well show that the difference between classes has narrowed.

### Summary

A socio-medical investigation of Aberdeen primiparae has shown that women who rise in social status at marriage tend to be of superior intelligence, education, and occupational skill. Such women also tend to be tall, to be in good health, and to have low prematurity and obstetric death rates. Conversely, women whose social status falls at marriage tend to have the opposite characteristics. Social status is defined in terms of the occupation of the patient's father and of her husband.

This selective interchange between classes at marriage tends to accentuate class differences, and is partly responsible for the continuing disparity between the mortality rates of the Registrar-General's social classes, despite the social and economic changes of recent years. These classes are changing in composition and size, and movement between them is highly selective, so that comparison of class mortality rates at different periods of time may give misleading results.

I am indebted to Professor D. Baird for help at all stages of this inquiry; to the almoners' department of the Maternity Hospital, and especially to Miss B. Thompson, for social data on hospital patients; to the Medical Officer of Health for Aberdeen, the matron of the Rubislaw Nursing Home, and the general practitioners of the city for data on women confined in their own homes or in the nursing-home; and to Mr. D. Allcorn, Mr. W. Z. Billewicz, Dr. J. N. Morris, Dr. I. M. Richardson, and Professor R. M. Titmuss for advice and criticism.

\*For a fuller discussion of these influences see Morris and Heady (1955).

### REFERENCES

- Baird, D. (1952). *New Engl. J. Med.*, **246**, 561.  
 — and Illsley, R. (1953). *Proc. roy. Soc. Med.*, **46**, 53.  
 Boalt, G. (1955). *Transactions of the Second World Congress of Sociology*, Vol. II. London.  
*Economist*, 1951, p. 1418.  
 Hollingsworth, D. F. (1955). *Proc. Nutr. Soc.*, **14**, 71.  
 Illsley, R., Billewicz, W. Z., and Thomson, A. M. (1954). *Brit. J. prev. soc. Med.*, **8**, 153.  
 — and Thompson, B. (1955). *Technical Problems of Social Classification Affecting Inter-class Movement at Marriage*. In preparation.  
 Logan, W. P. B. (1954). *Brit. J. prev. soc. Med.*, **8**, 128.  
 McKinlay, P. L. (1948). *Hlth Bull. (Edinb.)*, **6**, 8.  
 Martin, F. M. (1954). In *Social Mobility in Britain*, edited by D. V. Glass. London.  
 Morris, J. N., and Heady, J. A. (1955). *Lancet*, **1**, 554.  
 Registrar-General (1938). *Decennial Supplement, England and Wales, 1931. Part IIa, Occupational Mortality*. H.M.S.O., London.  
 — (1951). *Classification of Occupations, 1950*. H.M.S.O., London.  
 — (1954). *Decennial Supplement, England and Wales, 1951. Part I, Occupational Mortality*. H.M.S.O., London.  
 Scott, E. M. (1954). Ph.D. Thesis, University of Edinburgh.  
 Seers, D. (1948). *Bull. Oxf. Univ. Inst. Statist.*, **10**, 10, 309.  
 Thompson, B. (1954). *Med. Offr.*, **91**, 235.  
 Titmuss, R. M. (1943). *Birth, Poverty and Wealth*. London.

## NECROBACILLOSIS IN GREAT BRITAIN\*

BY

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The micro-organism most commonly known as *Bacillus necrophorus* causes various lesions in wild and domestic animals but is not often found in human infections, although in human beings it may produce a fatal suppurative.

The organism belongs to a group which has not yet been fully explored and which has come into existence through the realization that various bacteria described and named as the cause of different diseases in animals and in man are very similar. The first name given to the organism was bacillus of calf diphtheria, by which Loeffler (1884) called a bacillus which he saw in lesions in the throats of calves and in lesions in rabbits and mice which he infected by material from calves' throats. Other names which are now considered to be synonyms are *Bacterium necrophorum*, *Fusiformis necrophorus*, *Bacteroides necrophorus*, *Actinomyces necrophorus*, *Streptothrix necrophora*, Bang's necrosis bacillus, *Streptothrix cuniculi*, *Actinomyces cuniculi*, *Bacillus filiformis*, *Bacillus funduliformis*, *Bacteroides funduliformis*, *Bacillus pyogenes anaerobius*. Domesticated animals that are known to be subject to the infection include cattle, sheep, goats, deer, horses, pigs, rabbits, guinea-pigs, dogs, monkeys, and poultry; and various wild animals have also been found to be infected (Albrecht, 1929). The diseases may be epizootic or sporadic and local or widespread in the different species. Thus, as well as calf diphtheria, we have necrotic stomatitis of calves, lambs, and pigs, foot-rot of cattle and sheep, gangrenous dermatitis of horses and mules, multiple abscesses of lungs and liver of cattle and pigs, post-partum necrobacillosis of the vagina and uterus of cows, and navel-ill in calves born from them (Cunningham, 1930).

\*This paper was written because the following colleagues generously allowed the use of case notes or pathological reports: Dr. B. Barling, Mr. R. Blunden, Dr. A. B. Bratton, Dr. B. Freedman, Dr. A. L. Jacobs, Dr. E. D. Maunsell, Dr. K. S. May, Dr. D. M. Morrison, Mr. P. T. Savage, Mr. N. Tanner, Dr. W. M. Turtle. I am also grateful for the excellent bacteriological assistance of the late Mr. S. J. Denyer and of Mr. A. P. Wheeler.

### Types of Human Infection

Authenticated human cases have already been reported (in the approximate numbers given after each country) from Germany (94), U.S.A. (87), Hungary (24), Holland (15), Great Britain (10), France (31), French Indo-China (6), Scandinavia (2). This total of 269 cases does not include nine infections referred to but not described in the paper on one fatal case by Alston *et al.* (1938). The names of the authors of case histories from each country are given with the other references below. Most of the lesions recorded in human beings can be divided into four groups. The first, and smallest, comprises instances of infection of the skin or subcutaneous tissues. These cases include two men infected during experimental work with the organism derived from infected rabbits (Schmorl, 1891), a meat inspector who scratched himself on the tooth of a dead infected sheep (Stemen and Shaw, 1910), a farm worker who was bitten by an infected cow (van Wering, 1923), a veterinarian infected during manual removal of the placenta of a cow (Wohlstein, 1929), and a girl, employed in a butcher's shop, who had a deep infection of the right middle finger (van Gelder, 1930). Similar to these are infections of war wounds of 24 soldiers reported from Hungary (Bogdán, 1916), infection of a sailor reported in Britain (Forbes and Goligher, 1944), and an infection of a civilian at the site of a stab wound in the shoulder six months after the wound had healed (McVay and Sprunt, 1952).

The second, larger, group is of cases in which infection started in the region of the throat, sometimes after tonsillectomy or with sore throat or otitis media as a first symptom. Thirdly, there are many infections by way of the female genital tract, mostly after abortion or in the puerperium, and fewer, in either sex, by the alimentary tract, sometimes after surgical operation, or by the urinary tract. The fourth group has shown empyema—with or without lung abscess—as the first lesion. The reason for this comparatively large group is not clear, but it has been suggested that when lung abscess precedes empyema, inhalation from the mouth or throat may be the origin of the infection. In the last three groups general pyaemia and abscesses have occurred in very many situations.

In our experience of 12 cases there have been purulent meningitis, lateral sinus thrombosis, otitis media, peritonsillar and sublingual abscess, extensive suppuration of the neck and mediastinum, lung abscess, empyema, abscess of the spleen, abscess in the broad ligament of the uterus, abscess of knee-joint, and osteomyelitis of the femur. Bacterial endocarditis was found in two patients by Fisher and McKusick (1953); in a group of 20 cases. Smith and Ropes (1945) include peritonitis, prostatitis, salpingitis, and hepatic and renal abscesses; and several authors—for example, Harris and Brown (1927); Joe *et al.* (1937)—have recorded suppuration of the wound after caesarean section or of the uterus after vaginal delivery.

It is only in the first five patients of the first group that the origin was traced directly from particular infected animals. In the other groups, although a small number of the patients worked among animals or in rat-infested premises or handled meat, a source of infection from an animal was not demonstrated. There is not enough knowledge of the presence of the organism in healthy people. Joe *et al.* (1937) stated briefly that they had obtained the organisms from the cervical swabs of several presumably healthy patients. This has not been my experience in examining many anaerobic cultures from the cervix with this in mind and after examining carefully the tonsils from 50 cadavers without finding the organism. However, Dack *et al.* (1938) isolated 19 strains of the organism from the colons of patients with chronic ulcerative colitis, several of whom had an ileostomy; this is evidence of the presence of the organism in the intestine and of its increase in some abnormal conditions, but it is not generally accepted as showing a causative connexion between the bacillus and chronic ulcerative colitis. It indicates the source of infec-

tions which begin after operations on the intestine, and it may have a relation to the infection of the female genital tract.

Lemierre *et al.* (1938) reviewed more than 30 human infections due to *B. funduliformis* (*B. necrophorus*), and considered that the organism is an inhabitant, potentially pathogenic, of the throat and of the gastro-intestinal and urogenital tracts. Ernst (1951) cultured the organism from 86 patients who had chiefly pelvic infections due to it, and he thought that the organism might be saprophytic in the vagina. It is possible that the organism may enter the alimentary tract from animal sources and remain a saprophyte until a circumstance like tonsillectomy, operation on the intestine, or parturition offers a chance of invasion of the tissue.

The only cases in which an incubation period can be accurately known are four cases in the first group. In one, inflammation and severe pain followed within two days of scratching a finger on an infected sheep's tooth, and five days after the scratch there was coagulation necrosis on the back of the hand, with bullae, and the hand and arm to the shoulder were swollen. This contrasts with the slower course in the patient bitten by an infected cow, for it was three weeks before the first lesions—small abscesses—were seen at the site of the bite, and a week later lymphangitis began and involved the axillary gland. The veterinarian developed a lesion on his forearm three days after removing the cow's placenta, and it may be that the infection lay dormant for six months in the healed wound of the stabbed man.

After abortion or childbirth the period of incubation can usually be presumed, but one cannot be sure that infection may not have occurred later than the time of delivery or abortion, or of the interference to cause abortion. The same applies to infections following tonsillectomy. Harris and Brown (1927) described six puerperal cases; in one the temperature was raised when delivery was made, and in the others on the first, second, and third days of the puerperium, and suppuration followed in a few days in four cases. Beeuwkes *et al.* (1951) described a case of inflammation beginning in a laparotomy scar two years after the operation. One case in the present series twice had empyema at the same site, with ten years between the occasions, and the second illness is believed to have been due to latent infection in a sequestrum of bone. These examples show the great variation between almost immediate infection and long latency.

### Mortality

The illness also varies greatly in the rapidity of its course. In six fatal cases which we have observed, death occurred 6 days, 19 days, 3 weeks, 4 weeks (twice), and 7 weeks after the onset.

There were no fatalities from the local infections (other than war wounds) mentioned in the first group; but fatal cases have been reported in each of the other groups. An estimate of the case mortality rate of all reported cases cannot give a good picture of the seriousness of the infection because the literature consists to a large extent of small numbers of serious—often fatal—infections. However, those who have been on the look-out for the organism have collected cases of differing degrees of severity. McVay and Sprunt (1952) found 35 cases in five years: 21 patients (60%) were seriously ill and 6 (17%) died. These deaths were distributed over all the groups except the first group of skin infections, in which McVay and Sprunt had one patient, who survived. Among their patients were 13 in whom infection was by the female genital tract, and, of these, two died; to these 13 may be added the six puerperal cases of Harris and Brown (1927) with one death, making, in the two groups, 19 puerperal patients with 3 (16%) deaths. On the other hand, Lemierre *et al.* (1938) obtained from the literature (chiefly French) more than 30 cases of minor infections arising from the tonsils and the genito-urinary tract with very few deaths. Among 12 patients known to us in London there were 7 (58%) deaths; with 9 other cases of

infection in Britain recorded in the literature, the total of 21 infections in this country resulted in 13 deaths (62%). These figures may indicate that many less severe infections have been missed in Great Britain.

### Diagnosis and Prognosis

The diagnosis of this infection may be suggested by the peculiar odour—like Limburger or overripe Camembert cheese—of pus produced by it. If there is no pus, sputum, or uterine secretion to culture, and the organism is not found in a blood specimen, the infection is rarely suspected, and the illness is regarded as general bacterial toxæmia, septicaemia, or pyaemia. A polymorphonuclear leucocytosis occurs in most cases. Jaundice sometimes develops, and liver atrophy or Weil's disease may be suspected.

The prognosis in individual cases depends on the extension from the primary site and the multiplicity of metastatic lesions. Strains of the organism have been found to differ from one another in sensitivity to antibiotic drugs, but several were found sensitive to penicillin (Ruys, 1947), others to chlortetracycline (McVay *et al.*, 1949). Fisher and McKusick (1953) tested strains from 14 patients and found that 10 were sensitive to penicillin (from 0.1 to 50 units per ml.), 6 were sensitive to chloramphenicol (from 0.2 to 3.1 µg.), 7 to chlortetracycline (1.6 µg. or less), and 3 to oxytetracycline (0.1 to 1.6 µg.); 7 strains were comparatively or highly resistant to streptomycin. I found that the strain isolated from a patient in 1944 was sensitive to crude penicillin, and the strain from the same patient ten years later was sensitive to penicillin, streptomycin, chlortetracycline, chloramphenicol, and sulphonamide; and from the relatively few recorded cases treated with these drugs it seems clear that the prognosis is much improved by the possibility of this treatment (Cooper and Robson, 1947; Fisher and McKusick, 1953). Organisms, especially anaerobic streptococci, often accompany *B. necrophorus* in the lesions and may differ from *B. necrophorus* in drug sensitivity; they should be tested separately if necessary.

### Biological Features of the Organism

In pus *B. necrophorus* is found as a short, slender, Gram-negative, non-sporing bacillus, not acid-fast, 1.5–2 by 0.5 µ, often staining in a granular fashion and resembling very closely *Haemophilus influenzae*. The morphology in cultures is very variable and includes filaments of up to 50 or 100 µ, with fusiform or globular swellings. By electron microscope, the organism was seen to reproduce by multipolar germination from large round bodies, as pleuropneumonia-like organisms do (Smith *et al.*, 1948). There is

no confirmed evidence of branching of filamentous forms to justify the name of actinomyces or streptothrix, nor, in my experience, were tapering forms found to account for the name fusiformis.

For artificial growth, anaerobic conditions are relatively strictly necessary and plate cultures may die after exposure to air for 12 hours. Beveridge (1934) showed that anaerobic conditions for growth and survival are modified by the presence in cultures of certain other organisms, such as staphylococci. Growth is absent or poor in unenriched medium, and blood or serum is usually added to broth or agar. Cooked-meat medium under paraffin oil promotes growth and survival well. Growth takes two to four days to reach its maximum in most media. On solid media the colonies are by that time up to 2 mm. in diameter, transparent, and even in outline; later they may show a thinner extension of growth; many strains are haemolytic. In fluid media the growth is often flocculent but may be of even turbidity. All cultures have the strong, cheesy smell of the pus. If cultures on a solid medium are kept in the incubator for three weeks or more, darkening of the centre of colonies may occur. I do not know whether this is due to a pigment, but it serves to call to mind the organism of similar characteristics and pathogenicity to which Oliver and Wherry (1921) gave the name *Bacillus melaninogenicum*, on account of the production of melanin in the colonies.

From three of the patients in our series (Cases 1, 2, and 9) *B. necrophorus* was isolated in pure culture from all or some of the lesions, and these cultures remained pure on subculture; but from most of the others (Cases 3, 5, 7, 8, 10, 11, and 12) other bacteria—almost always streptococci—were found in the primary cultures in addition to *B. necrophorus*. It was found not only that there were colonies consisting entirely of streptococci but that it was impossible to be sure of obtaining a strain of *B. necrophorus* which remained free from streptococci in one or other of its subcultures. This association of symbiosis or, possibly, variation needs more study. The streptococci were not haemolytic and some were viridans.

Fermentation tests of different strains of *B. necrophorus* from human and animal sources have given different results, with some frequent patterns of reactions in some groups—for example, Beveridge (1934). I found that one human strain fermented lactose, saccharose, glucose, maltose, dextrose, raffinose, inulin, galactose, and laevulose, but not dulcitate, arabinose, adonite, inosite, or salicin; another strain fermented none of these.

Serological tests have shown that different serotypes exist among strains from various sources in human beings and animals (Orcutt, 1930; Beveridge, 1934; Walker and Dack,

### Cases of Necrobacillosis in Great Britain

Sex and Case	Age (Years)	Length of Illness (Weeks)	Principal Lesions	Result	Site of Entry	Year of Occurrence	Publication
F	18	14	Mastoiditis, extradural abscess	D.*	Tonsil		Franklin (1933)
F	5	4	Otitis media, empyema, lung abscess	D.	Throat		
F	28	7	Uterine sepsis, pyaemia, empyema	D.	Genital tract		Joe <i>et al.</i> (1937)
F	24	4½	Uterine sepsis, empyema	D.	" "		" " " "
F	—	—	Infection of caesarean section	S.*	" "		" " " "
F	—	—	—	D.	" "		" " " "
F	1	5	Meningitis, lung abscess, empyema	D.	Tonsil	1933	
M	2	27	Empyema	D.	?	1934	
F	3	29	Parametritis, splenic abscess, empyema	D.	Genital tract	1934	
F	4	19	Empyema	S.	?	1935	
M	5	32	—	S.	?	1935	
M	6	65	Arthritis of knee, pyaemia	D.	?	1935	
M	7	42	Abscess of neck	S.	? Tonsil	1936	
M	8	—	Peritonsillar and sublingual abscess	S.	Tonsil	1936	
M	9	29	Abscess of neck and of lungs, empyemata	D.	" "	1938	
M	10	36	Osteomyelitis of femur, pyaemia, pneumonia	D.	" ?	1938	Alston <i>et al.</i> (1938)
M	11	55	Abscesses, also carcinoma, of lungs	D.	?	1941	
F	12	17	Abscess of R. lung; L. empyema, gluteal abscess	S.		1944	
M	27	5	L. empyema and abscess of chest wall			1954	
M	24	12	Infection of wound of leg; lung abscesses	D.	Wound of leg		Forbes and Goligher (1944)
F	29	9	Abortion, thrombosis of iliac veins	S.	Genital tract		Jones (1944)
F	—	3	Normal delivery, toxæmia and septicaemia, anaemia	S.	? Genital tract		Cooper and Robson (1947)

\* D. = Died. S. = Survived.

1939). Unfortunately, most of our strains were lost during the war, before serological tests were successfully made.

Experimental infection of rabbits and mice has shown different degrees of virulence in strains isolated from human sources. I found that three cultures from separate patients produced large slow-growing abscesses at the site of injection in the hind legs of rabbits and small multiple abscesses in the peritoneal cavity of mice when injected intraperitoneally; in both species of animals there was not much general disturbance of health. Other workers have produced fatal pyaemic infection by intravenous injection.

In Great Britain I have found records, in the literature and in our own experience, of 21 patients suffering from this infection. (These include nine referred to, in summary, in the report of one case by Alston *et al.*, 1938.) Some details of these infections are given in the Table. Short accounts are given of 11 of the cases of the present series and a more detailed record of the patient (Case 12) who had two illnesses with an interval of ten years.

#### Outline of 11 Cases in which *B. necrophorus* was Isolated in Pure or Mixed Culture (see Table)

*Case 1.*—A girl aged 5 (Dr. D. M. Morrison, Highgate Hospital). The fatal illness began with acute otitis media on the left side four days after tonsillectomy. Acute meningitis followed ten days later, and the patient died after an illness of three weeks. Necropsy revealed left-sided otitis media and cerebral meningitis, and several small abscesses in one lung (Dr. A. B. Bratton). Bacteriological examination was made of cerebrospinal fluid, pus from left lateral venous sinus and left middle ear, and fluid in one lateral ventricle. In all of these *B. necrophorus* was found in pure culture. In a lung abscess the same organism was found in company with other bacteria.

*Case 2.*—A man aged 27 (Dr. B. Barling, St. Mary's Islington Hospital). The fatal illness was pleurisy and empyema of the left side, of one month's duration. Large amounts of fluid were aspirated on three occasions. This exudate was purulent and became thicker in consistency and more foul-smelling as the illness progressed. Bacteriological examination of the pleural exudate showed very large numbers of small Gram-negative bacilli, which on culture gave a pure growth of *B. necrophorus*. No post-mortem examination was made. The route of infection was not known.

*Case 3.*—A woman aged 29 (Dr. W. M. Turtle, St. Mary's Islington Hospital). The fatal illness lasted seven weeks. It was first diagnosed as influenza and rheumatism; and two weeks later a miscarriage occurred following patient's own efforts to produce it. A very toxic state with high temperature and offensive vaginal discharge followed. Empyema of the left pleural cavity developed, and a large amount of offensively smelling pus was removed from it by aspiration before death. At post-mortem examination a puncture wound was found in the right vaginal fornix and it communicated with an abscess in the right broad ligament. An abscess the size of a golf ball was present in the spleen, and there was extensive empyema in the left pleural cavity (Dr. A. B. Bratton). This patient had worked in the meat trade as a ham-washer, and after her death it was suggested that abortion may have been caused by a meat skewer. Bacteriological examination was made of the pus from the empyema (before and after death) and of pus from the abscesses in the spleen and broad ligament after death. In all of these *B. necrophorus* and *Str. viridans* were found by film and culture in large and approximately equal numbers.

*Case 4.*—A girl aged 19 (St. Leonard's Hospital). The illness was left-sided pleurisy, with empyema, and it lasted 17 weeks. Complete recovery was made. Large quantities of foul-smelling pus were removed by aspiration and, later, by drainage by tube. Bacteriological examination of pleural exudate showed filamentous bacteria in large amounts, and, although *B. necrophorus* was not obtained in direct culture,

abscesses were produced by subcutaneous inoculation of mice and *B. necrophorus* was obtained in culture from them.

*Case 5.*—A man aged 32 years (St. Leonard's Hospital). This illness was pleurisy and empyema of the left pleural cavity which began acutely, and recovery occurred in about a month, after aspiration and drainage by tube. The pus removed was copious and very foul-smelling. *B. necrophorus* and non-haemolytic streptococci were isolated in large and approximately equal numbers from the pleural exudate. The route of infection was unknown. The patient had cleaned stables for a time up to nine months before his illness.

*Case 6.*—A man aged 65 (Dr. K. S. May, St. Leonard's Hospital). This fatal illness was general pyaemia with purulent arthritis of a knee-joint from which *B. necrophorus* was isolated. (The full notes of this case were lost during the war.)

*Case 7.*—A man aged 42 (Mr. Norman Tanner, Highgate Hospital). This patient recovered from an abscess of the neck from which *B. necrophorus* and non-haemolytic streptococci were isolated.

*Case 8.*—A man (St. Mary's Islington Hospital). This patient had peritonsillar and sublingual abscesses from which *B. necrophorus* and many other organisms were isolated. He recovered.

*Case 9.*—A man aged 29 (reported by Alston *et al.* (1938), Bethnal Green Hospital). This acute fatal illness appeared to originate from a septic tonsil leading to a large abscess in the right side of the neck, followed by pulmonary abscess and bilateral pyopneumothorax. The abscess in the neck surrounded and invaded the internal jugular vein. *B. necrophorus* was obtained from the blood in pure culture during life, and in pure or mixed cultures from several specimens of pus at necropsy. The site of entry of the infection was almost certainly the tonsil, and gross dental sepsis and pyorrhoea may have had a part in the entry of the bacillus into the tissue.

*Case 10.*—A man aged 36 (Mr. R. Blunden, St. Mary's Islington Hospital). This fatal illness of six days began with acute arthritis of the right knee which was surgically drained, pus containing *B. necrophorus* being obtained. Blood transfusion was given, jaundice developed, and, at necropsy, osteomyelitis of the right femur and septic pneumonia and pyaemic abscesses were found. No site of entry of the organism was shown.

*Case 11.*—A man aged 55 (Dr. B. Freedman, Archway Hospital). This fatal illness lasted four weeks and was the climax of ill-health for more than a year, beginning with pneumonia which was followed by a "cough with an unpleasant taste" and grey sputum sometimes blood-stained. The patient was very ill and wasted. There were large and hard lymph nodes in the left axilla and dullness at the base of the right lung. *B. necrophorus* was cultured from pus at the right side of the chest posteriorly. At necropsy were found carcinoma of the bronchus in the lower lobe of the right lung, fibrosis and abscesses in the same lobe, and bronchopneumonia and abscesses in the left lower lobe (Dr. A. B. Bratton).

A fuller account follows of Case 12, the patient who had a recurrence of her illness.

#### Case 12

The patient was aged 17 at the time of her first illness, which began near the end of February, 1944, with cough, malaise, and pains in the back. She was admitted to an isolation hospital on suspicion of diphtheria and discharged after a few days with a diagnosis of tonsillitis. She came to St. Mary Islington Hospital (Dr. E. D. Maunsell) on March 9, when she was acutely ill and mentally apathetic; her temperature was 103.4° F. (39.7° C.). There were signs of pleural effusion on the left side and a small pressure sore

over the sacrum. On March 13 radiography showed a large cavity with a fluid level in the upper lobe of the right lung and a left pleural effusion. On March 15 and 17 the left pleural cavity was explored by needle, and each time thick offensive greenish pus was obtained. Large numbers of *B. necrophorus* were obtained in anaerobic culture of this, with a few colonies of *Staphylococcus aureus*. On the 18th radiography showed the abscess cavity in the right lung to be smaller and the effusion shadow on the left side had reached the seventh rib posteriorly. On the 21st Mr. Blunden resected a portion of the left tenth rib and inserted a tube for closed drainage. On the 28th Mr. Blunden opened and drained very offensive pus from a large abscess. From that time the temperature abated and recovery advanced.

On April 17 radiography showed a bronchopleural fistula in the left lung and a cavity still present in the right lung, and on May 11 pleural thickening on the left side and the cavity on the right side. She remained in hospital until May 18, when movement in the left side of the chest was good and the percussion note resonant; there was a small sinus at the drainage site. In hospital, she showed at first moderate anaemia of 70% haemoglobin, which improved to 86%, and polymorph leucocytosis of 26,900 and 18,400 per c.mm. *B. necrophorus* was not detected in sputum which was examined early in the illness. In June, as an out-patient, her sinus was seen to be healed; she had lost much of her hair. In November she was very well, had no cough, and her hair was growing again.

The patient remained well for ten years and married. She noticed an occasional twinge of pain in the left side of the chest.

When readmitted to hospital in the care of Dr. A. L. Jacobs, on April 17, 1954, she was 27 years old. She had been ill for four days with fever, headache, anorexia, and vomiting, and had a cough with yellow sputum. She was anxious, had slight dyspnoea, a flushed face, and labial herpes; her temperature was 101° F. (38.3° C.). The chest showed impaired movement on the left side and a dull percussion note at the left base posteriorly. Immediately above the old thoracotomy scar there was a swelling 5 cm. in diameter, red, tender, and slightly fluctuant. Radiography showed an encysted pleural effusion at the base of the left lung. Penicillin ("estopen," changed later to "distaquine" penicillin) was given from admission for 14 days and sulphadiazine, 1.5 g. daily, orally for the first five days, and then was replaced by streptomycin, 0.5 g. eight-hourly. On April 19 there was continuing fever and tachycardia, and 20 ml. of offensive thick yellow pus was aspirated. On the 21st Mr. P. T. Savage incised the old scar and evacuated 150 ml. of pus; he explored the empyema cavity by finger and found that it reached 3 in. (7.5 cm.) upwards and contained a small rib sequestrum; a tube for open drainage was put in.

Bacteriologically the pus showed a small number of Gram-negative bacilli and filaments and no acid-fast bacilli; aerobic culture gave no growth and anaerobic culture produced *B. necrophorus* and a Gram-positive coccus from which the *B. necrophorus* could not be separated. The organisms were both sensitive to penicillin, sulphonamide, chlortetracycline, chloramphenicol, and streptomycin. After drainage the temperature fell and the patient rapidly improved, being discharged from hospital on May 16. A radiograph in July showed only slight pleural thickening at the left base and a resected rib with regeneration. During this illness there was anaemia, with 77% haemoglobin, and the leucocytes numbered not more than 7,500 per c.mm. *B. necrophorus* was not found in the sputum.

### Summary

An account is given of the bacteriological features of *B. necrophorus* and of the types of suppurative infections in men and animals in which it has been found. Approximately 280 human cases have been found in the

literature and in the present series; 21 of the patients have been in Great Britain.

Among the 12 patients in our experience 7 died and 5 survived. One of the survivors had an empyema on the left side on two occasions—the second ten years after the first.

### REFERENCES USED IN THE TEXT OF THIS PAPER

- Albrecht, B. (1929). In Kolle, Kraus, and Uhlenhuth's *Handbuch der pathologischen Mikroorganismen*, 3rd ed., Band vi, 673. Jena.
- Alston, J. M., Baker, H., and Bratton, A. B. (1938). *A.R. Lond. County Coun.*, 1937, Vol. IV, Part III, p. 123.
- Beuwkes, H., Vismans, J. B. M., and Smeets, A. H. (1951). *Ned. T. Geneesk.*, 95, 1143.
- Beveridge, W. I. B. (1934). *J. Path. Bact.*, 38, 467.
- Bogdan, A. (1916). *Med. Klin.*, 12, 383.
- Cooper, T. V., and Robson, J. A. (1947). *Monthly Bull. Minist. Hlth (Lond.)*, 6, 10.
- Cunningham, J. S. (1930). *Arch. Path. (Chicago)*, 9, 843.
- Dack, G. M., Dragstedt, L. R., Johnson, R., and McCullough, N. B. (1938). *J. infect. Dis.*, 62, 169.
- Ernst, O. (1951). *Z. Hyg. InfektKr.*, 132, 352.
- Fisher, A. M., and McKusick, V. A. (1953). *Amer. J. med. Sci.*, 225, 253.
- Forbes, G. B., and Goligher, J. C. (1944). *Lancet*, 1, 399.
- Franklin, A. W. (1933). *Ibid.*, 2, 645.
- Harris, J. W., and Brown, J. H. (1927). *Bull. Johns Hopk. Hosp.*, 40, 203.
- Joe, A., Wyatt, J. M., and Cruickshank, R. (1937). *A.R. Lond. County Coun.*, 1936, Vol. IV, Part III, p. 31.
- Jones, H. P. (1944). *Lancet*, 2, 824.
- Lemierre, A., Gregoire, R., Laporte, A., and Couvelaire, R. (1938). *Bull. Acad. Méd. (Paris)*, 119, 352.
- Loeffler, F. (1884). *Mitt. Gesundheitsamt.*, 2, 489.
- McVay, L. V., jun., Guthrie, F., and Sprunt, D. H. (1949). *J. Amer. med. Ass.*, 140, 1150.
- and Sprunt, D. H. (1952). *Ann. intern. Med.*, 36, 56.
- Oliver, W. W., and Wherry, W. B. (1921). *J. infect. Dis.*, 28, 341.
- Orcutt, M. L. (1930). *J. Bact.*, 20, 343.
- Ruys, A. C. (1947). *J. Path. Bact.*, 59, 313.
- Schmorl, G. (1891). *Dtsch. Z. Tiermed.*, 17, 375.
- Smith, W. E., Mudd, S., and Hillier, J. (1948). *J. Bact.*, 56, 603.
- and Ropes, M. W. (1945). *New Engl. J. Med.*, 232, 31.
- Stemen, C. M., and Shaw, F. W. (1910). *J. Kans med. Soc.*, 10, 405. Quoted by F. W. Shaw, *Zbl. Bakt.*, 1933, 129, 132.
- van Gelder, R. I. (1930). *Derm. Z.*, 58, 368.
- van Wering, F. (1923). *Ned. T. Geneesk.*, 67, 2892.
- Walker, P. H., and Dack, G. M. (1939). *J. infect. Dis.*, 65, 285.
- Wohlstein, E. (1929). *Derm. Z.*, 56, 415.

### REFERENCES TO CASE RECORDS FROM SEPARATE COUNTRIES

- France*
- Desbuquois and Iselin (1945). *Bull. Soc. Méd. Hôp. Paris*, 61, 313.
- Lemierre, A., Gregoire, R., Laporte, A., and Couvelaire, R. (1938). *Bull. Acad. Méd. (Paris)*, 119, 352.
- French Cochinchina*
- Delbove, P., and Reynes, V. (1941). *Rév. Méd. Française d'Extrême Orient.* 19, 119. Abstract in *Bull. Hyg.*, 1941, 16, 659.
- Germany*
- Ernst, O. (1951). *Z. Hyg. InfektKr.*, 132, 352.
- Härders, H., and Hornbostel, H. (1951). *Zbl. Bakt., I. Abt.*, 156, 582.
- Kemkes, B. (1943). *Ibid.*, 151, 68.
- Lodenkämper, H. (1949). *Z. Hyg. InfektKr.*, 130, 260.
- Schmorl, G. (1891). *Dtsch. Z. Tiermed.*, 17, 375.
- Wohlstein, E. (1929). *Derm. Z.*, 56, 415.
- Great Britain*
- Alston, J. M., Baker, H., and Bratton, A. B. (1938). *A.R. Lond. County Coun.*, 1937, Vol. IV, Part III, p. 123.
- Cooper, T. V., and Robson, J. A. (1947). *Monthly Bull. Minist. Hlth (Lond.)*, 6, 10.
- Forbes, G. B., and Goligher, J. C. (1944). *Lancet*, 1, 399.
- Franklin, A. W. (1933). *Ibid.*, 2, 645.
- Joe, A., Wyatt, J. M., and Cruickshank, R. (1937). *A.R. Lond. County Coun.*, 1936, Vol. IV, Part III, p. 31.
- Jones, H. P. (1944). *Lancet*, 2, 824.
- Holland*
- Beuwkes, H., Vismans, J. B. M., and Smeets, A. H. (1951). *Ned. T. Geneesk.*, 95, 1143.
- Ruys, A. C. (1947). *J. Path. Bact.*, 59, 313.
- van Gelder, R. I. (1930). *Derm. Z.*, 58, 368.
- van Wering, F. (1923). *Ned. T. Geneesk.*, 67, 2892.
- Scandinavia*
- Henriksen, S. D. (1947). *Acta med. scand.*, 129, 352.
- Switzerland*
- Hartl, H. (1950). *Schweiz. med. Wschr.*, 80, 1136.
- U.S.A.*
- Buhler, V. B., Seeley, C. W., and Dixon, D. D. (1942). *Amer. J. clin. Path.*, 12, 380.
- Chandler, F. A., and Breaks, Virginia M. (1941). *J. Amer. med. Ass.*, 116, 2390.
- Cunningham, J. S. (1930). *Arch. Path. (Chicago)*, 9, 843.
- Fisher, A. M., and McKusick, V. A. (1953). *Amer. J. med. Sci.*, 225, 253.
- Greenblatt, I. J., and Greenblatt, A. P. (1945). *Ibid.*, 210, 596.
- Harris, J. W., and Brown, J. H. (1927). *Bull. Johns Hopk. Hosp.*, 40, 203.
- McVay, L. V., jun., and Sprunt, D. H. (1952). *Ann. intern. Med.*, 36, 56.
- Shaw, F. W. (1933). *Zbl. Bakt., I. Abt.*, 129, 132.
- and Bigger, I. A. (1934). *J. Amer. med. Ass.*, 102, 688.
- Smith, W. E., and Ropes, M. W. (1945). *New Engl. J. Med.*, 232, 31.
- Stemen, C. M., and Shaw, F. W. (1910). *J. Kans med. Soc.*, 10, 405. Quoted by F. W. Shaw, *Zbl. Bakt.*, 1933, 129, 132.
- Tullis, J. L., and Mordvin, O. E. (1946). *Amer. J. clin. Path.*, 16, 395.